A TEXT-BOOK

of

MEDICAL

DIAGNOSIS

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PREFACE TO THE SECOND EDITION.

THE revision of this edition has been in progress for the past two years.

The most efficient manner in which the authors can express their gratitude for favor shown to this work is by keeping it a fair exponent of its subject. Every effort has been made to bring the present volume abreast with our clinical and laboratory methods of diagnosis. It is earnestly hoped by the authors that all practical advances in diagnosis have been given consideration.

While our aim has been to make a thorough revision of this work, special mention may be made of the more important additions: Movements of the two halves of the chest; electrocardiograms; extrasystole; auricular fibrillation; sinus irregularity; succussion sounds audible over the abdomen; abdominal tension with original methods of determination; albuminous sputum; cobra-venom reaction in syphilis; the tick in transmitting relapsing fever; Rumpell-Leed phenomena in scarlet fever; inclusion bodies of Döhle in scarlet fever; sweating and its significance; Trichinella spiralis in the blood; MacEwen's sign and Brudzendski's sign of epidemic meningitis; Prendergast's reaction for typhoid fever; fatty emboli; pupillary reaction; drug eruptions; nitrogen content of the blood; respiratory movements in hiccough; colloidal nitrogen of the urine, and initial eruptions in measles.

Clinical tables have been added on the following subjects: Bloody sputum; dyspnea; hemorrhage from the mouth; abdominal enlargement; vomiting; ascites; splenic enlargement; hematuria, and bacteriuria. Among the subjects that have been rewritten are Stokes-Adams disease; blood-pressure; ulceration of the duodenum; Addison's disease, and anterior poliomyelitis.

We are especially indebted to Dr. Francis Ashley Faught for assistance in connection with the chapter on blood-pressure, and to the publishers, W. B. Saunders Co., for special courtesies, and, most of all, for their untiring devotions to the interest of this book; features which can only be appreciated by those responsible in the capacity of its authorship.

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PREFACE.

THE present volume is offered to the medical public at the repeated solicitations of both practitioners and undergraduate students. The special purposes of the authors have been primarily to furnish an improved method of determining the clinical features of disease, so that all of the more important symptomatic phenomena in a given case may be collected with ease and certainty, and to emphasize the importance of corollating symptoms with the structural changes on which they are dependent and their organismal etiology. It is confidently believed that a knowledge of the laws of disease thus gained, combined with personal experience, will prove the best guide to accurate diagnosis, and obviate the danger of being sidetracked by non-essential evidence. This method, which brings the entire organism under consideration, of investigating disease, as outlined in the introductory chapter of this work (vide infra), will render the question of individualization of cases, a prime requisite, free from serious difficulty. Moreover, it will provide a sure and proper basis for rational treatment.

The method herein advocated will forcibly encourage painstaking, thorough, and scientifically accurate investigation of disease, and it will more than compensate for the indifferent and embarrassing results of mere superficial observations of cases, which, be it remembered, can never carry an observer to eminence as a diagnostician. Additionally, the authors have aimed to present, consistently with a single volume text-book, the full modern resources of the art and science of medicine as related to medical diagnostics.

The new features, which it is hoped will commend themselves to professional favor, are the brief pathologic definitions of special diseases, the illustrative cases selected from those actually observed in the hospital and private practice of the authors, and the numerous diagnostic tables, designed to aid the student and practitioner in contrasting the distinguishing signs and symptoms of diseases which bear a close clinical resemblance to one another. Here should also be mentioned the sub-headings, *Summary of Diagnostic Features* and *Laboratory Diagnosis*, which occur in connection with the individual complaints described. The text is profusely illustrated with photographs and colored plates, with a view to facilitating the reader's grasp of the technic of the more refined methods of diagnosis.

Our best thanks are hereby extended to Dr. John M. Swan for kind aid rendered in connection with the task of proof-reading, to Dr. T. H. Weisenburg for preparing the section on diseases of the nervous system, and to Dr. George E. Pfahler, who furnished the subject-matter relating to Röntgenology. Finally, our thanks are due the publishers for much courtesy and kindly interest manifested while the volume was passing through the press.

JAMES M. ANDERS,
L. NAPOLEON BOSTON.

PHILADELPHIA, PA.

CONTENTS.

DISEASES OF THE LUNGS.

DISEASES OF THE PLEURA.

DISEASES OF THE CIRCULATORY SYSTEM.

DISEASES OF THE PERICARDIUM, HEART, AND BLOOD-VESSELS.

DISEASES OF THE PERICARDIUM.

DISEASES OF THE ENDOCARDIUM.

VALVULAR DISEASE.

DISEASES OF THE MYOCARDIUM.

DISEASES OF THE BLOOD-VESSELS, MEDIASTINUM, AND THYMUS GLAND.

THE BLOOD.

DISEASES OF THE DIGESTIVE SYSTEM.

DISEASES OF THE LIPS, TONGUE, AND MOUTH.

THE ESOPHAGUS.

THE STOMACH AND INTESTINES.

DISEASES OF THE STOMACH.

THE INTESTINES.

DISEASES OF THE PERITONEUM.

THE LIVER.

THE PANCREAS.

THE SPLEEN.

DISEASES OF THE URINARY SYSTEM.

THE BLADDER.

ACUTE INFECTIOUS DISEASES.

ANIMAL PARASITIC DISEASES.

CONSTITUTIONAL DISEASES.

DISEASES OF THE NERVOUS SYSTEM.

By T. H. Weisenburg, M.D.

INTRODUCTION.

GENERAL AND SPECIAL CONSIDERATIONS.

Diagnosis is the discrimination of diseases by their distinctive symptoms. In its legitimate scope, however, diagnosis appeals most strongly to pathology for an elucidation of morbid processes, and to general pathology—morbid physiology—for an interpretation of the symptoms or abnormality of function resulting from structural changes. Moreover, it takes account of normal anatomy, physiology, chemistry, and physics. With these varied aids, it attempts to decide the seat and nature of disease, as well as additional themes of inquiry, by establishing the connection between cause and effect—between special pathologic processes and their symptomatic manifestations. Diagnosis, therefore, inquires into many branches of medical science, and a divisional study of so complicated a subject is demanded to achieve satisfactory results.

In endeavoring to trace the clinical features of a given disease to their source, use is made of the physical signs and modern laboratory methods of investigating disease, as well as of all data relating to the anamnesis. The marks of disease are often decidedly obscure, and for their detection the diagnostician must call into requisition the various instruments of precision contributed by science, *e. g.*, the microscope, stethoscope, hemomanometer, and many others, as well as the helps furnished from the laboratory expert. A broad conception of the subject of diagnosis recognizes both clinical and laboratory methods and regards them as being equally important in the investigation of disease. While, in a given case, it may appear that the older clinical methods alone suffice for a correct diagnosis, the practical aid furnished from the laboratory renders the natural history and the clinical distinctions of the complaint in question more intelligible. Not only are knowledge and experience with chemical and biologic methods essential to the armamentarium of the broadly trained clinician, but he often finds the data available through such methods the only means of enabling him to reach an accurate conclusion, and in general they serve admirably as aids or checks. The equipment of the present-day diagnostician, therefore, comprises trained powers of observation and accurate clinical experience,—the corner-stone upon which diagnosis formerly rested,—coupled with the necessary acquaintance with the chemical and biologic methods to enable him to interpret their results. Any attempt, however, to replace a thorough anamnesis and careful, systematic physical examination by the laboratory findings, of whatever nature, is to be strenuously deprecated.

The principal symptoms of most diseases may be allied to numerous other morbid entities, so that the correct assigning of significance to indi-

vidual features presupposes a knowledge of the varied pathologic states from which it may proceed. Single symptoms must, therefore, be weighed in the light of the attending phenomena and their causes—either textural or functional. The department of medicine consisting in a study and description of the pathologic changes in disease cannot obviously be considered in this work, except brief reference to the same under the head of definition, for want of space, but it is the solid groundwork by which diagnosis is supported. With the thorough and vigorous methods to be carried forward at the bedside, therefore, studies in morbid anatomy in the deadhouse are to be constantly associated. The clinician who is at one and the same time a morbid anatomist is preëminently well equipped to clear up many obscure problems in clinical diagnosis.

A complete diagnosis also embraces the stage and variety of the disease in question and an explanation of the habits, occupation, age, and temperament of the individual. In other words, diagnosis in a broad sense discerns the status reached by the pathologic changes of the affection which has been recognized, and the complicating conditions which may be present. In this manner alone can individualization of special cases, which is the prime object of bedside diagnosis, be accomplished.

The physician is also called upon to make multiple diagnoses, in which instances it is often with exceeding difficulty that the primary and secondary affections are determined. Fortunately, the list of diseases of uncertain origin is steadily becoming smaller, thanks to the rapid advances of bacteriology, and multiple infecting microbes can be justly incriminated with causing the development of a primary and secondary affection in one and the same case. It is to be recollected that the most striking local and general features in such hybrids may be occasioned by some besetting complication, the underlying primary condition being either in great part or wholly veiled by the symptomatic disturbances set up by the secondary invaders.

To determine the primary disease in cases of mixed infection, it is helpful to recollect the fact that the process of secondary infection is often due to the streptococcus, while less commonly the streptococcus and the colon bacillus manifest their deleterious action under conditions of diminished resistance brought about by the original complaint.

Moreover, the testimony gained by a clear history, together with the data furnished by the laboratory, may suffice to put the clinician upon the right path. A critical analysis of the symptoms from the beginning, more especially if considered with reference to their histologic causes, may show two distinct pathologic conditions present, and indicate their development chronologically. In this connection it is to be observed that we are often called upon to deal with conditions presenting different pathologic forms rather than distinct pathologic entities. Facility to recognize special morbid processes, whether single or multiple, is acquired only through long, patient, conscientious study at the bedside, and the degree of success attained by the physician as a diagnostician is directly in proportion to the perfection of the technic employed, the extent of his knowledge of the scientific branches of medicine, and the proper utilization of the experience derived from previous investigations of similar diseases.

The collection of a sufficiency of data must be followed by a careful sifting of the clinically important features from the ensemble of data. Not uncommonly the experienced diagnostician evolves the diagnosis while carrying forward the details of his examination, but the deductions reached

in this manner need confirmation. It is safer, and it is here recommended, to defer the final conclusions until he has the completed fund of data for inductive purposes, except in those rare diseases in which a certain diagnosis rests principally upon a pathognomonic feature. There are a number of infective diseases that usually do not recur during the life of the individual, *e. g.*, typhoid fever, measles, whooping-cough, scarlatina, and variola. We should be guarded, therefore, in the matter of arriving at the diagnosis of second attacks of these diseases when the evidence of the occurrence of a primary attack in the past is convincing.

Hasty generalizations from partial or defective investigations of disease too commonly lead the observer to hold untenable ground in the field of diagnosis, and as certainly lead him to adopt disappointing lines of treatment. The scientific study of disease from the standpoint of diagnosis can never reach the high plane which its dignity and importance demand without receiving the continued impetus resulting from the combined application of all the known laboratory and clinical methods at our disposal.

There is demanded a close examination of the particular parts—a process of thought in which the value of each symptom, sign, or laboratory finding, as the case may be, must be estimated deliberately and weighed against other features. A judicious balancing of the important diagnostics is then required preparatory to final induction by correct reasoning. After the examiner has gained a certain amount of experience in the taking of an anamnesis he can, during the process of eliciting the facts pertaining thereto, discriminate the more essential phenomena and disregard those that are of least significance, in order that the details shall not become too massive for ready inference. On the other hand, the beginner should note all data, however insignificant, lest important clues to more or less obscure conditions present be overlooked. In not a few cases the most experienced clinician, after forming a provisional judgment from the anamnesis, is forced to abandon it for another after he has noted the pathologic physical signs and obtained all the available laboratory findings.

The physician is thus competent to recognize a disease—to evolve a diagnosis—but not with certainty until, with the aid of morbid physiology, he has correlated the symptoms with the bacteriologic and pathologic causes to which they owe their origin. A diagnosis formulated and founded in the manner indicated above becomes the sure foundation of rational prognosis and effective therapeutics.

It should be an invariable rule when the diagnosis is made to still consider the possibility of the existence of a combined condition or affection, so common are complications and associated diseases. Indeed, in many cases it is impossible for the physician to form a diagnostic judgment without making a study of the distinctions between a suspected or recognized disease and others that present points of striking similarity. Under these circumstances the pathognomonic diagnostics are golden, and should be searched for diligently. This phase of our subject, known as differential diagnosis, often occasions difficulty, and it is discussed at considerable length throughout the pages of this work in connection with individual affections.

There are a few methods of investigating disease which should be considered connectedly in this place; they are the Röntgen, cultural, chemical, and serum methods, and the opsonic index.

The Röntgen method has become almost universally applicable, and

in many cases will give the most accurate information that is obtainable in the living subject. It should, however, be looked upon simply as one of the means of diagnosis, and all other methods should first be utilized, as a rule, after which it can be used to the greatest advantage in clearing up obscure points in the diagnosis. It is usually a waste of time and energy to employ the Röntgen method as a routine practice.

The greatest field of usefulness of the *x*-rays in medical diagnosis is in studies of diseases of the chest and abdomen, although the method is by no means to be confined to these regions. In practically all diseases of the lungs some new light is afforded. In the study of the heart and blood-vessels the size, shape, and movements of these organs can be observed.

In diseases of the abdominal organs the size, form, position, motility, and mobility of the stomach can be determined, as well as the effects upon this organ of the movements of the diaphragm and the contractions of the abdominal wall; the time required for the food to pass through the small intestine can be recorded; the size, form, and position of the colon can be observed; and any form of obstruction along the alimentary canal can be studied and much information as to its character be obtained.

Renal, ureteral, and vesical calculi can be definitely located, and under favorable circumstances biliary calculi can be also photographed.

In all diseases caused by specific organisms the isolation of the special microbe and the determination of its biologic properties is the most certain and rigidly scientific method of arriving at a diagnosis. Unfortunately, in many infective complaints the cultural method is too difficult for ordinary clinical purposes. In a considerable number of diseases an accurate diagnosis is established by the mere demonstration of the presence of the organism, *e. g.*, malaria, lung tuberculosis, amebic dysentery, relapsing fever, lepra, diphtheria, Vincent's angina, gonorrhea, and the like. In other microbic affections the specific organism must also be isolated in pure culture, as typhoid fever, the paratyphoid fevers, pneumonia, tetanus, the plague, influenza, cholera, and many others. In some cases it is further necessary to prove the pathogenicity of the organism isolated by resorting to animal inoculations, although the necessity for this step has been in great part removed by the more recent introduction of serum methods. It is sometimes more convenient to inoculate an animal with a pathologic exudate or an excretion from the body of the sick (*e. g.*, pleural effusion) than to employ an isolated organism in the same manner. The feeding of an exudate, of tuberculous origin, to animals is also of service.

Serum diagnosis (like serum therapy) rests upon a knowledge of the bactericidal substances (antibodies) developed in the blood in the presence of a bacterial excitant. The antibodies are of two kinds: namely, those that defend the human organisms against the toxins of bacteria, *antitoxins;* and those that defend it against the bacteria themselves, *agglutinins* and *lysins*. The first application of the specific serum reaction to diagnosis was in typhoid fever, and in this disease the agglutinating property of the serum develops sufficiently early to be of great diagnostic value. A similar reaction is also of the highest importance in the recognition of so-called paratyphoid fevers. In the case of other bacterial diseases—*e. g.*, dysentery, tuberculosis, the plague, lobar pneumonia, glanders, Asiatic cholera—in which the serum method has been employed, it has not attained to an assured place as a diagnostic measure, either because the reaction is not sufficiently constant or reliable, or because it appears too late to be of diagnostic significance. It must be recollected, however, that sero-reac-

tions are not equivalent to the direct demonstration of the specific bacteria. On the other hand, on account of their ready application, the serum methods possess a distinct advantage over the cultural methods. But, though the diagnosis will usually have been made in these affections before a positive reaction is obtainable, the serum test, like the Röntgen rays, should be employed, even as late as the period of convalescence, to clear up dubious cases. Thus, it may serve to distinguish between typhoid fever or paratyphoid fevers and other febrile affections. Recent progress in serum diagnosis also includes reactions for the detection of hemolysis and conditions depending upon syphilis. The test recently suggested by Wassermann, and its modification by Hideyo Noguchi, for syphilis, deserve mention in this connection.

The rôle which the opsonic theory plays in medical diagnosis deserves brief notice in this connection. It has been known that the opsonins in the serum of healthy persons or those infected with microörganisms stimulate phagocytosis by making the bacteria more readily susceptible to inclusion in leukocytes, and the relative degree of phagocytosis bears a definite relation to the quantity of these substances present. Those cases of infective disease which on repeated examination show a lowered opsonic index to any organism—as the tubercle bacilli—are supposed to be infected with that organism. In case two or more organisms are present, that toward which the present opsonic index is lowest is probably the most important in causing the disease.

The opsonic index is further of practical aid in that it affords a clear idea of the patient's resisting powers and of the degree of his susceptibility to secondary infection; it thus likewise becomes of signal prognostic significance. For example, a staphylococcic infection showing a long-continued negative phase following injection gives little promise of being curable. Further improvement in the technic of determining quantitatively the opsonic power is greatly needed, with a view to diminishing the range of error, which up to the present has been rather extreme.

The employment of the opsonic method by physicians skilled in laboratory methods, however, may be regarded as a certain advance in diagnosis, although it is of vastly more value as a guide to therapy by vaccination methods.

Undoubtedly, the application of the chemical methods of investigation to the determination of diagnostic indications, particularly in diseases of the kidneys, stomach, and pancreas, has been decidedly helpful. The uses of this method, in relation to diseases of special organs, will be considered hereafter in appropriate connections in this work. Our modern knowledge of diseases of metabolism rests upon the results of the earlier chemical studies of the organic functions, and in this field of chemical investigation recent deductions have taught us practical lessons of far-reaching importance. Among these are to be noted the well-established fact that an increase of urinary uric acid is not due to increased excretion of that substance, but rather to urinary conditions antagonistic to the solution of uric acid and its compounds.

As regards carbohydrate metabolism, it may be said that chemical methods have assumed marked diagnostic importance in furnishing us the means of detecting sugar in the urine, and also those for distinguishing between sugar and other reducing bodies, as glycuronic acid, the alkapton bodies, and pentoses. Chemical methods have enabled us to appreciate the conditions resulting from a perversion of fatty metabolism. Thus, in some

diseases—*e. g.*, diabetes mellitus, the acute infections, and others—certain products of the katabolism of fat, as acetone, oxybutyric acid, and diacetic acid, are present in the urine. These substances may be met with in abnormal quantities due to increased destruction of fat, and may give indications of approaching complications of a serious character, which if recognized can often be obviated by appropriate therapy. It is a matter of keen regret, however, that the facilities for the application of reliable chemical studies to the investigation of disease are not so readily available for the benefit of the general practitioner as are bacteriologic methods.

Additional aids, furnished by the biologic method, are those known as deviation of the complement and bacteriolysis, but since their accuracy and value remain to be demonstrated, further discussion is deemed unnecessary. Among special biologic methods, blood culture is one of the most valuable. The bacteriologist can isolate specific organisms from the blood and thereby promptly establish an indubitable diagnosis; the method has been successfully applied to the differentiation of typhoid fever from the recognized forms of paratyphoid fever when the more usual distinctions fail. In general pneumonic infection and the various septicemic conditions, the results of blood culture may be the only available data by means of which the true nature of the condition can be established.

The most illuminating teaching of internal medicine is that which succeeds in demonstrating facts relating to the causes, symptoms, and physical signs, by laboratory study. This experimental method has already been applied for the verification of the physical signs in pleurisy (Opie), in the case of cardiac murmurs (McCallum and Thayer), and the accurate measurement of the blood-pressure in diseases or conditions artificially produced, *e. g.*, the exalted tension from the use of adrenalin, experimentally. There is need of more experimental laboratory work as applied to the analytic study of symptoms, with a view to supporting or refuting the conclusions based on clinical observation.

One of the principal objects of this work is to furnish a reliable guide to treatment in the widest sense. To this end a proper classification of the subjects treated is of the utmost importance from the viewpoint of both diagnosis and treatment. It is diagnostically useful to know that a certain disease belongs to a group of diseases presenting marked similarity in the main pathologic changes on which the symptoms depend. Such knowledge often points the way for the conduct of a judicial consideration of the differential diagnosis of the given case. In the same line of thought, a recognized member of the infectious class of diseases, more especially if atypical, calls for the closest scrutiny with reference to other affections belonging to the same category, as a rule. For example, if we were to regard typhoid fever as an intestinal disease, the general infections most likely to be confused with it, and needing to be considered in connection with the differential diagnosis, would be in danger of escaping our attention.

A classification based upon the nature of the pathologic process involved also enlightens us respecting the line of treatment to be pursued, *e. g.*, in any existing toxemia certain leading indications demand fulfilment, but if perchance an infective disease should be classed with a group of local complaints, these indications might not receive due attention. In general terms, then, the principle holds that an appropriate classification on a pathologic basis is to be adopted for both diagnostic and therapeutic reasons.

INVESTIGATION OF INDIVIDUAL CASES.

Coming now to the question of the investigation of individual cases with a view to the recognition of human diseases in their multitudinous forms, two things are essential: (*a*) Close observation and scrutiny of the symptoms and signs and the utilization of the laboratory resources, according to the most approved method of conducting an examination; and (*b*) the harmonizing inductively of the essential features and data with a mental picture of definite morbid states or entities.

(*a*) Nothing is more important for the student or practitioner than the adoption and rigorous enforcement of a method or system in taking up the study and examination of special cases. This mode of procedure insures the accumulation of the largest possible mass of clinical data and minimizes the necessity for making a diagnosis by mere inference—always an uncertain product. The clinician who, during the earlier years of his professional career, unswervingly adheres to a proper system of investigating cases will meet with a progressively smaller number in which questions regarding diagnosis must remain *sub judice*. The well-poised, calm, logical practitioner, skilled by a long process of self-imposed training, and armed with sharpened perceptivity and an extensive experience, may often infer a correct diagnosis from a limited number of essential facts, but this is a license which few have a right to enjoy.

For every induction is apt to be faulty that does not proceed from full information gained by a thorough and systematic objective investigation, coupled with the result of accurate clinical observation.

While it is impossible for any busy practitioner to keep abreast with the kaleidoscopic details of chemical and biologic technic, he should, with a view to becoming a trained clinical investigator, supplement bedside observation by familiarizing himself with the use of the microscope and other instruments of precision, as well as utilizing the advantages offered by even a small private laboratory. The clinician who is a trained microscopist and avails himself of the practical advantages of the bacteriologic laboratory can often make an etiologic diagnosis which no amount of clinical testimony can shake. At all events, every progressive physician must acquaint himself with the fundamental basis on which laboratory methods rest, since a ready interpretation of the results reported from public laboratories is a dominating necessity.

The method of noting down all data gained in study of a given case is conducive to precision of results and amplitude of view. The important matter of comparing one case with another is also thus facilitated.

As regards the best method of conducting an examination, it may be stated that the details of the procedure should be arranged under various heads.

It is one of the objects of this work to teach the method of conducting an examination of patients, and to that end we submit the subjoined scheme, believing that if its structural details are rigidly and systematically pursued, the results will be in a form that shall be of the greatest practical value to the examiner.

The object of the plan is not merely to enable the student and physician to render a diagnosis with precision, or to quicken his intellectual acumen, but to afford them an essentially practical knowledge whereby the indications—more especially those that spring from causative conditions and agencies—for the amelioration and cure of the complaints which are recog-

nized may be comprehended. The highest aim of diagnosis must be to furnish a key to the successful treatment of each case or disease studied. The early recognition of a disease is highly desirable, in order that the physician may be thereby enabled to forecast its probable course and issue.

Besides, the physician is under obligation to act solely in the interests of his patient.

Under an appropriate schedule the examiner is sure to discover facts which will necessitate remedial action, and this information must be carefully noted and subsequently utilized. In obscure cases reëxaminations are to be encouraged and advised, such secondary exploitations often resulting in a revision of opinion. Moreover, important light is often shed upon the diagnosis and treatment by the subsequent history and course of special cases.

In taking the anamnesis it has been found impracticable to follow a definite line of procedure in all cases. Among the most intelligent classes the patient may be allowed to tell the story of his illness, being merely guided by the examiner, but in most instances it is best to obtain the major portion of the historic data by appropriate questions. It is generally conceded, however, that leading questions are to be rigorously avoided, since it is an easy matter to modify the answer by the way in which the query is propounded. This form of questioning also invites exaggerated replies, especially in the case of such familiar symptoms as pain, cough, insomnia, and the like. We must also guard against erroneous answers, either wilfully (for the purpose of deception) or unintentionally made; although if we except markedly hysterical females and an occasional malingerer among males, feigned diseases are rarely encountered in routine practice. More commonly, perhaps, the possible exciting cause, as well as the condition itself, is intentionally kept *sub rosa* by females from motives of delicacy, *e. g.*, when suffering from carcinoma of the mammary glands, hemorrhoids, and uterine disorders.

But though case-taking at the bedside in private practice among patients who are extremely weak or ill is not practicable in perhaps the majority of the cases, the examiner should pursue the same general order of procedure in his interrogatories, simply omitting certain details so as to lessen the number of replies. The physical examination may have to be briefer than seems desirable, for similar reasons.

SCHEME FOR HISTORY-TAKING, PHYSICAL EXAMINATION, AND LABORATORY FINDINGS.

Name: *Address:* *Date:*

Family History. Previous History. Social History. Present Illness.

Physical Examination.

General Examination:

1. Posture:
2. Age—Actual: Apparent:
3. Weight: Height:
4. Skin and Mucous Membrane:
5. Edema:
6. Adiposity or Emaciation:
7. Glands:
8. Muscles:
9. Bones and Joints:
10. Psychic State:

Local Examination:

1. Head:
2. Eyes:
3. Mouth and Pharynx:
4. Nose:
5. Ears:
6. Larynx:
7. Neck:
8. Thorax:
   Lungs:
   Inspection: Palpation: Percussion: Auscultation:
   Heart:
   Inspection: Palpation: Percussion: Auscultation:
   Pulse:
   Blood-pressure:
9. Abdomen:
   Liver and Gall-bladder: Spleen: Stomach: Intestines:
   Kidneys and Bladder: Genital Apparatus:
10. Laboratory Findings:
11. *X*-ray Findings:

For purposes of clinical teaching it is useful to record the principal complaint of, and symptoms presented by, the patient, and to note the obvious physical signs on admission to the hospital, and from these phenomena to make a provisional diagnosis. This method often enables the student to appreciate more fully the data collated by the systematic examination which is to follow. In every-day practice, as with the undergraduate student, however, final judgment must rest upon a careful grouping of all data of diagnostic value afforded by a comprehensive description.

We shall now consider in detail the different elements entering into the above schematic outline, passing over any application of the same to diseases of special organs or systems of the body, since this phase of the subject will be treated of directly in connection with the various sections of the work.

Family History.—This aims at the detection of hereditary diseases in the antecedents or members of the immediate family. It is to be recollected that true inheritance of infectious diseases is rare. It is possible, however, that the toxins of such disorders or the effects of the growth of pathogenic organisms acting upon the germ-cells may influence the parental organism and lead to the offspring becoming modified in its development in one or other particular direction (Adami). After an individual begins its existence in utero, any modification is to be looked upon as of ante-natal acquirement.

Certain metabolic disorders, *e. g.*, gout, obesity, diabetes mellitus, and rheumatoid conditions, are probably attended with impaired nutrition of the germ-cells, thus affording an explanation of "the development and inheritance of diatheses."

Our inquiry should be extended to parents, the grandparents, brothers and sisters of the parents, and brothers and sisters of the patient. If the patient have children, inquiry regarding them is also to be made.

Previous History.—The inquiry should be directed first to any infectious diseases of infancy and childhood, more especially measles, whooping-cough, scarlet fever, diphtheria, and follicular tonsillitis; next in chronologic sequence to the previous occurrence of other infections, as variola, typhoid fever, malaria, erysipelas, tuberculosis, and syphilis; chlorosis in the female.

The age at which past complaints occurred, their duration, severity, complications, character of convalescence, and whether or not complete recovery ensued. Any previous illness like the present; if so, an analysis of the symptoms and the course. Antecedent injuries.

Social History.—Whether bottle- or breast-fed. Note the age, sex, married or single, profession or occupation (now and in the past), place of residence, temperament; if female, the condition of the menstrual function from the time of puberty to the present, giving details; if married, the number of pregnancies, childbed—normal or complicated, and if so, the character of the complication, and whether forceps were used; any sequelæ; version or operation (specifying), miscarriages, noting peculiarities and results; pelvic operations, if any; time and nature of same. *Habits* regarding exercise, its character and amount, whether systematic or irregular; habits of eating, as to mastication, regularity or irregularity of meal-time, character of the food employed, time allowance for eating; use of stimulants, as tea, coffee, malt and spirituous liquors, with details as to duration, quantities consumed, and hours of the day when taken.

Present Illness.—The first point of information to be gained is the date of onset; this will indicate to the mind of the examiner whether the patient is the victim of an acute or a chronic disorder. We next inquire as to what the patient thinks is the cause of the illness (catching cold, trauma, etc.), the precise mode of onset, sudden or gradual, noting first the initial symptom or symptoms, their character and order of development. The attention is apt to be directed to particular organs or systems of the body by the patient. These should be interrogated, and afterward the remaining systems in a similar manner seriatim, observing the points set forth below.

Nervous System.—Pain? Its location, nature, severity; whether constant or paroxysmal? Other sensory and motor disturbances: Headache? Insomnia? Emotional alterations? Depression of spirits? Any disturbance of the mental faculties?

Respiratory Apparatus.—Pain, its location? Excited by breathing, coughing, or movements? Dyspnea, constant or paroxysmal? Induced by exertion or other exciting causes? Rate of respiration (also during paroxysm)? Cough, constant or paroxysmal? Time and duration of coughing attacks? With or without expectoration? Character and daily amount of sputum? Its color, from admixture of blood? Consistence and other peculiarities?

Circulatory System.—Pain in precordium (left arm, neck, back)? Exciting causes of pain (exertion, mental excitement)? Palpitation, continuous or paroxysmal? Accompanied by mental apprehension, or pain, or dyspnea? Apparent excitants of paroxysms? Effect of tobacco? Effect of heavy meals? Any irregularity or intermittence (skipping) of heart-beats?

Stomach.—Pain, constant or intermittent? Precise time of onset of the pain, before or after food, or during the night? Effect of the ingestion of food on the pain? Its location, character, and radiations, if any? Appetite? Nausea, with or without vomiting? The appearance (blood, mucus, and undigested food), amount, and character of the vomitus? The precise time of vomiting, in relation to food taken? Eructations of gas or liquid (acid, bitter)?

Intestines.—Pain, its precise location, character, and particular radiation (whether downward to thigh, to back, etc.)? Constipation? Diarrhea? Number of bowel movements daily? Accompanied with pain, tenesmus?

Character of the discharges (mucus, pus, blood, scybala)? Color, amount, and consistence of feces?

Urinary System.—Pain, constant or paroxysmal in region of kidney or bladder? Primary seat and radiation of pain? Approximate daily quantity of urine? Frequency of urination? Accompanied with pain or tenesmus? Naked-eye changes of the urine? Is it turbid, smoky, or bloody? Amount and gross appearance of sediment on standing? Have stones or sand been passed?

Other Features.—Weakness? Loss of weight? Fever (temperature)? Night-sweats?

PHYSICAL EXAMINATION.

General Examination.—1. Posture. Sitting up, semirecumbent, or lying in bed. Decubitus (dorsal, ventral, or lateral). Whether position of body is fixed. Spine bent. Knees drawn up. Lying fixed on side.

2. Age—actual and apparent.

3. Weight and height.

4. Skin and mucous membranes: Among the points to be noted are the state of skin with reference to temperature (see fever), dryness, or moisture. If the cutaneous surface be moist, the degree of coldness should be recorded, since a cold, wet skin is ominous, especially if prolonged. The skin may be tense or relaxed and lying in folds even (*e. g.*, in diseases attended with marked emaciation). Note the color of the skin, whether pale, sallow, yellowish (as in jaundice); gray, caused by silver nitrate; abnormally red without cyanosis; also any marks, scars, swelling, or active eruptions which may be present. Cutaneous hemorrhages, as puncta, petechiæ, and ecchymoses occur. Small hemorrhages are most apt to appear at the hair-follicles. These fail to disappear upon pressure, and are thus distinguishable from a minute area of inflammatory redness.

The condition of the lymph-glands (size, consistence, degree of mobility, etc.). Bones and joints. Conditions of muscles (atrophy, hypertrophy, tonus of the muscles, trembling, chorea, athetosis); resistance of limbs toward passive movements (grasp, walking, standing, Romberg's phenomenon), reflexes (tendon reflexes, skin reflexes), condition of general nutrition (adipose, emaciation).

Psychic behavior, intelligence, consciousness (delirium, stupor, mental dullness, coma), speech (stuttering, aphasia).

Local Examination.—**Head.**—*Skull:* Note size and conformation; condition of hair.

Face: Expression, facial muscles, mobility of both halves, of eyelids, laughing, frowning.

Ears: Note condition as to hearing, any discharges, effect of pressure on mastoid process, and external ear.

Eyes: Conjunctiva (color, discharges); pupils (color, pupillary changes, reaction to light, convergence and accommodation, muscular action).

Nose: Note its size, shape, any obstructions in the nasopharyngeal ring, discharges.

Mouth and Pharynx: Condition of buccal mucosa, that of pharynx, of tonsils (ulcers, scars, swellings), masticating apparatus, and tongue (size, whether coated, protruded, straight, with or without tremor).

Larynx: Inspection with the laryngoscope if there be hoarseness or other symptoms pointing to involvement of this organ.

Neck: Length, circumference, thyroid gland. Note any tumor-masses, scars, eruptions, pulsations (whether venous or arterial).

Esophagus: Swallowing, obstruction to passing sound.

Thorax.—*Lungs:* Inspection: (*a*) The appearance of the external surface, evidence of emaciation, prominences, etc.; (*b*) the shape and size; (*c*) movements and degree of expansion (diseased side takes less part in breathing); and (*d*) fluoroscopic study.

Palpation: (*a*) The principal results of inspection are confirmed; (*b*) the tactile fremitus is elicited; this should be tested over every portion of the chest occupied by the lung; and (*c*) fluctuation may be detected, though rarely.

Percussion: (*a*) Immediate percussion; (*b*) mediate percussion, which is divided under three subheads: (1) Finger percussion, (2) finger-pleximeter percussion; (3) human-pleximeter percussion. (For the technic of these methods see Diseases of the Lungs, page 55.) Note results of percussion with reference to pitch, volume, length or duration, and quality of sound. (It is to be recollected that when the vibrations are slow, the pitch is low and vice versa.) Comparison of apices of the lungs; respiratory changes of the lung boundaries; interpretation of sounds—normal resonance (with modifications in health, according to age, the region percussed, and associated conditions), tympany, relative dullness; respiratory percussion (the patient holding the breath at full inspiration and full expiration); auscultatory percussion; palpatory percussion; amphoric resonance; special signs—bell tympany, cracked-pot sound, Wintrich's sign, Gerhardt's sign.

Auscultation: Methods—(*a*) immediate, (*b*) mediate (for advantages of each method and technic see Diseases of the Lungs, page 63); modifications of normal respiratory sounds (variations in vesicular breathing, bronchovesicular, bronchial or tubular, cavernous, amphoric); changes in vocal resonance—diminished or increased (bronchophony, egophony, pectoriloquy, amphoric whisper, etc.); adventitious sounds, râles (sibilant, sonorous, crepitant, subcrepitant, mucus, rattling sounds), friction rub.

Heart: Inspection: Seat, quality and rhythm of apex-beat; abnormal pulsations elsewhere over chest-wall, especially over first and second intercostal spaces; epigastric pulsation; cyanosis.

Palpation: Seat, quality, and rhythm of impulse; presence or absence of thrill.

Percussion: Note area of cardiac dullness (effects of respiratory movements, effect of change of position).

Auscultation: The mediate method to be preferred; note any modifications of normal heart sounds—first and second; if adventitious sounds be audible, note their point of maximum intensity, rhythm, area of transmission and quality; auscultate—(*a*) mitral area, (*b*) tricuspid area, (*c*) aortic area, (*d*) pulmonary area, (*e*) over vessels of neck, (*f*) over body of heart, (*g*) effect of exercise, (*h*) effect of change of position.

Pulse: (1) Condition of right and left radial artery (rigidity, tortuosity); (2) rate of pulse; (3) rhythm; (4) force; (5) tension; (6) size.

Blood-pressure.

Vascular System.—(1) Arteries; (2) veins; (3) capillaries; (4) lymphatics.

Liver and Gall-bladder.—Palpation and percussion (auscultation of gall-bladder if calculi are suspected).

Spleen.

Stomach and Bowels.—Inspection: Shape, depressions, and swellings.

Palpation: Tension, hernias, fluctuations, splashing sounds, painful spots, or diffuse tenderness.

Percussion: Variations from normal tympanitic notes (muffled tympany, dullness); size and outline of stomach (if necessary, after inflation with air or by *x*-ray examination); if necessary, stomach-tube and examination of stomach-contents; inflation of intestinal tract with air if necessary to detect tumor masses or obstructions and the like; examination of anus and rectum.

Kidneys.—Palpation and percussion of the kidneys (see Diseases of the Kidneys, page 635); palpation and percussion over the bladder (urination, retention).

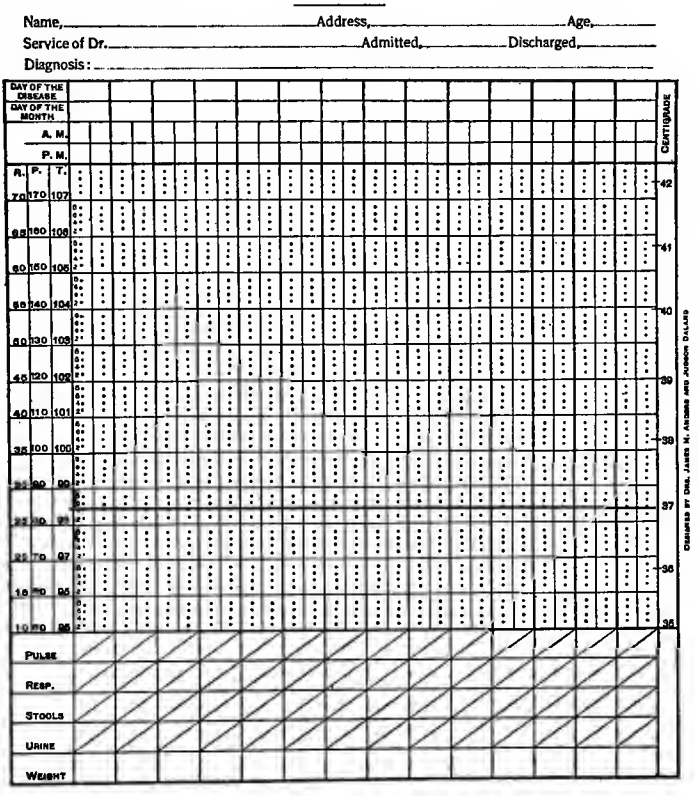


FIG. 1.

BEDSIDE OBSERVATIONS.

Additions to the findings on physical examination and bedside observation should be made from time to time under date; the temperature, pulse, and respiration being expressed in curves on the temperature chart; the daily quantity of urine, bowel movements, and weight at intervals are also to be recorded on this sheet. The subjoined temperature chart (Fig. 1) will assist the physician in keeping a complete record of further observations and examinations after the initial investigations into the case have been concluded.

Hand in hand with bedside observations must go certain laboratory investigations, for which the subjoined scheme may serve as a practical guide.

The Urine.—An examination of the urine is essential in all cases, independent of the character of the complaint in question. In every case the quantity for twenty-four hours, reaction, specific gravity, a microscopic examination of the sediment, and tests for albumin and glucose should be recorded. Should disease of the pancreas be suspected, Cammidge's reaction is needed; and in those suffering from obscure bone deformities, Bence-Jones albumose is to be considered. In suspected cases of typhoid fever the *diazo-reaction* of Ehrlich is a valuable aid.

In diseases of the kidneys the microscope is an all-important aid. A more complete analysis, *e. g.*, the estimation of the urea output, the detection of indican, acetone, diacetic acid, β-oxybutyric acid, bile pigment, and occult blood, and even a bacteriologic study of the urine may be of value in selected cases, *e. g.*, pyelitis, cystitis, tuberculosis, etc.

The Blood.—Generally speaking, an examination of the blood is demanded in those showing the general features of anemia, however slight. The determination of the number of corpuscles and of the percentage of hemoglobin is also of service in the diagnosis of obesity, pneumonia, and certain other sthenic maladies. A microscopic study of the individual red cells for the existence of poikilocytosis, irregularity in size and degeneration of the erythrocytes, is of great importance, and is best illustrated by the anemia of lead-workers. Important is it *to bear in mind that the number of red cells per cubic millimeter* may exceed the normal in persons suffering from an actual anemia *when there is associated cyanosis.*

In a study of the blood the most important information is obtained by the differential leukocyte count; next in order of value is the number of leukocytes in a cubic millimeter; and next, the hemoglobin percentage. Leukocytosis is found in pneumonia and the inflammatory processes generally; leukopenia in typhoid fever, malaria, and other diseases. In pneumonia the differential leukocyte count shows an increase of the polymorphonuclear neutrophiles; in infection with animal parasites, such as ankylostomiasis and trichiniasis, eosinophilia is frequently seen.

Cultural study of the venous blood will show pathogenic bacteria in acute ulcerative endocarditis, glanders, anthrax, typhoid fever, and other complaints. In certain diseases the sero-reactions are invaluable diagnostic acids—*e. g.*, typhoid fever. The sero-reaction of Wassermann is of the greatest diagnostic importance, especially in syphilis, which cannot be recognized by the ordinary examinations.

Sputum.—The naked-eye appearance of the sputum is of more value than that of any one other secretion or exudate, *e. g.*, bloody sputum is fairly characteristic of congestion or hemorrhage along the respiratory tract, the exceptions being where blood is cleared from the throat and from the posterior nares. Mention characteristic sputum of pneumonia, pulmonary cavity, bronchiectasis, etc. A clear watery (mucoid) sputum is fairly common in the incipient stage of pulmonary tuberculosis.

A microscopic analysis should be conducted whenever it is possible to collect an appreciable quantity of sputum, and in all cases in which there is cough (every sputum should be stained for the tubercle bacillus, and in suitable cases search for Curschmann's spirals, Charcot-Leyden crystals, and elastic fibers should be made). In all obscure cases a careful search for animal parasites should be made.

Gastric Fluid.—The presence and quantitative estimation of free hy-

drochloric acid, the total acidity, and reactions for the presence of lactic and butyric acids are always necessary. One or more tests should be employed for the detection of blood (occult bleeding), since bleeding may be so slight as to escape detection by other methods. An estimation of the combined hydrochloric acid should be made, as a rule, and the presence or absence of acetic acid, peptone, and bile should be noted. The quantity of mucus present should be carefully observed.

A microscopic study of the gastric fluid is of value in carcinoma of the stomach where it is customary to find *sarcinæ* and the *Boas-Oppler bacillus*, and indeed other microscopic findings of clinical value may be detected (*e. g.*, pus, erythrocytes). The degree of disintegration (breaking up) of starch cells after a test-meal gives a more or less accurate estimation of the activity of the salivary glands, and also of the degree of acidity of the gastric fluid; *the more hydrochloric acid present, the less are the starch cells broken.*

Feces.—The microscopic examination of the feces for the detection of intestinal parasites, their embryos, or their ova, and for the detection of protozoa is important. The passage of prepared meat balls through the alimentary tract to determine the destruction of nuclei of the meat cells should be conducted whenever pancreatic disease is suspected. A microscopic study for the detection of fat-globules in the feces is also necessary in the diagnosis of pancreatic disease. In typhoid fever, dysentery, gastric carcinoma, tuberculosis of the colon, gastric and duodenal ulcer, a positive reaction for occult blood is a most valuable sign. A bacteriologic study of the feces is of value in the determination of various pathogenic organisms.

Cultural studies are also necessary to recognize the bacillus of Shiga, streptococcus, staphylococcus, and other pathogenic bacteria.

Exudates.—Pus and all questionable exudates are only recognized through microscopic and bacteriologic studies. The determination of the cytologic elements of the sediment in pleural, peritoneal, and cerebrospinal fluids often gives important information. In view of our present knowledge of the modes of infection by the ankylostoma, a microscopic study of the exudate and scrapings from cutaneous ulcers may give valuable data.

DISEASES OF THE RESPIRATORY SYSTEM.

DISEASES OF THE NOSE AND THROAT.

LABORATORY EXAMINATIONS IN DISEASES OF THE NASO-PHARYNX.

Discharges from the nose and the throat may be studied as smears or by cultural methods. For the former method the mucus or pus, removed by a sterile swab or platinum loop through a speculum, is smeared in a thin layer on a clean glass slide, and allowed to dry in the air. After being fixed by passing three times through the flame of an alcohol lamp or a bunsen Burner, the smear may be stained with Loeffler's alkaline methylene-blue, by Gram's method, or by the method of demonstrating acid-fast bacilli. For the purpose of making cultures from either the nose or the throat, Loeffler's blood-serum is the most convenient culture-medium, although any of the standard media may be used. A small portion of the pathologic exudate is smeared on the surface of the medium with a sterile platinum loop or with a sterile cotton swab. Boards of Health issue outfits composed of a tube of Loeffler's blood-serum mixture and a sterilized cotton swab, contained in a pasteboard box, with a slip of cardboard for the necessary clinical data. These outfits are designed especially for the bacteriologic diagnosis of diphtheria and other pseudo-membranous inflammations of the throat; but they can readily be adapted to nasal discharges.

During health few bacteria are found in the nares, but in acute rhinitis the mucus contains numerous bacteria, many of which grow readily upon culture-media. The specific action of any particular bacterium in the production of acute rhinitis remains a question. Smears show many squamous epithelial cells, leukocytes, and at times red blood-cells. In virulent types of infection the nasal discharge may be composed principally of pus and epithelial cells, together with cocci and bacilli. During the course of certain specific infections the bacterium known to be the cause of the infection may be recovered from the nasal secretion. In cases of cerebrospinal meningitis an intracellular diplococcus is often found in the nasal mucus. The bacillus diphtheriæ is frequently found here in cases of active diphtheria and in cases of latent diphtheria. Bacillus pseudodiphtheriæ is often found in cases of acute rhinitis. Bacillus lepræ is said to be demonstrable in the nasal mucus in cases of leprosy before the other symptoms are sufficiently characteristic to permit of a positive diagnosis.

In chronic rhinitis the discharge is composed of yellowish or greenish crusts which have a fetid odor, and which are composed of epithelial cells, pus-cells, bacteria, and granular débris. The diplococcus ozænæ may be found in cases of ozena, and sarcinæ are sometimes present. In diphtheria (p. 861) and pharyngeal and laryngeal tuberculosis (p. 812) the characteristic organisms are found.

Suppurative inflammation of the accessory nasal sinuses is usually due to staphylococcus aureus, to streptococcus pyogenes, or to some other pus-producing organism.

In the diagnosis of diphtheria little confidence is to be placed on the examination of stained smears made from the pseudomembrane.

DISEASES OF THE NOSE.

ACUTE RHINITIS.

Pathologic Definition.—An acute catarrh of the Schneiderian membrane. This inflammation sometimes tends to involve the adjacent sinuses and passages. It is known to the laity as "cold in the head."

Predisposing and Exciting Factors.—Exposure to drafts and the influence of atmospheric vicissitudes that are especially prevalent during the winter and spring seasons are the most potent factors. Inhalation of irritants (physical, chemical, and biologic) is also capable of exciting inflammation of the nasal mucous surface. At times the disease may display some evidence of an infectious nature, and at such times may occur in epidemics. Direct extension from other mucous surfaces may be accountable for acute rhinitis.

General Complaint.—Sensations of chilliness, succeeded by feverishness, are common complaints. Frequent sneezing, headache, and malaise are prominent features, and there are apt to be experienced in unusually severe cases muscular pains in the back and extremities. Thirst is increased, anorexia complete, and constipation often attends.

One of the most annoying features to the patient is that of a somewhat profuse watery discharge from the nose, which is later followed by reddening and at times excoriation of the alæ nasi. Lacrimation is apparently increased, and is probably due to irritation of the mucous surface of the lacrimal canals. Adjacent mucous surfaces may also become involved, giving rise to conjunctivitis, catarrhal pharyngitis, laryngitis, and, in the severe forms, bronchitis. Nasolabial herpes may be present.

Physical Signs.—The pulse is frequent, the skin dry and unduly warm. The nasal mucosa is reddened and swollen, thus interfering with the sense of smell and taste, and with breathing. The color of the mucous surface is deepened. At first it is covered with an opaque mucus, and later with a muco-purulent secretion. As the affection progresses the secretion becomes more abundant and turbid.

Course.—In favorable cases all annoying symptoms begin to diminish from the third to the fifth days. The nasal discharge usually continues for a longer period unless controlled by medication.

Complications.—The nasal inflammatory process may extend to the pharynx, larynx, and at times to the trachea and bronchi, in which instance the signs and symptoms of involvement of these surfaces are conspicuous.

CHRONIC RHINITIS (CHRONIC NASAL CATARRH).

Pathologic Definition.—A chronic inflammatory process involving the nasal mucous membrane and consisting pathologically of two distinct forms: (*a*) *Hypertrophic,* in which there is enlargement of the lower turbinated bones, together with reddening and swelling of the nasal mucosa that may be general or limited either to the anterior or posterior nares. (*b*) *Atrophic,* in which there are atrophy and appreciable thinning of both

the nasal mucosa and underlying structures, which changes are followed by enlargement of the nasal cavities.

Principal Complaint.—(*a*) In the *hypertrophic* form nasal respiration is impeded, owing to the hypertrophy of the turbinated bones. The sense of smell is not acute, and there is a muco-purulent discharge from the nares, particularly the posterior, inducing "hawking."

(*b*) In the *atrophic* form the most conspicuous symptom is the peculiar odor of the nasal secretion, and the sense of smell is greatly diminished, or may even be destroyed.

Physical Signs.—These are obtained satisfactorily by the use of the rhinoscope, which reveals the actual condition of the mucous surface of the nasal fossæ, and upon this the diagnosis is based.

Exciting Factors.—The disease is thought to result from repeated attacks of acute rhinitis, or from acute involvement of the nasal mucous membrane that has not been successfully treated. Abel believes that the "bacillus mucosus ozænæ" is often an exciting factor in this condition.

DISEASES OF THE LARYNX.

ACUTE CATARRHAL LARYNGITIS (ACUTE ENDOLARYNGITIS).

Pathologic Definition.—An acute catarrhal inflammation of the mucous surface of the larynx.

Predisposing and Exciting Factors.—Exposure to cold and wet, excessive use of the voice, inhalation of irritating vapors, injury, excessive smoking, foreign bodies, and swallowing of corrosive substances are potent factors. The disease may be primary, but is more commonly associated with, and frequently follows, inflammation of the nose and pharynx.

Principal Complaint.—The voice is husky or completely lost; there is a sensation of tickling in the larynx, with a frequent dry cough; and there may be a feeling of a sense of pressure over the larynx and upper portion of the chest. Laryngeal spasm may be present in selected cases (see Spasmodic Laryngitis). Dyspnea is an annoying symptom in severe types of the disease.

Physical Signs.—The surface involved may be covered with a mucous secretion and is reddened and swollen. The vocal cords are swollen and reddened and lack their normal movements.

Thermic Features.—Fever may be slight, fluctuating between 99° and 101° F.

Diagnosis.—This is based upon the history of one or more of the predisposing factors, together with the characteristic changes in the voice. A laryngoscopic examination serves as a positive means of diagnosis.

SPASMODIC LARYNGITIS (LARYNGISMUS STRIDULUS; FALSE CROUP).

Pathologic Definition.—A spasmodic affection usually seen in children during the course of acute catarrhal inflammation of the laryngeal mucous surface.

Predisposing and Exciting Factors.—Conditions that predispose to acute laryngitis are also to be considered in connection with spasm of the larynx. It is sometimes excited by strong passion or emotion, and it may be associated with tetany. Rachitic subjects are especially liable. "The spasm of the adductors that causes the urgent dyspnea is probably reflex and due to peripheral irritation" (Anders).

Principal Complaint.—Two clinical varieties are to be distinguished:

(*a*) **Nervous type:** That in which the larynx is free from inflammation. This variety is characterized by sudden brief attacks of dyspnea either by day or night. General convulsions have been noted, but there is neither cough, fever, nor hoarseness. A repetition of these attacks may be experienced during the day.

(*b*) **Spasm of the larynx** associated with mild catarrhal laryngitis. The spasmodic attacks usually begin suddenly, upon awakening from a sound sleep. Positive evidence of the affection is afforded by the croupy, ringing cough, combined with the hard, stridulous breathing. A hoarse cough is often a precursor of the approaching spasm, as is also slightly stridulous breathing during sleep. Harsh breathing (stridor) is a vibrating noise produced by air passing in and out of the larynx or trachea, when one or both of these air-passages are partially obstructed. The following table is designed to set forth the various causes for this type of breathing:

Causes Within the Larynx or the Trachea.	
Foreign bodies.	Rupture of caseous glands.
Plugging by mucus.	Pus.
Affections of the Walls.	
Diphtheria.	Acute staphylococcal laryngitis.
Tuberculous ulceration.	Potassium iodid poisoning.
Posttyphoidal ulceration.	Syphilitic ulceration.
Acute edema.	Malignant ulceration.
Bright s disease.	Traumatic ulceration.
Acute streptococcal laryngitis.	Epithelioma of the vocal cords.
Acute pneumococcal laryngitis.	Fibroma of the vocal cords.
Stenosis after tracheotomy or cut throat.	Syphilitic stenosis.
	Epithelioma of the trachea.
Compression from Without.	
Thoracic aneurysm.	Malignancy of glands in the neck.
Mediastinal new growth.	Enlarged thyroid gland.
Epithelioma of the esophagus.	Enlarged thymus gland.

Physical Signs.—The respirations are seen to be altered, the neck is short and thick, and the auxiliary muscles of respiration are brought into action. The child prefers to sit or inclines slightly forward. Cyanosis may become extreme during the spasm and convulsive seizures may be observed.

Differential Diagnosis.—Spasmodic laryngitis is to be distinguished from laryngeal diphtheria, and the distinctive features are that laryngeal diphtheria develops more gradually and persists over a longer period than does spasmodic laryngitis. Prostration is also extreme in diphtheria, and moderate fever is present. (See p. 868.) The detection of a false membrane on the mucous surface of the nose or throat goes far to support the existence of diphtheria.

Prognosis.—A fatal termination is unusual, although repeated attacks are to be expected where spasm of the larynx develops in children.

TUBERCULOUS LARYNGITIS.

Pathologic Definition.—A subacute or chronic inflammation of the mucous surface of the larynx excited by the tubercle bacillus, and characterized further by congestion, edema, and ulceration.

Predisposing and Exciting Factors.—In the vast majority of instances tuberculous laryngitis develops secondary to pulmonary tuberculosis, certain authors regarding this form of tuberculosis as a complication of the pulmonary variety in from 18 to 30 per cent. of cases.

Principal Complaint.—The earliest symptom is that of hoarseness, which is followed by almost complete loss of the voice. After ulceration has become extensive and the surface of the epiglottis and pharynx are involved, swallowing is painful, and it is extremely difficult for the patient to take food.

Cough is decidedly painful, and may be more or less persistent. Cough is apt to be excited by talking.

Laryngoscopic examination shows the surface of the laryngeal membrane to be pale, and a variable number of broad, grayish, irregular, tuberculous ulcers are visible upon the posterior surface of the epiglottis and aryepiglottic folds.

Differential Diagnosis.—In ill-defined cases laryngoscopic examination is a necessary aid to distinguish between syphilitic and tuberculous laryngitis. The various tuberculin tests (p. 808) and the Wassermann reaction are deciding factors.

SYPHILITIC LARYNGITIS.

Remarks.—A variety of laryngitis developing during both secondary and tertiary forms of syphilis. It may appear in those where the luetic taint is inherited.

Principal Complaint.—Hoarseness is persistent and aphonia and dysphagia are also likely to develop. If it develops in secondary syphilis, the lesion is probably an erythema with superficial ulceration and a variable degree of catarrhal laryngitis.

During the tertiary form of syphilis the lesion of the larynx is apt to consist in small gummata. Rather deep-seated ulceration may develop in this form of the disease and may result in more or less extensive destruction of the laryngeal tissue. Laryngeal stenosis may result from syphilitic involvement of this organ where there are extensive cicatricial contractures.

EDEMATOUS LARYNGITIS.

Pathologic Definition.—An infiltration of the mucous membrane of the larynx by serum.

Predisposing and Exciting Factors.—Rarely it follows acute laryngitis, and develops during the course of erysipelas, diphtheria, scarlet fever, typhus and typhoid fevers, and acute phlegmonous inflammation of the adjacent structures; during the course of syphilis, acute and chronic nephritis, and chronic heart and liver diseases. Pressure from within the thorax may also cause laryngeal edema.

Principal Complaint.—The most prominent symptom is a rapidly developing dyspnea and huskiness of the voice, increasing from the onset. The respirations become stridulous.

Diagnosis.—The diagnosis is made immediately by drawing the tongue forward, when swelling of the glottis is apparent. Laryngoscopic examination is of service in selected cases. The clinical history is of moderate value in connection with laryngeal edema.

CHRONIC LARYNGITIS.

Pathologic Definition.—A chronic inflammatory process involving the mucous surface of the larynx, and characterized pathologically by thickening and congestion of the laryngeal mucosa, while in certain cases there may be a variable degree of ulceration.

Predisposing and Exciting Factors.—Chronic laryngitis follows repeated acute attacks, especially in those who speak much in public or in the open air; excessive smoking and chronic alcoholism are also potent factors in the production of this condition. Rarely it follows acute laryngitis, while nasal stenosis and chronic pharyngitis are occasional causes.

Principal Complaint.—The voice is husky, roughened, and in severe types of this trouble there is almost complete aphonia. Cough is the rule and may be either mild or severe, paroxysmal, and is usually preceded by a peculiar tickling sensation in the larynx. Pain is an occasional complaint.

Laryngoscopic examination reveals slight swelling with moderate reddening of the mucous membrane and prominence of the mucous glands of the epiglottis. Patches of superficial erosion may be detected.

TUMORS OF THE LARYNX.

Among the symptoms of laryngeal tumor should be mentioned hoarseness, cough (laryngeal in nature), and aphonia. Difficulty in swallowing and urgent dyspnea are also annoying symptoms where the tumor is unusually large. Laryngoscopic examination serves as a positive means of diagnosis.

DISEASES OF THE BRONCHI, LUNGS, AND PLEURA.

METHODS OF EXAMINATION.

DATA OBTAINED BY INQUIRY.

Probably in no other clinical division is history-taking of so great importance as it is in connection with affections of the pleuræ and lungs, and the method of obtaining clinical evidence from the patient will, therefore, be outlined.

Family History.—**Heredity** doubtless plays an important part, although with the advance of science the tendency at present is to regard heredity of less importance than it was considered twenty years ago. It should, however, hold first place in the findings obtained by inquiry.

It is important to know whether or not any members of the patient's immediate family have suffered from pulmonary or pleural diseases, and it is likewise equally important to ascertain whether the male or the female members of the family are the ones so afflicted. When the women of a household (particularly the one who does the cooking) are tuberculous, the disease is more likely to be conveyed to other members of the family than it is when the males are the afflicted subjects. The fact that asthma occurred in previous generations is of moderate importance, for in certain families both asthma and emphysema may exist for generations before tuberculosis becomes a family disease.

The general physique of the members of a family is quite an important fact to be ascertained, since tuberculosis and other diseases of the respiratory tract are to be expected in those cases in which narrow and contracted chests are family characteristics.

Personal History.—The patient should be questioned carefully as to his general physical condition for some years antedating his present illness, and in pulmonary affections it is of vital importance to ascertain the

patient's weight during health, and whether fluctuations in weight were observed during different seasons prior to the onset of the present malady. Should the patient's weight have been below the normal for one of his height, this should be taken into consideration and the cause for it ascertained whenever possible. A comparatively light weight with reference to height may be a family characteristic, and will then be of but little or no clinical significance. Loss of weight, especially when such loss dates from the onset of the affection and is progressive in nature, is highly significant of pulmonary disease.

Previous Diseases.—Those who have suffered from lobar pneumonia are greatly predisposed to pleurisy, bronchitis, and pulmonary tuberculosis. Rheumatism seems to bear an intimate relation to pneumonia and to diseases of the pleuræ. Intercostal neuralgia is also at times a precursor of pleurisy and of pulmonary disease. Children and even adults who have suffered from *adenitis* (glandular tuberculosis) are subject to pulmonary or other forms of tuberculous involvement later in life. Suppuration of the bones, hip-joint disease, etc., in early life are often expressions of tuberculosis. Valvular heart disease is to be taken into consideration in connection with diseases of the lung, although cardiac and pulmonary maladies occurring in the same individual are by no means common. Pulmonary symptoms (dyspnea, cough) may often be secondary to organic disease of the heart. (See p. 172.) Previous attacks of pleurisy are always suggestive of tuberculosis of the pleuræ, and are likely to be followed by tuberculous involvement of the lung substance.

Social History.—A general outline of the patient's mode of living and of his habits and customs is of great importance, and the present health of the other members of his family is to be considered in formulating a diagnosis.

Age and sex exercise marked influence, and will be discussed at length under each particular disease.

Occupation.—It is an established fact that persons exposed to the inhalation of particles of dust, *e. g.*, stone-cutters, instrument-makers, diamond-cutters, brass-finishers, miners, glass-workers, and those employed in foundries, are especially likely to develop pulmonary tuberculosis, asthma, bronchitis, and pleurisy. Persons following indoor occupations, who do not get sufficient exercise, such as bookkeepers, barbers, clerks, seamstresses, and cooks, are also likely to contract pulmonary afflictions. Occupations that necessitate exposure to cold and wet may at times contribute toward the development of pulmonary diseases, but, as a rule, those who live out-of-doors are less likely to become tuberculous than are those who are deprived of exercise and of invigorating air.

Source of Infection.—If a patient suffering from tuberculosis has been intimately associated with other tuberculous patients, it is to be presumed that the source of infection is that of contact. In the majority of instances tuberculosis is not transmitted directly from one member of a family to another, but may be conveyed by infected members or by food that has been handled by tuberculous persons, who, during coughing, would send their sputum in a spray about the room where the food was handled. The routes through which tubercle bacilli may enter the human body are manifold, and no one particular mode of infection need be emphasized here.

Cough.—Correlatively speaking, cough is reflex in origin. The mechanism is that of a deep inspiration, which is immediately followed by closure of the glottis, when an expiratory effort suddenly follows, the glottis is forced open, and the sound is produced by the forcible escape of the air.

Cough is a symptom of many pleural, pulmonary, and remote pathologic conditions, and may also occur as a hysteric manifestation. Cough of physiologic origin is seen during the early months of gestation.

Causes.—(1) Either acute or chronic irritation of the bronchial mucous membrane is sufficient to excite cough. The act of coughing may also be a physiologic process, serving to expel mucus, pus, and any foreign substance that may have collected in the bronchi. Among diseases in which cough is an almost constant symptom should be mentioned pleurisy, empyema, pulmonary tuberculosis with cavity formation, bronchitis, asthma, and emphysema. In diseases of the larynx cough is a cardinal symptom. Pressure upon the recurrent laryngeal nerve gives rise to cough and aphonia. (See Aneurism, p. 314.)

In thoracic aneurism the cough is quite characteristic, being harsh and rasping, and having a brassy or metallic ring. The cough of aneurism may be non-productive, or, as is often the case, paroxysms of coughing are followed by copious expectoration of mucopurulent material. Mediastinal and thoracic tumors may excite cough in persons in whom the lungs and pleuræ are healthy.

In organic heart disease, the result either of valvulitis or of myocarditis, cough not infrequently occurs as the result of imperfect circulation and venous stasis in the lungs. Incorrect posture in those of lowered vitality results in hypostatic congestion at the bases of the lungs, and such congestion, in turn, is often productive of cough. This variety of cough is commonly encountered in those suffering from acute and chronic febrile and afebrile maladies. The character of the cough, as previously stated above, is equally significant in pleurisy and in lobar pneumonia. The cough of pleurisy is short, non-productive, and hacking in character, and is accompanied by extreme pain in either side of the chest. In lobar pneumonia the cough is also short and harsh, but it is accompanied by slightly blood-streaked expectoration, and severe pain is present when the pleura is inflamed.

Reflex Cough.—A lesion of the brain involving the respiratory center at the floor of the fourth ventricle is another cause for cough. The cough of hysteria is in no way characteristic, but is readily detected by the efforts of the patient to produce this symptom and by the associated phenomena. The barking cough of hydrophobia is also readily detected in a neurasthenic. Irritation of the pneumogastric nerve produces cough.

Stomach Cough.—The experiments of Kohts do not prove that a cough may occur as the result of derangement of the stomach, yet patients having a decided cough are not infrequently also afflicted with gastro-intestinal catarrh. That certain coughs are due to gastric irritation is borne out by the fact that they disappear when the latter condition is relieved. In persons suffering from gastric catarrh a similar inflammatory process generally involves the pharyngeal and laryngeal mucous membrane, and this may explain the source of the so-called "stomach cough."

Ear Cough.—During an examination of the external auditory canal the patient frequently gives a harsh, hacking cough. Foreign bodies and abscesses in the ear may also excite a short, harsh, and fairly characteristic cough.

Tooth Cough.—The irritation from a diseased tooth may excite reflex cough in the adult, and in infants, during the process of dentition, cough is quite common.

Whooping-cough.—This cough may be non-productive or accompanied by expectoration. A peculiar sound (a whoop) is heard during inspiration, and occurs generally after the child has made several rapid attempts at

coughing. The whoop is usually followed by vomiting, and there may be epistaxis and even hematemesis. In whooping-cough the attacks of coughing are paroxysmal, purely spasmodic, and excited by violent exercise, talking, laughing, and the like.

The Cough of Diphtheria, Pharyngitis, and Esophagitis.—This is really the cough of laryngitis, and occurs when the diphtheric process extends to the vocal cords and larynx. (See Diphtheria, p. 867.) In pharyngitis and diseases of the esophagus cough may be a prominent symptom.

PHYSICAL EXAMINATION OF THE CHEST.

Landmarks.—In order to examine the chest properly all clothing must be removed, or the patient's chest and abdomen exposed from the clavicle to the umbilicus. When the patient is able to sit or to stand, the method of examination is quite simple, but when the patient is confined to bed, it becomes quite difficult to obtain certain physical signs.

Certain landmarks are always to be observed, regardless of the position of the patient. When examining the front of the chest, one always starts with the superior boundary or clavicles. These curved bones separate the supraclavicular from the infraclavicular regions on each side (Fig. 4), and, owing to the fact that they traverse the chest transversely, they are used at different points to mark certain lines required to divide the chest vertically. The sternum serves as an invaluable landmark, because it divides the chest vertically (Fig. 4), and its peculiar notch, which separates the ends of the clavicles at the top of the chest, furnishes a guide that seldom, if ever, changes as the result of disease.

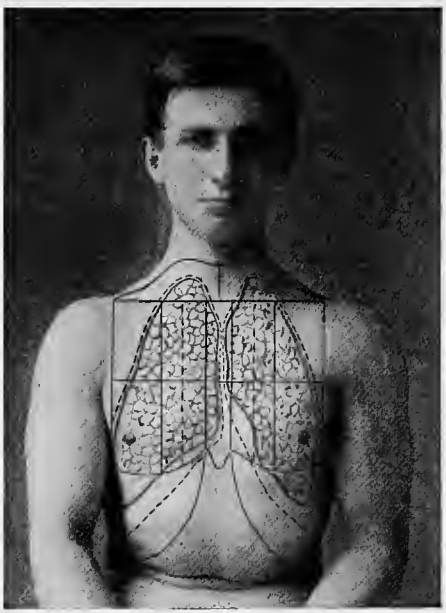


Fig. 2.—Relation of the Normal Lungs to the Pleuræ, Costal Margins, and Clavicles.

Again, it is of great importance to note that the suprasternal notch is at the level of the articular surface of the *second* and *third* thoracic vertebræ. About an inch below the suprasternal notch there is a distinct transverse ridge that marks the line of union between the first and second pieces of the sternum, and it is this sternal prominence that enables one to count the ribs.

Another method of counting is to regard the articulation between the clavicle and the sternum as the first rib. The sternal ridge is on a level with the *center* of the *body* of the *fifth thoracic vertebra*.

At the lower extremity of the sternum we observe the xiphoid cartilage. For the purpose of diagnosis the junction of the xiphoid cartilage with the greater portion of the sternum is on a level with the *articular surface* of the *ninth* and *tenth thoracic vertebræ*.

Counting of the ribs forms one of the most important steps in diagnosis, and is applicable to diagnosis both of the chest and of the abdomen. In

diagnosis we often speak of a certain rib or interspace as at some particular line. These lines will be described later. Fig. 57 shows the actual relation existing between the lung, liver, and heart, and the points at which these viscera are contiguous one with another, as described by the clavicles, sternum, and ribs.

Counting the ribs is further useful in dividing the chest transversely by imaginary lines at different levels, *e. g.*, a line encircling the chest at a level with the nipples would pass through the center of the sixth intercostal space at the midaxillary line; this is the point of election in aspiration of the pleuræ for the removal of fluid. A point of great diagnostic importance is the fact that the ribs pass obliquely downward as they leave the vertebræ, so that their sternal junction is on a lower level than their vertebral articulation, *e. g.*, the *articulation of the cartilage of the third rib* anteriorly is on a level with the body of the *sixth thoracic vertebra.* When studying the articulation between the third and the seventh rib, for example, the calculation is readily made by adding four to the number of the rib articulating with the sternum; thus, the seventh rib anteriorly corresponds to the eleventh vertebra.

Landmarks of the Back of the Chest.—1. *The Scapulæ.*—These bones are situated conspicuously at the top and back of the chest, and extend from the second to the seventh ribs inclusive. The inner end of the scapular spine is on a level with the spine of the third thoracic vertebra, and the inferior scapular angle is on a level with the spine of the seventh thoracic vertebra; consequently when the arms are permitted to hang at the sides and when both forearms are folded across the chest the seventh rib passes beneath the lower portion of the scapula.

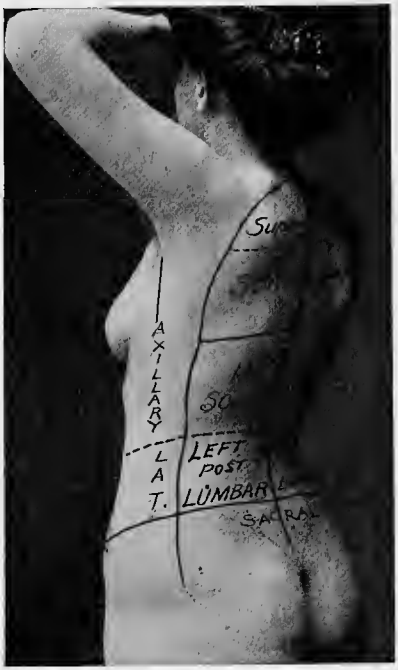


Fig. 3.—Arbitrary Divisions of Posterior and Lateral Surfaces of Chest.

2. *The Spine.*—The spinal column occupies the center of the posterior wall of the chest, and is outlined by a distinct groove. The spinous processes of the vertebræ are often visible as slight prominences along the column. In passing the hand from above downward over the spinal column, the processes are rendered more conspicuous and are readily palpable by directing the patient to bend forward. The tips of the vertebral spine also serve as landmarks, the one usually selected being that of the seventh cervical vertebra—the so-called "vertebra prominens." The spinous process projects obliquely outward and downward, so that the tip of the spinous process is on a level with the articulating surface of the rib below, *e. g.*, the second thoracic spine corresponds with the level of the third rib. The spinous process of the tenth vertebra is materially shorter than that of the others.

A fact to be remembered is that the first thoracic vertebra is in direct articulation with the seventh cervical vertebra; consequently the ribs

begin at this point. The second rib articulates with the second and third thoracic vertebræ, and this plan of articulation is continued downward to the tenth rib articulation. The eleventh and twelfth ribs articulate with the eleventh and twelfth vertebræ.

Epigastric Angle.—This angle is situated at the anterior portion and base of the chest; the apex is directed upward, and is formed by the xiphoid cartilage; its lateral boundaries are the converging cartilages of the ribs. During inspiration and expiration the degree of this angle is materially altered. At times it forms almost a right angle, whereas on deep inspiration an obtuse angle results.

Lines as Chest Landmarks.—In order to localize certain physical signs and to determine with ease certain definite points upon the chest-wall the chest is divided vertically by imaginary lines that transcribe definite arbitrary regions (Figs. 2 and 4). These are:

Anteriorly: (1) The mesosternal (midsternal) line, the middle line of the sternum. (2) The right and left *sternal lines*, corresponding to the lateral margins of the sternum. (3) The *parasternal* lines, midway between the border of the sternum and the nipple. (4) The *midclavicular lines*, usually passing through the nipples.

Laterally, the chest is divided by three imaginary lines: (1) The *anterior axillary* lines, which cross those points where the great pectoral muscles leave the chest-wall when the arms are raised to the horizontal. (2) The *midaxillary* lines, which pass through the center of the axilla, or midway between the anterior and the posterior margins of the axilla. (3) The *posterior axillary* lines, which extend vertically through those points where the latissimus dorsi muscles leave the chest-wall (Fig. 3).

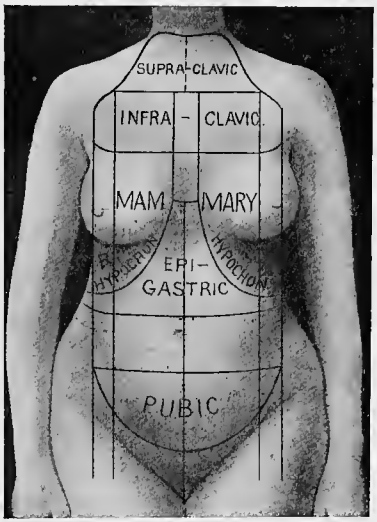


FIG. 4.—ARBITRARY DIVISION OF THE CHEST AND ABDOMEN.

Posteriorly, but two lines are generally recognized: (1) The *scapular* lines, which pass vertically through the angles of the scapulæ when the arm is allowed to rest by the side. (2) The *midspinal* line, which is drawn to correspond to the center of the vertebral column.

Regional Anatomy.—Anteriorly, the chest is divided into regions, and the relative size, location, and form of such regions are clearly shown in the accompanying illustration (Fig. 4). Therefore we will confine ourselves to naming the various viscera known to occupy each particular region.

The Supraclavicular Regions.—On each side we find the apices of the lungs, and a short section of both the subclavian and the carotid arteries, and also the subclavian and the jugular veins. The apex of the lungs rises, as a rule, to from one-half to one and one-half inches above the upper border of the clavicle, the left lung generally extending a little higher than its fellow. A portion of the floor of the supraclavicular space is formed by the first rib on each side. Immediately above the inner portion of the clavicle is the point at which pulsation from the subclavian artery may be felt.

The Clavicular Regions.—This region is small, and is bounded by the

margins of the inner two-thirds of the clavicles. Upon each side is found the apex of the lungs. On the right and underneath the sternal articulation of the clavicle is the bifurcation of the innominate artery, and just external to this is the subclavian artery. At the left sternal articulation both the carotid and the subclavian arteries are deeply situated.

The right infraclavicular region contains a portion of the upper lobe of the right lung, and beneath the right border of the sternum are the superior vena cava and the arch of the aorta. Underneath the second right costal cartilage the right bronchus rests.

In the **left infraclavicular region** we find the upper lobe of the left lung, and at the border of the sternum, the left pulmonary artery and a portion of the left auricle.

The Mammary Region.—In this region the two sides will be found to differ widely, the right side containing the lung, the dome of the liver, the extreme right portion of the heart, and the diaphragm, which fits snugly over the apex of the liver and extends well up into the lung—fourth interspace (Fig. 5). It is the intrusion of the superior border of the liver upon the lung that causes the lung tissue to rest superficially throughout this region, although a thin layer of the lower border of the lung extends as low as the sixth rib. The fissure dividing the upper and middle lobes of the right lung runs obliquely upward and backward from the fourth costal cartilage, while the fissure dividing the middle and inferior lobes of the lung arises at the fifth interspace (Fig. 5). The right side of the heart extends into this region, and a portion of both the auricle and the ventricle is covered by lung, and rests to the right of the sternum, between the third and the sixth cartilages.

The **left mammary region** contains the greater portion of the heart, which is partially overlapped by lung tissue. The outline of the heart in both health and disease is shown in the accompanying illustration (Fig. 57). A quadrilateral area of heart is uncovered by lung, and this portion corresponds to the right ventricle; the greater portion of the right auricle and of the left auricle and ventricle are deeply seated in this region. The apex of the heart ordinarily corresponds to the midclavicular line at the fifth interspace (Fig. 2). The fissure separating the superior from the inferior lobe of the left lung is situated at a point where the nipple-line crosses the sixth rib. The left lung also occupies this region.

The Inframammary Regions.—These extend downward from the sixth rib on each side to the margin of the false ribs, and from the sternum they are bounded externally by the costal cartilages. To the right of the median line we find a portion of the right lobe of the liver, the diaphragm, and, during the act of forced inspiration, the lower border of the right lung. The lower border of the liver is found by following the costal margin from the point at which the midclavicular or nipple-line crosses the costal cartilages.

The left inframammary region contains the lower margin of the left lung during inspiration, a portion of the left lobe of the liver, and the cardiac end of the stomach. It is somewhat difficult to separate the epigastric region from the two mammary regions, for many of the viscera lie in both. We have adopted an arbitrary division of the abdomen somewhat different from that ordinarily employed, thus simplifying, in a measure, the topographic anatomy of the inframammary region. The **right inframammary region** contains a portion of the right lobe of the liver.

The Sternal Regions.—That portion of the thorax underlying the sternum is divided into two parts:

In the *superior sternal region* are found the inner edge of the lungs at and below the second costal cartilages, the bifurcation of the trachea, the

aortic arch, the pulmonary artery, the left innominate vein, and the vena cava.

The *inferior sternal region* contains a portion of both the right and the left lung, the greater part of the right ventricle, the origin of the pulmonary artery, and the edge of the left ventricle, which is situated well posteriorly. The first part of the aorta, a portion of the right auricle, and a part of the liver are also found here, and within this area the pericardiac attachment of the diaphragm is located.

The Lateral Thoracic Regions.—*The Axillary Regions.*—These are bounded by lines that extend from the upper anterior portion of the axilla to a level with the lower margin of the mammary regions (sixth rib), and posteriorly by a line drawn from the upper portion of the axilla through the point where the latissimus dorsi muscle leaves the thorax when the arms extend horizontally from the chest (Fig. 6). Both axillary regions

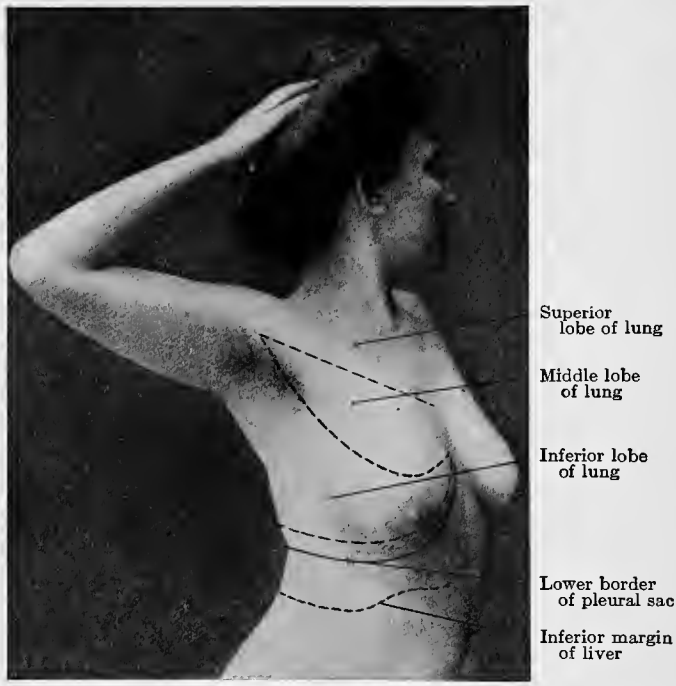


FIG. 5.—LATERAL VIEW OUTLINE TO SHOW THE RELATION OF THE RIGHT LUNG, PLEURA, AND LIVER.

contain lung tissue, and, more deeply seated, the bronchi and their smaller branches are found.

The Infra-axillary Regions.—These are bounded superiorly by the lower border of the axillary regions, and below by the margin of the ribs. Posteriorly, they are contiguous with the infrascapular regions (Fig. 6). In the right infra-axillary region the lung will be found to slope downward and backward as low as the eighth rib, at the point where it is bisected by the midaxillary line. The liver is also contained in this region. The left region contains, in addition to the lung tissue, a portion of the stomach and the spleen.

Posterior Regions.—The *suprascapular regions* (Figs. 3 and 6) contain the

apices of the lungs, and it is the portion of the lung that occupies this region that is most liable to be attacked early by tuberculosis; in studying incipient pulmonary conditions, therefore, the suprascapular region should be examined most carefully.

The *scapular regions* contain, for the most part, portions of the lungs, and the fissures dividing the pulmonary lobes are also situated in this region.

The Infrascapular Regions.—These regions are bounded superiorly by a line drawn across the inferior angles of the scapulæ, and below by the edge of the thorax, and extend in the median line downward to the eleventh vertebra. Anterolaterally these spaces are limited by the line bounding the infra-axillary region, which corresponds to the point at which the latissimus dorsi muscle leaves the chest-wall (Fig. 6). On both sides are the lungs, and their inferior margins extend downward as far as the eleventh ribs. On the right side, below the lung, is a small portion of the liver, and lying immediately in contact with the spinal column is the upper portion of the right kidney. To the left of the spine, passing from the median line outward, are the aorta, the left kidney, coils of intestines, and the spleen.

Fig. 6.—Arbitrary Regional Division of Back with Relation to Axillary and Infra-axillary, Suprascapular, Scapular, Infrascapular, and Lumbar Regions.

The Interscapular Region.—The size of the interscapular region may be somewhat increased by directing the patient to bend forward and to fold the arms over the chest (Fig. 29). Upon both sides of the spine are portions of the lungs. At the fourth thoracic vertebra is the bifurcation of the trachea. The bronchial glands are also situated near this point. To the left of the spine, and at the third or fourth thoracic vertebra, is the descending aorta, and in intimate relation with this are the thoracic duct and the esophagus.

The bifurcation of the trachea is nearly on a level with the third and fourth thoracic vertebræ, corresponding anteriorly to the angle of Louis or the second costal cartilage.

It must be remembered that the caliber of the right bronchus is considerably larger than that of the left, and that this canal passes in a horizontal direction immediately beneath the second rib. The left bronchus is situated slightly below the right, in the second interspace.

INSPECTION OF THE CHEST.

Preparation for Inspection.—It is impossible to obtain accurate information regarding the contour and movements of the thorax unless the patient is bared to the waist. Occasionally it is necessary to modify this general rule, but whenever such modification is made, there will always

be uncertainty as to whether or not the examiner has detected all existing abnormalities presented by the patient.

Position of the Patient.—During an examination of the chest the patient should preferably be standing or sitting, in order that the examiner may step from side to side and from front to back, viewing the chest from every aspect. When it is necessary to examine a patient in the recumbent

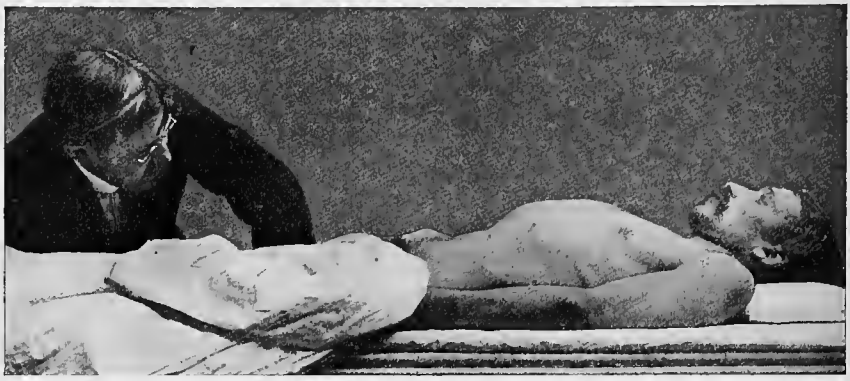


FIG. 7.—INSPECTING THE ABDOMEN AND CHEST.

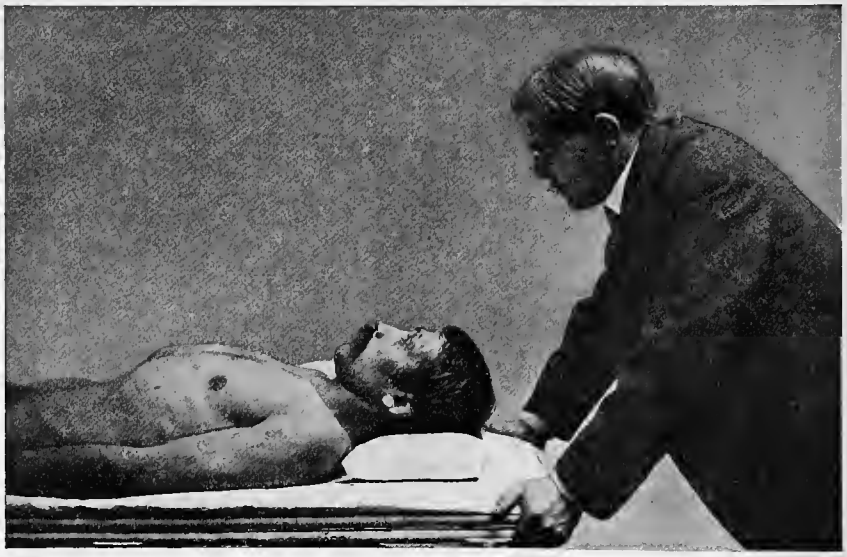


FIG. 8.—INSPECTION OF THE CHEST AND ABDOMEN.

posture, the examiner should stand first at the feet and then at the head of the patient, and note particularly the character of the chest movements (Figs. 7 and 8). The patient should then be turned first upon one side and then upon the other, and while this is being done the general expression, rapidity of respirations, and the degree of lividity or cyanosis are to be noted.

The general conformation of the chest, and its influence upon the various types of respiration, which will be described later, is of vital importance.

Light.—Whenever possible, the chest should be examined by daylight; for when this is done by artificial light, shadows, due to the various curves and prominences of the chest-wall, are likely to confuse the examiner.

What is to be Ascertained by Inspection.—(*a*) The appearance of the external surface, evidences of emaciation, etc.; (*b*) the shape and size; (*c*) movements and degree of expansion; and (*d*) fluoroscopic study.

The Chest-wall.—The characteristic appearance of the healthy skin is materially altered in disease; thus it is extremely pale in all diseases associated with anemia, and in vagabondism, Addison's disease, abdominal tumors, and jaundice it is pigmented. It also becomes pigmented as the result of tinea circinata, syphilis, multiple abscesses, and the like. Distention of the veins over the anterior surface of the chest is suggestive of pressure in the thorax, as from thoracic aneurism or enlarged bronchial glands.

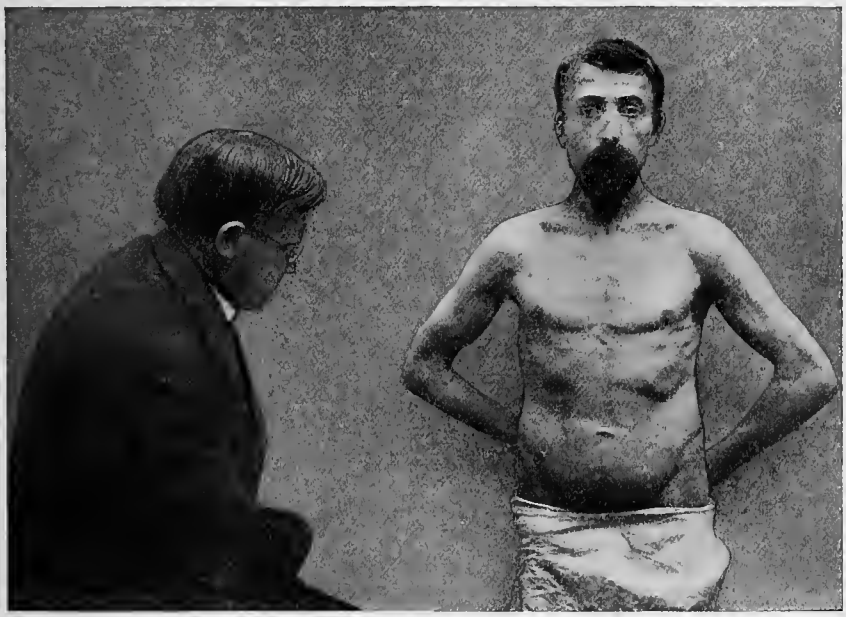


FIG. 9.—INSPECTION OF ABDOMEN (LATERAL VIEW) TO ASCERTAIN THE MOVEMENTS OF THE CHEST AND ABDOMEN.

In the female lactation is the most common cause of such distention. If the veins of the neck are enlarged, cardiac incompetency is to be suspected, and if they pulsate synchronously with the heart's action, tricuspid regurgitation is probably present. The veins over the chest-wall may also be enlarged as the result of pressure or obstruction to the thoracic vessels.

Thoracic edema is common in purulent exudates into the pleuræ. Subcutaneous emphysema may follow rupture of the lung, ulcer of the esophagus, and infection with gas-producing bacteria.

In order to estimate the degree of emaciation that has taken place the patient's present condition must be compared with that known to have existed in health.

Shape.—It is practically impossible to describe accurately the shape

of the chest, and a definite knowledge of its general conformation can be acquired only by making repeated examinations of the normal chest. Indeed, it is by this method alone that one can become familiar with the various types of chests to be found in healthy men, women, and children. Characteristic alterations in the conformation of the chests are seen in persons following certain occupations, as, for example, shoemakers, blacksmiths (unilateral overdevelopment), carpenters (funnel-shaped depression at xiphoid), coachmen (elevation of one shoulder), and those who have received training in military academies and gymnasiums (bilateral overdevelopment).

The chest in health is practically symmetric, its symmetry being due in a measure to the presence of subcutaneous fat.

The sternum projects slightly forward as it extends from above downward to the ensiform cartilage, the middle portion of the sternum being the most prominent part of the anterior surface of the chest. The ribs, upon their articulation with the cartilages that serve to connect them with the sternum, usually display a peculiar arched appearance, although not infrequently the anterior surface of the upper part of the chest is practically

a *b* *c*

FIG. 10.—NORMAL CHEST.
During: *a*, Forced inspiration; *b*, forced expiration; *c*, at rest.

flat, a condition referred to as the "flat type of chest." The xiphoid cartilage may be depressed or may project anteriorly in the healthy chest.

Bilateral Abnormalities of the Chest.—In athletes the chest is greatly enlarged, but this condition cannot be regarded as pathologic unless alterations in the viscera sufficient to cause the distention are present.

In the barrel-shaped chest of emphysema (p. 127) the transverse diameter is normal or decreased, whereas the anteroposterior diameter of the chest exceeds the transverse. The clavicles and upper ribs are usually conspicuously elevated, giving the patient the appearance of having an unusually short neck. The pathologic changes necessary to produce the emphysematous chest will be discussed at length under Emphysema. As the result of the changes that produce the emphysematous chest there is also engorgement of the veins of the neck; this tends to render the cervical region thickened, and, as the result of venous congestion, it may even pulsate. The bases of the chest may be greatly enlarged as the result of the presence of transudates or exudates into the pleural sacs and by

enlargement of the liver and spleen. The development of carcinoma in both lungs may also cause an abnormally large chest.

Unilateral enlargement of the chest is the result of pathologic changes causing an increase in the size of the viscera occupying one side of the chest, or is due to a lessening of the capacity of one lung, with compensatory emphysema of its fellow; thus, for example, following fibroid (adhesive) pleurisy of one side, the opposite side becomes abnormally enlarged in order to compensate for the lost breathing space.

A large pleural effusion causes an abnormal distention of the affected side of the chest. When such effusion is present, the lung of the opposite side becomes emphysematous, and, in consequence, the entire chest is enlarged; the emphysema may be so marked as to lead one at first sight to suspect the enlargement to be the result of a bilateral pathologic condition. Adhesive pleurisy, fibroid phthisis, a pulmonary cavity, and bronchiectasis may cause a lessening in the dimensions of one side of the chest, but the opposite side rarely changes its size greatly as the result of compensatory emphysema.

New-growths of the thorax—e. g., thoracic aneurism—most often causes a prominence of the sternum, although it is not uncommon to find an aneurismal tumor protruding from the back, through the scapula, or from any portion of the chest-wall. Tumor of the mediastinal glands is a frequent cause of prominence of the sternum. Carcinoma and sarcoma of the lung and pulmonary abscess may cause unilateral deformities of the chest. The rickety chest follows rachitis, and in this condition the chest may assume almost any shape. Adenoid disease in children may cause chest deformities that somewhat resemble those produced by rickets.

Movements of the Chest.—Inspection of the chest enables us to ascertain the frequency of respiration, the rhythm, the diaphragmatic phenomena, and the degree of expansion. In health, inspiration is an active process, whereas expiration is passive. Physiologically, the act of expiration is slightly longer than is that of inspiration, and bears a ratio of six to five. There is sometimes a distinct pause following expiration. Generally speaking, it may be said that the chest expands in all directions during inspiration and diminishes correspondingly in size during expiration. The character of the expansion during health can be learned only by inspecting the chest of several normal individuals.

In the normal male the respiratory movements vary between 16 and 24 a minute, whereas the adult female breathes from 20 to 22 times a minute. Breathing is much faster in children than in adults, and during the first year the average number of respirations is from 40 to 44 a minute, while at the age of five the child usually breathes 25 or 26 times in a minute.

Posture, exercise, excitement, digestion, disease, and certain drugs increase the number of respirations, whereas posture, lack of mental excitement, and drugs diminish the frequency of the respiratory act.

Types of Normal Respiration.—*The Costo-abdominal Type.*—This is frequently referred to as the diaphragmatic type of breathing, and is characterized by the fact that while the patient is breathing quietly the chest movements are more marked at the lower half than they are at the upper half of the thorax. This form of breathing is more common in males than in females. During inspiration the sternum rises slightly, and the ribs are elevated, and at the same time extended downward, forward, and outward. Both the anteroposterior and the transverse diameters of the chest are increased with each normal inspiration, and the epigastric angle

occupying the interval between the costal cartilages at the base of the chest is changed from an acute to an obtuse angle. In the diaphragmatic type of breathing the movements of the diaphragm are conspicuous, and the muscle acts conjointly with the muscles of the thorax; thus, as the diaphragm descends, there is a corresponding swelling of the upper abdominal hemisphere. During expiration the chest gradually assumes its original shape and size.

Costal Type.—In adult females the upper half or two-thirds of the chest moves more conspicuously than the lower portion, hence this variety of breathing is usually referred to as the upper thoracic type. In the clavicular regions, the upper portion of the sternum, and as low as the third rib, there is marked expansion with each inspiration, whereas the lower portion of the chest remains almost stationary, and the results of the movements of the diaphragm are but feebly, if at all, apparent through abdominal swelling with inspiration. The costal type of breathing is also seen in children and in men during sleep.

Movements of the Chest in Disease.—The chest movements are increased in practically all forms of difficult breathing, and the frequency of the movements is quite characteristic of certain affections—thus, a marked increase in the number of respirations may result from either pulmonary disease or other maladies. In children the movements are comparatively rapid. In fever and in nervous conditions the frequency of the chest movements are, as a rule, increased, whereas in coma and in certain cerebral diseases the respirations may be less frequent; indeed, this symptom follows certain toxic poisonings. From the degree of chest expansion we learn whether the respirations are deep or shallow; as previously stated, the ratio of the act of inspiration to expiration is as five is to six; in children, in most women, and in the aged, however, we find this ratio changed to from six to eight, the act of expiration being greatly prolonged. At times, where the degree of expansion and the duration of inspiration are increased, there is some obstruction in the upper air-passages,—*e. g.*, in the trachea and larynx,—and the exaggerated expansion affects chiefly the upper part of the chest, when there is a corresponding retraction of the flexible wall at the base of the thorax.

Dyspnea.—In dyspnea, or difficult breathing, the respirations, while deeper than normal, are not always increased in frequency. They may, however, be more frequent than normal. Dyspnea is a common symptom in pulmonary disease, but it does not follow that extensive disease of the lung is always accompanied by difficult or hurried breathing. Patients suffering from dyspnea are usually reduced in weight, move slowly, and lack the normal vigor of health.

Varieties of Dyspnea with Reference to its Exciting Cause.—Among the causes of dyspnea are:

1. Anything that lessens the normal amount of air intake required fully to oxygenate the blood—(*a*) Obstruction of the air-passages; (*b*) diminution of air-spaces from intra-thoracic (see Pleural Effusion, p. 142) and extra-thoracic (see Ascites, p. 564) exudates; (*c*) interference with the action of the muscles of respiration.

2. Maladies that are characterized by interference with the circulation through the lung.

3. Primary and secondary anemias.

4. Obstruction to the pulmonary circulation—*e. g.*, pulmonary embolism, lobar pneumonia, pulmonary infarct.

5. Interference with the nervous mechanism of respiration—cerebral tumor, cerebral hemorrhage, and the effects of uremic and other poisons upon the respiratory center.

6. A form of reflex dyspnea is occasionally seen in hysteria, gastric disturbances, and asthma. (See also Orthopnea, p. 173; Cheyne-Stokes respirations, below; cardiac dyspnea, p. 173.)

The normal rhythmic movements of the soft parts at the base of the chest are altered in practically all types of dyspnea, and in most pathologic conditions of the lungs and pleuræ.

The act of expiration is prolonged in emphysema, and is a characteristic feature of this disease. Again, whenever the expiratory act is prolonged, the accessory muscles of respiration are brought into action, and the patient assumes a posture that facilitates emptying the lung.

Cheyne-Stokes respiration is a disturbance in the rhythm of the respiratory acts characterized by distinct pauses. The respiratory acts forming the groups before and after a pause begin with a shallow inspiration; the inspirations gradually become deeper until the maximum of depth is reached; then they become more and more shallow until they cease. Each group is composed of from 10 to 30 respiratory acts; the pause occupies from thirty to forty-five seconds.

FIG. 11.—RESPIRATORY PHASES IN CHEYNE-STOKES RESPIRATION, GIVING THE RESPIRATORY TRACING FOLLOWING A PAUSE (Boston and Ulman).

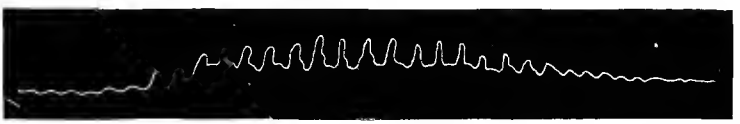


FIG. 12.—RESPIRATORY TRACING IN CHEYNE-STOKES RESPIRATION SHOWING COMPLETE RESPIRATORY MOVEMENTS, PRECEDED AND FOLLOWED BY A PAUSE (Boston and Ulman).

The cycle of Cheyne-Stokes respiration, which includes the ascending and the descending phase, together with the pause, usually occupies one minute, and may be as short as one-half or as long as two minutes. This type of respiration occurs after severe surgical shock, and late during valvular heart disease and kidney affections; it has been considered a symptom of nephritis (p. 661). Tumors of the brain, injuries, and hemorrhages involving the floor of the fourth ventricle frequently manifest Cheyne-Stokes respiration as one of their cardinal symptoms (Figs. 11, 12).

Unilateral Changes in Respiration.—The movements of the affected side of the chest are diminished in pneumothorax, large pleural effusions, and, in massive pneumonia, when, as the result of overwork, the movements of the opposite side are at the same time exaggerated. A large pericardial effusion may inhibit the movements at the base of the left chest, but here there is also exaggerated movement of the upper portion of the same side. Fibroid phthisis involving one lung causes a decrease in movements upon one side of the chest, but movements are exaggerated on the opposite side.

Local Abnormalities of Movement.—In this connection special attention is directed to the exaggerated movements of the bases of the lungs where the apices are affected with tuberculosis. Thoracic aneurism or thoracic tumor of whatever nature may give rise to unilateral abnormalities in the respiratory movements, such abnormalities varying with the location and the size of the tumor. Where tuberculous involvement of the apices of the lung is present, expansion may be slightly delayed at the affected point. An increased expansion is apt to occur over healthy lung whenever a large portion of the breathing space has been consolidated from any cause.

Pulsation of the chest may be the result of a dilated heart, or may be due to the heart being pulled out of its normal position by pleural adhesions. Epigastric pulsation is a symptom of cardiac dilatation. In empyema pulsation may be present over the affected pleura. During phonation bulging of the intercostal space overlying the pleural effusion is rarely detected. Bulging is best produced by directing the patient to close his nostrils tightly and then partially to stop the exit of air by placing the hand over the mouth while he is speaking.

PALPATION OF THE CHEST.

By means of palpation most of the results obtained by inspection are confirmed. In palpation, as in inspection, more accurate results are obtained by baring the subjects chest, although it is frequently necessary to palpate over a thin garment. Certain of the postures to be assumed

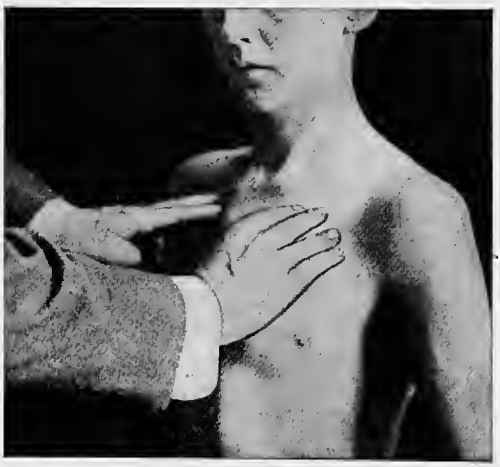


FIG. 13.—METHOD OF PALPATING OVER APICES OF LUNGS.

by the patient and the position of the operator during palpation are shown in the accompanying illustrations (Figs. 13, 14). In palpating the chest it is most important that the tactile fremitus should be elicited over practically every portion of the chest occupied by the lungs. The two sides of the chest must be studied comparatively, and palpation must be carefully performed over the axillary regions and bases.

Tactile Fremitus.—Generally speaking, the vibrations transmitted to the finger during the act of talking are more pronounced over the right than over the left apex, and in males they are decidedly more prominent

than in females. The thickness of the chest has a direct influence on eliciting the tactile fremitus, since persons with a muscular or fatty chest-

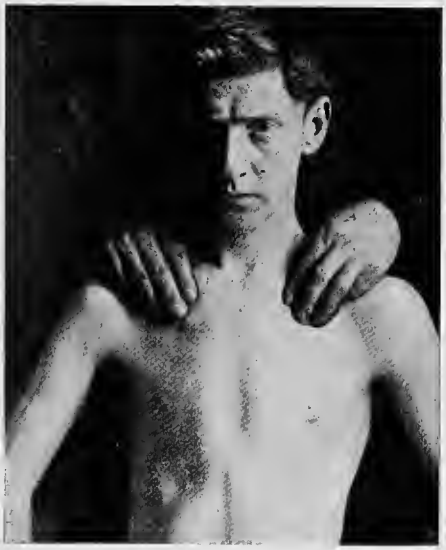


FIG. 14.—METHOD OF PALPATING APICES.

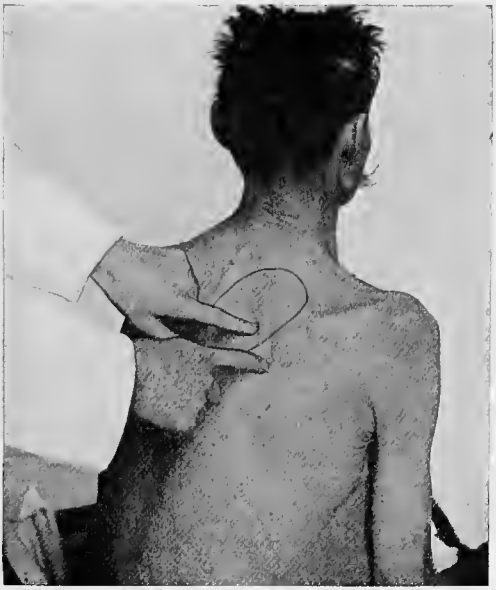


FIG 15.—DETERMINING THE EXACT BOUNDARY OF AN AREA OF PULMONARY CONSOLIDATION THROUGH THE DEGREE OF VOCAL TACTILE FREMITUS (Boston, N. Y. Med. Jour., Nov. 1, 1913).
The left index-finger recorded far more fremitus than did the right.

wall show an apparent diminution in the vibrations transmitted to the examining hand. Vibrations are also imperfectly transmitted in children.

Increased Tactile Fremitus.—Among the conditions that may give rise to an increase in the tactile fremitus are: (1) Lobar pneumonia;

FIG. 16.—PALPATION OF TWO SMALL AREAS WHERE FREMITUS IS INCREASED (Boston, N. Y. Med. Jour., Nov. 1, 1913).

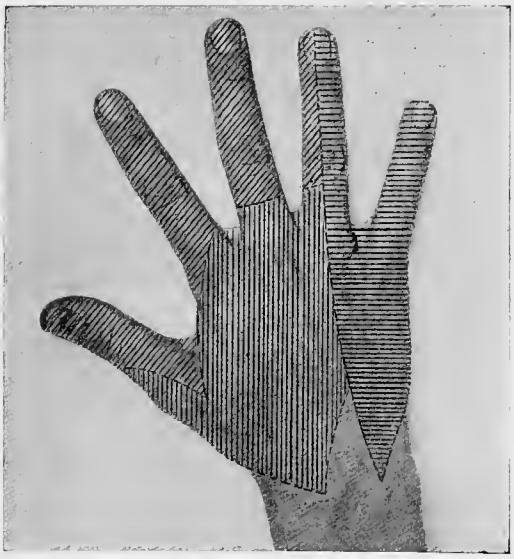


FIG. 17.—THE TRANSVERSE LINES REPRESENT THE AREAS SUPPLIED BY THE ULNAR NERVE, THE VERTICAL LINES OCCUPY THAT AREA SUPPLIED BY THE RADIAL NERVE, AND THE OBLIQUE LINES INDICATE AREAS SUPPLIED BY THE MEDIAN NERVE (Boston, N. Y. Med. Jour., Nov. 1, 1913).

(2) bronchopneumonia; (3) tuberculous cavity, with a dense wall; (4) tuberculous consolidation; (5) fibroid induration of the lung; (6) fibroid

tuberculosis; (7) hemorrhagic infarct; (8) atelectasis, and rarely a thickened pleura.

Diminished or absent fremitus may result from the following pathologic conditions: (1) Pleuritic exudates and thickened pleuræ; (2) bronchopneumonia; (3) dilated bronchi; (4) emphysema; (5) asthma. Fremitus is also absent in plugging of a bronchus, and limited areas over which it is impossible to detect fremitus may overlie thoracic aneurism, tumor of the lung, pneumothorax, and enlarged bronchial glands.

Fluctuations.—Fluctuation is seldom obtained over the chest, but when present, it is of great clinical importance. The commonest cause of fluctuation is aneurism—where, through pressure, there has been an erosion of the bones of the chest, a portion of the aneurismal sac protruding beyond the bony casing (Fig. 130, p. 315). In aneurismal tumor outside of the chest the opening through the bones is, as a rule, very small, the blood tumor expanding after it escapes from the chest. In rare cases fluctuation may be obtained over a large pleural effusion—a sign more common in children.

PERCUSSION.

In diseases of the chest, and especially in pathologic conditions of the lung, percussion offers very valuable clinical data. This method has been practised since its introduction, by Auenbrugger, in 1761.

Percussion consists in striking or tapping portions of the body in order to elicit vibrations; from the character of these vibrations it is possible to learn the conditions existing beneath the area percussed. In order to acquire skill in this method of diagnosis far more practice is required than in any one other clinical method.

Again, it is quite impossible to describe the practice necessary to accomplish the desired end. In order to become skilled in the art of percussion it is necessary—(1) that the tactile sense of the physician be well developed; (2) that he should be able to manipulate his hands and fingers with as much dexterity and ease as though he were playing a piano; (3) that he possess an acute faculty of distinguishing the degrees of vibrations. Whenever any one of these qualifications are lacking, the physician will never be able to obtain definite knowledge through the art of percussion.

Methods of Percussion.—(1) Immediate percussion; (2) mediate percussion. The latter is divided into three subheads: (*a*) finger percussion; (*b*) finger-plexímeter percussion; (*c*) hammer-plexímeter percussion.

1. **Technic for Direct (Immediate) Percussion.**—By this method physical signs are elicited by percussing the body-wall with the finger or fingers. Considerable practice is required to obtain success by this method, but when proficiency is attained, it is equally as valuable as indirect percussion. Direct percussion possesses one great advantage over other methods, since one is able to compare the notes obtained by percussion from each side of the body and from different points over the chest and abdomen. In hospital clinics it is our custom to strip the patient and to apply this method, beginning at the abdomen and continuing upward until the clavicles are reached; the same procedure is then applied to the back. By means of these methods one is often able to detect quickly the location of disease.

2. **Mediate Percussion.**—This method consists of placing a solid body (either the finger or a wooden or metallic substance) against the body-wall, and then striking it with the finger (Fig. 18) or with a hammer especially devised for the purpose. The medium placed against the chest-wall and

between it and the object with which the stroke is made is called a pleximeter. This should be of such size and form as to fit well between the ribs. When the hammer is used to strike the pleximeter, it is called a plexor. After considerable experience the plexor can be used with satisfactory results. Some examiners obtain better results by using the finger of the opposite hand than by employing the plexímeter and plexor. By placing the finger against the body and over the area from which sounds are to be elicited we obtain additional information, since the sensation that is offered by the patient's body to the operator's finger is often of great importance, and affords the physician a double method of ascertaining data—that is, by the sense of touch and of hearing. The further technic of mediate percussion is clearly shown by the accompanying illustrations (Figs. 18, 19).

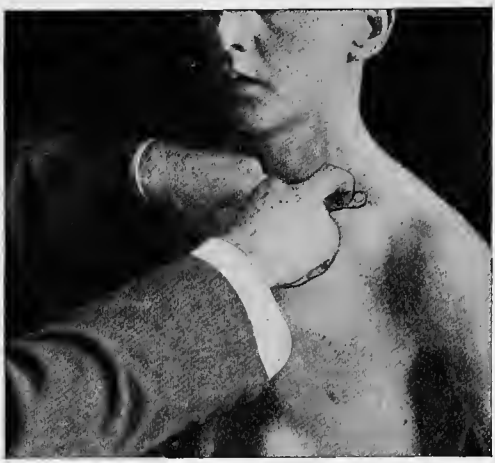


FIG. 18.—PERCUSSION OVER SUPRACLAVICULAR SPACE.

The position of the patient is an important factor in percussion. When the patient is able to stand, the examiner should insist that he stand as nearly erect as possible, and that he extend his chest to as near the normal as he can. Complete relaxation is a valuable aid in obtaining definite signs by percussion. Again, undue thickness of the chest-wall may materially interfere with this clinical observation.

In percussing the back of the chest it is well to direct the patient to fold the arms across the front of the chest, and to bend forward slightly, thus widening the space between the scapulæ (Fig. 20). The axillary region is readily exposed by directing the patient to lift both arms to near a level with his head, or to clasp his hands over his head. The patient should then direct his arms slightly backward, permitting them to hang along the posterior axillary line.

FIG 19.—APPLICATION OF FINGERS OVER APEX OF LUNG FOR PERCUSSION.

In a large proportion of cases one is compelled to percuss the chest-wall while the patient rests in bed, and it then becomes necessary to turn him from side to side. When percussing a patient in the recumbent posture, the physician must have clearly in mind the various positions the liver assumes as the result of posture. (See Diseases of Liver, p. 572.)

Analysis of Results Obtained by Percussion.—Sounds are usually distinguished by their pitch, volume, length or duration, and quality. The pitch is higher when the vibrations are rapid, and it will be noticed that the pitch will vary greatly over different portions of the chest and over the abdomen; consequently when the sound obtained is low in pitch, the vibrations are correspondingly slow.

Volume results from the amplitude of vibrations, therefore the degree of force exerted with each stroke of the percussing finger influences directly the volume of sound produced.

The accompanying illustration (Fig. 21) will serve as a diagrammatic representation of pitch.

Fig. 20.—Position of Patient for Percussion of Back between Scapulæ, Separating the Scapulæ and thus Increasing Interscapular Area.

Interpretation of Sounds.—Sounds can be correctly interpreted only after the operator has become thoroughly acquainted with the sounds to be elicited over different parts of the chest and over different organs during health. For example, it is impossible to describe with any degree of accuracy the note of pulmonary resonance or that of gastric tympany, tympany due to distention of the colon or to that of the small bowel, and it is likewise impossible to give a clear description of the note produced by hepatic dullness.

When the ear becomes familiar with these sounds, it will readily detect any deviation from the normal, which, in the majority of instances, indicates the existence of a pathologic condition.

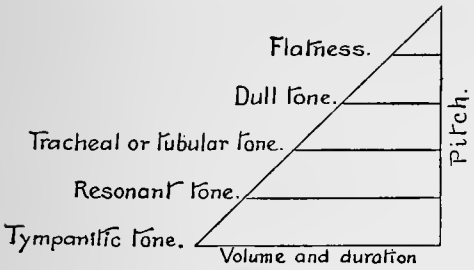


Fig. 21.—Diagrammatic Representation of the Character of Sound.
The perpendicular line shows the pitch; the horizontal lines, the duration and volume.

The sounds may vary considerably in pitch, volume, and tone during health, but such variations should never be confounded with those produced by disease. In order to detect the note of pathologic change, the examiner must first become thoroughly familiar with the sounds produced by extreme conditions during health.

Modifications in Health.—The degree of resonance is best exemplified by percussing over the upper axillary region, at the angle of the scapula and at the second rib anteriorly. At the second interspace a slightly higher pitched note is obtained on the right than upon the left side of the chest. The higher pitched percussion-note over the right than over the left apex is probably due to the larger diameter and higher position of the right

bronchus. The note is again modified by the thickness and the tension of the chest-wall as the result of muscular contraction, etc. There is always a lack of clearness in tones obtained in senile individuals, whereas in children the resonance is full and clear. The examiner must be thoroughly acquainted with the various sounds elicited from different portions of the chest, bearing in mind that the note obtained over the axillary region would be pathologic if it were elicited elsewhere, and that the note obtained over the apex of the lung differs greatly from that found at the angle of the scapula. The value of the evidence elicited through percussion is dependent upon three factors: (1) The area over which a certain sound is obtained; (2) the dexterity of the operator; (3) degree of muscular tension and thickness of chest-wall.

Tympany.—Pure tympany is obtained over a cavity or hollow viscus with smooth walls, and that is filled with air at the time of examination. The sound elicited is one of low pitch, great volume, and long duration.

During health tympany is elicited over the area of the stomach, but it must be remembered that this sound differs slightly from tympany the result of pathologic processes in the lung, *e. g.*, a pulmonary cavity. If the hollow viscus or cavity over which tympany is produced is unusually large, the note has a peculiar metallic character, best obtained when there is free air in the pleura (pneumothorax). (See p. 164.)

Caution.—Tympany over the base of the lungs posteriorly in children under two years of age is normal.

Dullness.—This sound is obtained as the result of percussion over that portion of the heart and of the liver not covered by lung. Areas of dullness where, under normal conditions, resonance should be obtained, is of pathologic significance. The peculiar types of dullness, that is, the pitch of the dull sound obtained and its duration and tone, are more or less intimately interwoven, so that a description here is scarcely practicable. In a word, dullness indicates that we are percussing over an organ that is practically airless, and, therefore, whenever this sign is obtained over the lung area, it signifies consolidation or an absence or diminution in the volume of air in that particular portion of the lung. Again, any deviation from the normal resonance that tends to approach the sound known as dullness shows that the volume of air in the lung occupying such area is less than that found under normal conditions.

Relative Dullness.—This type of dullness is obtained over structures that are airless, but where a portion of an air-containing viscus is interposed between the airless body and the chest-wall, *e. g.*, over that portion of the heart that is overlapped by lung tissue, relative dullness is obtained; the same sound is obtained over the dome of the liver (Fig. 234, p. 574). Absolute dullness is outlined with great ease by any method of percussion, whereas the determination of the area of relative dullness demands increased dexterity on the part of the operator and a greater cultivation of his auditory sense. Ability to determine the exact area of relative dullness is, therefore, one of the greatest achievements known to physical diagnosis, for in many instances in diseases of the lungs a positive diagnosis is based largely upon this finding.

Respiratory Percussion.—A physical sign that is especially applied to the difference of sounds over corresponding portions of the lung at the end of a full inspiration or a full expiration, the patient holding the breath after either act.

Superficial and Deep Percussion.—These terms are used to designate the force applied to the blow given in order to elicit sounds from certain tissue. Thus, deep percussion is required to outline the relative dullness

of the liver or of the heart, whereas, on the other hand, superficial percussion would be employed for outlining the areas of superficial or absolute dullness of these organs. Both superficial and deep percussion are required in an examination of the lungs, and are often of service in abdominal disease.

Auscultatory Percussion.—This physical method combines percussion with auscultation by means of the stethoscope (Fig. 22), and is by far the most practical method for the outlining of various diseased portions of the lung and for ascertaining the size of solid viscera. In determining the size of a viscus or an area of consolidation the stethoscope is placed near the supposed center of the area to be determined, and then percussion is made from distant portions of the chest toward the bell of the stethoscope, approaching the bell from all directions. Whenever the percussing finger reaches the margin of the solid body over which the stethoscope bell is

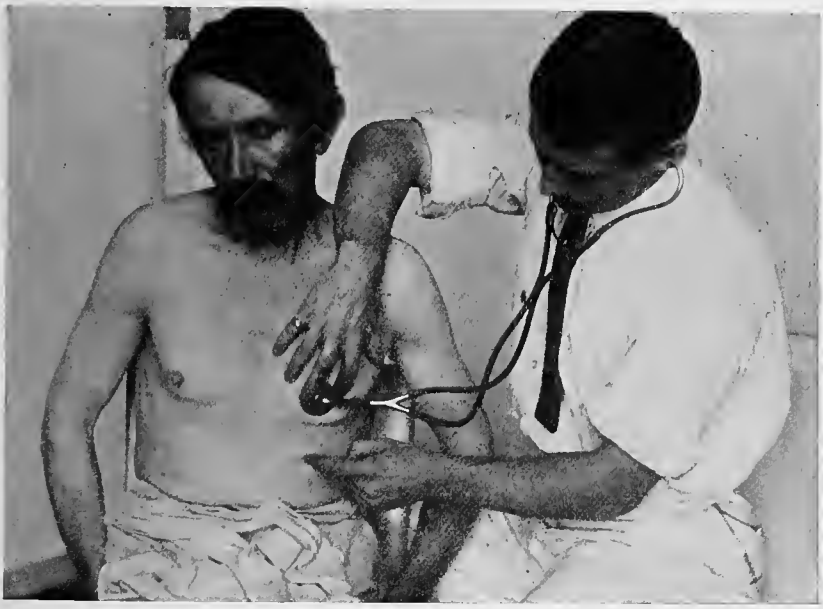


Fig. 22.—Method of Auscultatory Percussion Employed to Outline the Heart.

placed, a change of note will be audible. It is our custom to mark upon the chest-wall the point at which the note changes in percussing toward the bell of the stethoscope. When one has percussed in this way from practically every direction, encircling the bell of the stethoscope, the sounds have been carried to the bell along the lines corresponding to the spokes of a wheel. (See also Disease of Heart, p. 223.)

By means of this method the dullness of the liver is readily distinguished from that due to pulmonary consolidation of the right base, and the distinction between the flatness of pleural effusion and the dullness due to the liver is made with equal ease. Again, when examining the left side of the chest, this method is of great importance in determining the exact area of lung consolidation, and in differentiating such consolidation from heart dullness. Auscultatory percussion has been found a valuable aid in the diagnosis of tumors of the thorax and in outlining the heart. Lastly, better results are to be obtained through the use of auscultatory percussion by those having

but limited clinical training than by any of the other methods described here.

The note obtained by auscultatory percussion over the stomach is quite characteristic, as is also that audible when this method is applied over the colon and small intestine; nevertheless auscultatory percussion should not be employed to the exclusion of ordinary percussion in determining the size and location of the hollow viscera.

Palpatory Percussion.—This method consists in obtaining clinical evidence through two sources: (1) By an analysis of the type of sound that is produced by percussion; and (2) by an analysis of the character of sensation (resistance) offered to the finger that is placed against the chest-wall.

Diagnostic Significance of Special Signs in Disease.—The hyperresonant note, which approximates tympany, when found over both sides of the chest, is characteristic of emphysema (p. 124), but if the

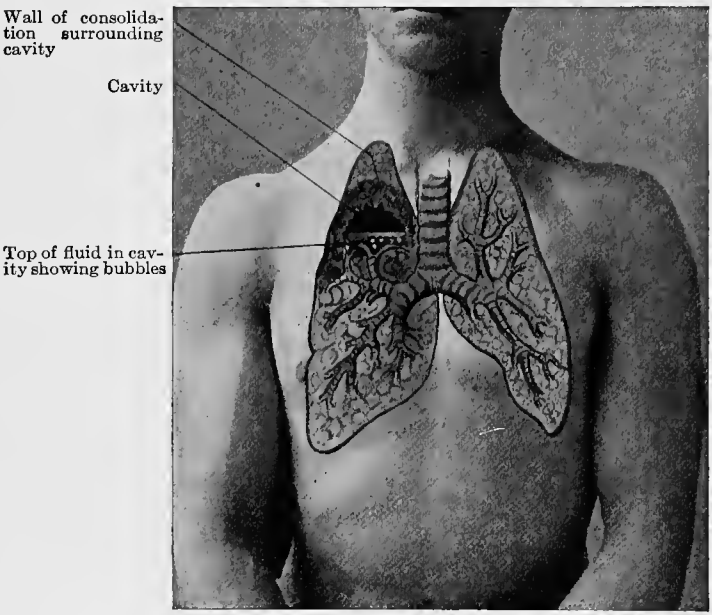


FIG. 23.—PHTHISIS WITH CAVITY FORMATION.

tension in the lung is extremely high, the tympanitic element of the note obtained is altered, and we have, instead, a variable or modified degree of dullness (wooden tympany).

Dullness at one or both bases, from whatever cause, is usually associated with hyperresonance at the apices (skodaic tympany, p. 128). Given a large pleural effusion, a skodaic note may be found in the region of the clavicle. Compensatory hyperresonance (skodaic tympany) is also found immediately surrounding areas of consolidation whenever the adjoining lung is healthy.

Dullness located at the apex of the lung deserves special consideration, since in the normal condition a variable degree of tympany—the so-called "bronchial percussion"—is present near the edge of the sternum and at the first and second interspaces. It is, therefore, necessary to percuss near the nipple-line in order to ascertain accurately the degree of hyper-

resonance surrounding apical consolidations. An increase in the area of cardiac dullness, when due to cardiac hypertrophy, dilatation, or the presence of pericardial fluid, may be accompanied by a hyperresonant note at the apex, in front and over the left scapular region posteriorly. This phenomenon exemplifies the important physical fact that whenever a portion of a lung is rendered incapable of functionating, either through disease of the lung itself or through intrathoracic pressure, portions of the healthy lung become emphysematous to compensate for such incapacity.

Tympany of the stomach has been discussed at length on p. 444. Some repetition is required, owing to the great importance that attaches itself to determining the actual boundaries of stomach tympany, which in the left anterior axillary line is a modified type of tympany at the fifth rib; when the stomach is distended, tympany may be found as high as the left fourth or even the third interspace. A localized area over which a tympanitic note is obtained signifies phthisis with cavity formation, bronchiectasis (dilated bronchus), pulmonary gangrene with cavity, and pulmonary abscess with cavity. The conditions that render it easy to elicit a tympanitic note over a small cavity are: (1) A thin chest-wall; (2) a relaxed condition of the chest; (3) a cavity situated near the chest-wall; (4) a cavity communicating with the bronchus.

Conditions exactly the reverse of those previously mentioned render it difficult to obtain tympany over a pulmonary cavity, and make forcible (deep) percussion necessary. At this point it is well to call attention to a pathologic condition that is prone to be neglected; *i. e.*, surrounding each cavity there is apt to be a thick band of consolidated lung tissue (Fig. 23), consequently upon moderate percussion dullness is obtained, whereas upon deep percussion tympany is elicited.

Amphoric resonance is the term applied to a variety of tympany to which is added a metallic quality; ordinarily it is somewhat high in pitch, but of slightly longer duration than tympany. Amphoric tympany is best obtained in pneumothorax when percussion is made over a pleura that is filled with air (p. 163). The degree of tension under which air is held in the pleura materially modifies the amphoric note obtained over such pleura; thus, when the tension is unusually high, a wooden note—the so-called "dull tympany"—is elicited. Amphoric resonance, when present over a large pulmonary cavity, indicates that such cavity rests near the ribs, has a firm wall, and that its inner surface is comparatively smooth.

Bell Tympany (Coin Percussion).—Whenever there is a suspicion of the presence of either general or localized pneumothorax, coin percussion should be applied; this is accomplished in the following manner: (1) Place a coin against the chest-wall and immediately over the center of the area of tympany; (2) place the ear against the opposite surface of the chest-wall, and then tap the coin gently with another coin or with some metallic substance. (See Fig. 54, p. 164.)

If there is air in the pleural cavity, an intensified metallic echoing sound is usually transmitted to the ear. Certain writers have referred to this peculiar note as bell tympany, and indeed the sound resembles that of a bell. This sound is rarely obtained over a large pulmonary cavity.

Cracked-pot Sound.—This is a variety of tympany to which is added a peculiar hissing and clinking sound. The clinking quality resembles that resulting from the tapping of a broken metallic vessel; hence the name, "cracked-pot sound." The hissing quality is apparently produced by air being forced through a small opening.

Caution.—In order to obtain this sound over a cavity several things are necessary: (1) The chest-wall must be relaxed and thin; (2) the cavity must rest near the surface of the lung; (3) it must be large and its wall thin; (4) the cavity must communicate with a bronchus; (5) the patient's mouth must be open; (6) the percussion strokes must be firm, but slow, giving the listener ample time to analyze each note produced. Percussion must be made during the act of expiration. Lastly, the sound is absent when a cavity is filled with liquid to a level above the communication with the bronchus (Fig. 23).

Peculiarities.—Theoretically, the cracked-pot sound should be elicited whenever the conditions just described are present, and, indeed, under such conditions this sound is obtained in the vast majority of cases. A peculiarity of this variety of tympany is that it is obtained over the chest of infants during health, and is invariably present when the child is crying. Occasionally, the cracked-pot sound is obtained over the chest of apparently healthy adults. A localized pneumothorax may communicate through the lung with a bronchus, and the cracked-pot sound may be one of its clinical manifestations. An extremely rare condition to find is a localized pneumothorax with an opening through the chest-wall. It is asserted by some that this variety of tympany may be elicited over that portion of the lung pushed well toward the apex of the chest as the result of a pleural effusion (p. 146), and that massive pneumonia may produce the same phenomenon.

Wintrich's Sign.—Here, percussion enables us to determine whether or not a cavity communicates with a large bronchus, in which case the percussion-note becomes louder and is raised in pitch when the patient opens his mouth, elevates his chin, and protrudes his tongue. *This sign is obtained at the end of inspiration, therefore the patient must inspire lightly and continue to do so until the percussion stroke is made.* By directing the patient to inspire deeply and then to hold his breath, as is at times recommended, a louder sound is produced, but, according to H. S. Anders, this destroys much of the characteristic element of the sign.

Exception.—If the cavity in the lung is filled with fluid to above a level of its communication with the bronchus, Wintrich's sign would be absent, but by changing the position of the patient the air in the vomica would communicate directly with the bronchus, when Wintrich's sign would be present—the so-called "interrupted Wintrich's phenomenon."

Percussion over the trachea and over a dilated bronchus causes a similar note under these conditions. Wintrich's note may also be obtained by percussing over a pleura distended by air, provided there is a large opening from the bronchus to the pleural sac. To obtain Wintrich's sign percussion is first practised, directing the patient to hold the mouth open and to protrude the tongue; later he is directed to close the mouth and lips tightly, and in this manner the operator obtains the greater variation in sound. In pleural effusion, where there is decided skodaic tympany above the liquid, a sound may be obtained by firm percussion that simulates closely Wintrich's phenomenon. In robust individuals Wintrich's note may follow firm percussion over the upper portion of the sternum. This sign is occasionally found in the presence of mediastinal tumors, owing to the fact that such growths occupy all the space between the surface of the sternum and the bronchi.

Friedreich's Sign.—Friedreich has called attention to a peculiar change in the degree of tympany when cavity in the lung is present; this observer

noticed that when the cavity communicated directly with the bronchus, the pitch of the note obtained was higher during and at the end of inspiration than during expiration. This sign may be of some value, but its presence is never necessary in order to determine the character of the lung condition, nor is it equally reliable with cracked-pot sound.

Gerhardt's Sign.—This phenomenon consists in a change in the pitch of the percussion sound obtained over a cavity with change in the position of the patient. Gerhardt's sign is obtained over a cavity in which one diameter is greater than the other, the cavity being partially filled with fluid. The change in the pitch of the percussion-note depends upon the alteration in shape of the air-containing portion of the cavity, as the result of change of position of fluid, and the lowest pitch is observed when the long diameter of the cavity is in the horizontal.

Biermer observed that the same phenomenal conditions observed by Gerhardt in pulmonary cavity were also present in pyopneumothorax.

AUSCULTATION.

Definition.—The method of listening to various sounds produced within the human body in health and during disease. Chief among the organs giving off these sounds are the heart, the lungs, the trachea, the gastro-intestinal tract, and the impregnated uterus. Abnormal sounds may be produced over any portion of the circulatory system as the result of pathologic changes.

As in percussion, so in auscultation, there are two methods of examination—the immediate and the mediate.

Methods.—In **immediate auscultation** the ear is placed directly against the wall of the patient's body, or separated merely by a thin towel. **Mediate auscultation** is a method in which an appliance, *e. g.*, the stethoscope, is used as an aid in conveying sounds to the ear. In examining the lung immediate auscultation is far more satisfactory than the mediate method, although the latter may be of value in certain special localized conditions. In examining the heart the mediate method should be applied, since the stethoscope enables one to recognize sounds and to outline their points of greatest intensity, as well as their areas of transmission with ease.

Advantages of Immediate (Direct) Auscultation.—Among these, special mention should be given of the following: (1) It is a ready method of making a rapid survey of the chest, as is often necessary in those who are extremely ill, who are unable to sit for any length of time, and who must be examined quickly, and while they are in the recumbent posture. (2) The value of the true respiratory sounds is better appreciated. (3) Slight alterations in sound are more likely to be detected. (4) When the ear is applied directly to the chest, the bruit of aneurism is more readily distinguished from the sounds of the heart. (5) Tactile sensations, conveyed to the examiner's ear when it is applied against the chest-wall, enable him to form a clear idea of the character of the chest movements. (6) The actual harshness of friction murmurs and voice vibrations are best appreciated when the ear is applied directly to the chest. (7) The exact time of action of the chest muscles is also appreciated, and this information is of great clinical value in acute fibrinous pleurisy.

Advantages of Indirect Auscultation.—Chief among these are: (1) It enables the listener to localize the point of greatest intensity of the sounds

to be analyzed. (2) A given sound may be followed over its various lines of distribution. (3) Pressure with the stethoscope over the thorax may cause certain sounds to disappear, whereas others are in this way intensified—a feature of some diagnostic importance. (4) It is possible to place the stethoscope over certain areas where it would be impracticable to employ the immediate method. (5) It is easier to obtain a knowledge of the condition of the lung in those patients who are too ill to sit while being examined. (6) It obviates direct contact with those suffering from contagious maladies. (7) In clinic and hospital work the operator may remain at a distance from his patient. (8) External sounds are excluded. (9) When combined with percussion, it serves as an excellent method for outlining viscera, areas of consolidation, tumors, etc. (See Auscultatory Percussion, p. 59).

Stethoscopic Auscultation.—While this is the most practical method for studying the condition of the heart, its field of usefulness is otherwise limited. The stethoscope does not give reliable results in examination of the lungs of children, and even in neurasthenic women. It is essential that the physician be thoroughly skilled in both the immediate and the mediate methods, and he must not use one method to the exclusion of the other.

FIG. 24.—METHOD OF HOLDING CHILD TO PERCUSS AND AUSCULTATE CHEST.

Technic.—It is preferable that the patient sit upright during the examination. While auscultating the front of the chest, the arms should hang carelessly by the side. When auscultating behind, the patient should fold the arms and lean slightly forward (Fig. 20). Both sides should have the same freedom of movement, which is attained only when the patient is sitting or standing.

It is necessary to listen over the lungs during forced inspiration, forced expiration, and to keep the ear in contact with the chest between these acts. Whenever possible, the chest should be bared, and a thin, unstarched towel placed between the examiner's ear and the chest-wall. In those cases in which it is not practical to remove all clothing, fairly good results may be obtained by auscultating through a thin unstarched garment. The room in which the examination is being made should, of course, be quiet. The best results are to be obtained by directing the patient how to inhale, speak, cough, clear the throat, and to whisper during this portion of a physical examination.

Normal Breath-sounds.—Under normal conditions there are three distinct sounds to be heard over specified areas of the chest, and if the student is thoroughly skilled in the recognition of these sounds, he will be able to detect disease of the lung or of its coverings whenever such sounds are heard over areas where they are not audible during health.

Bronchial Breathing.—This is a type of respiration heard nor-

mally over the trachea (Fig. 25), but pathologic whenever heard over the substance of the lung. By placing the stethoscope over the trachea immediately above the suprasternal notch, two distinct sounds are heard: (1) The one during inspiration; and (2) the one during expiration. These sounds are separated by a pause which is observed immediately before the end of inspiration. The sounds of both inspiration and expiration are practically of the same length, and the quality is harsh, blowing, or, as is often stated, tubular. The sound of bronchial breathing is loud and high pitched, although this may vary somewhat between inspiration and expiration. The sound during both acts, however, shows an elevation of pitch and in-

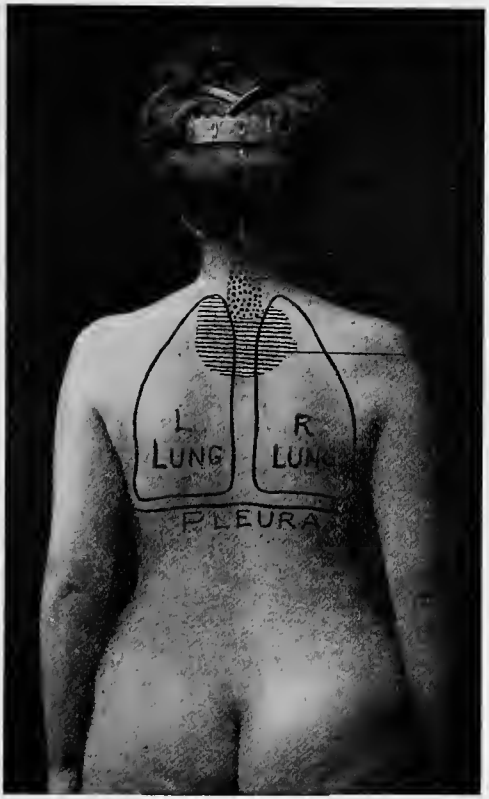


Fig. 25.—Area on Posterior Wall of Chest, where Bronchovesicular Breathing is Normally Present.

creased intensity. The areas over which bronchial breathing is to be found during health are shown in the accompanying illustrations (Figs. 25 and 26).

Bronchovesicular Breathing.—This breath-sound represents an imperfect type of bronchial breathing, as well as an exaggerated type of vesicular breathing. It is often referred to as a mixed type of breath-sound, since it displays definitely a certain amount of bronchial element, as well as an imperfect vesicular respiratory murmur. Bronchovesicular breathing is heard over a portion of the sternum, along the thoracic vertebræ, and over certain other areas of the chest, as is shown in the illustrations (Figs. 25, 26,

27 and 28). This type of breathing requires no description, for it may be obtained by auscultating the normal chest. Attention is, however, called to the fact that bronchovesicular breath-sounds are normal over the back of the chest as high as the vertebra prominens, and even to the third and fourth thoracic vertebræ. Owing to the anatomic formation of the right bronchus, this breath-sound is heard for some distance to the right of the spinal column.

Vesicular Breathing.—This is made up of a variety of breath-sounds heard over those portions of the lungs situated away from the areas over which bronchial and bronchovesicular breathing are normally present. Vesicular breathing has been described as resembling the sound produced by a soft breeze or as slightly sighing currents of air; by some it has been compared to the gentle rustling of the leaves of a tree by the wind. It is impossible to give a correct description of this sound, but a thorough

FIG. 26.—BRONCHIAL AND BRONCHOVESICULAR BREATHING DURING HEALTH.

acquaintance with it should be had by every student of medicine. A characteristic feature of vesicular breathing is the peculiar, breezy nature of the sound, which is practically continuous; in other words, it is heard during the whole of inspiration, and is immediately followed by the shorter sound caused by expiration. Vesicular breathing is modified by directing the patient to inspire deeply or to hold his breath.

During the act of inspiration the sound is moderately intense, but of low pitch, and is relatively three times as long as the expiratory murmur. During expiration the sound may not be audible or it may be present during but a portion of the act; it is less intense and of somewhat lower pitch, although during this act there is added a slight blowing quality—merely a soft puff of air.

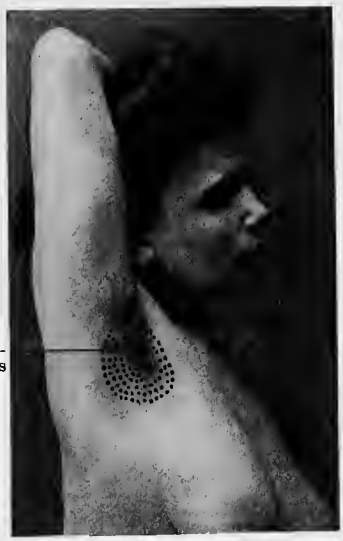
As has elsewhere been stated, the chest movements of inspiration and

expiration are to one another in time of duration as five is to six, although the sounds of vesicular respiration bear a ratio of three to one or four to one.

Variations in Vesicular Breathing.—It is important that the variations in vesicular breathing capable of being excited through normal conditions be thoroughly understood.

Breath-sounds normally show slight increase in sound

FIG. 27.—AUSCULTATION OF AXILLA FOR BRONCHOVESICULAR BREATHING.



Normal area where bronchovesicular breathing is heard.

FIG. 28.—AUSCULTATION OF AXILLA FOR BRONCHOVESICULAR BREATHING.

Age.—Up until the twelfth year the vesicular quality that characterizes vesicular breathing (in the adult) is markedly exaggerated, and the breath-sounds are harsher and louder than after puberty. At the other extreme of life, old age, the vesicular quality, while it retains this harsh sound is much more feeble than it is during early adult and middle life.

This condition is believed to depend upon a loss or weakening of the elasticity of the lung. Inspiration appears to be shorter, whereas expiration is slightly prolonged.

Sex.—The respiratory murmur is appreciably louder in the female than in the male. Auscultating over the upper and anterior portion of the chest, the breath-sounds are much increased in intensity in women. Certain anatomic conditions may, in selected cases, account for the softness of the respiratory murmurs in males—*e. g.*, the thickness of the chest-wall, firmness of the tissues, etc.

Regions Where Increased Sounds are Heard.—During health the breath-sounds are slightly more distinct and louder upon the right side, and this feature is most pronounced in the infraclavicular region. The thickness of the chest-wall influences the degree of sound conveyed to the listening ear, consequently the sounds are clearer anteriorly and in the axillary and infraclavicular regions than they are over the mammary and scapular regions. These variations in the intensity of the breath-sounds may be found in doubtful pathologic pulmonary conditions.

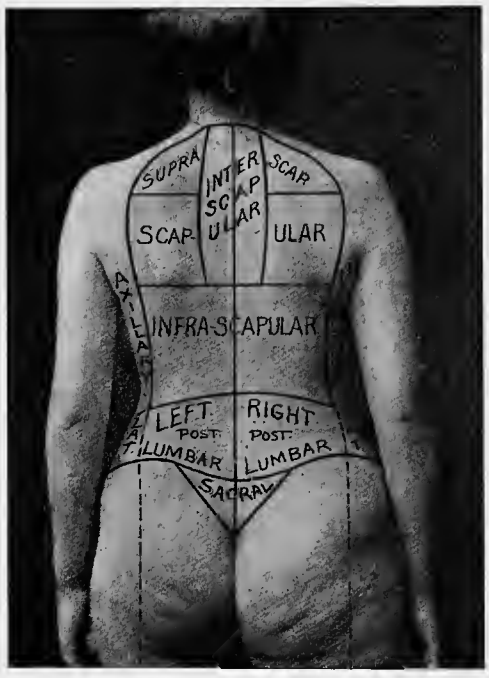


Fig. 29.—Arbitrary Division of the Back.

The appreciable weakness and almost absence of the vesicular murmur may also be physiologic when auscultating over thickened portions of the chest-wall, etc., for example, over heavy muscles, in obesity, in massiveness of the chest-wall.

Jerky (Cog-wheel) Respiration.—The so-called jerky or cog-wheel respiration is generally conceded to be an early sign of tuberculosis, but this peculiar interruption in the respiratory murmur is also an occasional feature during health. Jerky respiration when present is best seen in those who breathe slowly, and this type of respiration is, as a rule, better brought out by directing the patient to inspire deeply. This type of breath-sound is common in children while fretting and when crying, and in hysteric women. Physiologic irregularity in the respiratory murmur is audible over all portions of the lung, whereas irregularity due to incipient tuberculosis is heard only over isolated areas.

Systolic (Cardiac) Vesicular Breathing.—This respiratory murmur is characterized by a rhythmic exaggeration that is more or less jerky in character, and apparently influenced by the action of the heart. This peculiarity in the vesicular murmur is audible while the lung is expanding, and is limited to those portions of the lung overlapping the heart. The vesicular murmur gradually increases until the end of inspiration, after which there is an appreciable pause.

RÂLES (RHONCHI; RATTLES).

Râles are adventitious sounds heard over the lungs. They have received various classifications, and Page distinguished three great classes: (1) The dry; (2) the moist, and (3) indeterminate forms.

Dry Râles.—Of the several varieties of dry râles, the following are the ones that must be recognized for diagnostic purposes: (*a*) Sibilant and (*b*) sonorous.

Sibilant Râles.—These are high pitched and whistling in character, occurring with inspiration or with expiration, and may be present during both acts. The sibilant râle may be produced in the larynx or in the trachea, provided the caliber of either is sufficiently narrowed, and the same physical condition serves to explain the production of the sibilant râle in the larger bronchi. They are most commonly produced in the smaller bronchi, and result from the same mechanic conditions that give rise to coarse râles or from swelling of the mucous membrane. Sibilant râles are audible during the dry stage of acute bronchitis and in asthma.

Sonorous Râles.—These are loud, low-pitched, dry râles that accompany inspiration or expiration, and may even be heard during both acts. Ordinarily, the sonorous râle is produced in the larynx, the trachea, or the larger bronchi. Sonorous râles are produced in the larynx as the result of spasm of the glottis, hence they are a conspicuous sign in croup, whooping-cough, thoracic aneurism, mediastinal tumor, conditions that exert undue pressure upon the recurrent laryngeal nerve. A sonorous râle may originate in the trachea if this tube is either partially closed, from the pressure of external tumors and growths, or if its lumen is diminished as the result of new-growths upon its mucous surface. Inflammatory and edematous as well as cicatricial changes may also cause narrowing of the trachea.

Râles originating in the trachea are audible by the aid of the stethoscope over all portions of the lung, but are most distinctly heard nearest the site of their production. The sonorous râle may also be produced in the larger bronchi as the result of narrowing of the lumen of such pulmonary tubules, regardless of whether the condition results from external pressure, chronic inflammation, edema or spasm of the lining mucous membrane, and, indeed, the accumulation of thick, tenacious mucus may produce such râles within the bronchi. Sonorous râles are, as a rule, temporary, often disappearing after the patient coughs or clears his throat, a clinical evidence that suggests that they are possibly due to a varying spasmodic condition and to vibrating mucus that is dislodged by the act of coughing. Should the condition that favors the production of the sonorous râle be a permanent one, but few râles may be audible. During bronchitis the sonorous râle may accompany the sibilant variety.

Moist Râles.—These may be produced in the larynx, trachea, a pulmonary cavity, the bronchi, and in the air-cells. The moist râles that occur in the bronchi as the result of lowered vitality are an example of this particular type, and the sound thus produced is what is commonly known as the death-rattle. Moist râles are also heard over the entire surface of the chest in certain pulmonary conditions, but, as in the case of dry râles, it is possible to locate the area of their production by means of the stethoscope. In all instances where any type of râle is audible over the chest it is advisable to direct the patient to clear his throat or to cough, for in this way it may be possible to prevent temporarily the production of moist râles in the larynx and trachea.

Subvarieties of Moist Râles.—Certain subvarieties of the moist râle

are at times audible over the bronchi. For diagnostic purposes these have been divided into three subclasses: (1) Mucous; (2) submucous; and (3) subcrepitant.

Mucous Râles.—The mucous râle is a rather large, moist, bubbling sound, produced in the larger bronchi, and audible during the act of inspiration and of expiration. Mucous râles are materially modified by the act of coughing, and especially is this true when cough is accompanied by free expectoration. Fluid of whatever nature, when it accumulates in the larger bronchi, is likely to give rise to mucous râles; hence when a purulent, mucous, or bloody exudate exists upon the bronchial mucous membrane, such râles are present. Mucous râles frequently disappear after coughing, but may also be produced through the very act of coughing or clearing the throat. These râles may be heard over localized areas of the lung, or, as is more commonly the case, they are audible over the greater portion of both lungs.

Submucous Râles.—These are moist, bubbling râles, apparently smaller than mucous râles. They are probably produced in the medium-sized bronchi. Like mucous râles, they are heard during the respiratory acts, and are influenced by cough, expectoration, etc.

Subcrepitant (*Mucocrepitant*) *Râles.*—This variety represents the finest of the moist râles, and has its origin in the smaller bronchial tubes. This type of râle is heard chiefly on inspiration, and is not so readily influenced by coughing and clearing of the throat as are either mucous or submucous râles. This type of râle is probably caused by the inspired air forcibly separating the agglutinated walls of the finer ramifications of the lungs. In bronchopneumonia (capillary bronchitis) the subcrepitant râle is likely to be audible over the lower portion of both lungs posteriorly; it may also be a symptom of pulmonary edema, and is not infrequently detected during the stage of resolution in lobar pneumonia. The subcrepitant râle (crepitus redux) may be present in pulmonary hemorrhage, where the blood has escaped into the lung tubules, and in a similar manner pus may give rise to the subcrepitant râle. During the early stage of phthisis this variety of râle may be elicited immediately over the area of congestion, and although it is heard at other stages of the disease, when so heard it possesses less clinical significance.

Crepitant Râles.—These are produced in the air-cells, and are the only vesicular râles audible. They are characterized by an unusually fine, uniform, crackling sound, heard just at the end of inspiration. Another characteristic of the crepitant râle is that it is not influenced by coughing. The theory offered for their production is that the inspired air forcibly separates the agglutinated walls of the air-cells. Some observers believe that it is of pleural origin.

The crepitant râle is heard during the stage of congestion in croupous pneumonia. The crepitant râle may possibly have escaped notice during the first stage of pneumonia. It is absent during the second stage, while the air-cells are obliterated or filled with exudate. The râle heard in the third stage and known as the redux râle is in reality a subcrepitant râle.

Mucous Click.—This is a single, fine, high-pitched, moist, clicking sound, heard over both lungs, and but slightly, if at all, altered by coughing. The time of its appearance varies somewhat, but it is usually heard during or near the end of inspiration. The mucous click is quite commonly detected during the course of pulmonary tuberculosis, and over areas where there is incomplete consolidation.

Gurgles.—These are exceptionally large, moist, bubbling râles, probably originating in a pulmonary cavity or in an expanded bronchus that is par-

tially filled with fluid. They vary greatly in size, and are both high and low in pitch, depending upon the size of the cavity and upon the degree of consolidated tissue surrounding it. Gurgles are heard during both inspiration and expiration, but since inspired air enters the cavity with more force than is present during expiration, the sounds are louder during the inspiratory act.

Intrapleural Moist Râles (*Friction Murmurs*).—The pleuritic friction-sounds at times simulate closely those described as moist, bronchial, and vesicular râles, and some writers suggest that both the subcrepitant and the crepitant râles are intrapleural in origin. In selected cases the intrapleural murmur resembles both the mucous and the submucous râles, and it was suggested by the late J. M. Da Costa that the human ear could not always determine the origin of such râles. The following features, however, are characteristic of pleural râles: They are always localized, are audible over but one lung, are unaltered by coughing, unattended with expectoration, and strike the ear as being distinctly superficial.

"Intrapleural moist râles do not require actual inflammation of the pleuræ for their production." Alterations in the nature of a pleural exudate doubtless contribute toward the production of râles simulating other types previously described. (See also Pleural Friction-sound, below.)

Indeterminate Râle.—Under this head should be considered all other râles not included in the foregoing classification, and, generally speaking, they may be said to consist of crackling, grumbling, bubbling, and splashing sounds, that appear at first to be in part moist, partially dry, and heard during the acts both of inspiration and of expiration. These indeterminate râles are extremely frequent during the later stages of pulmonary tuberculosis, and are also a more common symptom in complicated lobar pneumonia, pulmonary abscess, and pulmonary gangrene.

Friction-sounds.—These are induced through pleuritic inflammation, and their significance was described by Honoré as early as 1819. The physiologic action of the pleuræ consists in a gentle gliding of the two layers, which is dependent in part upon lubrication of the pleuræ by a serous secretion. The pleural friction-sound is heard when inflammatory or other changes of sufficient gravity have taken place in the pleuræ, and have either roughened its surface or altered the character of the fluid which lubricates these surfaces; as a consequence, during the acts of respiration the dry, roughened surface gives rise to a rubbing, more or less grating, and crackling sound.

Pleural friction murmurs are heard distinctly during inspiration, and it is possible for several friction-sounds of varying duration to be present, which gives the impression to the listener's ear that the sound is interrupted. The pleural friction murmur is characteristic of the early stage of acute pleurisy, disappears during the stage of exudate, and frequently reappears following absorption or removal of the exudate by artificial means. In selected cases of acute pleurisy the diagnosis of a friction murmur is further substantiated by the detection of a friction fremitus by palpating over the area of greatest intensity of sound.

Splashing Sound (Succussion Splash).—The method by which such sounds are produced is termed succussion, and consists in shaking the patient while the ear is kept in direct contact with the chest-wall or the abdominal wall. (See Fig. 55.) The succussion sound may also be utilized in the diagnosis of diseases of the stomach (dilatation) and in intestinal obstruction. It is practically always possible to elicit a splashing sound in the case of pyopneumothorax and of hydropneumothorax. This sound is said by some observers to be audible over a large pulmonary cavity. The

splashing sound, when heard over the upper portion of the chest, is pathognomonic of either pyopneumothorax or pneumopericardium, the serous sacs involved containing a variable quantity of fluid. Cases have repeatedly been seen in which a distinct splashing sound was audible over the base of the chest in those suffering from subdiaphragmatic abscess (p. 595). A splashing sound present over the base of the chest, and particularly over the area of the stomach, is somewhat common in the negro. Succussion splash is a common sign in pneumothorax and in dilatation of the stomach, and it may rarely be encountered in connection with other conditions. The following table will set forth the possible causes of this sign when the sound is produced within the layers of the diaphragm or within the thorax:

1. Hydropneumothorax.
2. Pyopneumothorax.
3. Hemopneumothorax.
4. Subdiaphragmatic abscess, infected by the Bacillus coli communis.
5. Pyopneumopericardium.
6. Large pulmonary cavity.
7. Pleural effusion, infected by Bacillus coli communis.
8. Gastric ulcer (perforating the diaphragm and pleura).
9. Duodenal ulcer (perforating the diaphragm and pleura).
10. Hepatic abscess (perforating the diaphragm and pleura).
11. Cancer of the esophagus (perforating the pleura).
12. Traumatism with perforation of the pleura.
13. Diaphragmatic hernia.
14. Infection of pleura by Bacillus aërogenes capsulatus.

(See also p. 506.)

Metallic (Amphoric) Tinkle.—A peculiar tinkling sound displaying an initial amphoric quality, and heard over large cavities having a smooth inner surface. The following serves to explain this sound in a pulmonary cavity: Given a cavity of fair size with smooth walls, partially filled with liquid, and where the bronchus communicating with such cavity opens beneath the surface of the liquid: air entering from the bronchus when passing through the liquid produces an explosion or bubble, which, owing to the smooth cavity wall, is transmitted to the ear as a metallic tinkle. This sound is also believed to be produced by vibrations of viscid and semi-liquid substances contained within a cavity. The metallic tinkle is heard oftenest during inspiration, and may be produced by forced inspiration, speaking, coughing, and laughing. It is also heard in pyopneumothorax (see p. 164), in which conditions it is frequently heard following the succussion splash which is produced by shaking the patient. (See p. 164.)

Grunt.—The act of expiration may be accompanied by a distinct grunting sound, which in well-marked cases may be audible at some distance from the patient's chest, although it is usually elicited by placing the ear over the affected side. This sound is fairly characteristic of the stage of consolidation in lobar pneumonia.

Egophony.—Egophony is a variety of vocal resonance in which the sounds resemble the bleating of a goat. It is heard usually when there is a thin layer of fluid between the lung and the chest-wall. The most common seat of its production is at the angle of the scapula in cases of pleural effusion. It is also heard over superficial areas of collapse of the lung, and occasionally in cases of croupous pneumonia.

Compensatory Emphysema.—Whenever a portion of the once healthy lung has become incapacitated from any cause, its fellow and remaining healthy portions of the same lung are forced to do extra work. In auscultating over portions of compensating lung the vesicular element is exaggerated, and the sound obtained closely resembles that characteristic of the respiratory murmur of children. When the compensating lung is in close

proximity with a bronchus, the breathing is slightly more exaggerated, and is referred to as puerile respiration. This murmur, however, is usually detected over areas where bronchovesicular breathing is normally present.

Voice Sounds in Health.—By applying the ear or the stethoscope to the chest of a patient and directing him to turn his face away from the examiner and to speak in an ordinary tone of voice, counting one, two, three, a breezy noise is heard, but articular sound is absent. This sound is obtained over areas where only vesicular breathing is heard. Voice-sounds in health are influenced, first, by the character of the patient's voice, and, secondly, by the thickness of the chest-wall. The spoken voice apparently creates more and more sound as the listener approaches a large bronchus. The sounds heard over the lung have a similar significance to the vibrations transmitted from the larynx, trachea, bronchial air-columns, and substance of the lung and chest to the examiner's hand when palpating the chest. Vocal resonance consists of a form of vibrations that are appreciable only by the auditory sense.

The peculiarities of the voice-sound and its relation to disease will be discussed at length under each pathologic condition in which they form one of the physical signs. (See Pneumonia, Pulmonary Abscess, Tuberculosis.)

THE X-RAY EVIDENCE OF DISEASES OF THE BRONCHI, LUNGS, PLEURA, AND DIAPHRAGM.

By G. E. Pfahler, M.D.

General Remarks.—In general, the *x*-rays are absorbed by the tissues through which they pass in proportion to their density and thickness, and therefore will cast corresponding shadows upon the fluorescent screen or photographic plate. Any disease that will vary the density, outline, or position of tissues or organs can be demonstrated; likewise any disease which will modify the movements of an organ.

The shadows cast upon the plate or screen in any instance will vary much with the position of the tube in relation to the location of the lesion. Therefore, in order that the truest picture of the disease be obtained, it is important that the Röntgenologist have some general information as to the probable character and location of the disease preceding the examination. Likewise, so far as possible, the *x*-ray evidence must be interpreted in the light of the physical signs and clinical history.

The most transparent tissue of the body is the lung. Disease may increase this transparence or render it less transparent. Any condition which will increase the air-content of the lungs (asthma, emphysema) or decrease the thickness of the chest-walls (emaciation) will render them more transparent. Any condition which will diminish the air-content of the lungs (consolidations, neoplasms, etc.) or increase the thickness of the chest-wall (great muscular development, fat, edema, tumors) will decrease this transparency.

DISEASES WHICH INCREASE THE TRANSPARENCY ON THE LUNG.

Emphysema.—The increase in transparency is marked unless accompanied by edema or congestion. The interspaces are wider, the diaphragm lower, and its movement less.

Chronic asthma will, of course, give a similar appearance because of a secondary emphysema.

DISEASES WHICH DECREASE THE TRANSPARENCY OF THE LUNG.

Pneumonia.—In a typical lobar pneumonia the entire affected area presents a dense and almost uniform shadow, which is rather sharply outlined. At times, the consolidated area is not sharply defined, or there may be some extension of the process or congestion in the neighboring lung tissue.

At the beginning of the disease there is usually a faint shadow of consolidation in the region of the large bronchi. In a few hours, however, the shadow may extend over the entire lobe or lung and become absolutely and uniformly dense. This extension is usually from the root of the lung, but it may be from the periphery. When these early shadows are obtained, the physical signs may be entirely absent, because centrally located.

When complete consolidation has taken place, the density of the shadow may be so great as to obliterate the outlines of the ribs. Ordinarily, however, the consolidated area is sufficiently transparent to make the ribs visible, and this shadow is not uniformly dense. At the time of the crisis the consolidated area increases in transparency, usually beginning at the hilus (Rieder and Steyrer), or diffusely (v. Jaksch and Ratky). This increase in transparency keeps pace with the physical signs, but even after all physical signs disappear, shadows may be found, indicating incomplete resolution lasting several weeks (de la Camp). When such shadows are found, one must think of a recurrence, chronic pneumonia, tuberculosis, empyema, abscess, gangrene, and hydatid disease, though ordinarily they disappear without any further clinical evidence.

Localized empyema will usually give a dense, homogeneous shadow, with sharp outlines, which do not follow the outline of a pleural effusion.

Abscess and gangrene give very similar pictures, and neither one can be definitely differentiated by the *x*-ray evidence alone from tuberculosis. When a cavity has formed and the evidence is weighed with the clinical findings, a diagnosis can be made and the disease definitely located if operation seems advisable.

Adhesions to the pleura, diaphragm, or pericardium, which not infrequently follow pneumonia, and which may interfere with the free movement of the diaphragm or heart, can be demonstrated upon the fluorescent screen and the photographic plate.

Bronchopneumonia gives, as one would expect, shadows of smaller areas of consolidation, and in different parts of the same lung or both lungs. These shadows are less dense and more mottled.

Miliary tuberculosis may give a picture similar to that of bronchopneumonia.

Tuberculosis can be demonstrated in the lungs because the normal air-space is replaced by a more or less solid substance, which casts a denser shadow upon the fluorescent screen or photographic plate, and the disease can be demonstrated as early as such replacement occurs. Under favorable conditions tubercular deposits an eighth to a quarter of an inch in diameter can be shown, and, as a rule, the disease can be demonstrated before definite physical signs manifest themselves.

In contradistinction to the large and dense shadow cast in lobar pneumonia, in tuberculosis we have minute shadows, which may coalesce or overlie one another, and give a large area of the lung a mottled appearance. We seldom have the degree of density nor the uniformity seen in lobar pneumonia.

In *early cases* this mottled appearance is most likely to be found at the apices, and usually more on one side than the other, but it is often found early along the inner border or in the axillary region. As a rule, the disease is more extensive than is indicated by the physical signs. This is due to the fact that the deeper or centrally located lesions give less definite or no physical signs, while the shadows cast are equally as strong as the peripheral lesions.

One must be cautious in interpreting the general increase in shadow at the apices as tuberculosis, for an increase in the thickness of the overlying tissue on one side (muscle, fat, enlarged supraclavicular glands) or a lack of expansion (therefore, lack of air) may give a similar appearance. In such instances the shadows are not mottled, and in the lack of expansion it is likely to be bilateral.

Enlarged bronchial glands give isolated, rather dense, round or oval shadows in the region of the large bronchi. This examination is especially important in children when tuberculosis is suspected from the clinical symptoms, even though the physical signs are absent.

One must not misinterpret the radiating, rather large shadows in the region of the large bronchi, which are produced by the large blood-vessels.

Chronic Tuberculosis.—The infiltrations of chronic tuberculosis can be clearly shown. As a rule, the more chronic the process, the more dense will be the shadow of lesions of similar size and the more clearly outlined (partly because of the surrounding compensatory emphysema). In the *fibroid* variety one sees the shadows extending radially rather than in an irregular mottled fashion.

Cavities are recognized (when not filled with fluid) by their increased transparency, and consist usually of a light area surrounded by a dark wall of consolidation. Cavities the size of a pea have been recognized.

Bronchiectasis may be demonstrated when the cavities are empty or only partially filled with fluid, and especially when they can be found filled (dark area) in one instance and empty (light area) in another. Their transparency will be similar to tuberculous cavities, but the amount of surrounding shadow will depend upon the accompanying disease.

Syphilis of the lung has been demonstrated in a few instances, but the appearances are similar to those of tuberculosis. These appearances become important only when the clinical history is that of syphilis rather than of tuberculosis.

Anthracosis gives a similar appearance in many respects to that of fibroid phthisis, but a very much more dense shadow.

Neoplasms of the lung can, of course, be demonstrated, because there is a replacement of the transparent air-space by solid tissue. If this is a metastatic process and occurs in small multiple lesions, the appearance will be similar to tuberculosis, but the lesions seem to radiate more from the mediastinal region, and each lesion seems to have a more definite outline. When the lesion is large, it is more dense, more homogeneous in its shadow, and more clearly outlined than in tuberculosis.

THE DIAPHRAGM.

The Röntgen rays surely supersede all other methods in the examination of the diaphragm. Variations will occur in the outline, the position, and the movements. Here both a Röntgenoscopic and a Röntgenographic examination are useful.

The *normal* position and movements of the diaphragm vary very much,

and statements on this subject must be accepted as general and not taken as a standard. Measured *orthodiagraphically* in a healthy young subject with firm abdominal walls, anteriorly the right side is on a level with the upper border of the fifth rib, and the left side with the lower border of the fifth rib (Jamin). In quiet breathing the normal range of movement is 1 to 2 centimeters. With deep inspiration the diaphragm moves downward 2 to 4 centimeters. Practically, in healthy subjects, both sides should move equally, and therefore a difference in the degree of movement on the two sides will be important diagnostic evidence.

Early tuberculosis is likely to limit the degree of movement on the affected side (Williams' sign). Such limitation of the movement of the diaphragm on one side may be due to other conditions, such as painful affections above or below the diaphragm. The pain of pleurisy will therefore limit the movements of the diaphragm, and in the absence of physical signs, or when the pain is indefinite, it may be valuable evidence of a diaphragmatic pleurisy.

Pleuritic adhesions will limit the movements of the diaphragm, but this limitation is likely to be localized, and is evidenced by humps or peaks in the upper curve.

Hydatid cyst, if located on the upper surface of the liver, will give a localized elevation to the curve of the diaphragm, which is smooth in outline.

Subdiaphragmatic abscess will give an abnormally high position of the diaphragm on the affected side, will usually render it motionless (on account of pain), and will likely modify its contour.

Diaphragmatic Hernia.—In this condition there is an area of abnormal transparency above the line of the diaphragm in which the lung structure is absent, if large. If small, the overlying lung will cast some shadow. This condition must be differentiated from a *localized pneumothorax,* from a large *cavity,* and from eventration of the diaphragm.

If the patient "strains" with the abdominal muscles, as if at stool, holding the breath, a hernia will increase, while a pneumothorax, a cavity, and eventration of the diaphragm will remain stationary. Further, by giving bismuth mixture one may be able to trace the food through the hernia. *Eventratio diaphragmatica,* or congenital atrophy of the diaphragm, gives it a remarkably high position, uniformity in its outline, with limitation or absence in movement. The heart will be displaced.

In these differentiations, as in all others, much will depend upon the skill and resourcefulness of the Röntgenologist in eliminating error and arriving at a correct diagnosis.

THE PLEURA.

The normal pleura is not demonstrable by the rays.

Thickened pleura will produce a shadow in proportion to its thickness and extent. When very thick, 2 to 3 cm., it may be confused with a *new-growth.* When a new-growth is single, if viewed from all sides, it should give a more definite outline. If multiple, it should give a nodular and less uniform appearance. The thickened pleura can usually be differentiated from the shadow cast by *tuberculosis of the lung* because it is more homogeneous and fades gradually at its borders. If complicated by overlying tuberculosis, it cannot be differentiated.

Adherent pleura is recognized by its interference with the movements of the diaphragm or a portion of the lung, or when adherent to the pericardium, may interfere with the action of the heart.

Pleural effusion produces evidences according to its extent, its character, and the condition of the overlying tissue. If the lungs are congested, or the chest-walls thick, it is more difficult to recognize. Generally it can be demonstrated by the homogeneous shadow which it casts, which changes its level with a change in position, and by its displacement of the heart and diaphragm.

Pneumothorax is usually easily recognized by the very transparent area, together with the shadow of the visceral pleura and compressed lung, which is seen to be separated from the chest-wall; also by the displacement of the mediastinal organs.

Localized or interlobular pneumothorax requires great care in making a diagnosis, but usually this can be done.

Pyopneumothorax gives perhaps the most striking picture observed in diseases of the chest. One sees the very transparent area of the pneumothorax, with the thickened pleura or compressed lung on the inner side and the level of the fluid below, which waves up and down with each movement of the diaphragm. When one shakes the patient, a distinct splash can be seen, fluoroscopically.

THE SPUTUM.

COLLECTION.

In collecting the sputum for examination the ordinary vaselin bottle forms the most convenient receptacle. The bottle should, of course, be carefully cleansed to remove the fat-globules, and should be sterilized, if possible. This should be placed at the patient's bedside or in his room, so that when he coughs in the morning he may expectorate directly into the bottle. After the bottle is half full, it should be corked tightly and wrapped in heavy paper, upon which the name of the patient should be written.

Caution! Do not add water.

CHARACTERISTICS OF THE SPUTUM IN DISEASE.

Quantity.—The quantity of sputum ejected in pathologic conditions during twenty-four hours varies between a few cubic centimeters to 500 or even 1000 c.c., and bears a direct relation to the nature of the disease in question.

Copious expectoration occurs in pulmonary hemorrhage, pulmonary edema, bronchiectasis, tuberculosis, rupture of an abscess into the lung (diaphragmatic, hepatic, mediastinal), and rarely in pleural effusions. The so-called albuminous sputum, which is associated with pulmonary gangrene, is often profuse. In acute inflammatory processes involving the lung the sputum is generally scanty.

Odor.—The odor of the sputum can be regarded as characteristic in but two conditions—pulmonary gangrene and putrid bronchitis; in these diseases it has an offensive odor. At times the sputum of bronchiectasis gives off an unpleasant odor resembling that of gangrene. Sputum having a sweetish odor is characteristic of pulmonary ulceration, bronchitis, and empyema. An odor resembling that of rancid cheese suggests the addition of tyrosin, which results only when extraneous pus enters the bronchial tract.

Fluidity and Tenacity.—The density of the sputum will be found to correspond more or less closely to the quantity expectorated. The

density varies from that of a watery fluid to that of a gelatinous mass. Sputum placed in a cylindric glass will be found, upon standing, to separate into strata, there being a superior or frothy layer, a clear liquid layer, a third layer containing flocculi and particles of mucus, and an inferior stratum of rather dense, ropy material, often containing pus and blood. A creamy sputum is not infrequently seen. A liquid sputum is significant of edema of the lungs, tuberculous laryngitis, the early stage of pulmonary tuberculosis, or the perforation of an empyema or of a diaphragmatic or hepatic abscess. It also occurs in pulmonary abscess and gangrene. "Currant-jelly sputum" suggests malignancy, and "prune-juice sputum" appears where blood is derived from an edematous lung, as in adynamic lobar pneumonia.

Specific Gravity.—The specific gravity of sputum is dependent upon the general character of the expectoration, mucous sputum having a specific gravity of 1.003 to 1.010; purulent and bloody specimens, one between 1.014 and 1.025, whereas highly bloody sputum may reach 1.035.

Reaction.—Normal sputum has an alkaline reaction.

Color.—The sputum varies in color from that of a perfectly clear, transparent fluid, through the successive shades of gray, yellow, amber, orange, olive green, red, chocolate, and black. When the expectoration is entirely mucoid, it is colorless and nearly transparent.

Leukocytes render the sputum opalescent or turbid, according to the number of cells present; and, from the same cause, the color is first white, then yellow, and finally of a greenish hue. The presence of bile-pigments gives rise to a green sputum. A growth of bacteria (Bacillus pyocyaneus) may be accountable for a green color, and in cases of amebic abscess, whether hepatic or pulmonary, the sputum is chocolate colored. In paragonimus infection the sputum resembles anchovy sauce in appearance.

Black Sputum.—The sputum becomes gray after the inhalation of particles of carbon, whereas the sputum of coal-miners and of those residing in the mining districts is often dark and at times black, due to the presence of coal-dust (Plate I). Particles of iron may give the sputum a yellow or red color.

Bloody Sputum.—Sputum tinged with blood and studded with minute air-bubbles—"rusty sputum"—is characteristic of lobar pneumonia. Blood gives a red color to the sputum, varying in intensity with the amount present. It is most often encountered in pulmonary congestion and ulceration (phthisis). Hemorrhagic sputum may result from cardiac insufficiency. Whenever the blood is expectorated as soon as it escapes from the vessels, it is of a bright-red color. Bloody and dark-brown sputa are also observed in pulmonary abscess and in gangrene of the lung. The various types of hemoptysis, together with their clinical classifications as to source, exciting factors, etc., are given in the accompanying table:

DUE TO PATHOLOGIC CHANGES IN THE LUNGS.

1. Phthisis pulmonalis.
2. Pneumonoconiosis:
   - Metal workers lung,
   - Stonemasons lung,
   - Coal miners lung,
   - Plaster workers lung.
3. Cardiac disease, especially mitral stenosis.
4. Violent coughing:
   - Whooping cough,
   - Bronchitis,
   - Emphysema,
   - Asthma,
   - Cough accompanied by vomiting.
5. Traumatism:
   - Blows upon the chest-wall,
   - Fractured rib,
   - Exploratory punctures.

6. Lobar pneumonia (slight):
   Bronchopneumonia (slight),
   Septic pneumonia (slight),
   Pulmonary emboli,
   Hypostatic congestion,
   Abscess,
   Gangrene,
   Pulmonary thrombosis.
7. New growths of the lung:
   Sarcoma,
   Carcinoma.
8. Sporotrichosis of the lung:
   Aspergillosis,
   Actinomycosis,
   Streptothoracosis.
9. Aortic aneurysm by pressure on and by rupture into the bronchial tract.
10. Parasites:
    Hydatid cyst,
    Hepatic amebic abscess (bursting through the diaphragm into the lung),
    Primary amebic abscess,
    Filiariasis,
    Parongonimus Westermanni (tropical hemoptysis).

Due to Changes in the Bronchioles, Bronchi, or Trachea.

11. Acute bronchitis,
    Bronchorrhea,
    Bronchiectasis,
    Tracheobronchitis.
12. Ulceration of the trachea,
    Ulceration of a bronchus,
    Lymphosarcoma,
    Esophageal or other neoplasm,
    Invasion of a bronchus by a mediastinal sarcoma.

Due to Changes in the Larynx:

13. Acute laryngitis,
    Tuberculous ulceration,
    Syphilitic ulceration,
    Epithelioma,
    Sarcoma,
    Postdiphtheritic ulcer,
    Typhoid ulcer (rare),
    Traumatism,
    Variolous ulceration (rare),
    Leprosy of the larynx (rare).

Due to Hemic Changes:

14. Purpura,
    Scurvy,
    Splenomedullary leukemia,
    Hemophilia,
    Lymphatic leukemia,
    Pernicious anemia,
    Hodgkin's disease.

Uncommon Causes of Hemoptysis:

15. Interstitial nephritis,
    Arteriosclerosis,
    Vicarious menstruation,
    Varicose veins of pharynx.

Mucous Sputum.—This variety of sputum is clear, sticky, tough, and, during the early stage of bronchitis, scant in quantity. In the latter stage of bronchitis pus-cells are added, which render the sputum more copious and give it a yellowish or a greenish color.

Mucopurulent Sputum.—This is a variety of sputum seen in many forms of pulmonary disease. It is of clinical value in pulmonary tuberculosis, where, in the event of cavity formation, minute ragged clumps of mucopus, which are intimately surrounded by mucus, may be seen.

Nummular Sputum.—In this variety coin-like masses, often regarded as characteristic of cavity formation when first expectorated, float upon the surface, but the sputum may contain grayish-white masses and round or irregular particles varying in size from that of a pin's point to that of a millet-seed (caseous particles). These masses are usually precipitated from the liquid portions of the sputum, collecting at the bottom.

Serous Sputum.—A purely serous sputum is significant of edema of the lungs, and contains but few, if any, red blood-cells. When shaken, serous sputum displays a soapy froth having a faint pink hue when it is mixed with blood.

Albuminous sputum is seen in pulmonary tuberculosis, acute bronchitis, lobar pneumonia, and bronchiectasis. A highly albuminous sputum is always suggestive of pulmonary tuberculosis, even in the absence of tubercle bacilli and of definite physical signs.

MICROSCOPIC STUDY OF THE SPUTUM.

Organized Constituents.—Fibrinous Coagula.—During the course of certain pathologic conditions an exudate is deposited in the smaller bronchi, and after undergoing degenerative changes, this exudate results in the formation of a complete cast of a small bronchus. During the act of coughing a small amount of this coagulum is dislodged, and appears in the sputum as a gray, white, reddish-yellow, mahogany, or bloody particle.

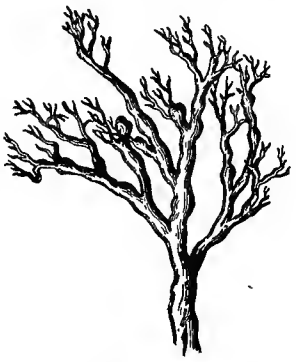


FIG. 30.—FIBRINOUS BRONCHIAL CAST.

Detection.—Fibrinous casts may be recognized by placing suspicious particles of the fresh sputum between two slides, and making rather firm pressure upon the upper slide. Fibrinous coagula are clearly seen when brought under a two-thirds inch objective (Fig. 30).

Significance.—Fibrinous casts are found in the sputum of fibrinous bronchitis, croupous pneumonia as the stage of resolution approaches, and in the presence of a diphtheric process in the finer bronchi.

Bronchial Spirals.—These bodies resemble bronchial casts (Fig. 31).

Detection.—Use the same technic directed for the detection of bronchial casts. At times it will be found necessary to use a high-power objective (one-sixth to one-eighth). Spirals appear as faint, translucent, elongated masses. A delicate white fiber runs longitudinally through the center of each spiral. Leukocytes, epithelial cells, Charcot-Leyden crystals, and, rarely, erythrocytes are entangled in the spiral mass.

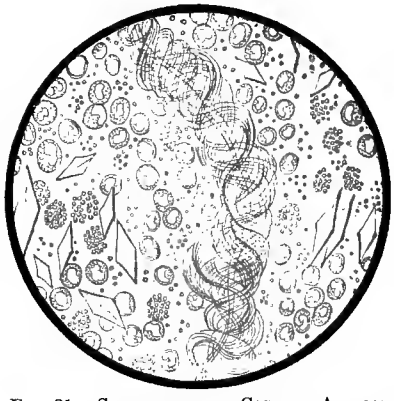


FIG. 31.—SPUTUM FROM A CASE OF ASTHMA, SHOWING CURSCHMANN SPIRALS, CHARCOT-LEYDEN CRYSTALS, LEUKOCYTES, AND NUMEROUS FREE EOSINOPHILE GRANULES (Jakob).

Spirals are common in the sputum of asthma. They are seen in croupous pneumonia, acute bronchitis, chronic bronchitis, pulmonary tuberculosis, and valvular heart disease. Spirals are suggestive of a catarrhal process in the bronchi.

Elastic Tissue.—Fibers of elastic tissue may occur in the sputum as single threads, arranged in a more or less perfect alveolar series, or, as is most usual, in small bundles. They are demonstrated in the manner described for the detection of fibrinous coagula, a one-eighth inch objective being used. Collect the fibrinous plugs from the sputum, place them in a solution of sodium hydroxid, and boil the soda

solution and its contained sputum until a gelatinous mass results. Add four times the total quantity of water; place the mixture in a conic glass, and allow it to stand for several hours. Centrifugalize the sediment, and place a portion of the second sediment thus obtained under a one-eighth or one-twelfth inch oil-immersion objective, when elastic fibers will be readily detected (Fig. 32). The presence of elastic fibers in the sputum indicates the existence of a destructive process in the lung. The true significance of these changes, however, is indicated only when they appear in the so-called alveolar arrangement, as they do in case of pulmonary cavity. Elastic fibers are rarely found in abscess of the lung, bronchiectasis, and pneumonia.

Animal Parasites of the Lung.—Paragonimus Westermanii.—This parasite has confined itself largely to Japan, Formosa, the Philippines, and Korea, although cases have been reported from other eastern countries.

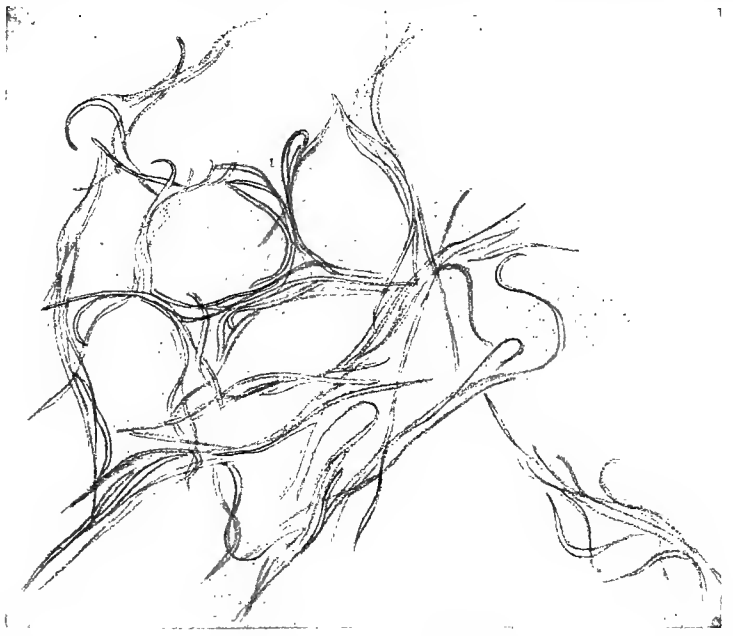


FIG. 32.—FIBERS OF ELASTIC TISSUE FROM SPUTUM IN A CASE OF PULMONARY TUBERCULOSIS OBSERVED AT PENNSYLVANIA HOSPITAL (obj. B. and L. one-eighth) (Boston).

Stiles and Hassall have recently recovered this parasite from the lungs of hogs raised in the United States, and MacKenzie has reported a case occurring in man from Portland, Oregon.

Sputum.—The sputum looks bloody, but the color is due to the presence of the ova of the fluke, although more or less altered blood-corpuscles may be present. The sputum may closely resemble that of lobar pneumonia in color.

Detection.—Place a portion of the bloody sputum upon a slide, apply a cover-glass, and study under a one-fifth or one-eighth inch objective. The color of the sputum is dependent upon the presence of numerous ova and red blood-corpuscles. The ova are oval in outline and are furnished with a distinct lid.

Trichomonas.—This type of parasite (Fig. 343) has been found in the sputum of pulmonary gangrene and in that from pus-cavities.

The **Balantidium coli** (Fig. 345) has been known to invade the respiratory tract of persons residing in the tropics, but the literature on the subject appears to contain but few authentic reports.

Bilharzia.—There are numerous reliable records of cases in which the ova of the Schistosomum hæmatobium have been found in the sputum. (See Hematuria.)

Amœba Coli (Entamœba Histolytica).—The ameba appears in the sputum when an amebic abscess of the lung is evacuated into the bronchial tract, or when an amebic abscess of the liver has ruptured through the diaphragm and communicated with a bronchus. (See Amebic Dysentery.) The sputum may at first be bloody, but later it may assume a yellowish or pearl-like color. Many epithelial cells are always present, and either pulmonary or hepatic tissue may at times be seen.

Filaria.—Filaria embryos have been known to appear in the sputum of persons affected with filariasis, and it was formerly believed that there was a special type of parasite concerned (Filaria bronchialis) when these parasites were found in the sputum. It is now known that the embryo filariæ retreat to the blood-vessels of the lung in the interval of their periodicity in the blood-current. In a concurrent destructive lung disease these parasites might be found in the sputum.

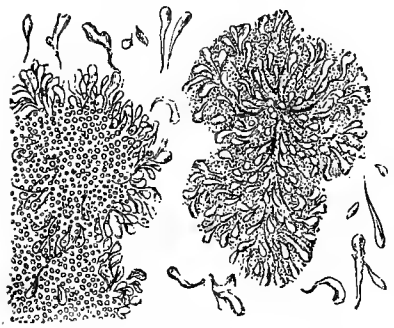


Fig. 33.—Actinomyces (after von Jaksch).

Tænia Echinococcus.—Whenever cysts of the dog tape-worm communicate with the respiratory tract, both hooklets and scolices of the parasite (Fig. 361) appear in the sputum.

Ascarides.—Both the adult parasites and their ova have been found in the sputum (Fig. 357).

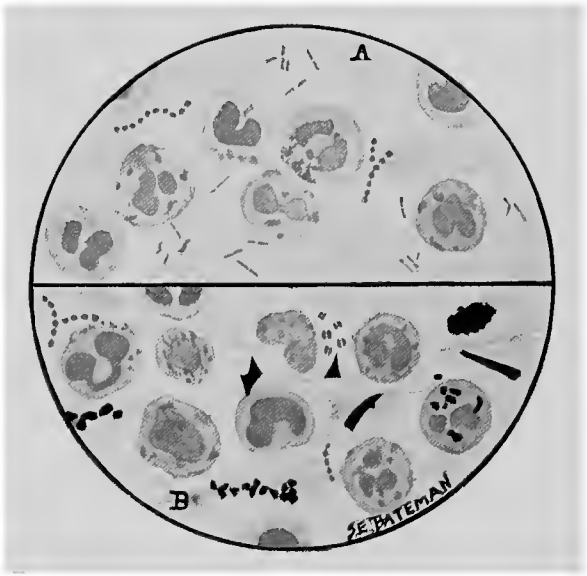
Fungi.—Among the fungi that are to be regarded as of pathologic interest, the most important are the actinomyces and the Aspergillus fumigatus.

Actinomycosis.—The detection of small granules and thread-like particles (mycelia) (Fig. 33) in the sputum verifies the diagnosis of actinomycosis of the respiratory tract. Actinomycosis may also involve the buccal cavity, in which case the ray-fungus is found in the sputum. Actinomycosis of the pleuræ usually causes a perforation of the chest-wall, and pus containing the fungus escapes externally.

Aspergillosis.—Foreign observers have found the Aspergillus fumigatus in the sputum of those suffering from pneumomycosis. Aspergillus is recognized in the sputum by the detection of many thread-like particles (mycelia) (Fig. 331). It is not uncommon to find sputum secondarily infected with the Aspergillus niger (Fig. 331), and it is difficult to distinguish microscopically between the mycelium of this fungus and that of Aspergillus fumigatus. This difficulty may be overcome by making a cultural study of these two fungi, when their identity will be readily discerned.

The **Mucor corymbifer** is also encountered in the sputum, and, in addition, many different molds may develop in the sputum after it has been exposed to the air.

PLATE I



A. Sputum showing tubercle bacilli stained with carbolfuchsin and Gabbet's methylene blue solution (obj. B. and L. one-twelfth oil-immersion).

B. Sputum of anthracosis, showing particles of coal-dust stained with methylene-blue (obj. Spencer one-twelfth oil-immersion). (Boston.)

Bacteria.—Numerous species of bacteria are present in the sputum, although in a comparatively small number of instances a definite micro-organism will be found associated with a certain disease. The streptococcus, a large diplococcus, the pneumococcus, Bacillus typhosus, Bacillus coli communis, Friedländer's bacillus, Bacillus tuberculosis, and the streptothrix deserve special mention.

Microscopic Study of the Bacteria of the Sputum.—Select from the sputum small caseous or bloody particles, place them upon a microscopic slide, crush, and spread into a thin layer. All sputa should be stained for the tubercle bacillus, and special stains are also necessary when searching for certain specific bacteria. As a rule, however, the method employed for staining the tubercle bacillus will be found also to stain satisfactorily many other microörganisms.

Staining for the Tubercle Bacillus.—1. Add a few drops of carbol-fuchsin (5 per cent. phenol, 90 parts; saturated alcoholic solution of fuchsin, 10 parts) to the specimen, and hold it above the flame until the staining solution begins to steam. Let this stain act for five minutes.

2. Wash in water, holding the forceps in such a manner that the stream strikes the slide near one end and then flows over the specimen.

3. Without drying, add to the specimen a few drops of Gabbett's methylene-blue solution (methylène-blue, 2 parts; solution of sulphuric acid (25 per cent.), 100 parts), and allow it to stand for two minutes; then wash in water and dry over the flame.

In searching for the diplococcus and organisms other than the tubercle bacillus equally satisfactory results may be obtained by staining with Löffler's alkaline methylene-blue for thirty seconds, washing, and drying.

Caution! Whenever the presence of tubercle bacilli in the sputum is suspected, the following method for their detection is to be recommended:

Place the sputum in an ordinary vaselin bottle, tie three or four thicknesses of gauze tightly over the mouth of the bottle, to prevent dust from entering it. Allow it to stand for several days, and then examine for the tubercle bacillus.

A still more satisfactory method is to smear the caseous particles of the sputum on slides and dry in the air. Fix either by passing directly through the flame or by keeping it upon a hot stage for from twenty minutes to one-half hour. Stain the fixed specimen by immersing it in a weak solution of carbolfuchsin (carbolfuchsin, ½ dram; water, 2 ounces) for twenty-four hours.

Differentiation.—Tubercle bacilli (Plate I) are to be differentiated from other acid-fast bacilli (by acid-fast bacilli are meant those that do not decolorize readily by acids or by alcohol), viz., the grass bacillus, the butter bacillus of Rabinowitch, Bacillus lepræ, and the smegma bacillus.

Significance.—Tubercle bacilli, when found in the sputum, furnish conclusive evidence of the existence of a tuberculous lesion along the course of the respiratory tract. Even a small ulceration of the bronchus may furnish a great number of bacilli, hence the presence of a profuse number of bacilli in a given sputum is no guide as to the extent of disease existing in the lung. Tuberculous laryngitis generally displays a large number of tubercle bacilli in the sputum, and the same is true of extensive ulceration and pulmonary cavities; nevertheless, a pulmonary cavity the result of tuberculosis may be present and no tubercle bacilli demonstrable in the sputum. This has been amply confirmed by us at autopsy.

Influenza.—The sputum contains slender bacilli that stain readily by

the ordinary anilin dyes. In order to cultivate the influenza bacillus, a special medium is necessary. Löffler's blood-serum will be found to serve well for this purpose, after the surface has been smeared with fresh blood.

The *clinical significance* that attaches itself to the detection of the influenza bacillus is still of doubtful value in the minds of many clinicians.

Diphtheria.—During the course of diphtheria the sputum often contains the diphtheria bacillus.

Acute Bronchitis.—The sputum displays many desquamated epithelial cells, which represent the various forms common to the respiratory tract. Leukocytes are always present in small numbers, and red corpuscles may be found. Later in the course of acute bronchitis the sputum becomes abundant, turbid, and yellowish or greenish in color.

Bacteriology.—The terms streptococcous bronchitis and staphylococcous bronchitis have been suggested for different types of the disease. In streptococcous infection the sputum contains innumerable streptococci, and when the form of infection is mild, it can be differentiated from a staphylococcic bronchitis only by a cultural study.

Chronic Bronchitis.—If expectoration is profuse, that is, if the sputum is expectorated in mouthfuls, the condition is known as bronchorrhea. This sputum is yellowish or yellowish-green in color, the color depending upon the number of pus-cells present and the stage of degeneration of such cells. A profusion of bacteria is present, but they bear no clinical significance to the type of bronchitis in question.

FIG. 34. — FRIEDLÄNDER'S BACILLUS IN PUS FROM PULMONARY ABSCESS (Boston).

Pneumonia.—The characteristic sputum of this disease is to be seen during the early stage of consolidation in lobar pneumonia, at which time it is scanty and tinged with blood (rusty), highly tenacious, and does not flow from the side of the sputum-cup. Red corpuscles and leukocytes are present, and when stained with hematoxylin and eosin or with a polychrome methylene-blue method, show many eosinophilic cells. Alveolar epithelial cells are also found, and many of these contain pigment and oil-globules.

Pneumococcus.—The pneumococcus is a small diplococcus that occurs in the sputum of lobar pneumonia, in which it is often the only organism present in great numbers.

The pneumococcus is well stained by Löffler's methylene-blue solution, but its characteristic contour (lance shape) is better demonstrated by the Gram method of staining. When carefully stained, each coccus is seen to be surrounded by a narrow hyaline space, which is bounded by a faint marginal band (capsule).

Encapsulated diplococci are always detected with difficulty. They are also commonly seen in the sputum of healthy persons, and are pathologic only when present in dense aggregations.

Bacillus of Friedländer.—This bacillus appears in great numbers in the bloody sputum of persons suffering from lobar pneumonia when the disease is due to the presence of this bacillus. This organism stains by the same methods given for the pneumococcus (Fig. 34). A number of encapsulated bacilli are also present in Friedländer's pneumonia. These bacilli may enter the blood, and are to be found in the pus from abscesses and from inflamed joints complicating an attack of Friedländer's pneumonia.

Bronchial Asthma.—At first the sputum is scanty, clear, grayish, or rarely reddish in color. It is always frothy, and is characterized microscopically by the presence of small, yellowish or grayish particles, "bronchial spirals" (Curschmann's spirals), Charcot-Leyden crystals (Fig. 31), and leukocytes. Many of the leukocytes show a special affinity for basic dyes, while the majority of them are decidedly eosinophilic.

Bronchopneumonia.—The sputum of this disease is not characteristic, containing many different bacteria—the pneumococcus, Staphylococcus pyogenes, bacillus of Friedländer, Streptococcus pyogenes, Bacillus pyocyaneus, Bacillus typhosus, diphtheria bacillus, Micrococcus tetragenus, and the meningococcus.

Pulmonary Abscess.—The fresh sputum from an abscess of the lung contains hematoidin crystals, fragments of lung tissue, and numerous crystals—cholesterin (Fig. 265), fatty acid, etc. Fibers of elastic tissue are not unusually present (Fig. 32).

Pulmonary Gangrene and Putrid Bronchitis.—The sputum of gangrene, when placed in a conic glass and allowed to stand for several hours, separates into strata: the inferior stratum is grayish yellow or brown and contains pus, small particles of a brown or greenish tint that vary in size from that of a millet-seed to that of a kernel of corn, and lung tissue. This sediment contains triple phosphates, leucin and tyrosin (Fig. 263, p. 658), and hematoidin crystals. Pus-cells and leukocytes are abundant, and the masses detected by the naked eye are found to be composed principally of pigment.

Elastic fibers, oil-droplets, crystals of fatty acids, and bacteria (Leptothrix pulmonalis, which stains bluish with Lugol's iodin solution) are present. The detection of particles of pulmonary tissue (elastic tissue) is the distinguishing feature between gangrene, where it is present, and putrid bronchitis.

The middle stratum of the sputum is transparent, and in it are suspended particles of mucus, while the superior stratum is usually of a dirty yellow color and is covered with a decided froth.

Pulmonary Tuberculosis.—The sputum from a case of pulmonary tuberculosis is but fairly characteristic.

Incipient Phthisis.—In this condition the sputum is scanty, grayish-yellow or whitish in color, frothy, and moderately tenacious. The larger portion is expectorated in the morning. As the disease advances the quantity increases, becoming copious and containing coin-like masses (nummular sputum) after cavity formation has taken place.

The detection of the tubercle bacillus is the only positive evidence of the existence of tuberculosis. Spirochætæ have been found in bloody sputum, although they probably have no connection with tuberculosis. Hemorrhagic sputum may form a rather dense clot, and the presence of even a small quantity of blood colors the sputum. Dark sputum is occasionally seen, and in cases in which the hemorrhage is severe, it is often difficult to ascertain its origin.

The accompanying table, modified from Boston, shows the points of differentiation between pulmonary and gastric hemorrhage:

Pulmonary Hemorrhage.	Gastric Hemorrhage.
1. Evidence of preëxisting pulmonary disease.	1. Referable to the throat, stomach, liver, heart, or develops in females near the time of puberty.

Pulmonary Hemorrhage.—(*Continued.*)	Gastric Hemorrhage.—(*Continued.*)
2. Preceded by thoracic oppressions and a saline taste.	2. Preceded by giddiness, faintness, and nausea.
3. Blood ejected by coughing when hemorrhage is small.	3. Blood ejected by vomiting or by clearing the throat.
4. In profuse hemorrhage and when ejected immediately blood is arterial in color.	4. Blood of gastric origin dark, as a rule; blood of pharyngeal origin, bright red.
5. Alkaline reaction.	5. Gastric blood acid, pharyngeal blood alkaline, in reaction.
6. Blood mixed with particles of mucopus.	6. May contain undigested food.
7. A pronounced beaded froth.	7. Froth less marked.
8. Microscopically, tubercle bacilli and possibly fibers of elastic tissue.	8. Microscopically, Sarcinæ ventriculi, starch-granules, particles of food, and, in the case of carcinoma, large nonmotile bacilli (Oppler-Boas) and, rarely, carcinomatous tissue.

Heart Disease.—In organic heart disease the sputum may be blood-stained. Such sputum, however, is distinguished from that of pneumonia and other acute congestions of the lung by the presence of epithelial cells filled with yellowish pigment (hemosiderin)—the so-called heart-disease cells.

Pneumonokoniosis.—This is a deposition of inorganic substances in the bronchial mucous membrane, with the appearance of such particles in the sputum.

Anthracosis.—Early in this disease, before the lung tissue has become seriously embarrassed by the deposit of dust, expectoration is slight, and takes place only upon rising and after a meal. When bronchitis is present, the sputum is copious in amount and contains large mucopurulent granules. Anthracotic sputum may present a variable degree of browning or an irregular distribution of black pigment.

The characteristic finding is small particles of coal-dust, which are readily detected under a one-fifth inch objective (Plate I).

Chalicosis.—This is a condition in which the sputum contains small particles of silica, and is present in persons who are more or less constantly exposed to such dust.

Siderosis.—The sputum in this condition resembles that of chronic bronchitis, although it may be brown or blackish. Alveolar epithelial cells and leukocytes are numerous, and brown or reddish pigment may be present. The addition of ammonium sulphid to sputum containing particles of iron turns it a blackish color, and upon the addition of hydrochloric acid and potassium ferrocyanid, the color is further changed to a Prussian blue.

Stycosis.—In this condition, which is most common in those working in plaster-of-Paris, about lime-kilns, etc., the sputum contains particles of lime. Cough and dyspnea are accompanied by free expectoration. It is more difficult to detect particles of lime than coal-dust, yet by careful focusing of the microscope it is usually possible to discover this fine pigment in the epithelial cells and leukocytes.

Stonemason's lung is a condition that follows the inhalation of particles of stone, which are detected in the sputum by chemic reactions, such reactions being also applicable to the detection of particles of lime. A microscopic study should always be made before resorting to chemic analysis of such sputum. Generally speaking, the sputum of stonemason's lung is that seen in chronic bronchitis (p. 91).

CHEMIC STUDY OF THE SPUTUM.

Organic Substances.—**Proteids.**—The sputa of pulmonary abscess, purulent bronchitis, croupous pneumonia, and cases in which many pus-cells are present contain peptone. Clinically, the detection of peptone in the sputum has thus far been found of questionable value.

Serum-albumin.—An excess of serum-albumin is found in the sputum of pulmonary edema whenever the albuminous properties of the blood are expectorated (albuminous sputum).

Sugar.—Glucose is rarely detected in the sputum. (See Glucose in Urine, p. 652.)

Ferments and Fatty Acids.—Analysis for the ferments and fatty acids present in the sputum is a matter of chemic rather than of clinical value, and for the technic of this analysis the reader is referred to special works upon chemistry.

DISEASES OF THE BRONCHI.

General Remarks.—Inflammatory diseases of the bronchi and those of the lung are differentiated chiefly by the wide diversity in the physical signs displayed by the two conditions. Bronchitis is an inflammation of the mucous membrane of the bronchi, either acute or chronic in character.

The inflammatory process may attack any portion of the bronchial tree—the larger, medium, or even the smallest bronchial tubes. Inflammation of the bronchial mucous membrane may be acute or chronic in nature, and may be either primary (infectious) or secondary to diseases of the heart, liver, kidneys, and lungs. It is also a symptom of certain of the acute infections—*e. g.*, measles, typhoid fever, and small-pox. (See Infectious Bronchitis, p. 90.)

ACUTE BRONCHITIS.

Pathologic Definition.—An acute disease characterized pathologically by congestion of the bronchial mucous membrane, which becomes covered with mucus or mucopus. Later there are desquamation of the ciliated epithelial cells, edema, and, in severe cases, infiltration of the mucosa with leukocytes.

Exciting and Predisposing Factors.—In the majority of cases acute tracheobronchitis results from an inflammatory process that has extended from the upper air-passages, *e. g.*, nares, pharynx, or larynx, and is secondary in nature. The bronchi may be the site of a primary acute catarrhal process, but such cases are by no means common. Mechanic, chemic, and biologic irritants that are known to act directly upon the bronchial mucous membrane may in themselves produce primary bronchitis.

Among the predisposing factors are:

(1) **Age.**—Bronchitis is most common at the extremes of life.

(2) **Lowered Vitality.**—The debilitated are especially prone to this affection.

(3) **Occupation.**—Those exposed to the inhalation of irritating dusts (lime, foundry dust, silicates) are predisposed to bronchitis.

(4) **Climate.**—Those residing in sections where the temperature is known to fluctuate greatly within a short period are more susceptible to

the disease than are those living in sections having a more stable temperature. Humidity has also been shown to exercise some influence upon the development of bronchitis.

(5) **Season.**—The greater number of cases is seen during the fall and winter months, and at periods when colds are prevalent. Epidemics of any nature appear to increase the frequency of bronchitis, and this is especially true of epidemics of influenza, measles, and scarlatina. (See Infectious Bronchitis, p. 90.) Acute bronchitis often develops as a complication of both acute infectious and chronic maladies. It is an almost constant feature of typhoid fever, small-pox, and certain other of the infectious diseases, and is a serious late complication of organic heart, liver, or kidney disease and the anemias.

Principal Complaint.—The patient usually states that he has contracted a severe cold, and that the first symptoms recognized by him were repeated chilly sensations, sneezing, moderate coryza, sore throat, and hoarseness. In children there may be a history of one or more convulsions at the onset. The patient declares that he is tired and indisposed. There is some soreness in the muscles of the back and limbs, and, at times, headache is present.

When the attack is well developed, substernal soreness is a complaint, the patient often stating that there is a raw or burning sensation beneath the sternum upon deep inspiration; in the more severe cases substernal pain is present. *Cough* develops during the first few hours following the initial symptoms, and if severe, gives rise to intercostal soreness and aching, with soreness about the diaphragm. The character of the cough is quite significant, being at first harsh and non-productive, whereas later, *i. e.*, on the second or third day, free expectoration occurs. There may be paroxysms of coughing with or without expectoration, such paroxysms often being excited by a change of position—*e. g.*, from sitting or standing to the recumbent posture, and upon rising after a night's rest.

Thermic Features.—The temperature is somewhat irregular, and may range between 100° and 101° F.; in severe types of infection it may reach 103° F. It is highly important that acute bronchitis be recognized when it develops as a complication of other febrile conditions, since it may cause an additional rise of temperature.

Physical Signs.—**Inspection.**—The results of inspection are negative in adults, but in children rapid respirations are common. An examination of the larynx discloses the fact that the laryngeal mucous membrane is reddened and covered with an exudate. The fauces may also be congested.

Percussion.—In the vast majority of cases percussion is negative. Slight impairment of resonance is rarely observed beneath the angles of the scapulæ, and is present only when a large amount of mucus with probably partial occlusion of the bronchi exists. This sign must not be confounded with a similar impairment due to an acute pneumonic process; a feature of importance in this connection is that in pneumonia the impairment of resonance is unilateral, whereas that due to acute bronchitis is bilateral.

Auscultation.—The breath-sounds are increased in intensity, and over the anterior portion of the chest and near the apices the respiratory murmur may be harsh. Numerous râles are heard over both the apices and over other portions of the lung; some are high-pitched and squeaking in character, others being of the sibilant and sonorous variety. (See p. 69.) After a few days, both large and medium-sized mucous râles are audible.

The râles may disappear after the patient clears his throat or coughs, but if he is examined one or two hours later, these moist râles will again be present. In order to distinguish between a murmur originating within the lung and one that is pleural in origin the patient should be directed to cough, and the effect upon any questionable râle carefully noted. (See Intrapleural Râles, p. 71.)

Laboratory Diagnosis.—Sputum.—At first, especially during the first twenty-four hours of the disease, the sputum, while scanty, is decidedly viscid; later the expectoration becomes more profuse and mucopurulent in character, and by the third day, in severe cases, the ejecta is almost pure pus and may be of a greenish color. *Microscopically*, shreds of mucus and epithelial cells, a few being ciliated and others showing evidence of degeneration, are seen. Bacteria are numerous, the predominating organism varying in each particular case; thus at times it may be a large diplococcus, a streptococcus, the staphylococcus, and again the influenza bacillus. (See Infectious Bronchitis.) At least one slide should always be stained for the bacillus of tuberculosis.

Illustrative Case.—K. J., male, age nineteen, a teamster by occupation, gives the following history: Has been healthy during the past five years, and during all this time there has been a gradual increase in his weight until he now weighs 145 pounds (height, 5 feet 8¼ inches). There is no history of having consulted a physician since the age of fifteen.

Upon the day preceding his appearance at the medical clinic he suffered undue exposure to both cold and wet, and during the night following such exposure he awakened several times suffering from violent frontal headache and a sense of chilliness. Upon rising in the morning the mental condition was dull and cough was distressing, being accompanied by slight expectoration. When seen, the patient complained bitterly of a sense of constriction over the anterior surface of the chest, and also of substernal discomfort. He experienced a moderate amount of soreness at the base of the chest, on a level corresponding to the insertions of the diaphragm. Constipation was present.

When first seen, the patient was sitting, and he then gave no evidence of having difficulty in respiration, although it was found that exercise and talking induced cough. The chest was apparently normal, and the number of respirations a minute was slightly accelerated. The cutaneous surface appeared normal except for a moderate flushing of the cheeks. There was a mild congestion of the conjunctivæ, and the pharyngeal mucous membrane was also reddened. Auscultation revealed the presence of many fine, crackling râles over those portions of the chest immediately overlying the large bronchi.

When first examined the temperature was 101° F., and continued fluctuating between 99° and 102° F. for a period of three days during his stay in the hospital.

Summary of Diagnosis.—A diagnosis is made from the following symptoms: (*a*) Slight fever; (*b*) cough, which is at first dry and later productive; (*c*) the character of the expectoration, together with the acuteness of the onset, which is accompanied by chilly sensations; (*d*) the undue harshness of the respiratory murmur, numerous dry râles during the first twenty-four hours, and the development of moist, bubbling râles later—all of which signs are audible over the entire chest. A history of exposure or the development of the condition during the course of a febrile or an afebrile malady always points strongly toward acute bronchitis.

Differential Diagnosis.—Pleurisy with effusion differs from acute bronchitis in that flatness is present over the base of one pleura. Upon auscultation the signs of bronchitis may be present over that portion of lung above the fluid and over the unaffected side. The fact that pleural effusion is seldom bilateral, and that even when both pleuræ are involved the signs of bronchitis are absent at the bases (patient erect), serves to dif-

ferentiate these conditions. The history of a sharp, stabbing pain in the pleura is absent in acute bronchitis, as is also movable dullness.

Bronchopneumonia may follow an attack of acute bronchitis, in which event the following additional symptoms and signs appear: increase in the number of respirations, cyanosis, frequent pulse-rate, and an elevation in temperature of one or two degrees. Here the aim is not to differentiate between bronchitis and bronchopneumonia, but rather to determine the time at which pneumonia develops.

Incipient tuberculosis is distinguished from acute bronchitis by the fact that in tuberculosis the physical signs of bronchitis are distinctly localized, and are usually found at one or both apices, whereas in acute bronchitis the peculiar respirations and types of râles are heard over the entire lung surface.

Miliary tuberculosis (pulmonary type) may exhibit the physical signs of acute bronchitis, but the general condition of the patient and the fact that he does not develop the characteristic sputum by the second or third day serve to differentiate bronchitis from this type of tuberculosis. (See Miliary Tuberculosis, Differential Diagnosis, p. 801.) In miliary tuberculosis a culture of the blood may disclose the presence of tubercle bacilli, which finding will serve early in this disease to make the diagnosis positive.

Clinical Course.—Acute bronchitis runs a varying course, depending largely upon the condition to which it is secondary. For example, if it follows direct extension from congestion of the upper respiratory tract, the course varies from a few days to two weeks, and rarely, indeed, does the disease continue for a longer period. A somewhat protracted course is commonly encountered in those who are debilitated, in the aged, and in persons suffering from either the gouty or the tuberculous diathesis. In tuberculous subjects there is at times a tendency for the bronchial congestion to extend to the finer tubules, and in these the general symptoms are severe. When bronchitis complicates one of the acute infections, its severity is more or less directly dependent upon the severity of the primary infection.

INFECTIOUS BRONCHITIS.

General Remarks.—In addition to the acute form of bronchitis known to extend from inflammatory processes of the upper air-passages, and that form which develops during the course of certain acute infectious fevers and afebrile conditions that are characterized by asthenia, the disease may form one of the leading and at times chief clinical features of certain acute conditions, such as hay-fever, measles, whooping-cough, typhoid fever, influenza, and acute "infectious" colds.

Hay-fever.—In this disease a variable degree of bronchitis is fairly characteristic, and may develop with or even antedate the coryza, which is a characteristic feature. The general clinical picture of hay-fever has been described at length on p. 794, but for our present purpose attention is directed to the fact that the physical signs of acute bronchitis are invariably present, and, as a rule, their severity varies in direct proportion with the degree of irritation of the nasal mucosa. The bronchitis of hay-fever shows less tendency to terminate in early recovery than do other types of this malady, and this peculiarity of the disease warrants its separate classification.

Influenza.—In the respiratory type of this infection (acute infectious colds), acute bronchitis is an early and prominent symptom.

Laboratory Diagnosis.—Both the sputum and the nasal secretion contain the bacillus of influenza.

Measles.—The symptoms and signs of acute bronchitis are among the earliest clinical manifestations of measles, and, indeed, may continue for days and, in unfavorable cases, even for weeks. The more severe the type of bronchitis present, the more serious is the case, and the more likely is it to develop pulmonary complications (bronchopneumonia).

The physical signs differ in no way from those detailed under Acute Bronchitis.

Laboratory Diagnosis.—The sputum is scanty at first, and free expectoration seldom occurs until the eruption begins to fade. A microscopic analysis of the sputum gives no positive information with reference to the type of infection.

Whooping-cough.—An early evidence in this disease is the onset of acute bronchial catarrh, which is at first mild, but gradually increases in severity. Acute bronchitis extends over a period of weeks before the development of the whoop which characterizes the disease. The more severe the type of bronchitis, the more likely is the child to develop bronchopneumonia. Late during the course of whooping-cough the bronchial condition assumes a subacute nature, and chronic bronchitis commonly results. The expectoration reveals nothing diagnostic of this disease.

CHRONIC BRONCHITIS.

Pathologic Definition.—A chronic inflammatory process involving the bronchial mucous membrane, and characterized by the occurrence of destructive changes in the superficial epithelial layer, with thinning of the mucous membrane of the larger tubes as the result of atrophy of the muscular coat. The mucous glands are destroyed, and there are localized areas of infiltration (thickening) and dilatation of the bronchial tubes.

Varieties.—The disease is rarely primary in origin, the vast majority of cases developing as the result of preëxisting acute or chronic maladies.

(1) There is a special type, commonly seen in men past middle life, who either display a gouty diathesis or have been sufferers from emphysema, organic heart disease, general arterial sclerosis, or renal disease.

(2) Another variety is that known as **dry catarrh,** which is also observed in elderly individuals and almost always follows emphysema. This form of chronic bronchitis is characterized by a paroxysmal cough that may occur once or twice or oftener during the twenty-four hours, and is accompanied by scanty but highly tenacious expectoration.

(3) Special attention has been called to the chronic bronchitis of young females, and this type is characterized, first, by the class of individuals it attacks, and, second, by the fact that it does not materially impair the general nutrition.

(4) **Bronchorrhea** is a condition in which the leading symptoms of chronic bronchitis are present, and, in addition, there is a profuse watery and at times mucopurulent expectoration. Bronchorrhea should be considered in connection with putrid bronchitis and with bronchiectasis.

(5) **Fetid Bronchitis.**—In this condition the sputum gives off an odor resembling that of decomposing animal tissue, which forms the characteristic clinical manifestation in this type of the disease. It should be re-

membered, however, that fetid expectoration is also a feature of pulmonary gangrene (p. 119), pulmonary abscess (p. 122), and dilatation of a bronchus (p. 101). In making the diagnosis, therefore, these conditions should be carefully excluded.

Predisposing Factors.—Age is not without influence, since the majority of cases occur after the fortieth year. The disease is seen, however, in children, particularly after an attack of whooping-cough or of one of those diseases of childhood characterized by malnutrition.

Season.—Cold appears to predispose to attacks of chronic bronchitis, and it is common to find that those free from the disease during the summer months are again attacked as the cold weather approaches, the condition continuing until spring or even summer returns. During the autumn months the pollen of certain plants appears to excite a chronic form of bronchitis in selected cases.

Other Predisposing Conditions.—The disease frequently follows repeated attacks of acute bronchitis, and is especially likely to develop after an attack of influenza, measles, whooping-cough, small-pox, or scarlet fever.

Alcoholism, chronic rheumatism, gout, and pulmonary tuberculosis frequently antedate chronic bronchitis. Organic disease of the heart, chronic obesity, emphysema, repeated attacks of asthma, and chronic nephritis manifest chronic bronchitis as a late symptom.

Exposure predisposes to the development of chronic bronchitis in that it gives rise to repeated attacks of the acute variety. Those exposed to irritating dusts (coal-miners, workers in foundries and factories, hat-makers) are especially likely to develop chronic bronchitis after prolonged exposure to such mechanical irritants.

Principal Complaint.—There is often a history of repeated attacks of acute bronchitis, or of the patient having at some time experienced an attack similar to that from which he is now suffering. Generally speaking, this condition resembles closely that described at length under Acute Bronchitis (p. 88), except that all the symptoms are less severe. The patient may complain of a sense of substernal constriction, but pain is rarely, if ever, experienced. Should the *cough* be pronounced, as it often is, the patient suffers from considerable discomfort and soreness about the base of the chest, and a sense of distress and even soreness is felt in the epigastrium and along the margin of the ribs. More or less cough is likely to be present continuously, but repeated paroxysms of coughing form the chief and most distressing complaint. Where expectoration is scanty and highly tenacious, cough is more severe and decidedly more distressing than where expectoration is free, with probable involvement of only the larger bronchial tubes.

Physical Signs.—Inspection.—The neck is short and somewhat thickened, and there may be evidence of cyanosis, both of which conditions result from the associated emphysema. Other signs obtained by inspection are usually due to a coexistent condition, and are not in themselves dependent upon chronic bronchitis. Should chronic bronchitis continue for years, emphysema follows.

Palpation.—A distinct fremitus may be transmitted from the larger bronchi to the palpating finger in those cases in which a quantity of mucus has collected in the bronchial tube. The respirations are often somewhat hurried, and the degree of expansion may be limited.

Percussion yields a clear and rather hyperresonant note over the entire

lung. Rarely, indeed, during acute exacerbations of a chronic bronchitis there may be moderate impairment at the bases posteriorly, a sign due to pulmonary congestion or possibly to localized pulmonary edema.

Auscultation.—The respiratory murmur is less distinct than normally, and if there is associated emphysema, an appreciable prolongation of the expiratory murmur is heard. Both large and small bubbling râles or rhonchi are heard over the entire chest, and these are particularly audible at the angle of the scapula, over the bases, and at the junction of the third ribs with the costal cartilages. In the so-called "dry catarrh" the râles are often high pitched and wheezing, and at certain times may be accompanied by moist râles.

The heart-sounds are normal at first, but when the disease has continued for years, both the first and the second sound becomes altered, and there is generally accentuation of the second pulmonic sound, as the result of increased blood tension in the lung. (See Emphysema.)

Laboratory Diagnosis.—In the ordinary type of chronic bronchitis there is cough with free expectoration upon rising in the morning, and, as a rule, one or two similar paroxysms during the day. The sputum displays no characteristics, but is a thick mucoid or mucopurulent fluid, and at times almost pure pus is ejected. In the so-called "dry catarrh" there is little, if any, expectoration.

Microscopically, the sputum will be found to contain many cocci and bacilli, but none of these is known to be pathogenic in nature. Epithelial cells from the lining of the bronchial tubules are always present, and some show evidences of degeneration. Leukocytes and pus-cells are also common, and shreds of fibrinous coagula (see p. 80) may rarely be detected. In selected cases of chronic bronchitis the sputum displays an abnormally high percentage of cells that stain by eosin. The eosinophilic granules in the leukocytes found in the sputum are not clearly outlined, as they are in the leukocytes of the circulating blood. When there is an associated asthmatic condition, Curschmann's spirals (Fig. 31) and Charcot-Leyden crystals (Fig. 31) are commonly present.

Summary of Diagnosis.—Chronic bronchitis is readily recognized from the following group of symptoms, which is unusually prominent: cough, expectoration, an absence of fever, and loss of strength and of weight. The fact that chronic bronchitis is, as a rule, a secondary condition is to be remembered, the heart, liver, lungs, and kidneys being studied carefully in order to determine the site of the primary disease.

Differential Diagnosis.—Pulmonary Tuberculosis.—In those cases of bronchitis in which the disease has continued for months or even years it must be carefully differentiated from pulmonary tuberculosis. The leading differential points are given in the following table:

Chronic Bronchitis.	Pulmonary Tuberculosis.
1. There is often a history of chronic heart, liver, or kidney disease.	1. History of tuberculosis in family or associates common.
2. Occupation may be that of a coal-miner, glass-blower, stone-cutter, or worker in foundries.	2. There may or may not be a history of working in dust.
3. Attacks recur with the approach of cold weather, and are mild or disappear during the summer months.	3. Apparently follows an acute cold or an attack of pleurisy or influenza.
4. There have often been periodic attacks of asthma, which occur at certain seasons—*e. g.*, fall and summer.	4. Absent.

Chronic Bronchitis.—(*Continued.*)	Pulmonary Tuberculosis—(*Continued.*)
5. Sputum does not contain tubercle bacilli, but a profusion of other bacteria (cocci and bacilli) are present.	5. Tubercle bacilli present.
6. Curschmann's spirals commonly seen.	6. Rare.
7. Pulmonary elastic tissue seldom, if ever, observed.	7. Common after cavity formation.
8. Physical signs obtained over both lungs.	8. Localized to one lung or to apices, rarely at one base.
9. Absent.	9. Pulmonary hemorrhage common.
10. Moderate loss of weight and strength.	10. Progressive weakness and emaciation.
11. Absent.	11. Fever present. After ulceration there is an evening temperature of from two to four degrees, whereas during the morning hours the temperature is normal or subnormal.

Fetid bronchitis is to be distinguished from other pulmonary conditions in which the sputum emits an offensive odor. Thus, in abscess of the lung the sputum contains shreds of lung tissue, elastic fibers, crystals of hematoidin, cholesterin, and amorphous blood-pigments, all of which substances are absent from the sputum of fetid bronchitis. Again, the physical signs in abscess are localized. Fever, which is a feature of abscess, is absent in fetid bronchitis.

In **pulmonary gangrene** the odor of the sputum is one of its most prominent symptoms. Lung tissue may be present, although at times elastic fibers are dissolved by certain questionable ferments present in the sputum. Blood-pigment is also present. In gangrene the physical signs are either absent or distinctly localized, whereas in bronchitis the characteristic signs are heard over both lungs.

Bronchiectasis is seldom bilateral, consequently the physical signs are localized at one or both apices. This subject will be further discussed under Bronchiectasis. (See p. 100.)

Clinical Course.—This varies greatly in different cases, but in the majority of instances complete recovery is unusual, although there are exacerbations of the condition, followed by prolonged intervals during which the leading symptoms, cough and expectoration, subside. The majority of cases terminate, after a course of months or years, in emphysema. In those cases in which the bronchial catarrh results from cardiac, liver, or kidney disease, the course is influenced entirely by the preëxisting disease.

FIBRINOUS BRONCHITIS (Plastic Bronchitis; Croupous Bronchitis; Mucous Bronchitis).

Pathologic Definition.—This is a rare condition, characterized by either an acute or a chronic catarrhal inflammation of the bronchial mucosa, together with the production of a fibrinous exudate and the formation of fibrinous casts of the bronchial tubules.

General Remarks.—The causes of fibrinous bronchitis are obscure. Autopsy has revealed the existence of such conditions as pneumonia, chronic pleurisy, and pulmonary tuberculosis.

Varieties.—(1) The *acute form,* in which the attacks are unusually severe and lead one to look for an almost immediate fatal termination. (2) The *chronic type,* in which the attacks are mild, but occur somewhat regularly.

Predisposing and Exciting Factors.—No positive exciting factor is known, but various bacteria are often present in the sputum.

Sex.—Males are affected in 66⅔ per cent. of all cases.

Age.—Practically all ages may suffer from this affliction, yet the majority of cases are seen during the second and third decads.

Season plays an important etiologic rôle, spring furnishing the greatest number of cases. Rarely, indeed, a series of cases occurs in the same locality, and Pichini regards the disease as epidemic. Chronic diseases of the lung, as well as diseases of the skin (pemphigus, impetigo, and eczema), appear to predispose the patient to the development of fibrinous bronchitis.

Principal Complaint.—In acute fibrinous bronchitis the disease is ushered in by a severe *rigor*, which is directly followed by *high fever*, *urgent dyspnea*, and *paroxysmal cough*. The patient states that after severe coughing he is often able to expectorate a small quantity of material that contains one or more bronchial plugs. Following a paroxysm of coughing there is generally blood-streaked expectoration. The most dangerous symptoms are dyspnea, general oppression, a severe cough, with little or no expectoration, and a tendency toward asphyxia.

In chronic fibrinous bronchitis the attacks are less severe than in the acute variety, and an important feature is that these attacks occur at irregular intervals, varying from one week to one or more years. Rarely, cases are seen in which an attack of paroxysmal coughing with the expectoration of fibrinous exudate may occur daily for an indefinite period. Generally speaking, the symptoms of chronic fibrinous bronchitis are those seen in chronic bronchitis, with the aforementioned exceptions.

Thermic Features.—In the acute variety fever is to be expected, and in the chronic form there may be a mild febrile period. The fever is often the result of the preëxisting condition, and is but slightly, if at all, influenced by the bronchial trouble.

Physical Signs.—Inspection.—The attitude of the patient is that of one suffering from an asthmatic attack. (See p. 97.)

Palpation.—When a portion of the bronchial tree is plugged by the fibrinous exudate, it is impossible for air to enter that section of the lung, and, as a consequence, fremitus and expansion are diminished over such limited area.

Percussion over the affected area is often negative, yet there is usually a perceptible hyperresonant note over the surrounding healthy lung tissue, and in those cases in which large portions of the bronchial tree are involved, the percussion-note may be impaired over the affected section; but after dislodgment of the bronchial cast, however, normal resonance is restored.

Auscultation.—The breath-sounds are the same as those of chronic bronchitis, and although both harsh and hurried respiratory murmurs and dry râles are heard over different portions of the lung, they are in no way characteristic of this affection.

Diagnosis and Laboratory Diagnosis.—These rest entirely upon the finding of fibrinous coagula in the sputum. Such coagula, when spread thinly on a slide and studied under a low-power objective (two-thirds), must correspond in form to the peculiar arrangement of a section of the bronchial tubes. In true diphtheria a fibrinous exudate may appear in the sputum, but it has seldom been formed in the bronchial tubules. In order to determine the actual nature of the fibrinous exudate the cast must be hardened, sectioned, and studied for the bacillus of diphtheria. Fibrinous coagula from the bronchial tubules, when teased under

the microscope, will display a laminated structure, whereas the membrane dislodged as the result of infection by the Klebs-Löffler bacillus never presents this characteristic.

ASTHMA (Bronchial Asthma).

Pathologic Definition.—A chronic condition characterized by hyperemia of the bronchial mucosa and the presence of a mucous exudate, or by a neurosis of reflex origin, with the changes peculiar to chronic bronchitis, including emphysema, hypertrophy, and dilatation of the right heart. Spasmodic constriction involving the mucous membrane of the bronchial tree may also be present. Asthma, according to certain writers, may be excited by arterial constriction.

Remarks and Clinical Types.—In certain instances there appears to exist a constitutional susceptibility to spasm of the local muscular fibers. Among the conditions which appear to excite asthma and which stamp, more or less clearly, the various clinical types of the disease should be mentioned:

(*a*) **Acute bronchitis,** in which there may be isolated sections of true inflammation of the bronchial mucosa. It should be borne in mind, however, that where there is inflammation, bronchitis may in itself be excited by asthma.

(*b*) The **inhalation of certain irritants**—*e. g.*, gas and other vapors, tobacco smoke, dust from the street or that containing the pollen of plants—and even the odor of certain animals have been known to excite attacks of asthma. During the summer and autumn months many city dwellers, upon removing to the country, develop severe attacks; indeed, when the reverse obtains and those living in the country come to the city, similar attacks may be induced.

(*c*) **Secondary Asthma.**—Periodic attacks of asthma often occur during the course of such chronic maladies as organic heart disease, nephritis, rheumatism, gout, syphilis, and emphysema, as well as in lesions located in the medulla.

(*d*) **Nervous Asthma.**—Not infrequently a neurotic element is the causative factor in the production of asthma, severe attacks frequently following emotional excitement, disappointment, and grief.

(*e*) **Reflex Asthma.**—Asthma may be reflex in origin, as is seen in those suffering from obstruction to the upper air-passages due to nasal polypi, spurs, and disease of the teeth.

(*f*) **Gastric asthma** may follow dietetic errors, and constipation may result in the production of typical seizures (intestinal asthma). Some writers believe that in those suffering from asthma due to gastric derangements the diaphragm is abnormally contracted, and the base of the lung elevated above its normal position.

Predisposing Factors.—**Heredity** plays an important part, and is shown to figure in at least 50 per cent. of all cases.

Sex exercises great influence, practically two-thirds of all cases being seen in males.

Season is of special importance in those particular cases in which asthma results from the irritation offered by the pollen of certain plants; consequently the greater number of these cases occur during late summer and autumn months. Most cases of asthma, however, develop during the winter and spring months, and this is probably explained by the fact that many

cases follow an attack of hay-fever from which the patient suffered during the autumn.

Principal Complaint.—In about 50 per cent. of cases the patient complains of one or more of the following prodromal symptoms: irritability of temper, mental buoyancy, or, on the other hand, mental hebetude, vertigo, dull headache, gastro-intestinal irritation (dyspepsia), and an increased frequency of urination.

Paroxysm.—This may appear at any time during the twenty-four hours, but most often attacks the patient during the night, and frequently after he has enjoyed several hours' sleep. There is a tendency for the attack to return at the same time on successive nights. The onset is frequently sudden, the patient being awakened from a sound sleep by extreme dyspnea and paroxysmal cough, although, as a rule, a certain amount of thoracic constriction and moderate dyspnea is experienced for a short time—a few hours—preceding the attack. Suddenly the patient feels that he is smothering, sits up in bed, inclines forward, grasps his knees or some firm support in order to assist him in elevating his shoulders and bringing the accessory muscles of respiration into action. During a severe attack he frequently rushes to the open window. A favorite attitude is for the patient to sit on one chair and rest his arms upon another, again endeavoring to assist respiration by the use of the muscles of the neck and chest. On attempting to expel the air the patient gives a harsh, high-pitched, or rattling cough.

Thermic Features.—The temperature rapidly falls to below the normal and remains subnormal during the height of the attack.

Physical Signs.—Inspection.—*General.*—The patient sits inclined forward and grasps the knees or some firm object. The skin of the face is pale, whereas that of the fingers, lips, and eyelids, as the result of defective oxidation, is decidedly livid; the mucous membrane of the lips, mouth, and tongue may be cyanosed. The expression is anxious.

Local.—The chest is enlarged, and in those who have suffered from repeated attacks covering a period of months or years the chest is more or less barrel-shaped (Fig. 37, p. 127), due to inability on the part of the patient to expel the air. There is limited expansion of the chest, and the respirations number 10 to 14 a minute. A characteristic feature is alteration in the respiratory rhythm, inspiration being short, and immediately followed by a prolonged expiratory effort. The epigastrium is, as a rule, unusually prominent, due to lowering of the diaphragm. The neck is seen to be thick, and appears unusually short; the vessels of the carotid region stand out prominently, and pulsation over the right carotid region is common. The clavicles, as well as the shoulders, are elevated. The head is held in a somewhat fixed position. Speech is slow and interrupted.

Palpation.—Pulsation of the vessels of the neck is often detected, and not uncommonly there is a throbbing in the sternal notch. In those cases in which there is associated emphysema with dilatation of the right heart, distinct pulsation is detected in the epigastrium. The apex-beat may be absent, when this portion of the heart is covered by the emphysematous lung. The pulse is weak, thready, and rapid. The chest expansion is greatly restricted, and the movements, while not frequent, are jerking in character.

Percussion.—A hyperresonant note is obtained over both lungs, and in chronic cases, where there is associated emphysema, the percussion-note may be somewhat tympanitic in character. The area of absolute cardiac dullness is diminished, and if there is marked emphysema, this is absent.

A limited area of impairment may be detected in the epigastrium as the result of a dilated right heart.

Auscultation.—The inspiratory murmur is short and unusually feeble, whereas the expiratory sound is distinctly prolonged and accompanied by a low wheezing that may be heard even at some distance from the patient. Numerous dry râles are audible, and the majority of these are high-pitched, squeaking, and both sibilant and sonorous in character. The character and location of certain râles may vary at any time during or after an attack of asthma, and a certain type (see Râles, p. 69) may be present for but a short time during the entire attack. As the patient's condition improves and when the attack is about to terminate, moist râles are heard over both lungs. If moist râles are heard during the paroxysm, this is an indication that the patient is suffering from an associated bronchitis.

Laboratory Diagnosis.—Early during an attack of asthma the sputum is scanty, clear, beaded with froth, and at times grayish or tinged with red, due to the admixture of blood-corpuscles. Numerous grayish, mucoid particles are visible to the naked eye, and when these are studied under a one-sixth inch objective, they display a characteristic spiral formation (Curschmann's spirals, see p. 80). Charcot-Leyden crystals (Fig. 31) are an almost constant finding, and the sputum is always rich in leukocytes. Sputum stained with an alcoholic solution of eosin demonstrates that many of the leukocytes contain eosinophilic granules, whereas when stained with basic dyes, many of the white blood-cells show basic granules.

Blood.—Owing to the extreme cyanosis, the number of red cells in a cubic millimeter will be found to be far above the normal, ranging between 5,000,000 and 10,000,000. Because of imperfect oxidation, the color index is high, and it is usually difficult to match the true blood color with the shades of the hemoglobinometer.

During the prodromal stage the urine is increased in quantity, pale, and of low specific gravity.

Illustrative Case.—Peter H., male, aged forty-six; height, 5 feet 7 inches; weight, 150 pounds.

Family History.—Father living at the age of sixty-eight, but has had gout for several years. Mother died at the age of forty-five, cause unknown. A sister, aged forty, suffers from periodic attacks of hay-fever, and a brother, aged thirty-six, is reported as enjoying good health. One younger sister died of meningitis (?) before puberty.

Previous Medical History.—Patient had the diseases of childhood, including diphtheria at the age of twelve years, following which he claims there was a purulent discharge from the right ear; and for a number of years after, whenever he would contract a cold, the ear trouble returned. Ten years ago he suffered from stomach disorder (gastritis), and at this time he also had jaundice, which, as nearly as he could remember, lasted about three weeks.

Social History.—Occupation, foreman in sales department of a retail clothing store. Married at the age of thirty; has one son and two daughters who are in good health. Another daughter became afflicted with infantile paralysis at the age of three and one-half years. Two years ago he took a sea voyage, leaving August first and remaining at sea for a period of ten days, when he landed at Liverpool, England. He set sail for America five days later, and did not experience any asthmatic symptoms until he reached home, when he had sensations referable to his old disturbance, but no distinct attack was experienced.

Present Illness.—At the age of thirty-six he took a brief vacation in the country during the month of August, and states that he traveled over the hills of a farming district for eight or ten days, when suddenly he noticed that his nose discharged freely and that he had several severe attacks of sneezing. Coryza continued for a period of several days, when he was awakened during the night, suffering from what his physician termed an asthmatic paroxysm. He has contracted asthma during the autumn

months of each year since his initial attack, nine years ago, and has further observed that each attack is more severe than that of the preceding season. The attack appears during the night, although he states that he feels uncomfortable and has some aching of the muscles and moderate tightness over the chest one or two days before a distinct paroxysm develops. With the onset of each attack, if indoors, he feels that it is practically impossible for him to breathe, and sits up in bed, and at times is compelled to rush to an open window. He says that there is a severe sense of constriction over the chest. The attacks begin in August or September, and usually continue for from two to four weeks. Their course does not appear to vary, regardless of the treatment instituted. He has also found that he is especially liable to develop an attack of asthma upon changing his residence from the city to the country, but such attacks are milder than are those previously described.

Cough continues throughout the attack, and during the paroxysms it is harsh and seldom accompanied by expectoration; when, however, the pulmonary symptoms, especially the dyspnea, begin to subside, the expectoration becomes more copious.

The temperature is approximately normal during the greater part of the twenty-four hours, but when a violent paroxysm ensues, the temperature may be subnormal for a short period.

Physical Examination.—*General.*—When seen during the attack, the patient's expression is anxious. He sits with his body inclined forward, and grasps his knees or some firm object with his hands. He is extremely irritable, shows evidence of great fear, and refuses to move.

Local Examination.—The neck is shortened and appears swollen. The chest is unusually large, and expansion is very slight. The lips and even the tongue and mucous membrane of the mouth may show distinct cyanosis, and a similar condition may affect the ears, finger-tips, and lower extremities during the paroxysm.

Palpation.—This confirms inspection with reference to the movements of the chest. The apex-beat of the heart is often feeble, and at times scarcely perceptible. The pulse is small, but of fair tension, registering 90 to 100 beats a minute.

Percussion.—There is hyperresonance over the surface of both lungs, and the area of cardiac dullness is appreciably diminished.

Mensuration.—The circumference of the chest at the nipple-line is greater than normal, and varies only from one-half to one inch either on deep inspiration or on forced expiration.

Auscultation.—The breathing is harsh, and can be heard several feet away from the patient. The breath-sounds over the base of both lungs are accompanied by fine, squeaking râles. Over the upper portion of the lungs numerous high-pitched, piping, and at times bubbling, râles are heard. At the intervals between the paroxysmal attacks the expiratory murmur appears to be prolonged and is low in pitch.

Laboratory Diagnosis.—The sputum is scanty at times, or again may be copious. Microscopically, it contains many fine, plug-like particles, which, when examined under a one-sixth or one-eighth inch objective, display a peculiar spiral network (Curschmann's spirals, Fig. 31). When stained with eosin and methylene-blue, the sputum shows a great number of eosinophilic cells. Various bacteria, bacilli, and cocci are also present.

Diagnosis by Induction from Clinical Data.—The diagnosis was based upon the periodic attacks of spasmodic dyspnea, together with the characteristic physical signs of the disease.

Course of the Disease.—The attack which develops each autumn generally continues for from two to five weeks. At such times, when the patient was seen immediately after the onset of an attack, the paroxysms were distinctly lessened in severity as the result of treatment. When, however, the condition existed for from seven to fourteen days before a physician was consulted, treatment gave but little if any relief. By October of each year he is no longer distressed by the disease, although at practically any time during the year râles more or less characteristic of asthma are audible over both lungs.

Summary of Diagnosis.—The diagnosis is based almost exclusively upon the character of the paroxysm. The history of previous asthmatic seizures or of the existence of renal or cardiac disease, and a possible history of dietetic error, are also of value. The character of the sputum, which is scanty during the early stage, and the detection of Curschmann's spirals, as, also, the absence of fever, constitute valuable evidence on which to formulate a diagnosis.

Differential Diagnosis.—Laryngeal affections may be distinguished from asthma by the following clinical features: in disease of the larynx (edema of the larynx, spasm of the glottis) the voice is altered and aphonia generally ensues, both of which features are lacking in asthma. Again, the physical signs of asthma that are audible over both lungs are absent or greatly modified in laryngeal disease. In the latter condition the patient cannot inspire, whereas in asthma he is unable to expire and to rid his lung of air. Further, in laryngeal stenosis the chest is of normal size, whereas in asthma the dimensions of the chest are increased.

Emphysema is distinguished from asthma first by the fact that in the former the dyspnea is continuous, and, secondly, that in emphysema there are certain characteristic physical signs that are not observed in uncomplicated cases of asthma. (See Emphysema, p. 127.)

Clinical Course and Duration.—In mild cases of asthma there may be one or at most four or five nocturnal paroxysms, with or without cough during the day. In still another class of cases the patient suffers but little, if any, inconvenience for a period of days or weeks, when suddenly and without apparent cause he develops a paroxysm. The cough, which is distressing during and immediately after a seizure, may be more or less continuous for a few days. Dyspnea, accompanied by cough and free expectoration, is present. Even in this last class of cases paroxysmal attacks occur with a varying degree of periodicity.

BRONCHIECTASIS.

Pathologic Definition.—This condition may be either congenital or acquired. The latter variety is characterized by atrophy of the various layers of the wall of the bronchus, and by both cylindric and sacculated dilatation of the bronchial tubes. Dilatation may affect either the large, medium-sized, or the comparatively small bronchial tubes, and may be more or less generalized or localized, bilateral or unilateral. In the congenital type localized expansion of the bronchial tubes is also present.

Predisposing and Exciting Factors.—Age and Sex.—Bronchiectasis is most common in early adult and in middle life, and males are more often affected than females.

Given a previously weakened bronchial wall, the extra strain of violent coughing is sufficient to produce dilatation; and however slight the dilatation, the accumulation of an exudate following such sacculation serves, by reason of its own weight, to favor the process of further dilatation. Again, as the result of the weight of the exudate in the sacculated portion and the strain of coughing, the elasticity of the lung becomes more and more impaired.

Bronchiectasis is usually a secondary condition, complicating chronic bronchitis, bronchopneumonia, and whooping-cough. Pressure of a thoracic aneurism upon a bronchus may in time weaken the bronchial wall and result in bronchiectatic expansion. Diseases of the pleuræ in which there are marked fibroid change and inhibition of the respiratory function also favor the development of bronchiectasis. A bronchus often becomes dilated in those cases in which the surrounding lung tissue has undergone fibroid changes (fibroid phthisis).

Principal Complaint.—The patient complains bitterly of severe paroxysmal coughing, which attacks him in the morning, upon rising, and probably once or twice during the day. The paroxysms are brought on by

change of position, such as turning from side to side while in bed. The cough is always accompanied by the expectoration of from four fluidounces to one pint of sputum during the twenty-four hours. There is usually a history of chronic bronchitis, asthma, emphysema, whooping-cough, or chronic disease of the lungs. Extreme prostration has not been observed, nor does decided loss in weight take place.

Dyspnea is dependent upon the degree of dilatation of the bronchus, and may become extreme during paroxysmal coughing.

Thermic Features.—Uncomplicated cases of bronchiectasis run an afebrile course.

Physical Signs.—These are dependent upon three conditions: (1) The size and location of the dilatation; (2) the histologic condition of the surrounding lung tissue; and (3) the thickness and general relaxation of the chest-wall.

Inspection.—If the bronchial dilatation is large and is situated near the anterior surface of the chest, there will be an appreciable retraction of the chest-wall. The chest-wall is also retracted in those cases that have followed fibroid pleurisy and fibroid phthisis. The expansion of the chest is restricted over a large dilatation of a bronchus.

Palpation.—Tactile fremitus is increased where the surrounding lung tissue is consolidated, but where the dilated portion of the bronchus comes into direct contact with the chest-wall, fremitus may be diminished or absent. The degree of fremitus is dependent upon the amount of solid substance that lies between the chest-wall and the wall of the dilated portion of the bronchus.

Percussion.—The percussion-note is not influenced by the size or location of a dilated bronchus, but the alteration is dependent, as previously stated, upon the location of the sacculated bronchus and upon the condition of the surrounding lung tissue and pleuræ; consequently, given a markedly dilated bronchus situated near the chest-wall, the note may be hyperresonant or even cavernous in nature; whereas, on the other hand, a dilated bronchus with much partially consolidated lung surrounding it would give a decidedly dull note upon moderate percussion, but here too deep percussion will elicit a cavernous (semitympanitic) note. The percussion-note is dull when the sacculated portion of the bronchus is filled with exudate, and immediately after coughing and free expectoration a tympanitic note is often obtained over the same area.

Auscultation.—As a rule, the breath-sounds are markedly exaggerated, and in many instances bronchial breathing is audible. The various types of râles (see p. 69) are extremely common, and may display a metallic quality. Over a dilatation situated immediately beneath the pleuræ the breath-sounds, in addition to being harsh, possess an amphoric quality that is practically indistinguishable from the so-called cavernous breathing—*i. e.*, a distinct pause occurs between the inspiratory and the expiratory murmur.

Laboratory Diagnosis.—The sputum, as a rule, is grayish or brown in color and mucopurulent in consistence. At times it gives off a sour odor, and again it may be fetid. A somewhat characteristic feature of the sputum is that, upon standing, it separates into three strata: (1) a superior layer, composed of brown, frothy material; (2) a middle stratum of watery or serous consistence; and (3) an inferior layer of thick, granular débris.

Microscopically, the sputum contains many pus-cells, Charcot-Leyden crystals (Fig. 31, p. 80), and crystals of the fatty acids (Fig. 31, p. 80). Many bacteria (bacilli and cocci) are present. Mycelial threads (fungi) are occasionally seen. Rarely, indeed, fibers of elastic tissue are present,

but their presence is dependent entirely upon destructive changes in the lung substance. In those cases in which there is actual destruction of the lung tissue with congestion or ulceration the sputum may contain red blood-corpuscles.

Summary of Diagnosis.—This is based largely upon the history of preëxisting maladies that materially favor the development of bronchiectasis, and upon the fact that there has been no decided loss of strength and of weight. When the disease is running an afebrile course, this points strongly to the existence of bronchiectasis, and excludes a diagnosis of pulmonary tuberculosis. The character of the sputum and the fact that the recurrent paroxysmal cough is not accompanied by hemorrhage from the lung strongly suggest dilatation of the bronchus. The physical signs so closely resemble those present in pulmonary disease with cavity formation that while their presence is necessary in order to establish a diagnosis of dilated bronchus, such diagnosis cannot be based solely on the existence of these signs.

Differential Diagnosis.—The following table sets forth the distinctive features that separate bronchiectasis, pulmonary cavity, and thoracic aneurism:

Bronchiectasis.	Pulmonary Cavity.	Thoracic Aneurism.
1. History of asthma or pertussis of long standing.	1. History of tuberculosis.	1. Heavy lifting, high living, and previous attacks of acute endocarditis, rheumatism, or syphilis.
2. There is but moderate emaciation, without decided weakness.	2. Marked progressive emaciation with weakness.	2. Not characteristic.
3. Characteristic fetid sputum.	3. Nummular sputum containing tubercle bacilli.	3. Large quantities of sputum, often blood-tinged, are expectorated when the aneurism rests upon and causes partial obstruction of a bronchus.
4. Pulmonary hemorrhage absent.	4. Common.	4. Rare.
5. Physical signs of cavity without impairment of the percussion-note at apices.	5. Impairment at apices an almost constant finding, except where cavity involves the base of one lung.	5. Flatness is obtained over aneurism.
6. Dullness, which may change to a semi-tympanitic note after coughing.	6. Same.	6. Area of dullness not altered by coughing or by posture.

Circumscribed empyema, when it communicates with a bronchus through a fistulous opening, may somewhat resemble bronchiectasis, the differential features being that empyema is always marked, at some time in its course, by high temperature and leukocytosis, with an increase in the number of polymorphonuclear leukocytes. Emaciation and weakness characterize empyema.

Actinomycosis of the thorax may communicate with a bronchus or, less often, may perforate externally. The distinctive feature between actinomycosis and bronchiectasis is that in the former condition actinomyces fungus is present in the sputum.

Clinical Course.—This is, as a rule, favorable as to life, although a

permanent cure seldom follows, the condition continuing for years without marked interference with the patient's general nutrition.

BRONCHIAL STENOSIS.

Pathologic Definition.—A condition characterized by partial occlusion of the lumen of a bronchus, either as the result of disease or of foreign bodies, etc., within the bronchus itself, or, more commonly, from pressure from without, as the result of thoracic aneurism, enlarged bronchial glands, and the like.

Principal Complaint.—The most urgent complaint is that of dyspnea, which may be so pronounced as to bring into action the accessory muscles of respiration. Cough and expectoration are usually present.

Thermic Features.—Moderate fever is the rule, though by no means a constant finding.

Physical Signs.—Inspection.—In well-marked cases the skin and mucous surfaces are cyanosed. Chest expansion is unequal on the two sides, and there is often retraction of the interspaces on the affected side during the inspiratory act.

Tracheobronchoscopy.—By means of this special method it is possible, with the aid of the bronchoscope, to inspect the upper and lower air-passages and ascertain the existence of stricture and of disease of the mucous lining of the bronchial tubes. Stenosis is recognized by this method, and it is possible to distinguish between stenosis the result of external pressure and that resulting from changes in the bronchus itself. For a more detailed description of this method the reader is referred to special works upon this subject.*

Palpation.—Tactile fremitus is diminished and often absent, over the area of lung supplied by the diseased bronchus.

Percussion.—During the early stages of bronchial stenosis no positive evidences are elicited by percussion, but atelectasis may occur as a late complication, and in this case dullness is obtained over the area of lung involved. A fact to be considered in connection with every case of bronchial stenosis is that the area of lung affected is often small and completely covered by surrounding healthy, but emphysematous, lung tissue, which tends to obscure the evidences of disease.

Auscultation.—The vesicular murmur is feeble over the affected area, due to the diminished volume of air entering the peripheral portions of the lung. Numerous râles, both sibilant and sonorous, are present over the site of the obstruction.

Summary of Diagnosis.—Auscultation offers the most positive diagnostic sign—the detection of sibilant and sonorous râles at a point corresponding to the position of a bronchus. A history of thoracic tumor, particularly if such tumor is aneurismal in character, should always create suspicion of the existence of bronchial stenosis. The fact that at some time the bronchus was wounded by the lodgment of foreign bodies, etc., is also of great importance in formulating a diagnosis. Retraction of the interspaces is a valuable sign in those cases in which it is possible to eliminate the preëxistence of pleurisy with adhesions.

X-Ray Diagnosis.—The *x*-ray will serve to demonstrate the presence of foreign bodies when present.

* Chevalier Jackson, of Pittsburg, has produced a monograph giving the technic, etc., for the use of the bronchoscope, and has also devised an instrument (Fig. 180, p. 448) that has proved satisfactory in the hands of many investigators.

DISEASES OF THE LUNGS.

CONGESTION OF THE LUNGS.

Varieties.—(1) **Active congestion,** a secondary condition, which accompanies such pulmonary affections as pneumonia, tuberculosis, bronchitis, and pleurisy. Some authors claim, and correctly so, that primary pulmonary congestion, while rare, may occur, and is probably the forerunner of pulmonary edema. During pulmonary congestion the bronchial mucosa is also involved.

(2) **Passive congestion** may be present either as a general passive hyperemia of the lung tissue, mechanic in nature, or as a localized hyperemia (hypostatic congestion). In the mechanic variety of passive congestion (brown induration) the lung tissue is distended and crepitation is appreciably diminished. Even the interstitial connective tissue may be edematous, and an extravasation of blood-pigment may have taken place into the alveolar cells. The hypostatic variety seen in acute infectious diseases produces a condition known as hypostatic pneumonia.

Predisposing and Exciting Factors.—Active Hyperemia.—Generally speaking, this condition is a symptom that accompanies some form of pulmonary disease in which there is an active inflammatory process. The inhalation of irritating substances—*e. g.*, gases, foul air, flame, and hot air—may also give rise to active hyperemia. Rarely congestion follows violent exercise, such as running and other athletic feats, and it has been known to follow the excessive use of alcohol.

Passive Hyperemia.—Mechanic passive hyperemia results from an interference with the current of blood flowing between the right and left heart through the lung. The commonest causes are mitral stenosis, mitral regurgitation, and dilatation of the right ventricle. It occasionally follows traumatism to the head, apoplexy, and cerebral tumor.

Hypostatic congestion may develop as a complication of other febrile or afebrile maladies in which there is an appreciable enfeeblement of the heart's action, as in the aged and in such maladies as typhoid and other fevers; it may also occur as a late complication in cardiac disease, liver affections, the anemias, tuberculosis, and malignant disease. Hypostatic congestion is favored in those cases in which the patient rests for a long time in one position, and particularly when he lies upon his back.

Symptomatology of Pulmonary Congestion.—The symptoms of pulmonary congestion are vague. There is *cough,* accompanied by free *expectoration.* The *sputum* is covered with a thick froth, and contains shreds of mucus. In those cases in which the degree of congestion is marked, the sputum is blood-streaked, and may contain alveolar epithelial cells in which particles of blood-pigment are deposited.

Physical Signs.—Inspection.—The respirations are increased in frequency and shallow, and movement of the nostrils is distinctly perceptible. There is cyanosis of the lips and finger-tips, mild or intense, according to the degree of congestion present, and there may be lividity of the face.

Palpation often reveals a slight increase in the tactile fremitus over the bases, and when congestion develops during the course of acute fever, this sign is most often obtained posteriorly.

Percussion.—The note is impaired over the congested area, but seldom

sufficiently to be regarded as dullness. A comparative study of the upper and the lower portion of the lung is necessary in order to detect pulmonary congestion, either passive or active, since the condition is a bilateral one that attacks the bases first.

Auscultation.—The breath-sounds over the congested lung are somewhat increased (bronchovesicular), and rarely, indeed, is true bronchial breathing audible.

Summary of Diagnosis.—This is based largely upon the heart's action, a feeble heart always giving rise to a suspicion of the existence of pulmonary congestion. The presence of mitral or tricuspid disease is also of importance in formulating a diagnosis. The physical signs obtained by palpation and percussion, when sufficiently distinct, are almost positive evidence of the presence of pulmonary congestion. The frequency and character of the respirations are the only constant features, and, indeed, these may be absent when pulmonary congestion follows traumatism to the brain, paralysis, or cerebral tumor.

Clinical Course.—This is dependent almost entirely upon the pre-existing condition. In valvular heart disease pulmonary congestion often subsides after the administration of cardiac stimulants and sufficient rest. In those cases resulting from exposure to irritating gases, etc., the condition tends rapidly toward recovery. The course is likely to be more protracted in the cases of hypostatic congestion resulting from acute infectious and debilitating disease.

PULMONARY EDEMA (EDEMA OF THE LUNGS).

Pathologic Definition.—An effusion of serous fluid into the interstitial lung tissue, and an exudation of such serum into the air-vesicles.

Etiology and Varieties.—Pulmonary collateral edema is rarely, if ever, a primary condition, but is secondary to other inflammatory processes—*e. g.*, lobar pneumonia, bronchopneumonia, pulmonary infarct, hypostatic congestion, and abscess. This form of inflammatory edema attacks only the lung tissue surrounding an acute inflammatory process, a variable degree of emphysema being adjacent to it.

General Edema.—This process usually begins at the bases, but in marked cases may have invaded the entire lung substance of both sides. The mode of production of pulmonary edema is questionable, the following conditions apparently being in intimate relation with it: (1) Increased tension of the blood-vessels of the lung, from whatever cause—mitral, pulmonary, or tricuspid disease. When aortic disease produces an obstruction to the escape of blood from the left heart, this in turn causes damming back of the blood into the lung, increasing the blood-pressure there. (2) An increase in the fluidity of the blood. (3) Disease of the pulmonary vessels—*e. g.*, impaired nutrition—may also interfere with the circulation through the lung, and this pathologic state probably explains the pulmonary edema, seen in both acute and chronic Bright's disease. (4) Pulmonary edema may develop in profound septic conditions and acute and chronic maladies in which toxic poisoning is a marked feature, the edema being due to the action of such toxins upon the heart muscle (weak heart) or upon the nervous system. (5) Pulmonary edema may also follow irritation of certain portions of the vasomotor system, which in turn encourages a relaxation of the pulmonary tissue. (6) General pulmonary edema is of common

occurrence late during the course of unfavorable cases of lobar pneumonia and in all pulmonary inflammations. It may also appear as a terminal condition in both the essential and the symptomatic anemias, hepatic cirrhosis, brain tumor, cerebral hemorrhage, valvular heart disease, pulmonary tuberculosis, and acute infections in which exhaustion is prominent.

Symptoms.—Except in well-marked cases, these are, as a rule, vague. Cough is always present, and is accompanied by free expectoration of frothy, serous fluid (bronchorrhea). The patient usually complains of cold extremities and of extreme dyspnea, which latter is increased upon the slightest exertion.

Thermic Features.—Pulmonary edema does not cause a rise in temperature, therefore any febrile symptoms that are present should be regarded as indicative of the preëxisting condition.

Physical Signs.—Inspection.—The skin of the face and extremities becomes livid, and the lips and tongue are distinctly cyanosed. In selected cases the skin may be covered with beads of perspiration, but this is by no means a constant finding. The movements of the chest are feeble, and the respirations are rapid.

Palpation.—The pulse is weak and rapid, and later becomes dicrotic. The skin of the extremities is cold, whereas that covering other portions of the body is clammy.

Percussion.—The note is usually impaired at the bases, and there may be a variable degree of impairment over the greater portion of both lungs. Rarely, indeed, there is dullness over localized areas of the lung surface, a feature more common in localized than in general pulmonary edema.

Auscultation.—The true vesicular quality of the respiratory sound may be absent, and bronchovesicular breathing be audible in its place. Numerous small moist râles are to be heard over the entire chest, but are usually more marked as the bases of the lungs are approached. In those cases in which there is an associated bronchitis, large piping râles are heard at the apices, along the sternal border, and are often audible at the angles of the scapulæ. The heart-sounds are increased in frequency, and an appreciable accentuation of the second pulmonic sound is heard early. Late in pulmonary edema it is not uncommon to find cardiac dilatation, in which case both the first and the second sound of the heart resemble those of the fetus.

Summary of Diagnosis.—This is based largely upon the physical signs, particularly the increase in frequency of the respirations, the weak, rapid pulse, and the presence of numerous moist râles over the greater portion of both lungs. The absence of fever is a favorable clinical feature when pulmonary edema complicates an afebrile condition.

Differential Diagnosis.—Hydrothorax, when it complicates valvular heart disease or pulmonary disease, may be distinguished from pulmonary edema by the fact that in hydrothorax there is flatness at the bases (Fig. 45), and the upper level of this flat note changes with the position of the patient (see Pleurisy, Fig. 49), a phenomenon that does not occur in pulmonary edema. In pulmonary edema numerous moist râles are present over the area where the percussion-note is impaired, and the breath-sounds are slightly intensified; in hydrothorax, on the other hand, no râles are audible over this area, and the breath-sounds are absent. The sputum in hydrothorax is scanty, as a rule, while in pulmonary edema bronchorrhea exists.

BRONCHOPNEUMONIA (CAPILLARY BRONCHITIS; CATARRHAL PNEUMONIA).

Pathologic Definition.—An inflammatory pulmonary consolidation, often developing secondary to bronchitis, various infectious diseases, and as a terminal infection. It is characterized by the presence of an acute inflammation of the smaller bronchi and air-vesicles, with isolated areas of consolidation in both lungs (Fig. 35). The size of the consolidated areas varies from that of a pin's point to that of a pea, and these minute consolidations may coalesce, thus causing consolidation of a variable portion of one lobe. Surrounding each area of consolidation there are evidences of an attempt at compensatory emphysema.

Clinical Varieties.—(*a*) **Suffocative Catarrh.**—This term was employed by the earlier writers to describe that type of disease, due to exposure to certain poisonous gases, in which the cerebral centers were affected and the patient remained in a somewhat stuporous condition. Dyspnea and cyanosis are constant features and increase rapidly. Cough, which may be present early, disappears as the stupor increases. The respirations become more and more rapid and very shallow. Large, moist râles are audible over the entire chest; the heart-sounds become weak, and finally acute dilatation of the right heart results, which presages a fatal termination.

(*b*) **The Primary Form of Children.**—In children under two years of age the disease frequently begins abruptly, the fever rising rapidly to from 102° to 104° F. It is possible at times to localize isolated areas of pulmonary consolidation with the aid of the stethoscope and the employment of auscultatory percussion. (See p. 59.) This type of bronchopneumonia in many respects resembles true lobar pneumonia, although its clinical course is, as a rule, somewhat different.

(*c*) **The Primary Form of Adults.**—Where the patient is seen early, the signs and symptoms are those of a severe acute bronchitis, although the high fever, racking cough, increasing dyspnea, and profound prostration are more marked than in bronchitis. The sputum is scanty, and the general clinical picture resembles that of lobar pneumonia, differing only in the fact that definite physical signs—*i. e.*, consolidation and bronchial breathing—are lacking.

(*d*) **Bronchopneumonia with Remittent Fever.**—This type of catarrhal pneumonia is doubtless more common than is generally believed, and, because of its peculiar temperature-curve, may occasionally be mistaken for some other condition. The febrile peculiarities are more often seen in children than in adults, although they may occur in the aged. This variety is of special interest, since it serves to explain the peculiar intermittency of the temperature during the course of many chronic febrile and afebrile maladies.

(*e*) **Cerebral Type.**—In certain selected cases bronchopneumonia developing in children may be ushered in with severe nervous symptoms, such as intense headache, convulsions, delirium, stupor, or even coma. In these cases, also, gastro-intestinal symptoms, such as nausea, severe vomiting, and diarrhea, with abdominal pain, are not infrequently present. The abdominal symptoms are often so severe that bronchopneumonia may not be recognized. A careful analysis of the character of the respirations, the temperature, and the increasing rapidity of the heart's action, with a lessening in the volume of the pulse, is often necessary in order to recognize this type of catarrhal pneumonia.

(*f*) **Ordinary Type.**—Catarrhal pneumonia may develop during the course of acute, subacute, or chronic bronchitis, as well as during convalescence from such acute infections as measles, whooping-cough, scarlet fever, diphtheria, typhoid fever, and influenza, or during the course of certain chronic maladies, such as nephritis, hepatic cirrhosis, carcinoma, and the anemias. This distinctly secondary type of the disease often develops insidiously, and the first evidence of its existence is had when the patient displays an irregular type of temperature, or when the temperature that was present in the primary condition becomes higher. The clinical features of this type of disease will be discussed below.

Exciting and Predisposing Factors.—**Bacteriology.**—It is extremely difficult to draw deductions from the pathologic evidence found at autopsy in those dead of bronchopneumonia. Weichselbaum and other observers have found in the minute areas of consolidation, and by both the direct and cultural methods, streptococci, pneumococci, staphylococci, and the influenza bacillus. These and other bacteria have also been recovered from individual cases that have come under our notice. When bronchopneumonia has complicated typhoid fever, the typhoid bacillus is not an unusual finding. Colon bacilli are commonly cultivated from the area of consolidation, the rule being, however, to find more than one organism; as previously stated, this makes it impracticable to estimate the pathogenicity of any organism cultivated from the areas of consolidation in bronchopneumonia. A bacteriologic study of the sputum, therefore, will reveal a number of types of bacteria, many of which are known to be pathogenic.

(*a*) The disease may occur by direct extension of an acute inflammatory process due to the inhalation of some irritating substance, such as gas or ether. (*b*) It may follow the inhalation of particles of mucus, which not uncommonly occurs in laryngeal diphtheria and other conditions with obstruction in the larynx, producing the so-called "inspiration pneumonia." (*c*) It may arise as the result of the entrance of liquid or semiliquid food into the trachea, an accident that seldom, if ever, takes place unless paralysis of the muscles of deglutition or impairment of sensation of the larynx and trachea is present. Inhalation pneumonia may also result from the inspiration of blood during surgical operations upon the larynx, mouth, and tonsils, and is also a frequent complication arising during the course of carcinoma of the throat and of the esophagus. In the new-born pneumonia may follow the inspiration of amniotic fluid or of mucus from the birth-canal. It has also been found that, during the administration of ether to certain selected cases,—*e. g.*, syphilitics,—there is likely to be a hypersecretion of mucus from the upper air-passages and buccal cavity, and particles of such mucus are occasionally inspired.

Symptomatology.—Clinically, two quite distinct forms of catarrhal pneumonia may be said to exist—primary and secondary.

Primary Bronchopneumonia.—This type is found more often among adults than among children, and is characterized by a somewhat acute onset, the symptoms being those common to acute bronchitis—*i. e.*, pronounced dyspnea, severe cough with but slight expectoration, an irregular temperature, varying between 99° and 102° F., rarely reaching 104° F. In severe types of the disease the temperature may assume the continuous type for from two to four days, declining, as a rule, by lysis. The cough is always accompanied by a moderate amount of expectoration, which is at first glairy or frothy, and rather tenacious, and in occasional cases may be tinged

with blood, the latter being more common when the pneumonia has been preceded by valvular heart disease.

Secondary bronchopneumonia is a catarrhal pneumonia that develops during the course of some primary malady, the symptoms of pneumonia being frequently obscured by those of the primary affection. This type of catarrhal pneumonia seldom manifests itself until inflammatory changes in the bronchial mucosa have taken place. Secondary catarrhal pneumonia is readily recognized when the physician is thoroughly acquainted with the condition of his patient, and suddenly observes that the respirations are increased in frequency and become more and more rapid until, within the course of twenty-four to forty-eight hours, they may number from 30 to 60 or even 80 a minute. The patient, as the result of the primary disease from which he is suffering, is unconscious of the development of any acute symptoms that mark the onset of catarrhal pneumonia—*e. g.*, chill, nausea, muscular pains, etc. The first manifestation observed by the patient is cough, with difficulty in breathing. Occasionally he complains that cough evokes a pain about the base of the chest, and that although he expectorates freely, he does not obtain any relief from the dyspnea.

Thermic Features.—In both primary and secondary bronchopneumonia the temperature is controlled largely by the preëxisting condition, and is in no way characteristic, although, as a rule, it continues of an irregular remittent type.

Physical Signs.—Inspection.—In those cases in which there are numerous isolated pneumonic areas throughout the lungs the skin becomes dusky, the lips and finger-tips are cyanosed, the nostrils move quickly, and the respiratory movements are rapid, although the degree of chest expansion is somewhat limited. Extensive consolidation may follow as the result of coalescence of numerous small consolidated areas, in which case inspiratory retraction of the lower ribs and of the lower portion of the sternum has been observed, and is indicative of imperfect lung expansion.

Palpation confirms inspection as to the limited expansion of the chest. Tactile fremitus may be increased in those cases in which areas of consolidation are situated immediately beneath the pleural surface, provided the patient's chest-wall is thin. The pulse becomes rapid during the first few hours after the onset of catarrhal pneumonia, reaching 120 to 140 beats a minute. As the disease advances the tension of the pulse is lowered, the wave becomes feeble and the rhythm irregular, and, as a late feature, the pulse is dicrotic and compressible. When catarrhal pneumonia develops during the course of certain maladies in which a pulse of high tension is a characteristic symptom, such as nephritis or scarlet fever, this change in tension and frequency may be a valuable prognostic guide.

Percussion.—The percussion-note is influenced entirely by the location and size of the area of consolidation; thus, if there are a number of small pneumonic areas near the chest-wall, the percussion-note will be hyperresonant, owing to the fact that each pneumonic area is surrounded by a zone of emphysema. On the other hand, where several of these isolated pneumonic patches coalesce to form one larger area of consolidation (Fig. 35), distinct dullness is obtained, but surrounding this large area of pneumonic lung there is also a band over which hyperresonance is obtained. It is practically impossible to elicit impairment or dullness over a small area of consolidation unless this area is situated immediately beneath the pleura and the chest-wall is thin.

Auscultation furnishes most valuable data, revealing, as it does, the

presence of numerous fine crepitant râles over the pneumonic portions of the lung. The respiratory murmur will be found to have lost its normal quality and is distinctly bronchovesicular, whereas in those cases in which several small areas of consolidation have united to form a large hepatogenous mass, true bronchial breathing is audible. Most important in connection with auscultation is the fact that alterations in the breath-sounds are always detected at the base of the chest, and occasionally at the apex, but are the same over both lungs.

Laboratory Diagnosis.—The sputum is scanty at first, but in those cases in which catarrh of the respiratory tract has previously existed, it may be profuse. As a rule, it is frothy, glairy, and, in certain instances, may be streaked with blood. The sputum is tenacious, but not to the degree observed in the sputum of lobar pneumonia. A bacteriologic study of the sputum shows the presence of numerous bacteria. (See p. 108, Bacteriology.)

FIG. 35.—BRONCHOPNEUMONIA, SHOWING ISOLATED AREA OF CONSOLIDATION.

Leukocytosis may or may not be present, its presence or absence depending upon the general vitality of the patient, the nature of the preëxisting disease, and the type of organism that has excited the pneumonic process. When present, it is merely an expression of the patient's reaction against the disease.

Illustrative Case of Bronchopneumonia Complicating Influenza.—S. W. W., male, age fifty-seven; height, 5 feet 9½ inches; weight, 167 pounds.

Family History.—Father died at the age of eighty-four; cause unknown. Mother died of pneumonia at the age of sixty-one. Three younger brothers living. A sister died during childbirth at the age of thirty years. No history of malignancy or of constitutional disease in the family.

Previous History.—Does not recall having any of the diseases of childhood except measles, at fourteen years of age. Had typhoid fever at the age of thirty-four, and an attack of some stomach disorder at the age of forty-three years. Since then he does not recall having consulted his physician except for an occasional cold.

Social History.—Married, and has two daughters living. He is a business man, and although he spends most of his time indoors, he has always devoted a certain portion of the day to outdoor exercise, *e. g.*, driving, horseback riding, and walking.

Present Illness.—Began five days ago with headache, chilly sensations, pains in the loins and limbs, extreme prostration, cough, and constipation. The weakness was progressive until, by the second day, he was unable even to sit up in bed. There were frequent attacks of sneezing, marked coryza, and lacrimation.

Headache and muscular pains were intense during the first twenty-four hours, but subsided somewhat by the second and disappeared on the third day. The cough was harsh, occurring often in severe paroxysms, but was accompanied by slight, if

any, expectoration. On the third day, despite the fact that the patient's condition with reference to the respiratory tract seemed less favorable, the cough subsided.

The patient was unusually nervous from the onset of the disease. On the night of the third day there was mild delirium, and on the fourth and fifth days low muttering delirium was present, and continued throughout the course of the disease. Later there was muscular twitching, and the patient picked at the bed-clothes.

At the onset the temperature varied between 99° and 101.5° F., but on the fourth day the physical signs of bronchopneumonia developed, and the general symptoms became intensified. The temperature rose to 102.5° F., and afterward continued to run an irregular course throughout the disease.

Physical Examination.—*General.*—At the time of onset the patient's expression was anxious. Upon attempting to move it was at once apparent that extreme prostration was present, even the voice being weak. After the fourth day the patient rested upon his back, and when placed in one position, showed no inclination to move. The face and the skin in general were of a more or less dusky hue.

Local Examination.—The face was flushed. The lacrimal secretion was excessive, and he was also greatly annoyed by the discharge from the nose. The mucous membrane of the nasal cavity, throat, and conjunctivæ was congested. After the fourth day of the disease marked cyanosis of the lips, finger-tips, and tongue was present. The respirations were now rapid, and there was distinct working of the nostrils.

Palpation.—Palpation confirmed inspection with reference to the rapidity of chest movements, and it was found that there were two small areas over the left side of the chest and one on the right, through which the spoken voice-sound was unusually well transmitted.

The action of the heart was weak and rapid—about 120 beats a minute. The pulse was small, weak, readily compressible, and showed a tendency to become dicrotic. After the fourth and especially upon the sixth day it was practically impossible to count the radial pulse.

Percussion over both lungs was negative, except over the small areas where vocal tactile fremitus had been found increased, and here, upon deep percussion, a variable degree of impairment of the percussion-note was observed. Upon the fifth day of the disease the percussion-note was impaired posteriorly over the bases of both lungs.

Auscultation.—The breath-sounds were harsh, and during the first two days of the illness were accompanied by a few scattered dry and moist râles. During the third and fourth days numerous râles, chiefly subcrepitant, were heard over various portions of both lungs, and the breath-sounds over the small areas showing impaired resonance were bronchovesicular. Upon the fifth day the breath-sounds were accompanied by large numbers of bubbling râles, and at this time pulmonary edema was probably present. This type of respiration continued until the end.

The heart-sounds, while rapid, showed a lowering of the muscular quality, and during the fourth and fifth days of the illness the first sound had practically lost its booming quality, and the so-called "fetal" heart-sounds were audible.

Laboratory Findings.—As soon as the symptoms became distinctive, specimens of the secretion from the nose, throat, and conjunctivæ were stained, with a view to detecting the presence of the influenza bacillus. They showed the presence of many slender bacilli, morphologically identical with the bacillus of Pfeiffer. Cultures from the same source made upon blood-serum that had previously been treated with hemoglobin also showed the influenza bacillus. The urine became scanty during the third day, and at the fifth day anuria developed. The urine gave a well-marked reaction for albumin, but casts were not found until the fourth day of the disease, and were of the granular variety.

Course of the Disease.—When first seen by the physician, the clinical picture was that of influenza, and a diagnosis of this disease was made and was later confirmed by the laboratory findings. The case continued to be one of uncomplicated influenza until the third or the beginning of the fourth day of the illness, when the symptoms of bronchopneumonia were added. After the fourth day the patient's general condition progressed from bad to worse, and on the fifth day pulmonary edema developed. Death occurred on the sixth day.

Summary of Diagnosis.—The diagnosis is made first from a thorough study of the preëxisting condition, or from a history of exposure to the exciting causes—(*a*) inhalation of gas, anesthesia, and operations upon the mouth, nose, and throat. (*b*) The previous existence of a disease that has had delirium as one of its clinical manifestations should always arouse suspicion

of bronchopneumonia, and should encourage a careful physical examination of the chest. (*c*) While the preëxistence of bronchitis is also of importance in formulating a diagnosis of catarrhal pneumonia, in addition there must be detected isolated areas of pulmonary congestion or consolidation. (*d*) Dyspnea and cyanosis are among the most constant symptoms of this affection, and are dependent upon the number of areas of consolidation and the extent of their distribution throughout both lungs. (*e*) The somewhat prolonged duration of the course, the fact that the febrile manifestation does not terminate by crisis, and the irregular (remittent) type of fever are important factors in the diagnosis. (*f*) The heart's action, as shown by the increased rapidity and decided weakness of the pulse, is to be considered in making a diagnosis of bronchopneumonia, and there is, as a rule, a variable degree of dilatation of the right heart.

Differential Diagnosis.—Occasionally bronchopneumonia may be mistaken for **lobar pneumonia** that is tuberculous in origin. The rule, however, is that tuberculous processes involve the apices rather than the bases of the lungs, but this is by no means a constant finding. Occasionally cases of so-called simple catarrhal pneumonia are seen in which the apex of one or of both lungs is involved. During the early stages it is quite difficult to distinguish between these two conditions, but as the disease advances the physical signs of a tuberculous process, including cavity formation, become distinct, and a microscopic examination of the sputum may disclose positive evidence of the existence of tuberculosis. In acute tuberculosis the feces often contain tubercle bacilli.

The accompanying table, modified from Anders, sets forth the distinctive features between bronchopneumonia (catarrhal pneumonia) and lobar pneumonia:

Bronchopneumonia.	Lobar Pneumonia.
Etiology.	
1. Presence of pathogenic organisms* (streptococci).	1. Presence of the Diplococcus pneumoniæ.
2. Usually secondary to bronchitis and acute infectious diseases, *e. g.*, measles, whooping-cough.	2. Usually a primary disease.
Clinical History.	
3. Onset gradual, without rigor.	3. Onset abrupt, with rigor; previous health generally good.
4. Fever is governed by the extent of the inflammation, is of irregular type, and after a variable duration, declines by lysis.	4. Fever is high, of continuous type, and falls by crisis between the fifth and ninth days.
5. Sputum glairy, tenacious, and in adults may be blood-tinged.	5. Sputum characteristic (rusty or prune-juice and very tenacious).
6. Dyspnea and cyanosis prominent.	6. Dyspnea and cyanosis relatively less marked; countenance anxious.
7. Physical signs of generalized bronchitis always marked, and usually preponderating over those of consolidation.	7. Signs of bronchitis generally absent; those of lobar consolidation always preponderating.
8. Consolidation commonly bilateral.	8. Commonly unilateral.
9. Duration indefinite, often extending over many weeks.	9. Duration definite, as a rule; convalescence follows crisis.
10. Consolidated areas likely to become the seat of tuberculous infection.	10. Far less likely to become the seat of tuberculous infection.

* The discovery of streptococci in the sputum is of questionable diagnostic value, since numerous other organisms have been found in the sputum of bronchopneumonia when streptococci were absent. The Streptococcus pneumoniæ of Weichselbaum has also been found in a number of cases of lobar pneumonia.

Clinical Course.—This is governed entirely by the degree of dissemination of the minute areas of consolidation in each lung. In those cases in which primary bronchopneumonia follows the inhalation of irritating and foreign substances the clinical course is rapid and reaches its height within from twenty-four to seventy-two hours. In the mild forms of primary catarrhal pneumonia recovery generally ensues in from seven to fourteen days.

In secondary pneumonia the clinical course and prognosis are governed largely by the preëxisting disease, and when this has been of a mild type, a favorable termination may be expected in the course of two or three weeks. In those suffering from a severe type of typhoid fever, influenza, scarlet fever, etc., the catarrhal pneumonia is also likely to be of a severe grade, and continues for three or more weeks unless a fatal termination ensues. Death occurs in from 28 to 50 per cent. of such cases.

LOBAR PNEUMONIA

(CROUPOUS OR FIBRINOUS PNEUMONIA; PNEUMONITIS; LUNG FEVER).

Pathologic Definition.—An acute infectious disease caused by the Diplococcus pneumoniæ, which produces an acute inflammation of the substance of the lung. It is characterized pathologically and clinically by three stages: (1) The stage of congestion; (2) the stage of red hepatization, and (3) the stage of gray hepatization. (See Infectious Diseases, p. 770.)

CHRONIC INTERSTITIAL PNEUMONIA

(FIBROID INDURATION; CIRRHOSIS OF THE LUNG).

Pathologic Definition.—A primary or secondary, subacute or chronic, disease, characterized by the overproduction of pulmonary connective tissue.

Varieties.—**Local interstitial pneumonia** is a term used to describe a condition in which but a portion of one lobe or of one lung has become sclerotic and contracted; **diffuse interstitial pneumonia** differs from the localized type only in the fact that in the former there is an overproduction of the pulmonary fibrous connective tissue in both lungs, such fibrous growth generally involving the greater portion of the entire pulmonary tissue.

Predisposing and Exciting Factors.—Interstitial pneumonia is occasionally encountered as an apparent primary condition, but in the majority of cases it is secondary to some prolonged inflammatory process involving the lung tissue. Pneumonokoniosis, or continuous exposure to the inhalation of certain irritants,—*e. g.*, dust, particles of steel, lime, and the like,—may produce chronic interstitial changes in the lung tissue. Localized interstitial pneumonia not infrequently complicates acute pneumonia, pulmonary abscess, and, less often, it is the result of pulmonary tuberculosis, syphilis, disease of the pleuræ, cysts of the lung, and emphysema.

Diffuse interstitial changes in the lung may occur: (1) After low-grade types of lobar pneumonia or bronchopneumonia in which resolution has been delayed. (2) They may develop as a sequel to influenza in which there have been indefinite symptoms of pneumonia. (3) They may occur in cases in which a portion of the lung becomes atelectatic, due either to disease of the lung itself or to pressure from without. (4) Interstitial changes may take place in and about the pneumonic areas of those recovering from bron-

chopneumonia, and after some time has elapsed the entire lung may become highly sclerotic. (5) Bronchopneumonia excited by the tubercle bacillus often shows a special tendency toward the development of fibroid changes in the lung, and this condition has been designated pulmonary tuberculosis with the production of fibroid tissue. (6) In certain cases of pleurisy there is a decided tendency toward the formation of connective tissue, and this pathologic change may extend from the pleura to the lung tissue, until finally the greater portion of the lung is bound down by dense, fibroid bands, or may be penetrated by fibrous tissue that extends deeply into the lung substance. (7) As a rule, both the lung and pleuræ are affected by the fibrous change, but a high grade of fibrosis of the lung may occur without coincident change in the pleura, or the reverse condition may obtain.

Principal Complaint.—There is a definite history of a continuous loss of flesh and of strength, which may have covered weeks, months, or even years. The patient usually complains bitterly of cough, which grows progressively worse, and is always accompanied by a mucoid or mucoserous and, at times, bloody expectoration. If the disease is due to pneumonokoniosis, the sputum may be discolored by the substance inhaled. Dyspnea occurs upon the slightest exertion. Pain may or may not be present, but in those cases in which there are many pleural adhesions it is an annoying symptom. Owing to the high grade of interstitial change that frequently takes place, constriction of certain portions of a bronchus, with an appreciable expansion of another portion of the same bronchus, may occur, and, as a sequence, the patient may exhibit symptoms of bronchiectasis.

Thermic Features.—The temperature is normal in uncomplicated cases of chronic interstitial pneumonia.

Physical Signs.—**Inspection.**—According to whether the condition is bilateral (rare) or unilateral the chest-wall is retracted, and this retraction is usually most conspicuous at the apices, although there may be basilar contractions with overdistention of other portions of the chest. Generally, however, the contour of the chest in chronic interstitial pneumonia is irregular. By inspection certain portions of the chest will be seen to expand freely, while other portions remain unchanged or expand but feebly. In those cases in which the left lung is most affected the heart is likely to be displaced upward and probably to the left. In right-sided chronic interstitial pneumonia it is not uncommon to find the heart drawn well to the right of the sternum.

Palpation confirms inspection with reference to the expansion and contour of the chest. Tactile fremitus is, as a rule, increased, and particularly is this the case over those portions of the chest-wall that have sunken as the result of fibrous pulmonary changes. Occasionally, as the result of certain changes in the pleuræ, the tactile fremitus is found to be decreased.

Percussion.—The percussion-note will vary within wide limits over different portions of the same lung; thus, dullness and almost flatness may be obtained where marked sclerotic change in the lung tissue and decided thickening of the pleura have taken place, while but a short distance away from this point the percussion resonance may be hyperresonant or even tympanitic in character, the latter note depending upon the existence of compensatory emphysema. The area of cardiac dullness may also vary with the character and degree of fibrous change present (retraction of the lung).

Auscultation.—The breath-sounds are in no way characteristic. If fibrous bands connect a bronchus directly to the surface of the chest, the breathing will often be bronchial in character, although but slight actual

consolidation may be present. If a bronchus is expanded, amphoric or even cavernous breathing may be elicited. As a rule, the breath-sounds at the base of the lungs posteriorly are feeble. A friction-sound is often detected, and may be present over a period of several days or weeks. Râles are also present, but are of limited clinical significance.

Summary of Diagnosis.—A diagnosis is attained, first, from a careful analysis of any preëxisting disease of the lung and pleura. Next, the physical signs are of importance; thus deformity of the chest, marked retraction of the affected side, and irregular expansile movements are among the most positive findings of chronic interstitial pneumonia. Cough and dyspnea with moderate emaciation are additional evidence of the existence of this disease.

Clinical Course.—This is chronic throughout, the patient growing slightly, although progressively, worse for a period of three, five, or more years. A fatal termination seldom results directly from interstitial pneumonia, but is due usually to an intercurrent malady.

PNEUMONOKONIOSIS

(ANTHRACOSIS; CHALICOSIS; SIDEROSIS).

Pathologic Definition.—A variety of chronic interstitial pneumonia due to the inhalation of small particles of a solid substance, such as lime, stone, iron, or coal. The various forms of the disease derive their names from the character of the substance inhaled. Thus:

(*a*) When interstitial change in the lung follows the inhalation of coal-dust, the disease is known as **anthracosis.** A macroscopic study of the lung shows it to be brown or black in color, its pleural surface presenting a mottled appearance. Upon incising the organ the knife encounters a gritty, stone-like resistance. Microscopically particles of black pigment are to be found within the pulmonary tissue, and there may also be an increase in the fibrous connective tissue, although in those suffering from the so-called miner's asthma undue distention of the air-cells may be apparent.

(*b*) A similar pathologic condition presenting the same clinical symptoms is seen in stone-cutters. This is termed **chalicosis.**

(*c*) Employees of factories and foundries in which the air is laden with metallic particles often develop sclerotic changes in the lungs, the condition being known as **siderosis.**

Principal Complaint.—The symptoms develop somewhat insidiously, and within the course of a few months the patient complains of chronic bronchitis. This condition, however, does not exist until after several months or even years of more or less constant exposure to the irritating substances. After the symptoms of asthma (p. 97) have been present for weeks, months, or possibly years, the patient develops emphysema (see p. 124), which may be either localized or general, and at this time the symptoms he complains of are practically those of emphysema.

Physical Signs.—These are not at all distinctive, nor are they constant in any series of cases. Generally speaking, the physical signs of anthracosis are the same as those of chronic interstitial pneumonia (*q. v.*), the exceptions to this general rule being that in selected cases emphysema develops early, and that instead of the chest being retracted, it is abnormally distended.

Laboratory Diagnosis.—This offers the most reliable clinical evidence. Regardless of the cause of the disease in question, the sputum is copious, and each paroxysm is followed by profuse expectoration. In an-

thracosis the sputum may be black or brownish in color, and when studied under a $\frac{1}{8}$ or $\frac{1}{12}$ inch oil-immersion lens, small particles of coal will be seen to be present (Plate I). Not uncommonly leukocytes containing small black particles of dust are seen, and, indeed, dust is often apparently embedded within certain epithelial cells.

The sputum of those suffering from stone-cutter's disease or of those who have been exposed to the inhalation of plaster-of-Paris or of lime shows the presence of many small white or grayish particles of silica. The macroscopic appearance of such sputum, however, is in no way diagnostic. In siderosis, as in anthracosis, the sputum is characteristic, being rusty colored or reddish, and upon microscopic study presenting fine particles of metallic substance.

The sputum of pneumonokoniosis always contains many degenerated epithelial cells, leukocytes, pus-cells, and a profusion of bacteria. The tubercle bacillus is a common finding.

Summary of Diagnosis.—A history of exposure to coal, stone, iron, or metallic dusts is of great importance, and should always suggest pneumonokoniosis. The symptoms of bronchitis, the physical signs of interstitial pneumonia, and the characteristic microscopic findings in the sputum leave no room for doubt as to the nature of the condition.

Course and Duration.—This form of chronic fibroid pneumonia is of prolonged duration, the patient growing progressively worse from year to year, the condition, as a rule, terminating in pulmonary tuberculosis.

PULMONARY TUBERCULOSIS.

Pathologic Definition.—An infectious disease caused by the Bacillus tuberculosis. It may be acute or chronic in form, and is characterized by the formation of small tubercles in the lungs and in other portions of the body. When infection is localized to the lung, large areas of consolidation occur, which eventually break down and result in cavity formation. When general infection takes place, the so-called miliary tuberculosis results. In the latter condition microscopic tubercles may be found in the muscle tissue, choroid, and in practically all the viscera. (See Infectious Diseases, p. 803.)

PNEUMORRHAGIA (PULMONARY APOPLEXY).

Pathologic Definition.—The escape of blood from the pulmonary vessels into the air-cells and connective tissue of the lung, either with or without appreciable laceration of the pulmonary tissue.

Varieties.—There are three varieties—the circumscribed, the pneumonic, and the diffuse.

Predisposing and Exciting Factors.—The most common cause of pulmonary apoplexy is **rupture of a thoracic aneurism** after it has become firmly adherent to the visceral pleura. The conditions that favor the accident are those that favor the rupture of thoracic aneurism—*e. g.*, traumatism to the chest, heavy lifting, and morbid states that increase the heart's action.

Sex is a predisposing factor, the majority of cases occurring in adult males.

Clinical Features.—As a rule, the history and physical signs of aneurism are present prior to the development of pulmonary hemorrhage, and the general clinical picture is that described under thoracic aneurism (p. 312), the most prominent characteristics of which are hemoptysis, dysp-

nea, cyanosis, subnormal temperature, and a tendency toward circulatory collapse.

Physical Signs.—If the patient survives the initial hemorrhage, the physical signs of pulmonary consolidation may be elicited in selected cases.

Laboratory Diagnosis.—The sputum is almost pure blood, is expelled with but slight coughing, and does not contain tubercle bacilli or fungi.

PULMONARY EMBOLISM

(HEMORRHAGIC INFARCTION; EMBOLISM OF THE LUNGS).

Pathologic Definition.—A condition caused by thrombosis or by embolism of a branch of the pulmonary artery, resulting in a wedge-shaped infarct, the base of which is directed toward the pleura. The involved portion of the lung becomes firm, airless, and dark in color. This condition may be single or multiple, the area of pulmonary tissue involved rarely exceeding the size of a walnut.

Varieties and Etiology.—(1) **Non-septic embolism** occasionally occurs during the course of chronic organic heart disease, and is said to follow mitral stenosis and, less often, mitral regurgitation. Thrombi may also be generated in the right auricle or in the large vessels, and finally become lodged in the branches of the pulmonary artery. Venous stasis involving the pulmonary tissue also predisposes to pulmonary infarct.

(2) **Fat Emboli.**—The pulmonary vessels are plugged with emboli of fat. There may be minute hemorrhages into the interstitial tissue surrounding the lesion, and the small arteries are filled with fat.

Predisposing Factors.—Traumatism to the adipose tissue and fractures.

Symptoms.—In this type of fat-embolus are seen extreme dyspnea, cardiac failure, and temperature ranging about 102° F. Symptoms usually progress from bad to worse.

(3) **Septic emboli** are carried to the lung from septic processes elsewhere in the body, and may complicate gangrenous or suppurative conditions involving remote organs.

Principal Complaint.—In non-septic cases both the history and the general clinical features are those of valvular heart disease. Following the pulmonary embolism acute symptoms develop—*e. g.*, pain in the chest, dyspnea, expectoration of blood-streaked mucus, followed by syncope. Hemoptysis, while not a constant feature, is of clinical significance when it develops in a patient suffering from mitral disease, and when it is accompanied by severe pain and cough, is still more significant of pulmonary embolism.

Physical Signs.—Unless the area of lung involved is comparatively large, percussion and palpation are negative. If several emboli are lodged in different portions of the lungs, the character of the respiration is that of bronchopneumonia (p. 109).

By **palpation** it is at times possible to detect areas in which the tactile fremitus is increased and the **percussion-note** impaired.

Auscultation.—Moist râles are audible over the greater part of the affected lung, and the breath-sounds are intensified. When a large area of lung collapses as the result of an embolus, bronchial breathing may be audible. It must be borne in mind that the physical signs are in part, at least, the result of hyperemia of the surrounding pulmonary tissue, and, indeed, in many instances the actual condition is obscured by the emphysema which surrounds the diseased portions of the lung.

Laboratory Diagnosis.—The sputum is often bloody, the blood being equally distributed throughout. It is easily expectorated, is always frothy.

Clinical Course.—Septic cases run a rapid febrile course, and the prognosis is that of pyemia. In non-septic emboli the course is also rapid, but the condition may exist for days or even weeks and recovery follow.

PULMONARY GANGRENE.

Pathologic Definition.—A condition produced by the action of the organisms of putrefaction upon a devitalized section of pulmonary tissue. The extent of the destructive changes in the lung tissue varies greatly in different cases. The gangrenous area is surrounded by a zone of congestion.

Varieties.—(*a*) **Diffuse gangrene** is rarely seen complicating lobar pneumonia. In this condition there may be occlusion of the larger branches of a pulmonary artery. This process may be so extensive as to involve an entire lobe; cases have been reported in which an entire lung was destroyed.

(*b*) **Circumscribed gangrene** may be unilateral or bilateral. Circumscribed gangrene may follow embolus of an artery, or, as is more often the case, it may be the termination of an acute inflammatory process.

Exciting and Predisposing Factors.—(1) Gangrene occurs when the pulmonary tissue, devitalized from any cause, is attacked by the organisms of putrefaction. Pathogenic bacteria may also be present, although the part played by this class of micro-organisms is questionable. The exact degree and nature of the changes in the pulmonary tissue necessary for the development of saprophytic bacteria (organisms producing gangrenous changes) have long been the subject of controversy; suffice it to state here that both saprophytic and parasitic bacteria are likely to be present in the sputum of pulmonary gangrene.

(2) Acute inflammation of the lung,—*e. g.*, lobar pneumonia,—bronchopneumonia, tuberculous cavity with secondary infection, acute bronchitis, stab wounds and traumatism of the lung, contusions of the thorax, as well as inflammation resulting from perforation of the diaphragm or of the esophagus by carcinomatous or ulcerative processes, are all conditions that predispose to the development of pulmonary gangrene. In fact, it is by one or more of these processes that the vitality of the pulmonary tissue is lowered.

(3) Pulmonary hemorrhagic infarction, emboli derived from gangrenous tissue elsewhere in the body and from purulent tissue, with the production of pulmonary abscess, may at times be the exciting causes of gangrene. Pulmonary gangrene frequently develops from an embolus that probably originated in a suppurative process in the middle ear or the mastoid cells.

(4) Foreign bodies—*e. g.*, particles of food, metallic substances, etc.—entering the lung by way of the trachea are prominent exciting factors in pulmonary gangrene. (See Bronchopneumonia, p. 108.)

(5) Thoracic tumor, either aneurismal or glandular, may, by continual pressure upon the lung, give rise to gangrene.

(6) Those suffering from certain acute infectious maladies—*e. g.*, noma—are especially likely to develop gangrene. In protracted febrile conditions pulmonary gangrene may occur as a complication, but seldom develops until convalescence sets in.

(7) In such afebrile conditions as diabetes mellitus and chronic valvular (mitral) heart disease gangrene of the lung is not of infrequent occurrence. (See Pulmonary Embolism, p. 117.)

Principal Complaint.—In those cases in which the area of gangrenous involvement is small and in which secondary infection with pyogenic bacteria has not occurred, there are few, if any, constitutional symptoms, and the patient complains only of *cough* and the *expectoration* of fetid material. In this class of cases the physical signs are negative.

In those suffering from more extensive pulmonary gangrene or gangrene following abscess-formation, pneumonia, the inspiration of foreign substances, etc., there are progressive weakness and loss in weight. *Anorexia* develops early, and continues throughout the disease. The most annoying symptom is *cough*, which is paroxysmal. The patient may cough between the paroxysms, but, as a rule, a violent spell of coughing occurs every two to six hours, particularly after awaking from sleep. During each attack of coughing a large quantity of sputum may be expectorated and emit a characteristic gangrenous odor. (See Laboratory Diagnosis, p. 120.) In the majority of instances the patient's breath also gives off this offensive odor, although this is not a constant finding, having been absent in a case observed by one of us, in which the gangrenous process was found at autopsy to have no direct communication with a bronchus.

Pain may be present, but is seldom an annoying symptom unless the areas of lung that are involved are superficially situated and an associated pleurisy is present. *Vomiting* may be an annoying symptom, and is probably excited by the offensiveness of the material expectorated. The patient's *general condition* usually progresses from bad to worse, until finally he is unable to leave his bed. Pulmonary hemorrhage, although by no means a common symptom, may follow ulceration of the pulmonary artery, and profuse hemorrhage may rarely cause a fatal termination.

Thermic Features.—Early in pulmonary gangrene the fever becomes irregular, fluctuating between 99° and 102° F. In those cases in which sepsis becomes profound or gangrene develops secondarily to a suppurative process elsewhere, the temperature is governed largely by the preëxisting condition, and is often of the continued type; an exception to this rule is seen in pulmonary gangrene complicating pulmonary tuberculosis with cavity formation, in which there is an evening rise in temperature followed by a morning remission.

Physical Signs.—**Inspection.**—In cases showing constitutional symptoms there is extreme pallor, and later cyanosis of the mucous membrane, finger-tips, and feet develops. Swelling of the ankles may also occur late during the course of the process. Profound emaciation is always present in this class of cases.

Palpation.—The pulse becomes weak, rapid, irregular, and often dicrotic. These and other characteristics of the pulse are dependent on the degree of prostration. If gangrene follows pulmonary abscess or pulmonary tuberculosis, the evidence obtained upon palpation will be the same as that obtained in these conditions. (See Tuberculosis, p. 803.) Generally speaking, the tactile fremitus will be found increased whenever there is an associated consolidation of the pulmonary tissue that extends to a point near the chest-wall. Gangrenous areas that are located centrally, however, manifest no definite physical signs. The chest movements are frequent, and may be jerking in character.

Percussion gives negative results except in those cases in which there is consolidation, when resonance is impaired. A hyperresonant note may be obtained over the lung immediately surrounding the gangrenous process.

Auscultation.—The heart-sounds are weak and rapid, and there is

often evidence of valvular disease, which is particularly common when pulmonary gangrene follows embolism. During the stage of consolidation the breath-sounds over the affected regions are harsh and may be bronchial in character. Both fine and coarse moist râles are audible, and late in the disease the breathing may be that heard in the presence of pulmonary cavity. If the pulmonary inflammation extends to the surface of the lung and the visceral pleura becomes involved, a pleural friction murmur may be audible. (See Pleurisy, p. 139.)

X-Ray Diagnosis.—It is possible not only to locate accurately the site of the gangrenous process, but this affection gives a fairly characteristic picture. The shadow will be found to vary greatly, depending upon the size and duration of the process. (See p. 74.)

Laboratory Diagnosis.—The **blood changes** are those of secondary anemia; at times leukocytosis may be present. Cultures from the venous blood are likely to give positive results when gangrene follows septic processes.

The **sputum** is of a greenish or bloody color, and is frequently said to be "prune juice" in character. When placed in a conic glass and permitted to stand for several hours, the sputum will be found to have separated into three quite distinct strata: (1) A superior layer, which is frothy, opalescent, and of a greenish yellow color; (2) a middle stratum, which has the appearance of water; and (3) an inferior layer, which is composed of a greenish or brown sediment, showing macroscopically many shreds of mucus and necrotic tissue; rarely this layer is bloody. Microscopically, many bacteria are present, and portions of degenerated lung tissue, fibers of elastic tissue (rare), pus-cells, fungi, and both red and white blood-cells are seen.

Summary of Diagnosis.—This is determined largely by the characteristic odor of the breath and of the sputum. The fact that the sputum separates into the characteristic layers when permitted to stand for several hours, and the detection, in the inferior layer, of lung tissue, go far toward confirming the diagnosis. Rapid emaciation and progressive prostration are prominent features in pulmonary gangrene.

Differential Diagnosis.—Fetid Bronchitis.—In this condition the sputum, while highly offensive, does not have a gangrenous odor, and emaciation and prostration are often lacking and are never profound. Fever, which is common in pulmonary gangrene, is not marked in fetid bronchitis.

Clinical Course.—This is dependent, first, upon the extent of the pathologic process, and, secondly, upon whether or not such devitalized tissue becomes infected with pyogenic organisms. In those cases showing marked constitutional symptoms—*e. g.*, fever, prostration, etc.—the clinical course is rapid, extending over a period of weeks, or at most months. Repeated hemorrhages from the lung render the prognosis less favorable.

PULMONARY ATELECTASIS

(COLLAPSE OF THE LUNG; COMPRESSION OF THE LUNG).

Pathologic Definition.—A condition in which a portion or the entire volume of air is removed from the air-cells of a portion of the lungs.

Predisposing and Exciting Factors.—(1) This condition is most commonly encountered in the new-born and in prematurely born infants. In either case it may be the result of feeble breathing power on the part of the child or of malformation of the respiratory tract. (2) Acquired atelectasis may follow inflammatory processes with the production of mucus in

the smaller bronchial tubes. (3) Compression of the lung from whatever cause (pericardial effusion, pleural effusion, thoracic aneurism, new-growths of the thorax, and pneumothorax) may also give rise to atelectasis. (4) Enfeebled respiratory efforts markedly predispose to the development of atelectasis, and may result from cerebral hemorrhage, interference with the nerve supply to the lung, pressure upon the diaphragm from large abdominal tumors, peritoneal fluid, and tympanites. (5) Late during the course of bronchopneumonia and lobar pneumonia portions of the lung may become atelectatic, as has been shown by autopsy.

Symptoms and Signs.—As this condition is practically always secondary, the symptoms and signs are those of the preëxisting disease, appreciably intensified, however, by atelectasis.

Inspection.—The movements of the chest are, as a rule, limited to the apices. Respiration is labored and unusually frequent.

Course.—The patient rapidly approaches a state of collapse, the pulse becomes weak and rapid, the skin cold and clammy, vitality diminishes, and death soon follows.

ABSCESS OF THE LUNG
(Suppurative Pneumonitis).

Pathologic Definition.—An acute localized infection in which destruction of the pulmonary tissue and a circumscribed accumulation of pus within the lung occur. Surrounding an acute abscess there is an area of consolidation, and still further beyond the pulmonary tissue is congested for a considerable distance. The abscess may communicate with a bronchus or may rupture into the pleura.

Exciting and Predisposing Factors.—(1) **Bacteria.**—Streptococci are commonly present, but are not the only direct excitant. The diplococcus of pneumonia and the bacillus of Friedländer are not infrequently seen, as are also other pyogenic organisms—*e. g.*, staphylococci, Bacillus pyocyaneus, Bacillus coli communis.

(2) An **acute localized inflammation of the lung,** such as is seen in both lobar and lobular pneumonia, may terminate in abscess-formation; hence the conditions that predispose to these types of pneumonia also predispose indirectly to the formation of abscess.

(3) **Penetrating wounds of the lung** from without, perforation of the lung from ulcer or carcinoma of the esophagus, abscess of the liver, gastric ulcer, etc., are also among the exciting causes of pulmonary abscess.

(4) The **aspiration of foreign substances** that may carry with them pyogenic bacteria is also productive of abscess.

(5) In **pulmonary tuberculosis with cavity formation** abscesses are common, and isolated abscesses may be found in different portions of the lung.

(6) Metastatic abscess of the lung may develop during the course of **septic processes** elsewhere in the body and during septicopyemia. Septic emboli from whatever source frequently find a lodging-place within the pulmonary tissue and give rise to circumscribed abscess there. In this particular type of infection of the lung the abscess is usually situated near the pleural surface and is often egg-shaped.

The history is of considerable importance in making the diagnosis. Trauma to the chest may be an exciting cause. The tendency to the development of pulmonary abscess is greatly increased while a patient is under

treatment for septic conditions elsewhere or for acute ulcerative endocarditis.

Principal Complaint.—*Pain* may be a prominent feature in those cases in which the abscess is situated near the surface of the lung, and consequently excites pleuritis. *Cough* may be an annoying symptom, and is usually accompanied by the free *expectoration* of purulent material. *Chills*, followed by an elevation in the temperature and later by profuse sweating, are a conspicuous feature in a fair proportion of all cases. *Prostration* is progressive, and there is also a steady loss of weight. As a rule, the patient becomes nervous and irritable, and delirium is to be expected when fever is a conspicuous factor.

Thermic Features.—The temperature is somewhat influenced by the preëxisting condition; however, an irregular fever, 101° to 104° F., is to be expected.

Physical Signs.—**Inspection.**—Emaciation is well marked. The skin and mucous surfaces are unusually pale, and if the abscess is large, the lips and finger-tips may be cyanosed. The movements of the chest vary greatly, depending upon whether or not the pleura is involved; in any case the respirations are increased, and if the abscess is large, the two halves of the chest may expand unequally.

Palpation shows an increase in the tactile fremitus over the consolidated areas. The heart's impulse is weak and rapid, and abnormal pulsation is often detected above the right clavicle. Unless the abscess cavity is large and superficial, palpation may be negative.

Percussion.—A variable degree of impairment is elicited over the abscess, and surrounding it for some distance there may be hyperresonance, due to compensatory emphysema.

Auscultation.—The breath-sounds may be those heard in the presence of a cavity, yet this finding is unreliable in many instances. Owing to the inflammatory changes and edematous condition of the lung, large and small moist râles are heard in the region of the affected area. Bronchial breathing is not infrequent.

X-Ray Diagnosis.—Here the clinical features of pulmonary abscess are comparatively clear, and its location is possible through this means of diagnosis (p. 74).

Laboratory Diagnosis.—This probably furnishes the most reliable data in the diagnosis of pulmonary abscess. The sputum is purulent, yellow, or frequently greenish or brownish-yellow, in color, and at times it may be streaked with blood. The odor of the sputum is, as a rule, offensive, but at times sweetish, being distinctly different from that emitted from the sputum of pulmonary gangrene and of putrid bronchitis. Microscopically, it will be found to contain particles of lung tissue (elastic fibers), pus, red blood-cells, and granular tissue débris.

Summary of Diagnosis.—(*a*) A history of the existence of some condition or an accident that predisposes to the development of pulmonary abscess is of great importance. (*b*) Diagnostic value is to be attached to the examination of the sputum and the detection of great numbers of elastic fibers. (*c*) The physical signs of cavity-formation, when present, go far toward confirming the diagnosis, but it is impossible to base the diagnosis upon the clinical history and the findings obtained by physical examination. The *x*-ray is important in this connection.

Clinical Course.—In abscesses following the development of pyemic processes elsewhere the course is rapid, and the general clinical picture is

that of septicemia plus the characteristics of pulmonary abscess. Abscess arising as a sequel of lobar pneumonia may run a protracted course, terminating favorably in from six to twelve weeks.

Complications.—If the abscess is situated near the visceral pleura, it is likely to perforate the pleura, giving rise to a purulent pleuritis (p. 150), empyema, or *pyopneumothorax* (p. 161).

NEW-GROWTHS OF THE LUNGS.

General Remarks.—The most common tumors of the lung are carcinoma and sarcoma. In rare cases these growths may be primary, and when this is the case, a unilateral development is observed. In the majority of instances, however, malignancy of the lung is a secondary condition, and both lungs are involved, numerous foci being seen.

Carcinoma of the Lungs.

Pathologic Definition.—A carcinomatous growth, often secondary, involving the pulmonary tissue and resulting in consolidation of the organ. The carcinomatous process may extend by contiguity to the pleuræ and other thoracic structures.

Varieties.—(1) Primary pulmonary carcinoma; (2) carcinoma secondary to carcinomatous growth involving remote portions of the body (head, rectum, or extremities); this is, as a rule, bilateral. (3) Secondary carcinoma resulting from direct extension of a carcinomatous process from the esophagus, stomach, liver, or mammary gland; this is frequently unilateral.

Principal Complaint.—This will be found to vary in accordance with the location and extent of the lesion. *Pain* is an early symptom, and in those cases in which the pleuræ are involved, is agonizing. The general symptoms of bronchitis—*e. g., cough, expectoration, dyspnea*—are quite constant. If the growth becomes large, symptoms referable to pressure upon the heart and upon the large thoracic vessels are also present. Late during the course of pulmonary carcinoma pressure upon the esophagus may give rise to dyspnea; and should the recurrent laryngeal nerve become involved, hoarseness and aphonia ensue. (See symptoms of aneurism, p. 313.) Pulmonary tumor may also exert sufficient pressure upon a bronchus to cause the signs and symptoms of bronchial stenosis. (See p. 103.)

Physical Signs.—Inspection.—In those cases in which there may be a large new-growth in the lung the thorax will be somewhat prominent and fixed over the site of the tumor. Cases are recorded in which a carcinomatous mass protruded through the chest-wall. The interspaces are, as a rule, widened, and the cutaneous veins are distended. The right supraclavicular region is unusually prominent, and often shows decided pulsation as the result of intrathoracic pressure. Edema of the face, neck, and even of the thorax and arms is a late symptom in pulmonary carcinoma. The axillary and cervical lymph-nodes are generally enlarged.

Palpation confirms inspection as to the movements of the chest, enlargement of glands, and edema, and, in addition, reveals the fact that the tactile fremitus is altered and in many instances absent over certain portions of the lung, whereas at other points it may be normal or increased as the result of pleuritic adhesions or localized areas of consolidation. If the pleura is attacked by the carcinomatous process, an effusion into the pleura generally

follows, in which case the physical signs of serofibrinous pleurisy (see p. 142) are present in addition to those of pulmonary carcinoma.

Percussion.—The note is impaired over all portions of the lung invaded by the tumor, and the degree of impairment varies with the degree of pulmonary consolidation present.

Auscultation.—Where there is extensive carcinomatous involvement of both lungs, the respiratory murmur may be greatly diminished, and indeed absent, over certain localized sections; but in those cases in which the tumorous growth is localized along the course of a large bronchus, bronchial breathing is audible, and, indeed, the breath-sounds may resemble those heard when a pulmonary cavity is present. Numerous large and moist râles may be present over one portion of the chest, while at other portions the breath-sounds may be absent. So varied is the evidence obtained by auscultation that this method offers but little valuable data in the study of this disease.

Laboratory Diagnosis.—Free expectoration is an almost constant symptom; at the same time, during the course of the disease, the sputum resembles currant-jelly, or again it may be bloody, or perhaps green in color, depending upon the character of the changes that have taken place in the carcinomatous tissue. The sputum commonly emits an offensive odor.

Microscopically, pus-cells, leukocytes, red blood-cells, granular débris, and occasionally clusters of epithelial cells—the so-called "cancer clusters"—are found; too great an importance should not be attached to finding the last-named elements. Crystals of hematoidin are occasionally seen.

The *hemic* changes are those of secondary anemia. (See *x*-ray Diagnosis, p. 75.)

Clinical Course.—Carcinoma of the lung progresses from bad to worse, terminating fatally within a few weeks or months.

SARCOMA OF THE LUNG.

Remarks.—Sarcomatous disease frequently invades the glandular tissue at the root of the lung, although secondary sarcoma of the lung proper may also be met. The diagnosis of sarcoma is based largely upon the clinical history and the preëxistence of a sarcomatous growth elsewhere. The *symptoms* and *signs* closely resemble those of pulmonary cancer (*q. v.*). Two cases of pulmonary sarcoma have developed in patients under the care of one of us at the Philadelphia General Hospital, and both of these followed sarcoma of the knee.

PULMONARY EMPHYSEMA.

Pathologic Definition.—This is a chronic disease, characterized by an abnormal thinning and loss of power of the pulmonary air-cells, with overdistention of such cells by air, and possibly escape of air into the interlobular connective tissue. The bronchial mucous membrane is usually the seat of a chronic inflammation. (See Remarks and Pathologic Characteristics, p. 125.)

Compensatory emphysema, however, is not a pathologic process, but consists in physiologic dilatation of the air-cells secondary to pathologic processes in other portions of the lung.

Varieties.—(1) **Interlobular emphysema** is a condition in which

an air-cell has ruptured and a portion of its contained air has escaped into the surrounding connective tissue.

(2) **Vesicular emphysema** is an abnormal dilatation of the alveoli and finer air-passages. There are three varieties: (*a*) Compensatory; (*b*) hypertrophic; and (*c*) atrophic.

Interlobular Emphysema.

Etiologic Factors.—These include: (*a*) Injury to the lung, penetrating wounds made by fractured ribs, violence, etc. (*b*) Paroxysmal coughing, as, *e. g.*, in whooping-cough, and the inhalation of irritating gases; indeed, this condition may rarely follow violent muscular exercise, convulsions, and labor. This type of emphysema selects by preference the upper lobes and anterior surface of the lung. Interlobular emphysema may rarely be found as an associated condition in advanced stages of vesicular emphysema.

Vesicular Emphysema (Compensatory Emphysema).

This variety is limited to certain localized pulmonary regions, and, as its name implies, occurs as the result of pathologic conditions in other portions of the viscus that prevent, or at least inhibit, lung expansion during the act of inspiration. Compensatory emphysema, therefore, is not a pathologic condition, but a vicarious one, demonstrating the capability of the air-cells in one portion of the lung to expand sufficiently to do the additional work of a diseased part. Among the diseases in which compensatory emphysema occurs are pulmonary tuberculosis, lobar pneumonia, chronic tuberculosis with cirrhosis of the lung, and extensive disease of one lung.

A good example of compensatory emphysema is seen in pleurisy with effusion, where one pleural sac is nearly filled with fluid, and in pyopneumothorax. If the greater part of one lung is incapacitated by disease, as in lobar pneumonia, the remaining portions of the diseased organ and its fellow display general emphysema. It is, therefore, seen that compensatory emphysema, while probably a physiologic process, is nature's method of obtaining compensation for the loss of a portion of lung by any pathologic condition.

Hypertrophic Emphysema.

Remarks.—In this condition pathologic changes have resulted in a diminution in the retractility and elasticity of the lungs, as the result of overdistention of the individual air-cells, in consequence of which the lungs become permanently enlarged (air-cells expanded). In those persons who develop true emphysema early in life it is fair to presume, at least, that the retractile lung energy was deficient, possibly as the result of a congenital condition.

Pathologic Characteristics.—Macroscopically, large air-cells can be distinguished immediately beneath the pleuræ, and air-sacs as large as a walnut, and even larger, may project above the lung's surface, a series of air-blebs being commonly seen at the anterior border.

A microscopic study shows that the dilatation originates in the infundibular and alveolar passages. The septa are partially obliterated; the alveolar walls are thinned and finally perforated, and in consequence of these

changes the air-cells communicate with one another. The process is an atrophic one, the elastic fibers disappearing, whereas the larger ones become less conspicuous and often rupture. Following atrophic changes the capillaries disappear, and the epithelium of the air-cells undergoes fatty degeneration.

Ordinarily, the bronchial mucous membrane is the seat of a chronic inflammation. The diaphragm is appreciably lowered, and the liver and spleen are correspondingly depressed.

Physiologic Pathology.—The right side of the heart shows well-marked changes: the cavities are dilated and the walls slightly hypertrophied, owing to obstruction in the pulmonary circulation. The pulmonary artery and its branches are enlarged and the seat of an atheromatous degeneration.

Exciting and Predisposing Factors.—(1) This affection is most often a secondary one, developing during the course of other diseases of the lungs, *e. g.*, whooping-cough, chronic bronchitis, and asthma. When it develops under such conditions, emphysema is directly attributable to the mechanic influence and strain put upon the alveolar walls during the act of coughing. There is also interference with the escape of air from the air-cells and smaller bronchi. Many of these primary affections result in an increase in the intra-alveolar air-pressure, and, as a consequence, the cell itself becomes permanently expanded.

(2) In both whooping-cough and bronchial asthma the condition is at first that of a temporary emphysema, but numerous recurrences produce permanent overdistention of the lung.

(3) That *occupation* is not without influence is seen in the case of musicians who play wind instruments, and who, not infrequently, present a variable degree of emphysema. Violent muscular exercise also tends to produce permanent dilatation of the air-cells, consequently emphysema is common in stevedores, stokers, athletes, and those who do heavy lifting. The disease is one of the working classes, and *males* are more often affected than females.

(4) *Heredity* plays quite a prominent part in the etiology of this disease, and many members of the same family may suffer from emphysema; indeed, it is occasionally seen to extend through several generations, affecting one or more in each.

(5) *Age.*—After the age of fifty the elasticity of the lung tissue is diminished, and in consequence a variable degree of emphysema develops. At the other extreme of life, as previously mentioned, congenital emphysema may be present.

Organic heart disease and any other condition that causes a permanent congestion of the lungs markedly predispose to emphysema.

Principal Complaint.—Emphysema develops insidiously, and the patient is, as a rule, unaware of his actual condition, complaining of the symptoms of chronic bronchitis, asthma, or whatever other pulmonary disease was originally present. In those cases in which emphysema occurs as the result of occupation the condition develops slowly, but in whooping-cough the lung becomes emphysematous in the course of a few days. In the former class the history shows that the patient has suffered from a gradual loss of strength and of flesh for some years, and in many cases he is conscious of the deformity of his chest (Fig. 36).

The most marked symptom is *dyspnea*, together with paroxysmal *cough*, the severity of these symptoms varying in proportion to the degree of distention of the pulmonary air-cells. In this pulmonary condition mild

dyspnea is constant, but upon physical exertion it often attains an extreme degree.

Late during the disease the patient's speech is somewhat characteristic, and his sentences are interrupted. As the disease advances the respiratory symptoms become more and more distressing, until, at length, as the result of increased blood tension in the lung, cardiac symptoms develop.

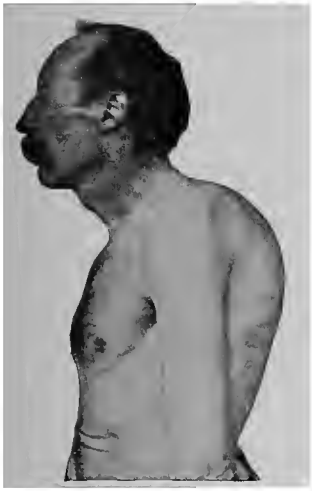


FIG. 36.—EMPHYSEMATOUS CHEST (Dr. W. H. Smith, Massachusetts General Hospital).

Cough is believed to be due to the presence of an *associated bronchitis*, and is particularly annoying during cold weather. Indeed, there is a type of emphysema that appears in young adults and affects them most during the winter months, when they display all the characteristic features of this disease.

Thermic Features.—Fever is absent throughout the entire course of uncomplicated cases of emphysema, and a subnormal temperature is by no means uncommon.

Physical Signs.—**Inspection.**—In advanced cases there is lividity of the skin and mucous surfaces. The contour of the chest is characteristic; it is often barrel-shaped, the anteroposterior diameter being markedly increased, whereas the transverse diameter remains nearly normal (Fig. 37). The sternum is often decidedly bulging, the neck short, the back arched, and the head tilted forward. The infraclavicular and supraclavicular regions are abnormally prominent, whereas the episternal notch is deepened. The clavicular and other accessory muscles of respiration stand out prominently, and this is responsible in part for the apparent shortening of the neck. The intercostal spaces are widened, and the ribs approach the horizontal plane more nearly than they do in health. It is not uncommon to find the external veins of the chest markedly dilated. The movements of the chest are vertical in direction, the chest moving en masse instead of displaying the normal expansile respiration, and during the act of inspiration there may be a retractile instead of an expansile movement of the base of the chest. Respiration may be more frequent than normal, although this is by no means an essential feature. The labored effort at respiration seeks to expel rather than to inspire air, whereas the rhythm is not only altered, but in extreme cases is actually reversed, inspiration being short, while expiration is greatly prolonged. (See Auscultation, p. 128.) In extreme cases the apex-beat of the heart is not discernible. Epigastric pulsation is quite common, and pulsation at the second or third interspace, in the midclavicular line, may also be observed in selected cases, while venous pulsation is commonly seen in the right carotid region.

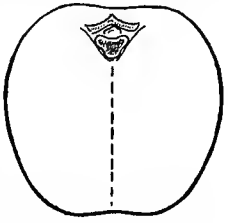


FIG. 37.—TRANSVERSE SECTION OF AN EMPHYSEMATOUS THORAX.

Palpation.—In addition to confirming the character and the degree of expansile movement of the chest, palpation reveals the fact that the tactile fremitus is markedly decreased. The apex-beat of the heart is always feeble, and in extreme cases it may be imperceptible. The *pulse*,

although normal at first, soon becomes weak and thready, but is not decidedly increased in frequency even late in the disease. As the result of overdistention of the right heart, which is caused by increased blood tension in the lung, a systolic shock is often detected in the region of the ensiform cartilage, and epigastric pulsation is the rule.

Percussion yields somewhat characteristic findings, since everywhere over the surface of the lung hyperresonance is elicited. The character of the hyperresonant note is controlled by the degree of expansion of the air-cells. Indeed, in extreme cases of emphysema the note may give off a wooden tone. The area over which a hyperresonant note is obtained is larger than normal, and this feature is particularly conspicuous in the clavicular regions. In advanced cases the area of cardiac dullness is greatly diminished, and the heart is often covered by emphysematous lung; in the latter case cardiac dullness is absent. The diaphragm having been depressed by the greatly distended lungs, the area of liver dullness is, as a rule, situated some distance below the normal level.

Auscultation.—The characteristics of the respiratory sounds of emphysema are that inspiration is short and feeble, whereas the expiratory murmur is appreciably lengthened, the normal ratio—inspiration, four; expiration, three—being disturbed—it may be one to one. Prolonged low-pitched expiration, accompanied by a wheezing sound, is probably the most valuable diagnostic sign of emphysema; at the same time the inspiratory murmur is often lower in pitch than the normal.

In exceptional cases the respiratory murmurs are harsh, owing to the extreme effort upon the part of the patient to expel air. As a rule, various râles are heard over practically the entire chest, and, indeed, the râles are not infrequently those heard in pulmonary asthma, chronic bronchitis, and pulmonary tuberculosis. A peculiar rubbing sound is occasionally detected, and is believed to result from friction of the large air-cells against the pleuræ. In the interlobular variety a crumbling sound is occasionally audible, and is of doubtful significance. Laennec called attention to the presence of a sound that closely resembled the subcrepitant râle (see p. 70), but this is by no means a constant finding.

Theoretically speaking, the voice-sounds should be poorly transmitted through the distended lung tissue; but as emphysema commonly develops as a secondary condition, the transmission of the voice-sounds is of but limited clinical value in certain cases.

In advanced cases of emphysema the right heart at first hypertrophies and later dilates; when the latter occurs, a tricuspid regurgitant murmur is audible at the ensiform cartilage. Early in the disease accentuation of the second pulmonic sound is not infrequently heard.

Laboratory Diagnosis.—The sputum is practically the same as that found in chronic bronchitis. (See p. 93.)

X-Ray Diagnosis.—In typical cases the most striking feature is the unusual degree of transparency that is universal through both lungs. (See p. 73.)

Illustrative Case.—E. C., male, aged twenty-four years; stevedore by occupation. The patient has suffered from repeated attacks of bronchitis and of bronchial asthma since early adult life. During the past ten years he has noticed that he suffers from extreme shortness of breath upon the slightest exertion, and that exertion also excites cough. He complains bitterly of violent attacks of coughing, and after moderate exertion dyspnea is so prominent a feature that speech is interrupted. The appetite has been good throughout his illness; in fact, his present condition developed so insidiously

that he is unable to fix the date on which he first experienced permanent difficulty in breathing. Moderate emaciation has occurred, but extreme weakness is not present.

Local Examination.—The characteristic barrel-shaped chest is seen, the neck appears to be short, and the supraclavicular and infraclavicular spaces are unduly prominent. There is distinct throbbing of the vessels at the right side of the neck, and marked pulsation occurs in the epigastrium, while the apex-beat of the heart is imperceptible. The lips and finger-tips are cyanosed.

Tactile fremitus is greatly decreased, even over the upper portions of the chest, and is very feeble at the bases of the lungs. The note everywhere is hyperresonant to percussion, whereas auscultation reveals a short, feeble inspiration and a prolonged expiration, subcrepitant râles being present, particularly over the bases posteriorly.

Summary of Diagnosis.—A history of the presence of conditions that markedly predispose to emphysema—*e. g.*, asthma, whooping-cough, and violent exercise—goes far to support a diagnosis. The typical barrel-shaped chest is positive evidence of the existence of emphysema, except in those cases in which it is associated with disease of the spine. The gradual development and prolonged duration of the condition and the typical symptoms and signs are most characteristic of emphysema.

Differential Diagnosis.—**Thoracic tumor** (aneurism) may produce a deformity of the chest that resembles in certain respects the deformity seen in emphysema. The distinguishing features, however, are that in aneurism the chest is not symmetrically enlarged, but an undue prominence occurs over some localized portion—most often the sternal or scapular regions. Again, aneurism gives a localized area of dullness, while surrounding this area, as the result of compensatory emphysema, a hyperresonant note is obtained. In aneurism there is likely to be a disturbance—an inequality—in the pulsations of the two radials, and a bruit, as well as a marked diastolic shock, may be present over the area of consolidation.

Pneumothorax may cause an enlargement of the chest that is not unlike that seen in emphysema. The clinical features that distinguish pneumothorax from emphysema are: (*a*) Pneumothorax develops acutely, and with lancinating pain on one side of the chest; (*b*) immediately following the pain a condition of shock ensues, from which the patient rallies in from two to twelve hours; (*c*) the coin test is obtained over the affected side, and breath-sounds, if present over this half of the chest, are metallic in character; (*d*) after sufficient time has elapsed for the effusion of fluid into the pleural sac to take place, the succussion splash is present; the temperature ranges between 101° and 104° F. and is septic in character. The signs and symptoms here outlined are unknown in emphysema.

Clinical Course.—In those cases of emphysema resulting from whooping-cough recovery occurs in from three to six months. That type of the disease seen during adult and after middle life assumes a chronic course, extending over a period of several years, and showing no tendency toward improvement. After the pulmonary condition has sufficiently interfered with the circulation through the lung, cardiac embarrassment becomes evident, and the clinical picture is that of emphysema plus the symptoms of cardiac disease.

SENILE EMPHYSEMA.

A condition in which, as the result of advanced years, the elasticity and retractility of the lung are diminished, and atrophy of the lung tissue has occurred—the so-called *small-lunged emphysema.* As a result of

the senile changes that have taken place in the wall of the alveolar cells, coalition of many of the air-cells occurs, with the production of rather large air-sacs.

In senile emphysema the volume of air in the lung is seldom above the normal, and, indeed, as a rule, the total volume of air is found to be diminished, a condition in striking contrast to that previously described under Hypertrophic Emphysema.

FUNGOID DISEASE OF THE LUNG.

Pulmonary Actinomycosis.

Pathologic Definition.—A chronic infectious disease, common in bovines, and occasionally seen to attack man. It is excited by the ray fungus (actinomyces), which develops in the pulmonary or pleural tissues, resulting in consolidation and possibly, later, in ulceration. (See Infectious Diseases, p. 920.)

Aspergillosis of the Lungs (Pseudotuberculosis).

Pathologic Definition.—A primary or a secondary mycotic disease of the lung caused by the Aspergillus fumigatus, and characterized pathologically by consolidation with cavity-formation. (See Infectious Diseases, p. 927.)

Pulmonary Blastomycosis.

Pathologic Definition.—A secondary disease caused by the blastomyces, and characterized pathologically by the condition known as pneumomycosis (pulmonary consolidation). (See Infectious Diseases, p. 923.)

Streptothricosis.

Pathologic Definition.—A disease caused by the streptothrix, and characterized pathologically by pulmonary consolidation, caseation, and cavity-formation, with a tendency toward metastatic involvement of other viscera and of the lymph-nodes. (See Infectious Diseases, p. 923.)

PARASITIC DISEASES OF THE LUNGS.

Echinococcic Disease of the Lungs.

Pathologic Definition.—A disease induced by infection with the dog tape-worm (Tænia echinococcus), and characterized pathologically by the presence of scolices, hooklets, and shreds of cyst membrane in the sputum. (See Parasitic Diseases, p. 986.)

Amebic Abscess.

Pathologic Definition.—This condition is caused by the Entamœba histolytica, and is usually secondary to amebic abscess of the liver. It is characterized pathologically by destruction of the pulmonary tissue with cavity-formation. (See Parasitic Diseases, p. 988.)

Endemic Hemoptysis.

Pathologic Definition.—A disease caused by infection of the lung by Paragonimus Westermanii. The parasites make small cavities in the

lung tissue, and here, in a peculiar exudate, deposit their ova, which eventually escape with the sputum. (See Parasitic Diseases, p. 988.)

DISEASES OF THE PLEURAE.

MOVEMENTS OF THE TWO HALVES OF THE CHEST.

This clinical method was introduced in 1913 by Boston and Ulman, who gave a preliminary report after the study of 50 cases.

A correlative study of the pneumograms from the two halves of the chest renders immediately apparent the fact that organic lesions of the lung and pleura and both liquid and air in the pleural cavity give unmistakable evidences through this method.

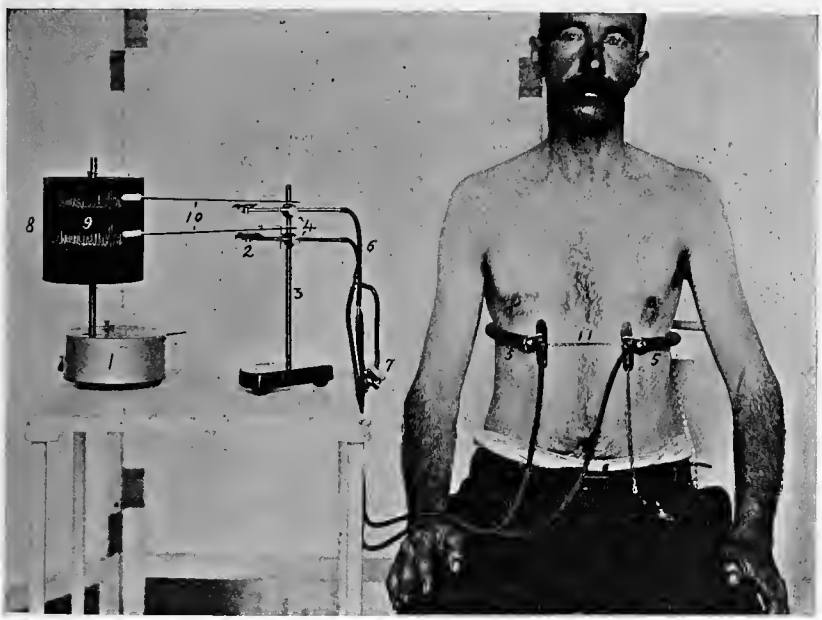


FIG. 38.—RECORDING MOVEMENTS OF THE TWO HALVES OF THE CHEST. INSTRUMENT IN OPERATION (Boston and Ulman).

Consideration.—Certain alterations in the pneumograms may result from either excessive or diminished muscular development, and also from unusual freedom of the respiratory movements; but these features do not, however, produce any difference in the writings of the two halves of the chest. This method of study has been found to be of inestimable value in all clinical forms of pleurisy, unilateral consolidations of the lung, pulmonary cavity, cardiac dilatation, cardiac hypertrophy, and diseases accompanied by increased abdominal tension. It likewise shows a distinct difference in the movements of the two halves of the chest where there is a unilateral loss in muscular tone, a feature best exemplified in hemiplegia.

Technic employed by one of us (Boston) in collaboration with Dr. Ulman:

The apparatus (Fig. 38) consists of (1) kymograph, (2) two Marcy tambours, (3) metal stand, (4) two clamps, and (5) two pneumographs (modified Ellis). The pneumograph consists of a rubber tube, 8 inches long, distended by a spiral wire spring. One end of the tube is closed, while the other end has an opening attachment to connect

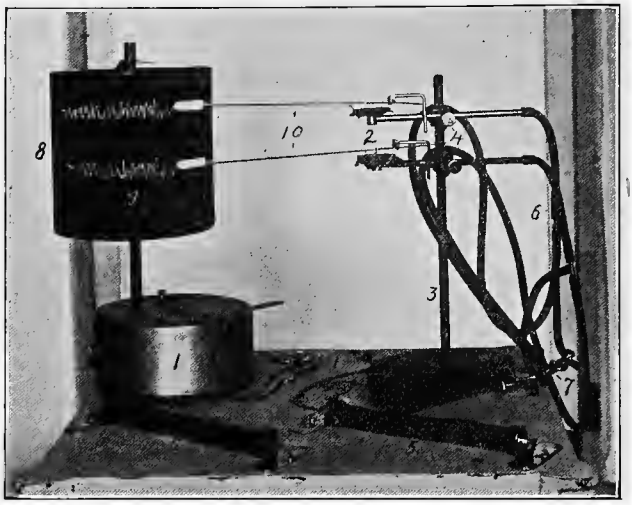


FIG. 39.—APPARATUS SHOWING SEPARATE PARTS (Boston and Ulman).
(*5*) Pneumographs, (*11*) connecting chains.

with rubber tubing (6) to the tambours. A bivalve (7) is interposed between each pneumograph and tambour to prevent rupture of the rubber membrane of the tambour.

Changes in the air pressure in the pneumograph is transmitted to the Marey tambours (2), which writes the respiratory movements upon the smoked paper on the drum of the kymograph (8), and produces the pneumograms (9).

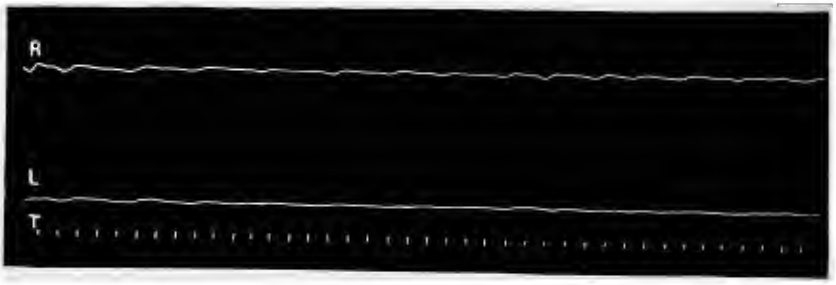


FIG. 40.—BILATERAL PNEUMOGRAM FROM A PATIENT SHOWING LARGE PLEURAL EFFUSION OF THE LEFT SIDE (Boston and Ulman).
T, Time indicator, rate 60 per minute; L, curve shows limited movements of the left half of the chest.

The pneumographs (5) are held in position upon the lateral parts of the chest by means of two small chains (11). One connects them across the back and the other across the front of the chest. Care must be taken not to have the chains too tight, as this will inhibit the respiratory movements of the chest. They are best applied on a level with the sixth ribs, so that the anterior part of the pneumographs, which contain

the opening for connections to the tambours, is situated about the nipple line. The distance separating the pneumographs over the back will vary greatly, depending upon the size of the chest.

The apparatus can be applied to the patient whether in the erect, sitting, or reclining posture. Have the small bivalves (7) open to prevent undue pressure on the tambours while adjusting the apparatus, preparatory to taking tracings. When ready to take

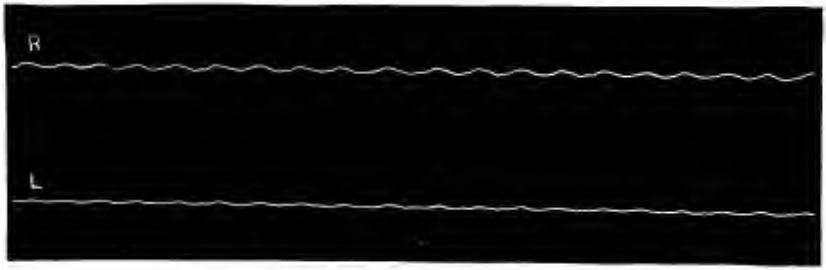


FIG. 41.—BILATERAL PNEUMOGRAM FROM A MALE AGED TWENTY-FOUR, SHOWING THE PHYSICAL SIGNS OF A LARGE TUBERCULOUS CAVITY NEAR THE APEX OF THE LEFT LUNG (Boston and Ulman).
There were also present evidences of an old pleurisy of the left side. L, Curve shows lessened expansion of the left half of the chest.

the tracings these bivalves are to be closed. White glazed paper, 6 inches wide, is placed on the drum (8) of the kymograph and smoked evenly, though not too heavily, by the flame from a coal-oil lamp or a gas burner.

Be careful to bring the writing points of the two levers (10) of the tambours in the same vertical line, and with just sufficient pressure against the smoked paper, on the drum, to prevent binding. The distance between the two levers is not constant, but depends upon what type of tracing you desire to take. Usually from $1\frac{1}{4}$ to 2 inches apart

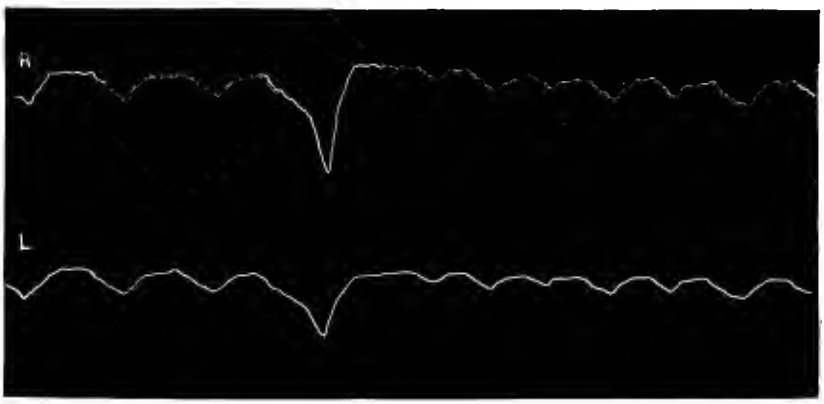


FIG. 42.—BILATERAL PNEUMOGRAM FROM A CASE OF RIGHT HEMIPLEGIA (Boston and Ulman).
Note especially the marked irregularity in curve R; probably dependent upon lack of muscular tone of the paralyzed side. The extreme downward amplitude of the curves result from forced inspiration, curve R descending much further than does curve L, a feature probably also due to diminished muscular tone of the right half of the chest.

will suffice. The tension of the rubber membranes of the tambours (2) must be equal. Should the patient cough (Case V), yawn, sneeze, or laugh during the taking of the record, these acts cause undue amplitude in the curves of the pneumogram. Figure 39 shows the separate parts of the apparatus.

The time-marker can be placed at the base of the drum and this record may be made at the time the respiratory movements are recorded (Fig. 40). The time record may be

taken after the pneumogram, but in such cases care must be taken that the speed of the revolving drum is the same as it was when the pneumogram was made.

The degree of pressure within the pneumographs is increased by inspiration (causing the downward curve of the pneumogram), while expiration lessens this pressure and corresponds to the upward curve of the pneumogram.

The accompanying bilateral pneumograms will serve to show how disease causes variations in time and amplitude of the writing of one

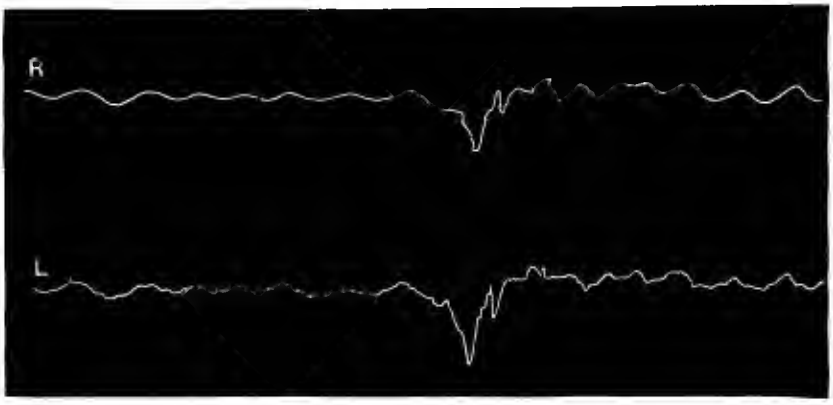


FIG. 43.—BILATERAL PNEUMOGRAM FROM A CASE OF EXTENSIVE CHRONIC PLEURISY OF THE LEFT SIDE; ALSO SMALL PULMONARY CAVITY AT THE LEFT APEX (Boston and Ulman).
Extreme downward amplitude of the curves resulted from the patient coughing.

side of the chest. The upper tracing of the bilateral pneumogram represents the movement of the right half of the chest, while the lower tracing is produced by the movements of the left side of the chest.

In certain forms of pleurisy, pleural effusion, and pneumonic consolidation the movements of the affected side are greatly diminished, as com-

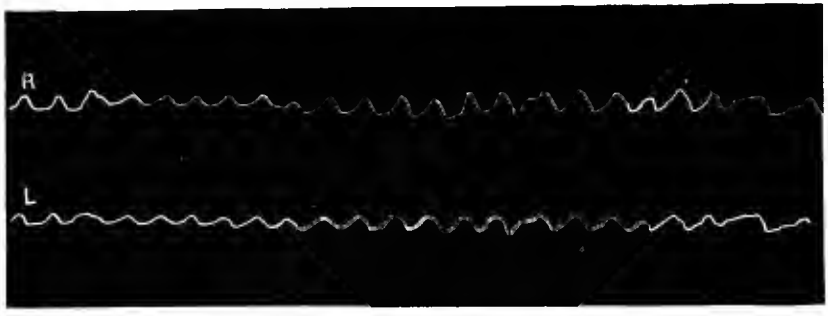


FIG. 44.—BILATERAL PNEUMOGRAM FROM A CASE OF EXTREME DYSPNEA—RESPIRATIONS 60 PER MINUTE (Boston and Ulman).
In extreme dyspnea the two curves are likely to show such differences.

pared with those of the unaffected side (Figs. 40 and 41). Again, the movements may be widely different either at the upper portion or at the base of the chest, such variations depending upon the character and location of the lesion present. Unusual amplitude of both the right and left

curves are rather characteristic of fluid in the abdominal cavity. The pneumograms are also affected by pneumonia, pulmonary cavity, chronic pleurisy, hemiplegia, and condition accompanied by dyspnea. (See Figs. 42, 43, and 44; also Mitral Regurgitation, Serofibrinous Pleurisy, Chronic Nephritis (Exudative), and Cheyne-Stokes Respiration.)

HYDROTHORAX (DROPSY OF THE PLEURAE).

Pathologic Definition.—A secondary condition in which there is an accumulation of transudate in one or both pleural sacs, without the existence of inflammatory changes in the pleuræ. Usually the condition is bilateral.

Exciting and Predisposing Factors.—Hydrothorax is in reality not a disease, but merely a symptom of a pathologic change that is remotely situated; nevertheless it is necessary to describe the clinical features of this symptom.

Varieties and Causes.—(1) **Hemorrhagic Hydrothorax.**—Under this heading are considered those conditions in which, as a result of impoverishment of the blood, a blood-stained transudate accumulates in the pleura; among these are leukemia, pernicious anemia, amebic dysentery, malignant disease, malaria, scurvy, chronic suppuration, and syphilis.

(2) **Local pathologic changes** may also give rise to the development of hydrothorax, and most of the unilateral cases belong to this class. Among the local exciting causes are: pressure upon the superior vena cava, pressure upon the thoracic duct, enlargement of the heart (dilated right auricle), thoracic aneurism, enlarged mediastinal glands, and carcinoma of the pleuræ.

(3) **Renal Changes.**—Renal disease is commonly concerned in the production of bilateral hydrothorax, and it will readily be understood that here there are two conditions that favor the accumulation of fluid within the pleura: (*a*) Increased work upon the part of the heart; and (*b*) impoverishment of the circulating blood. In hydrothorax of renal origin the diagnosis is confirmed either from a history of Bright's disease or from the laboratory diagnosis.

(4) **Cardiac disease** is a frequent cause of bilateral hydrothorax, and in those cases in which there is cardiac enlargement, which in turn exerts pressure upon the thoracic vessels, unilateral hydrothorax may result. Cardiac hydrothorax is recognized by the detection of organic disease of the heart.

Principal Complaint.—This is usually dependent on the preexisting disease, of which hydrothorax is but an additional symptom. After the fluid has accumulated in the pleuræ, there generally occurs a variable degree of aggravation of the original symptoms of the preëxisting disease, and, in addition, the patient complains of other symptoms certain of which are more or less characteristic of hydrothorax: Dyspnea becomes more and more marked, depending upon the quantity of fluid present in the pleural sacs, and if severe, cyanosis is present; paroxysmal coughing and asthmatic seizures are common, and symptoms referable to enfeebled circulation are apparent—*e. g.*, coldness of the extremities.

Physical Signs.—In bilateral hydrothorax (Figs. 45, 46) the physical signs are identical with those seen in pleurisy with effusion (*q. v.*), unless there is a thickened pleura, when a variable degree of dullness is found by percussion above the level of the fluid. It is important that the examiner keep in mind the fact that bilateral pleural effusions are uncommon, whereas

bilateral hydrothorax is the general rule. The history of the patient will often enable one to interpret correctly the physical signs obtained in a case of hydrothorax.

Previous Attacks.—One attack materially predisposes to subsequent seizures.

Clinical Course.—This depends upon the exciting factors, the length of time a transudate has remained in the pleural sacs, and whether or not it is possible to institute judicious treatment. In those cases due to cardiac and renal disease a prognosis of the preëxisting disease is readily made, but when hydrothorax is the result of hemic degeneration, selected cases may yield to treatment.

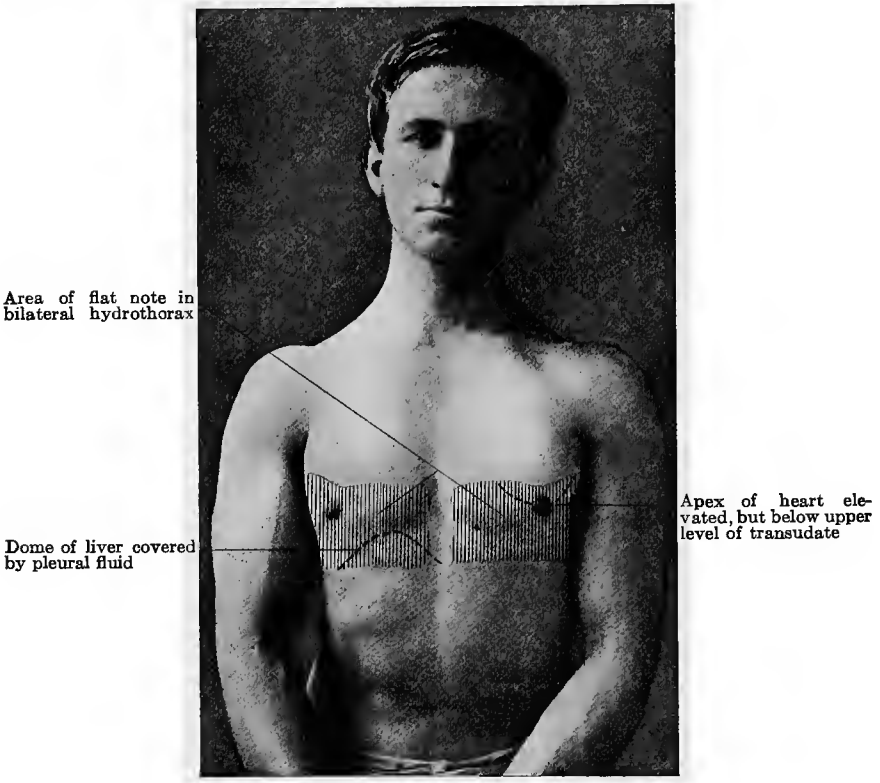


FIG. 45.—BILATERAL HYDROTHORAX.

PLEURISY (PLEURITIS).

Pathologic Definition.—A disease characterized by the presence of either a local or a general inflammatory process of the pleuræ. (See Pathology of Special Varieties.

Varieties.—These are: Acute plastic pleurisy, serofibrinous pleurisy (pleurisy with effusion), purulent pleurisy (empyema), tuberculous pleurisy, subacute pleurisy, chronic adhesive pleurisy, diaphragmatic pleurisy, encysted pleurisy, intralobular pleurisy, cancerous pleurisy, and hemorrhagic pleurisy.

The **predisposing and exciting factors,** as well as the clinical picture of each of the subclasses, will be described at length under their respective headings.

Acute Plastic Pleurisy (Dry Fibrinous Pleurisy).

Pathologic Definition.—An acute inflammation of the pleuræ, characterized by congestion and the formation of a fibrinous exudate that covers the affected surface of the pleura. The pleura loses its normal luster, and the area involved is devoid of the normal glistening surface. Minute ecchymoses may be seen, and as the exudate accumulates upon the pleura a shaggy, roughened appearance results. Owing to the friction induced by the rubbing of the two layers of the pleura the exudate at times becomes greatly thickened, the involved surface of the pleura presenting a yellowish

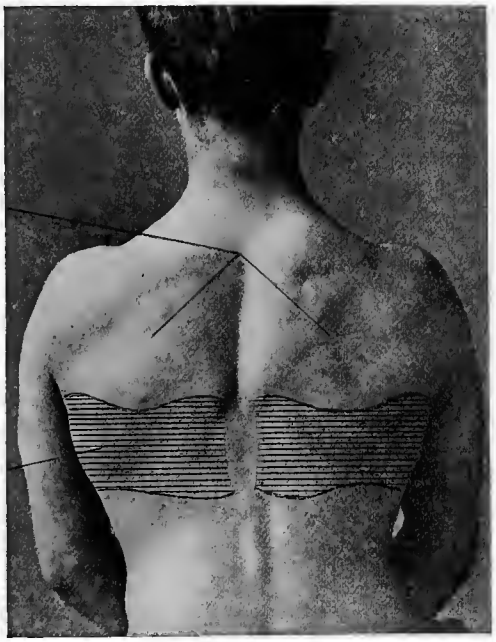


Fig. 46.—Bilateral Hydrothorax.

or reddish-gray appearance. The disease may advance to the formation of pleural adhesions; in mild cases, however, they are absent, and the products of the exudate undergo fatty degeneration and are later absorbed.

Varieties.—(1) **Primary plastic pleurisy** is said to occur when, prior to the onset of the pleuritic condition, the patient enjoyed health. Aschoff, in a careful study of 200 cases of acute plastic pleurisy, found but 41 cases in which the disease developed in previously healthy individuals.

(2) **Secondary plastic pleurisy** results from the extension of either an acute or a chronic inflammatory process to the pleura—*e. g.*, the associated pleurisy of lobar pneumonia.

PRIMARY PLASTIC PLEURISY.

Exciting and Predisposing Factors.—Bacteria.—In both acute and chronic pleurisy the direct cause is a pathogenic micro-organism or the chemic product of such organism. Among the bacteria commonly concerned in the production of plastic pleurisy should be mentioned the Bacillus tuberculosis, Streptococcus pyogenes, Staphylococcus pyogenes albus, Staphylococcus pyogenes aureus, pneumococcus, colon bacillus, and the Bacillus typhosus. These organisms may also be present after suppuration has taken place in the pleura, and any one of them may be the exciting cause of acute pleurisy. Other bacteria may enter the pleural cavity late in the course of dry pleurisy, or after a serous exudate has accumulated in the pleural sac. The ray fungus has been recovered from the pleura. Animal parasites—*e. g.*, Entamœba histolytica—have been known to cause acute inflammation of the pleura.

Predisposing Factors.—Primary pleurisy not infrequently follows undue exposure to cold and wet.

Sex.—The disease is slightly more common in men than in women, and is frequently seen to follow injury to the thorax.

Age figures prominently as a predisposing factor, the majority of cases developing during early adult and middle life.

Season is not without influence, the winter and spring months providing the greatest number of cases.

Rheumatism.—Those afflicted with a rheumatic or gouty diathesis are most likely to be attacked, and persons showing a predisposition to the development of pulmonary disease, as, for example, tuberculosis, frequently develop acute pleurisy as the initial manifestation of infection. Every case of so-called idiopathic pleurisy should be studied carefully, having in mind that the great majority of such cases are either tuberculous or rheumatic in origin.

SECONDARY PLASTIC PLEURISY.

General Remarks.—Secondary plastic pleurisy results from direct extension of the inflammation, hence it is associated with pulmonary abscess, pulmonary gangrene, superficial pulmonary cavity, lobar pneumonia, bronchopneumonia, hemorrhagic infarct, and hepatic, diaphragmatic, or mediastinal abscess. Plastic pleurisy may develop as a complication during the course of acute rheumatism. Conditions characterized by malnutrition—*e. g.*, chronic nephritis—are also likely to develop acute pleurisy as a complication. The pleura may be attacked secondarily by a similar inflammatory process involving other serous membranes—*e. g.*, synovial sacs, the meninges, or the pericardium.

Principal Complaint.—As a rule, the symptoms are well marked, although there are exceptional cases in which they are so mild as to be disregarded by the patient.

Pain Referable to the Chest.—The patient is suddenly seized with a sharp, stitch-like pain, generally near the nipple. This pain is increased on deep inspiration, and on movement of the arm of the affected side.

Cough is troublesome, and is accompanied by a lancinating pain.

Among the *general complaints* is that of chill or a series of chilly sensations, which may have been slight or so severe as to constitute a distinct rigor. The patient declares that he was feverish at the onset, and that he sweat profusely.

The *appetite* is poor, thirst somewhat increased, and constipation obstinate. Headache, while by no means constant, may be an annoying symptom.

Thermic Features.—In the average case the temperature ranges between 100° and 103° F., whereas in severe types of infection it may reach 104° or even 106° F.

Nervous Symptoms.—Mild cases may run their course without developing any nervous manifestations other than those that have been previously mentioned.

Physical Signs.—Inspection.—*Attitude.*—In sitting, the patient inclines toward the affected side, his arm being clasped tightly against the chest. He may also stoop slightly forward, and there is drooping of the shoulder. Within a few hours after the initial pain the face becomes flushed. The movements of the chest are limited, the respirations are rapid and jerky in character, but on the affected side there is an absence of movement.

The *tongue* becomes coated within the first thirty-six hours, and the lips are dry.

Palpation.—Palpation confirms inspection with reference to the limited movements of the chest. A friction fremitus can be felt in a small percentage of cases.

Percussion.—Throughout the entire stage of acute plastic pleurisy the percussion-note is normal over both the affected and the healthy side. The exception to this rule is that, if the plastic exudate is extensive, moderate impairment of resonance will be detected. Percussion frequently is quite painful, however. After a patient has suffered from repeated attacks of acute pleurisy, the pleura may be appreciably affected, and impairment of the ordinary resonance obtained by percussion will follow.

Auscultation.—A friction murmur is heard over a limited area on the affected side of the chest. This murmur is audible both by the direct and by the indirect method of auscultation. It consists of a peculiar grazing or grating sound, which is most distinct at the end of inspiration. The point of greatest intensity of the friction murmur depends entirely upon the area of pleura involved, and the murmur may be detected over any portion of the lung. With the appearance of a fibrinous exudate upon the pleura the friction-sound becomes somewhat less distinct and appears to be masked by numerous, fine, crackling râles that are heard during both inspiration and expiration. After the fibrinous exudate has accumulated a distinct friction-rub is usually audible when the patient is directed to inspire deeply. If the plastic exudate is extensive, as rarely occurs, the lung is somewhat compressed, and the breath-sounds become somewhat bronchial in character, thus making the differential diagnosis between adhesive pleurisy and pneumonia rather difficult.

Laboratory Diagnosis.—In those cases that display high fever, the urine is lessened in quantity and may contain a trace of albumin. In severe inflammation of the pleura the number of leukocytes will be above the normal.

Illustrative Case of Pleurisy.—James D., a cigar-maker, aged twenty-seven years. Height, 5 feet 10¼ inches; weight, 152 pounds.

Family History.—Parents and three younger sisters living, one of whom suffered from an attack of pleurisy at the age of seventeen years, and now, at the age of nineteen, her physician has directed her to live in a mountainous district. She is reported as having a severe cough, and although there is no positive evidence that she is afflicted with tuberculosis, yet the patient's description of her condition suggests strongly the existence of this disease.

Previous History.—Had measles at the age of nine, but does not recall having had

the other diseases of childhood. At the age of twenty-one he suffered from an attack of pneumonia, and states that he was confined to bed for a period of six weeks and was unable to return to his work for several months. Before this attack he was unusually robust, but since he has never regained his original weight. For the past three years he has suffered from periodic attacks of tonsillitis.

Social History.—The patient is married, and has two children living, both apparently healthy. He is a mechanic, employed in a cutlery factory, and the room in which he works is not well ventilated; there is also a large amount of dust.

Present Illness.—Two days before consulting a physician he was seized with what he believed to be a severe cold, and when seen soon after, he stated that during the night he had had repeated attacks of chilliness, following which he felt feverish. Anorexia and constipation were also present.

There was some cough the evening before definite symptoms developed, and during the night the cough became more severe, harsh, short, and was not accompanied by expectoration. The cough further caused intense lancinating pain in the region of the left nipple; this symptom became so severe that the patient was compelled to restrain his coughing in order to obviate the pain. Headache was present, and there was also some soreness of the muscles of the back and limbs, although the patient's chief complaint was of pain in the chest. Both pain and cough were relieved when the patient assumed a certain position—*i. e.*, when he inclined toward the affected side and held the chest firmly with the hand. Relief also followed strapping of the left side of the chest. By the end of the first week the pain had subsided markedly, and by the third week it suddenly disappeared. (See Serofibrinous Pleurisy, p. 142.)

Soon after the initial symptoms appeared fever developed, and was of an irregular type, fluctuating between 99° and 101.4° F. for a period of two weeks, when it gradually fell to near the normal.

Physical Examination.—*General.*—When first seen, the patient was sitting with his body inclined well toward the affected side, and grasping the left side of the chest with the hand. The skin was bathed in perspiration.

Local Examination.—Movements of the chest were much diminished and the affected half appeared almost motionless. The head was inclined to one side and speech was interrupted. Slight movement of the body was followed by an expression of pain. The mucous surface of the throat was reddened. The face was flushed, the conjunctivæ reddened, and the expression was that of pain.

Palpation.—Tactile fremitus was decreased over the base of the left lung in the anterior axillary region. The movements of the same side of the chest were decidedly restricted.

Percussion.—There was a moderate degree of impairment of the percussion-note over an area about two to three inches in diameter, and the center of this area corresponded to the point where the friction murmur was heard with greatest intensity.

Auscultation.—During the acts of respiration a distinct to-and-fro, harsh, respiratory (friction) murmur was audible over a small area immediately below and outside the left nipple. Few crackling râles were also heard over this area. By the third day of the illness the friction murmur had disappeared, but râles were still audible.

Laboratory Findings.—The urine was slightly decreased in quantity, from 20 to 35 ounces being usually voided during the twenty-four hours; it was high colored, but contained neither albumin nor sugar, and a microscopic examination was negative.

Diagnosis by Induction from Clinical Data.—The history of suspected tuberculosis in another member of the family and the previous attack of pneumonia, following which he never recovered his usual weight, were factors that gave rise to the suspicion that the pleural condition was tuberculous in nature. Occupation was also regarded as a possible predisposing factor, and the fact that the disease began as a severe cold, during the course of which there was chilliness, followed by fever, cough, and a lancinating pain in the region of the nipple, was considered highly suggestive of pleurisy. Other evidences of the existence of acute pleurisy were that pain was intensified by deep respiration, and that the patient inclined toward the affected side and held his hand firmly over the site of the pain. The fact that the pain disappeared suddenly further supported the original view that the patient had been suffering from *acute plastic pleurisy*, and at this time effusion was probably accumulating in the pleural sac. Fever, which was not extremely high at any time, was decidedly irregular, a fact that strongly suggested the existence of pleurisy. Among the physical signs detected during the early stage of the illness and considered pathognomonic was a to-and-fro friction murmur, synchronous with respiration.

Differential Diagnosis.—At the onset the pain suggested the possible existence of intercostal neuralgia, from which the existing condition was differentiated by the following facts: (*a*) There was no distinct soreness upon palpation over the affected

side of the chest; (*b*) pain was not distributed along the course of the intercostal nerves; and (*c*) a pleural friction murmur was present. (See Differential Table, p. 151.)

Course of the Disease.—At the end of the first week the fever had fallen considerably, registering 100° F. during the evening hours. Pain had disappeared and the appetite was somewhat improved. During the second week the patient was permitted to sit up in bed and was allowed dry foods—an attempt being made to restrict the taking of liquids. Three weeks later dyspnea developed quite acutely, and within forty-eight hours following its appearance the physical signs of effusion into the left pleura were present. Fluid continued to accumulate in the pleura until its upper level was found at the superior border of the third rib anteriorly, and the fluid remained at this level for two weeks, when it was deemed advisable to remove at least a portion of it by aspiration. Following the removal of about twenty ounces of serous fluid (see Serofibrinous Pleurisy), the patient's general condition continued to improve until the seventh week, when he was permitted to leave the house.

Summary of Diagnosis.—A history of exposure to cold and wet, or of a tendency toward the development of pulmonary disorders, is of great value in formulating a diagnosis of acute pleurisy. The occurrence of a chill, followed by moderate fever, and acute lancinating pain in the chest are among the most valuable symptoms detailed by the patient. A moderate increase in the frequency of the pulse, together with immobility of the chest, further strengthens the diagnosis. The one positive sign of acute pleurisy, however, is the occurrence of a friction murmur, which develops early and remains, though slightly modified, until an effusion is poured into the pleural sacs.

Average Duration.—The milder types of the disease tend toward a favorable termination in from four days to three weeks. Severe types—*e. g.*, those ushered in by a rigor and high fever—may terminate fatally. After repeated attacks there is a tendency for the pleuræ to become markedly thickened and for adhesions to form. In the latter event the patient may suffer from pleuritic pains for an indefinite period. Acute plastic pleurisy occurring during either an acute or a chronic disease shows less tendency to terminate favorably than when it attacks those in apparent health. Acute pleurisy developing during the course of pulmonary tuberculosis assumes a protracted course. It is to be remembered that in a large percentage of all cases an accumulation of serum in the pleural sacs (serofibrinous pleurisy) takes place.

Serofibrinous Pleurisy (Pleurisy with Effusion; Subacute Pleurisy).

This condition is merely the second stage of an acute pleurisy, in which a serous or a serofibrinous exudate has escaped into the pleural sac. Serofibrinous pleurisy, like acute pleurisy, may be either primary or secondary in nature.

Varieties.—Among the special varieties encountered are encysted pleurisy, partial pleurisy, and encapsulated pleurisy.

Predisposing and Exciting Factors.—The etiology of the disease is the same as that of acute plastic pleurisy (*q. v.*), since, as previously stated, serofibrinous pleurisy is but a second stage of the disease in its severer forms.

Infection with the tubercle bacillus is said to be the exciting factor in 75 per cent. of cases, and the general belief is that the tubercle bacillus attacks primarily the pleura; this subject, however, is still unsettled. That tubercle bacilli invade the pleuræ secondarily to a similar involvement of the lung cannot be doubted.

Rheumatism.—An accumulation of fluid in the pleural sac not unusually occurs as a complication of acute articular rheumatism. A similar condition may follow typhoid fever, scarlet fever, and epidemic meningitis. In lobar pneumonia the pleura of the affected side may be the seat of a serous effusion.

Bacterial Infection.—A bacteriologic study of the fluid obtained from the pleuræ is often negative, although many instances have been reported in which different bacteria have been recovered. A most satisfactory method of ascertaining which pathogenic organism is present is to inoculate an animal with a portion of the fluid; this method is almost essential in order to detect tubercle bacilli in the pleural exudate.

Principal Complaint.—The history and symptomatology are the same as those previously outlined under Dry Pleurisy. (See p. 139.) After the patient has suffered from an acute lancinating pain in the side for several days, the character of the pain becomes gradually altered.

Pain.—After a copious effusion has been emptied into the pleural sac, the pain becomes of a dragging or tearing character. Its intensity is not dependent on the quantity of fluid that is present, for not infrequently we find a pleura nearly filled with fluid where a moderate amount of pain is present. When the pleura is well filled, the pain ceases to be localized, and may be absent; more or less soreness, however, is always present over the entire half of the chest. Pain along the margin of the ribs or in the midsternal region may be distressing, and is likely to mislead the physician. In those cases in which a copious pleural effusion is present and pain is not a prominent symptom, it may be excited by directing the patient to cough, to bend from side to side, or to inspire deeply. To avoid error we repeat that the acute pain of dry pleurisy diminishes with the appearance of the effusion, but that the pain may be continuous even though the pleura is well filled with fluid; conversely, this symptom is seldom, if ever, absent throughout the entire course of the serofibrinous stage of pleurisy.

Cough may continue from the dry stage throughout the greater part of the course of an attack of serofibrinous pleurisy. The character of the cough changes as the effusion accumulates, and the harsh cough, which may have been accompanied by slight expectoration, now becomes less racking and expectoration is more free. When serofibrinous pleurisy terminates in recovery, expectoration is more profuse during the stage of absorption, and the cough, which may continue for some weeks, is either dependent upon an associated catarrhal bronchitis or upon the irritation resulting from the reëxpansion of the lung.

Dyspnea, as previously stated under Dry Pleurisy, is present, and the respirations are shallow and jerking or irregular. Inspiration is often made up of a series of short inspiratory efforts, and the act may be interrupted at any time. Whenever there is a copious effusion in one pleura, or when both pleural sacs are half filled with fluid, dyspnea becomes pronounced, and the patient may be unable to rest in the recumbent posture; cyanosis is usually well marked. In those cases in which the effusion has accumulated rapidly dyspnea is more intense than in those in which a much longer time was consumed in collecting an equally large quantity of fluid. It is not uncommon to find a patient with one pleural cavity two-thirds filled, the fluid having accumulated slowly, display little evidence of embarrassed respiration.

Gastro-intestinal Symptoms.—*Anorexia*, while mild during the dry stage of pleurisy, becomes well marked whenever the amount of effusion is large. *Nausea and vomiting* may occur at any time during the disease, and *constipation* is an almost constant symptom during all stages of pleurisy.

Thermic Features.—Fever is present throughout the greater portion of an attack of serofibrinous pleurisy, the temperature ranging between 100° and 103° F. Near the end of the second or third week there is generally an appreciable decline in the temperature, and by the fourth week it has often reached the normal. Not infrequently, in the more severe cases, a continued type of fever (101° to 104° F.) runs through the second and third weeks of the disease. On the other hand, there are mild types of pleurisy in which the temperature never exceeds 101° F. A hectic temperature, with evening exacerbations and morning remissions, is suggestive of infection of the pleural fluid with some pus-producing organism, but it is to be remembered that this is by no means a positive sign. The axillary temperature of the affected side may be from one-half to two degrees higher than that of the opposite side, but this peculiarity of the temperature is not a constant finding, and its clinical significance is questionable.

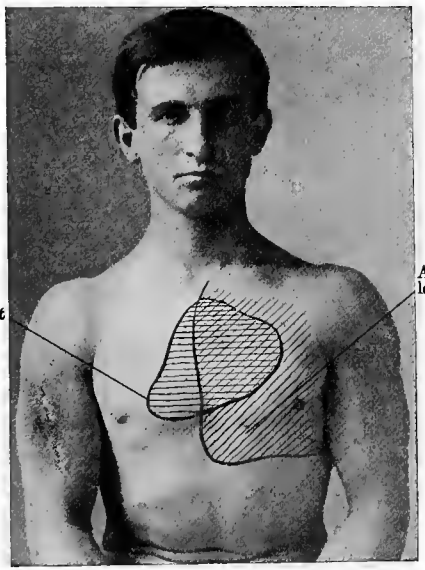


FIG. 47.—LARGE PLEURAL EXUDATE ILLUSTRATIVE OF DISPLACEMENT OF THE HEART.

Cardiac Symptoms.—These form so prominent a group of symptoms occurring during the course of serofibrinous pleurisy that they have been considered under a separate heading, and out of the regular order employed for their description. As soon as fluid begins to accumulate in the pleura the heart-beats increase in frequency, and as the accumulation proceeds the pulse-rate may show a corresponding increase, ranging between 100 and 130 beats a minute. If the accumulation of fluid is large and the heart is greatly

displaced to one or the other side of the chest, the pulse becomes irregular both in rhythm and in volume. There is some controversy as to whether this irregularity in the pulse is the result of pressure of a pleural effusion upon the heart or of pressure upon the great vessels in the thorax. In the judgment of certain observers pressure upon both the heart and the vessels is responsible for this irregularity of the pulse.

Owing to embarrassment of the heart and to torsion of the great vessels at the base of the organ (large left pleural effusion, Fig. 47), cyanosis develops and often becomes extreme. Dyspnea, as previously mentioned, may result in part from embarrassed circulation following pressure upon the heart.

Physical Signs.—These are directly dependent upon the quantity of exudate present in one or in both pleural sacs. The three factors that figure most prominently among the physical signs of serofibrinous pleurisy are: (*a*) The signs present when the effusion is at its height; (*b*) displacement of thoracic and abdominal viscera; and (*c*) the signs present during the stage of absorption.

Stage of Effusion.—*Inspection.*—If one pleural sac is only partially filled, say to the fourth rib, inspection reveals but slight bulging at the base of the chest on the affected side, and, indeed, in muscular and obese individuals no alteration in the contour of the chest may be perceptible. If one pleural sac is filled to the third rib, a distinct bulging of the affected side is apparent, and the chest movements over such a large effusion are limited, and often confined entirely to the apex. The abdominal type of respiration becomes more and more prominent as the quantity of fluid in the pleural sacs increases, and where the effusion is large, the respirations are rapid and often shallow (Fig. 48). There may be but little difference apparent in

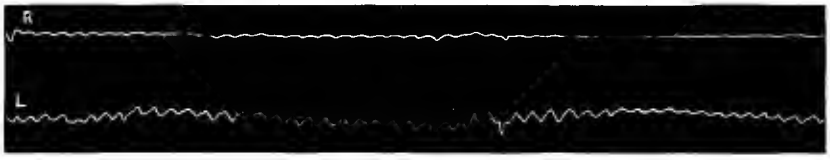


FIG. 48.—BILATERAL PNEUMOGRAM FROM A CASE OF RIGHT PLEURAL EFFUSION.
Respirations, 48 per minute. Curve R represents movements of the right half of the chest. Curve L represents movements of the left half of the chest. Note irregularity in the general course of curve L. Case studied at the Philadelphia General Hospital. (See Bilateral Movements of Chest, p. 131.)

the two sides of the chest, even though one pleura is practically filled with fluid, this condition being due to compensatory emphysema of the lung of the unaffected side.

The apex-beat of the heart is always displaced when a large pleural effusion is present. If the effusion occupies the left pleura, the heart's impulse may be seen to the right of the median line, and in extreme cases it may be observed at the fourth and fifth interspace, in the right axillary region. In right-sided effusion the heart is displaced to the left. Whenever there is but a moderate effusion in the left pleura (the sac being half filled), the apex-beat of the heart is elevated, and a distinct pulsation may be seen about the third or fourth interspace inside the left nipple-line.

Absence of the apex-beat may be dependent upon the fact that the apex of the heart is lodged behind the sternum (Fig. 47), the result of pressure

from a left-sided pleural effusion; it may not, however, indicate the position of the apex of the heart, since, because of cardiac embarrassment, there is frequently undue pulsation of the right auricle.

On inspection of the base of the chest anteriorly there is noticed undue fullness at the margin of the ribs on the affected side; the epigastrium is also seen to be prominent. In the case of a large pleural effusion the prominence of the epigastrium may extend from the affected side beyond the median line. In thin subjects it is often possible to observe the outline of the lower margin of the liver when it is decidedly displaced by a large right-sided effusion.

Mensuration.—When a large unilateral effusion is present, the measurements of the affected side of the chest are increased, and its contour is altered most at its base. Inequality in the measurements of the two sides of the chest may be found during health, and in right-handed individuals the right side is slightly larger than the left; consequently, a moderate effusion into the left pleura of such an individual would not be detected by mensuration. The degree of expansion of the healthy side during the act of inspiration gives positive evidence of the existence of pleural effusion, since expansion is absent at the base of the chest on the affected side.

The horizontal measurements of the chest are also altered by fluid in one pleural sac, the distance from the clavicle to the margin of the ribs being greater on the affected side.

Palpation.—By pressing the two hands upon the chest and directing the patient to inspire deeply, the limited range of expansion of both sides of the chest may readily be appreciated. The chest-wall over a large pleural effusion is practically fixed, whereas the opposite side is observed to move more rapidly than in health. The intercostal spaces are found to be prominent over a large effusion. Rarely, indeed, the tissue of the chest-wall may pit upon pressure, and fluctuation is said to occur.

Tactile fremitus is absent over an effusion, the exceptions to this rule being in the case of an infant—*e. g.*, a child crying—and when pleural adhesions were present prior to the accumulation of the fluid, such adhesions still anchoring the compressed lung to the chest-wall. Absence of tactile fremitus is of less clinical significance in women than it is in men, and, owing to the character of the female voice, these patients should be instructed to pronounce distinctly and to assume a masculine tone of voice during chest examinations.

The impulse of the apex-beat of the heart is always displaced in large pleural effusions, and where such displacement is sufficiently great to embarrass the circulation, pulsation of the right auricle may be palpable. Pulsation at the right side of the neck and in the sternal notch may also be detected. When pushed down by a pleural effusion, the liver is felt below the costal margin (Fig. 50), and the spleen is likewise displaced downward when the left sac is well filled. Whenever either the spleen or the liver is palpable below the costal margin, it is necessary to determine, by both percussion and auscultatory percussion, whether or not the viscus in question is actually enlarged.

Percussion.—As soon as the effusion begins to accumulate in the pleura the percussion-note is impaired beneath the angle of the scapula, and with the increase in the quantity of fluid exuded, this impairment changes to dullness, and eventually to flatness, which is present over the entire base of the chest upon the affected side when the patient is standing or sitting.

When the pleura is more than half filled with fluid, the area of flatness extends across the median line (paravertebral area of dullness), giving a peculiar arched-like line of flatness, extending from the upper level of the fluid to the base of the pleura, and for from one and one-half to two and one-half inches beyond the median line (Fig. 49). The paravertebral angle is altered by the position of the patient (Fig. 49).

Second in importance is the sensation offered to the pleximeter finger, such resistance being augmented over a large effusion and where a flat note is obtained. The note obtained over the fluid is always flat, and there is a variable degree of impairment for some distance above the fluid if the pleura is half filled. One of the characteristics of pleural fluid is the change of level of the fluid (Fig. 49) with the change in position of the patient. In extensive pleural adhesions movable dullness is not present.

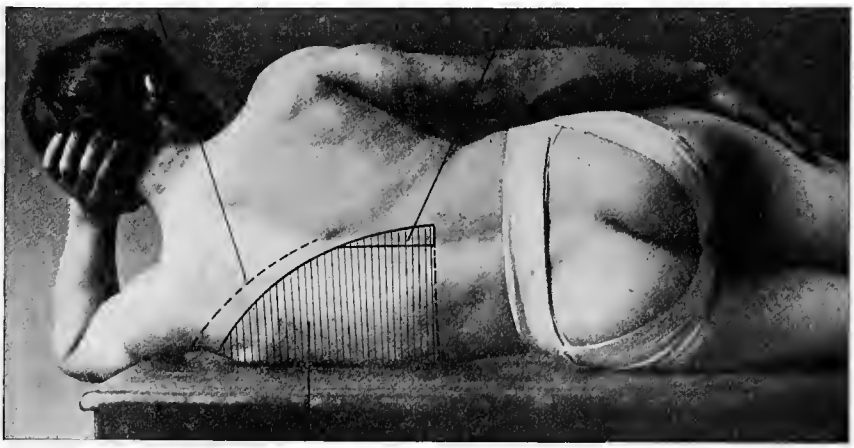


FIG. 49.—EFFECT OF POSITION OF THE PATIENT ON A LARGE PLEURAL EFFUSION.

In those cases in which the exudate fills one pleura to the level of the third rib the note obtained by percussion over the apex and above the third rib is hyperresonant (skodaic resonance) and often tympanitic in character. Modified skodaic tympany is also found above an effusion that almost fills the pleura to the fourth rib, and a hyperresonant note is elicited above the level of the fluid when both pleuræ are partially filled. In the case of a large effusion, in which the pleuræ is practically filled with fluid, firm percussion may elicit the so-called "cracked-pot sound"; to obtain this, however, certain conditions must exist: (*a*) The compressed lung and its bronchus must be forced against the chest-wall anteriorly; (*b*) the chest-wall must be thin and relaxed; (*c*) the patient must breathe with the mouth open; (*d*) firm percussion is required.

The upper limit of pleural fluid or the line of flatness is not horizontal when the patient is sitting or standing, but is slightly higher near the spine, and becomes gradually lower as the anterior surface of the chest is approached. If the quantity of fluid present is moderate, a reverse condition is found, and

the lower level of the fluid, as indicated by the level of the note of flatness, is found posteriorly, rising highest in the axillary region, and descending slightly as we pass anteriorly. This curved line of flatness has been referred to by Garland and others as the **S**-line.

It is only by careful percussion and by a keen appreciation of the sensation offered to the finger (pleximeter), as well as by a careful analysis of the note produced, that the distinction between the lower border of the pleural fluid and the upper margin of the liver can be made. Again, it must be borne in mind that, owing to the anatomic relation of the pleura to the top of the liver (Fig. 50), a certain portion of the latter must be overlapped by

FIG. 50.—AREA OF FLATNESS IN LARGE RIGHT PLEURAL EFFUSION.

the distended pleural sac, which makes it necessary to employ deep percussion, and even then the height to which the top (dome) of the liver rises is difficult to determine.

A large accumulation of fluid in the left pleural sac causes an obliteration of Traube's semilunar space, and produces an alteration in the percussion-note obtained over this area.

Auscultatory Percussion.—In order to insure accuracy in determining the level at which the flatness of pleural effusion and that of hepatic dullness unite, auscultatory percussion is invaluable. This method of combined auscultation and percussion is also of service in determining the position of the spleen when there is an effusion into the left pleura.

Auscultation.—Whenever sufficient fluid has accumulated in a pleural sac to cause a separation of the parietal and visceral pleuræ, the friction murmur described under acute pleurisy (p. 139) disappears, and the breath-sounds become weak and distant over the fluid. After the pleura is at least half filled with fluid, the breath-sounds over the affected side above the fluid may acquire a bronchial quality. Breath-sounds are absent over the pleural fluid except in those cases in which the patient has suffered from previous attacks of pleurisy, in consequence of which pleural adhesions are present which convey the sound from the visceral to the parietal layers of the pleura. Again, when an enormous quantity of fluid is present in the pleura and the lung is compressed tightly beneath the clavicle, distinct bronchial breathing is audible over this compressed lung. Seldom, indeed, the bronchial quality of the breathing is heard over the entire chest on the affected side. The breath-sounds may display an amphoric quality, and, indeed, the breathing at times resembles that heard in pulmonary cavity. Râles are also present over the affected side, and in children their presence is of but limited diagnostic value.

When one pleural sac is only partially filled, bronchovesicular breathing is audible above the fluid and over the unaffected lung, and in proportion to the quantity of fluid in the pleura the breath-sounds are exaggerated over the unaffected side.

Vocal resonance is absent over a pleural effusion, and, owing to compensatory emphysema of the healthy lung, it is also diminished over the unaffected side and above the level of the fluid. If firm pleural adhesions exist below the upper level of the fluid, breath-sounds may be altered or even exaggerated over an area where a flat note is obtained by percussion. In those cases in which there is a large effusion into the pleural sac and compression of the lung occurs, the voice-sounds may be exaggerated and simulate closely the sounds heard over a superficial tuberculous cavity.

Egophony.—By placing the ear at a point level with the junction of the fluid with the lung the voice-sounds resemble the bleating of a goat when the patient speaks (egophony). In small pleural effusions egophony is most likely to be heard over the scapular region.

X-Ray Diagnosis.—Williams states that, given a large pleural effusion, the rays do not readily pass through it, and, as a consequence, the outline of the diaphragm, ribs, and solid viscera is obliterated on the affected side. By means of fluoroscopic examination it is possible to detect displacement of the heart, and Bergoine and Carrie, by a fluoroscopic study, observed the changes in the pleural fluid resulting from the position of the patient and also from the action of the diaphragm. Fluoroscopy is a method of great diagnostic value, since the heart may be markedly displaced, and yet, owing to the fact that the organ is covered by emphysematous lung (compensatory), such displacement may be undetectable by percussion. (See *x*-ray findings, p. 77.)

Paracentesis.—Aspiration of the pleural sac is of inestimable value in determining both the type of fluid contained in the sac and the variety of bacteria that may serve as etiologic factors. The operation is, comparatively speaking, free from danger if done under antiseptic precautions.

Caution.—Always test the aspirator by removing fluid from a cup or bottle, through it, before inserting the needle into the pleura.

Technic.—The accompanying illustration (Fig. 51) will show the posi-

tion in which the patient is preferably placed. Those cases where it is impossible to sit the patient erect are aspirated with decided difficulty, since it is impossible to get far below the level of the fluid. In any event the hand of the affected side should be placed on the opposite shoulder in order to widen the interspaces. The points of election are just below the angle of the scapula and in the mid-axillary line. Insert the needle in the sixth interspace on the right, and in the seventh interspace when aspirating the left pleura. In selected cases the needle may be inserted one interspace lower. Estimate approximately the thickness of the patient's chest-wall and grasp the needle firmly at a point allowing just sufficient of it to reach the pleura. Insert the needle immediately above, and hug closely to the superior surface of the rib to avoid wounding the intercostal

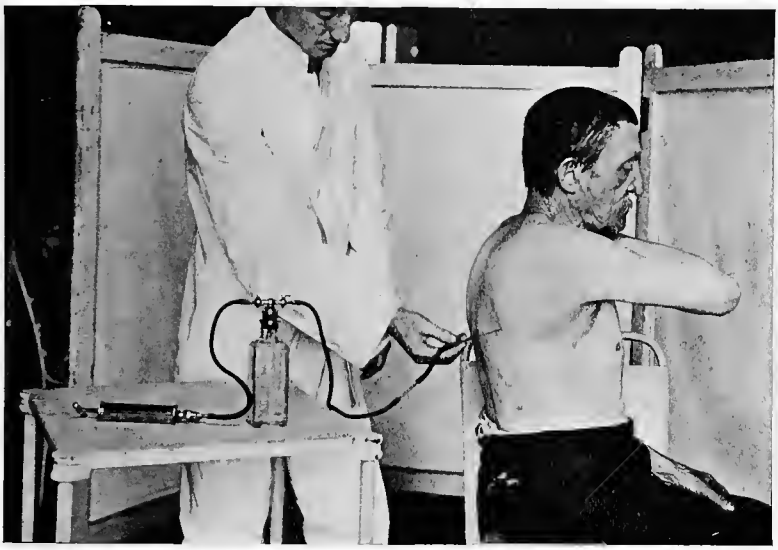


FIG. 51.—ASPIRATION OF THE RIGHT PLEURA

artery. Immediately upon having entered the pleura elevate the outer portion of the needle in order to direct its point downward and from the lung.

Stage of Resorption.—*Inspection.*—The abnormalities observed when the disease was at its height gradually disappear with the resorption of the fluid, and when a large pleural effusion has become almost completely absorbed, the chest and respiratory movements are approximately normal. After repeated attacks of pleurisy, and, indeed, rarely after a single attack, permanent retraction of the affected side may follow. During the stage of absorption the widened intercostal spaces become narrowed, the elevated shoulder of the affected side assumes its normal level or droops slightly, and slight curving of the spine toward the affected side may rarely be detected. For weeks or even months after an attack of serofibrinous pleurisy the scapula of the affected side may project further from the chest than does its fellow. Permanent shrinking of the thorax may follow serofibrinous pleurisy, but

such deformity is usually compensated for by abnormal expansion of other portions of the thorax. Chest deformities occurring as the result of pleurisy are discussed at length under Empyema (p. 127).

Palpation.—Tactile fremitus, which was absent over the fluid, is now discernible, and is situated at a lower and lower level from time to time as the fluid is being absorbed. There are few exceptions to this general rule, and these are dependent upon the formation of pleural adhesions and extreme thickness of the pleura. As the fluid disappears the expansion of the affected side more and more closely approximates that of the normal, although after the process of absorption is completed, the expansile movement of the affected side of the chest may show moderate restriction.

Mensuration.—Some months after an attack of serofibrinous pleurisy a limited expansion of the affected side of the chest generally occurs, and when at rest, the measurements of this half of the chest are slightly below those of the opposite side.

Percussion.—With the absorption of the pleural fluid the flat note gradually disappears, giving place to normal percussion resonance, which progresses from above downward. Normal percussion resonance is not obtained for weeks and often months after an attack of serofibrinous pleurisy. With the absorption of the fluid areas of dullness, due to displacement of the heart, liver, and spleen, gradually disappear, to reappear at their normal sites.

Auscultation.—During the process of absorption of a large effusion breath-sounds that were formerly heard only at the apex of the lung are now heard at a lower and lower level from time to time until the fluid has been completely absorbed, when they become audible at the base. As the fluid disappears the breath-sounds, which at first were extremely weak and distant (at the base), gradually assume their normal tone.

The friction-sound, described under Acute Pleurisy (p. 139), may reappear when the fluid is almost completely absorbed, and occasionally it persists for days, weeks, or even months after the patient has apparently recovered and is free from pain. In rare instances the lung does not expand sufficiently to follow the upper surface of the fluid during the process of absorption, in which case breath-sounds are heard only over the upper portion of the pleura.

The heart-sounds, which were rapid and may have been greatly altered by the presence of a large effusion, return to normal with the disappearance of the pleural exudate.

X-Ray Diagnosis.—The best results are to be obtained by placing the patient in the erect posture for the plate posteriorly. This means of diagnosis is of great assistance in determining the localized areas of consolidation due to encysted pleurisy. (See *x*-ray diagnosis, p. 77.)

Laboratory Diagnosis.—While the quantity of fluid in the pleural sac is increasing, the urine becomes scanty, of high color and high specific gravity, and may contain albumin. During the stage of resorption the flow of urine is increased, and it becomes pale and of low specific gravity unless the patient is placed upon a dry diet.

Fluid obtained from the pleural sac is serous in character, has a specific gravity of 1.018 to 1.030, and is rich in albumin. Microscopically endothelial cells, leukocytes, and, rarely, bacteria (tubercle bacilli) are present. An examination of the sediment obtained by centrifugation of a pleural fluid shows a preponderance of lymphocytes if the process is tuberculous,

of polymorphonuclear cells if it is septic. In order to determine whether or not a pleurisy is tuberculous in character, a healthy guinea-pig should be injected with a small quantity of the pleural exudate, when, if tuberculosis is the causal factor, the animal will develop tuberculosis in from four to eight weeks. Cultures from pleural effusions seldom show the presence of the bacillus of tuberculosis unless a large amount of fluid is used, and, indeed, such cultures usually remain free from bacteria.

Summary of Diagnosis.—In serofibrinous pleurisy there is a history of preceding dry (acute) pleurisy, except where malignancy extends to the pleuræ. The diagnosis is strengthened by the presence of movable flatness, the absence of breath-sounds over the affected area, and increased respiratory murmurs over the compressed lung and over the unaffected side of the chest. Cyanosis and limited movements of the affected side further confirm the diagnosis, whereas aspiration of the pleura makes the diagnosis positive, and serves as the distinguishing feature between serofibrinous pleurisy and empyema. The *x*-ray findings are also of great value.

Differential Diagnosis.—In those cases in which the patient has been seen during the attack of **acute dry pleurisy,** differentiation is easy, and is based largely upon the preëxisting condition. Pleural effusion is to be distinguished from other pathologic conditions in which the lung is *consolidated, retracted,* or *compressed;* and from *new-growths* of the *thorax, pericardial effusion, acute cardiac dilatation, hydrothorax,* and *lobar pneumonia.*

General Tuberculous Infiltration of the Lung.—When a large portion of one lung is involved by an acute tuberculous process that has spread rapidly from an initial pulmonary focus, the associated pleurisy resulting from such inflammatory process may point toward the existence of *serofibrinous pleurisy.* These two conditions may, however, be differentiated by the physical signs, since over a consolidated lung dullness is obtained, and not flatness, and the resistance offered to the pleximeter finger is less intense than it is over a pleural effusion. Bronchial breathing is heard over a lung consolidated from any cause, but is usually absent over fluid; the breath-sounds are also intensified over consolidation or even over a partially consolidated lung, whereas over fluid they are usually diminished or absent. Movable dullness is characteristic only of pleural effusion. The adjacent viscera—the heart and liver—are not displaced by pulmonary consolidation as they are by pleural fluid. The detection of tubercle bacilli in the sputum goes far to support a diagnosis of tuberculous infiltration.

New-growths of the pleura may compress the lung, and when this occurs, the signs of pulmonary consolidation are present; but here again the adjacent viscera are not displaced, and the measurements of the affected side of the chest are seldom, if ever, greater than those of the unaffected side.

The accompanying table, amplified from Anders, shows the leading differential points between acute **croupous (lobar) pneumonia** and **pleurisy with effusion.**

Pleurisy with Effusion.	Primary Lobar Pneumonia
Symptoms.	
1. Onset marked by chilliness persisting for a few days.	1. Onset acute, with rigor, lasting one hour or longer.
2. The pain is sharp, "stitch-like," and strictly localized.	2. Acute pain (similar), but soreness more diffuse.

Pleurisy with Effusion.—(*Continued.*)	Primary Lobar Pneumonia.—(*Continued.*)
3. Cough is irritating; no expectoration, or, if present, catarrhal in character.	3. Cough more marked, and accompanied by rusty or bloody, tenacious expectoration.
4. Sputum negative; tubercle bacilli rare.	4. Dense aggregations of pneumococci present.
5. Moderate fever of continuous type; decline by lysis.	5. Fever, 102° to 104° F., falls by crisis.
6. Prostration moderate.	6. Prostration extreme.
7. Herpes does not appear.	7. Herpes labialis quite common.
8. Leukocytosis absent or moderate.	8. Leukocytes number 15,000 to 40,000 per c.mm.
Physical Signs.	
Inspection.	
9. Unilateral distention of the thorax.	9. Absent.
10. Countenance pale and anxious.	10. Mahogany-colored flush of cheeks.
11. Limited expansion at base of chest on the affected side.	11. Degree of expansion slightly, if at all, inhibited.
12. Patient, when in bed, rests upon or inclines toward the affected side.	12. Most likely to rest upon the back.
Palpation.	
13. Tactile fremitus diminished or absent.	13. Increased over area of consolidation.
14. Expansion limited on the affected side.	14. Not detectable.
15. Interspaces bulging at base of chest.	15. Absent.
16. Traube's semilunar space is usually prominent.	16. Absent.
Percussion.	
17. Flatness, with great resistance to the plexímeter finger.	17. Dullness with less resistance, and sometimes a tympanitic note.
18. Shows displacement of adjacent viscera.	18. Absent in uncomplicated cases.
19. If the sac is partially filled, the line of flatness changes with the position of the patient.	19. Absent.
20. Upper level of flatness extends from the sternum to the spine (patient sitting or standing).	20. Outline of dullness irregular and usually limited.
Auscultation.	
21. Diminished or absent breath-sounds over effusion the rule. Respiratory murmur diffuse, distant, and generally unaccompanied by râles. Bronchial breathing may be present over the entire affected side of the chest.	21. Harsh bronchial breathing and presence of râles in first and third stages, unless a bronchus is plugged.
22. Vocal resonance diminished or absent; egophony heard at junction of lung and upper level of fluid.	22. Bronchophony (loud), unless a bronchus is occluded.
23. Friction-sound heard in early and late stages.	23. No friction murmur; râles present.
Aspiration.	
24. Serum is recovered from pleura by aspiration.	24. Negative or yields a few drops of thick blood.

Cysts of the liver or of the lung, or even abscess of the liver, may push the diaphragm and lower border of the pleura to a sufficient height to give rise to physical signs that may be confused with those of pleural effusion. The clinical history of hepatic disease differs widely from that obtained in

pleurisy. In the former dullness may be extreme, and may even simulate the flatness of pleural effusion; but movable dullness is absent in hepatic disease, whereas it is constant in pleural effusion. An exploratory puncture will furnish decisive evidence in distinguishing between pleural effusion and disease of the liver.

Hydrothorax.—The physical signs presented by unilateral hydrothorax are identical with those seen in pleural effusion, except that in the former the friction murmur is absent. As a rule, however, hydrothorax is bilateral, which fact serves to distinguish it, in the majority of instances, from acute pleural effusion. Bilateral accumulation of fluid in pleurisy, while uncommon, may occasionally be found. In hydrothorax there is no history of acute pleurisy, but, on the contrary, one of cardiac, hemic, hepatic, or renal disease is the rule. Aspiration of the pleura serves as a valuable distinguishing point, since a transuded pleural effusion (the fluid of hydrothorax) is of low specific gravity,—never above 1.015,—whereas a pleural exudate the result of acute pleurisy has a higher specific gravity.

Pericardial Effusion.—A large pericardial effusion may be mistaken for fluid in the left pleura. In pericardial effusion dyspnea is a more prominent symptom than in pleural effusion. In the former the heart is not displaced to the right (Fig. 47), as in pleural effusion. In pericardial effusion percussion shows the area of flatness to be circumscribed, and to be most marked in the axillary region. Along the posterior margin of the left pleura normal pulmonary resonance is obtained, whereas in pleural effusion a flat note is elicited over this region. In pericardial effusion the heart-sounds are distant, feeble, or muffled, while in pleural effusion the quality of the heart-sounds is unaltered.

Dilatation of the Heart.—In acute cardiac dilatation the area of cardiac dullness may be sufficiently great to occupy the greater portion of the anterior and axillary surfaces of the chest, as high as the fourth rib. A circumscribed area of dullness the size of a silver dollar is often found near the angle of the scapula. On deep percussion normal pulmonary resonance is obtained on a level with the base of the pleura and near the spinal column, and relative dullness (over the portion of the dilated heart overlapped by lung) is detected anteriorly and in the axillary region. Marked pulsation of the epigastrium is a prominent sign in acute dilatation, and is but feebly manifest or absent in pleural and in pericardial effusions. The sounds of the heart are weak, rapid, irregular, and lacking in muscular element in cardiac dilatation.

Clinical Course and Duration.—These are dependent entirely upon the exciting cause. The prognosis, regardless of the causal factors, is guardedly favorable. The course is divided into two stages—the febrile stage, which corresponds to the time when the exudate is accumulating, and the afebrile, which corresponds more or less closely to the stage of resorption. Generally speaking, the febrile period continues for from seven to twenty-one days, whereas the afebrile period varies greatly in duration and is dependent upon the presence or absence of complications. In selected cases the pleural exudate appears to accumulate rapidly, and in these same individuals rapid absorption often takes place. Certain mechanic hindrances may delay absorption of the fluid, in which case the final course of serofibrinous pleurisy becomes subacute.

A fatal termination may result from extreme pressure upon the heart and upon the great vessels.

Complications and Sequelæ.—The prognosis is far less favorable

in those cases in which the serous fluid becomes infected with pyogenic organisms (streptococci and staphylococci). Infection with other bacteria—*e. g.*, the typhoid bacillus, the pneumococcus, the colon bacillus, etc.—also tend to make the prognosis more grave, and will be considered at length under Empyema (p. 158). Chronic adhesive pleurisy may follow an attack of the acute serofibrinous variety, and is a complication that results in permanent lessening of the air-space of the lung on the affected side. Empyema and chronic bronchitis may be a sequel of serofibrinous pleurisy. (See p. 156.)

SPECIAL CLINICAL FORMS OF PLEURISY.

Carcinomatous Pleurisy.—This clinical form of pleural irritation usually results from direct extension of malignant disease from adjacent structures—*e. g.*, the esophagus and lung.

The symptoms are quite similar to those of acute dry pleurisy, except that they continue for a longer period. A serous or bloody effusion is likely to result where carcinoma involves the pleura.

Hemorrhagic Pleurisy.—Under this heading are included all types of pleurisy in which, in addition to an exudate of serum, blood-corpuscles and hemoglobin are also present in the pleural fluid. The depth of color of a pleural exudate is entirely dependent upon the quantity of blood that has extravasated with the serum.

Etiology.—Among the conditions capable of exciting a hemorrhagic pleural exudate are: traumatism and fracture of the ribs, carcinoma of the pleura, tuberculous pleurisy (either circumscribed or general), superficial pulmonary cavity, chronic interstitial nephritis, secondary infection of the pleura in acute infectious conditions (pneumonia, anthrax), and a right-sided hemorrhagic pleural exudate may follow atrophic hepatic cirrhosis; it is also rarely seen during the course of hepatic hypertrophy. The general arterial sclerosis characteristic of old age and alcoholism is also accepted as a possible cause for the accumulation of bloody fluid in the pleura.

Hemothorax.—This is an accumulation of bloody fluid in the pleura, with or without disease of the pleura itself. The etiologic factors in this condition are practically identical with those described in hemorrhagic pleurisy.

Tuberculous Pleurisy.—Acute serofibrinous pleurisy may be of tuberculous origin, but the majority of such cases doubtless develop during the course of pulmonary tuberculosis, and are the result of direct extension from a superficial pulmonary cavity or consolidation. When tuberculous pleurisy follows tuberculosis of the lung, its development is less acute than is that of acute plastic pleurisy, and there is a tendency toward chronicity. Chronic adhesive pleurisy results in more or less obliteration of the pleural sac by a thickening of the pleura and by adhesive bands.

Owing to adhesions and to consequent retraction of the lung on the affected side, certain physical signs are observed over the affected pleura; these include retraction of the interspaces, limited expansion, abnormal tactile fremitus, and impairment of percussion. The unaffected side of the chest is unusually prominent, and may display compensatory emphysema. It is occasionally found that both sides of the chest have been affected by chronic adhesive pleurisy, in which case there are localized areas of retraction on both sides, whereas other portions of the chest are unduly prominent. Tuberculosis of the pleura, when primary, and, indeed, occasionally when secondary, is followed by tuberculosis of the pericardium. Tuberculous peritonitis is also often found as a complication.

Acute tuberculosis of the pleura is one of the common causes for the accumulation of bloody pleural exudate. (See Hemorrhagic Pleurisy, p. 154.) Tuberculosis of the pleura may terminate in recovery, although a large proportion of all cases is followed by the development of pulmonary tuberculosis. If the condition assumes the chronic adhesive form, the patient may live for many years, although he is never restored to perfect health.

Encapsulated Pleurisy.—This is a variety of pleurisy in which the pleural exudate is held in one position by firm adhesions (Fig. 52). Anatomically, it is not infrequent to find more than one small sac of fluid that is practically isolated from the general pleural sac. Encapsulated pleural effusion may be found over any portion of the lung, and in this event the

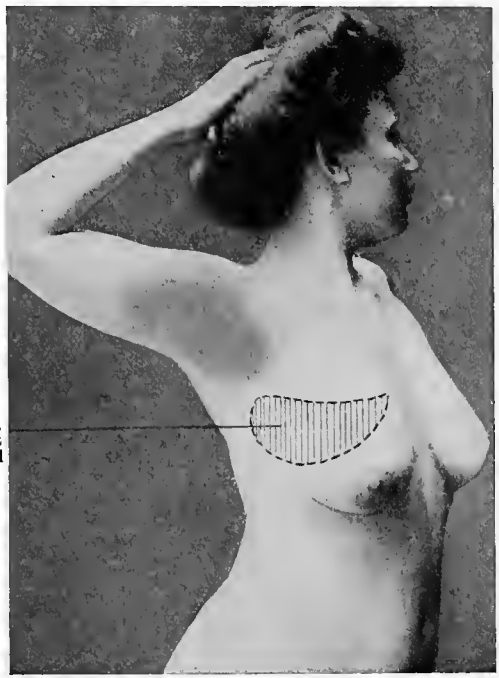


FIG. 52.—ENCAPSULATED PLEURAL EXUDATE.

physical signs are those of consolidation, although these are often so modified that the diagnosis of encysted pleurisy is made only with difficulty.

Interlobar Pleurisy.—This is a special variety of serofibrinous pleurisy in which the exudate is more or less completely encapsulated. It is due to the presence of recent or of old pleural adhesions, the pleural fluid being retained between the pulmonary lobes.

Interlobar pleurisy is more common upon the right than upon the left side, and the encapsulated fluid is oftenest found near the root of the lung, and between the superior and middle lobes. Although it usually follows acute and chronic pleurisy, interlobar pleurisy may also develop as a complication of lobar pneumonia. The encapsulated exudate may rarely become infected with pyogenic organisms, and instances are recorded in which such

purulent material has gained access to a bronchus and been ejected with the sputum. The quantity of fluid in the capsule is usually small—not exceeding a few drams or, at most, a few ounces.

Upon inspection the physical signs common to the presence of a large pleural effusion are lacking, and, on the contrary, the interspaces of the affected side are either normal or deepened, the quantity of fluid being too small to cause bulging.

It is extremely difficult to obtain a sufficiently clear history and to elicit the satisfactory physical signs necessary to make a positive diagnosis of interlobar pleurisy. In patients in whom the chest-wall is thin, a diagnosis is more readily attained.

Diaphragmatic Pleurisy.—When the inflammatory process first attacks that portion of the pleura covering the diaphragm, and if the inflammation is localized, the condition is referred to as diaphragmatic pleurisy.

Pain is a most constant feature, and extends along the tenth rib and across the upper portion of the epigastrium to the articulation of the sternum with the xiphoid cartilage. In severe cases the pain may be reflected slightly over the abdomen. Deep inspiration and movements of the chest and abdomen increase the pain. When an effusion collects at the base of the pleura, the pain diminishes and finally disappears. Nausea, paroxysmal coughing, and vomiting are occasionally seen, and the symptoms of peritonitis may be present.

Fever is always present, and is slightly higher than in the ordinary type of serofibrinous pleurisy. The effusion in diaphragmatic pleurisy is said to be more likely to become infected with pyogenic bacteria than that resulting from other forms of pleural irritation. (See Empyema, p. 158.) The physical signs, with the exception of the friction murmur, which may persist during the initial stage, are negative, unless the accumulation of fluid is large.

Chronic Pleurisy (Adhesive Pleurisy).

Pathologic Definition.—A chronic inflammation of the surface of the pleura, with or without effusion.

Chronic Pleurisy with Effusion.—This commonly follows serofibrinous pleurisy (see p. 154), although it may develop insidiously. After a moderate amount of effusion has collected the physical signs are practically those of acute serofibrinous pleurisy (p. 144). This type of pleurisy differs markedly in certain particulars from the acute form, *e. g.*: (*a*) Dyspnea is but slight, owing to the slow accumulation of the fluid in the pleura; (*b*) fever is generally absent, and, indeed, a subnormal temperature is not unusual; (*c*) the accumulated fluid shows little or no tendency to disappear, and may remain for weeks, months, or even years.

Chronic pleurisy with effusion becomes more serious when it is found in those under ten years of age, for there is a special tendency for such exudate to become infected with pyogenic bacteria. (See Empyema, p. 158.)

Dry Chronic (Adhesive) Pleurisy (Thickened Pleura).

Remarks.—Reference has already been made to pleural adhesions above, and the rule is that this type of pleurisy follows the serofibrinous variety of the disease after the exudate has been absorbed. Owing to the

slow absorption of the serofibrinous exudate, the fibrinous constituents of the fluid become further organized into layers of connective tissue. Further changes take place, and both the visceral and parietal layers of the pleura become coated with the fibrinous elements of the exudate, and, probably owing to irritation of the pleura, an actual proliferation of the pleural covering occurs. In all events the pleura becomes markedly thickened. Adhesions and even thickening of the pleura are likely to be most pronounced at the base of the chest, although they may extend over the entire pleura and materially incapacitate the lung. This condition may follow empyema, and instances are reported in which the pleural exudate has undergone calcareous degeneration.

Principal Complaint.—A history of one or more attacks of acute pleurisy and of serofibrinous pleurisy is usual, although many cases follow a chronic course from the onset; the latter are nearly always tuberculous in origin. There are vague and sometimes *acute pains* over the affected pleura, the patient becomes *dyspneic* and *exhausted* upon slight exertion, but there are never any definite, rational symptoms that point conclusively to the existence of this form of pleurisy.

Physical Signs.—Inspection.—The patient is usually emaciated, the chest movements are restricted, and depression of the interspaces is common.

Mensuration shows that there is atrophy of the affected side. The apex-beat is often displaced as the result of pleural adhesions, and may even be seen to the right of the median line. When there are many dense adhesions and the pleura has become markedly thickened, certain vasomotor symptoms, due to pressure upon the sympathetic nerves, are observed—*e. g.*, unilateral sweating (usually limited to the head and chest), unilateral flushing of the face, and inequality of the pupils.

Palpation.—Tactile fremitus is, as a rule, markedly decreased over the affected pleura, and most commonly at the base of the chest; but in the event of the presence of dense pleural adhesions extending from the parietal pleura to the lung, localized areas in which the fremitus is increased may be found.

Percussion may be negative, although in those cases in which there is decided thickening of the pleura the percussion resonance is greatly impaired, and a firm stroke is necessary to obtain a note suggestive of underlying lung tissue. The area of cardiac dullness may also be altered. When there are marked thickening of the pleura at its base and many adhesions, it is customary to obtain a hyperresonant note over the apex of the same lung, and if the disease is unilateral, a hyperresonant note is elicited over the unaffected side.

Auscultation.—The breath-sounds are diminished, feeble, and in some instances indistinct. A friction murmur, although not constant, is by no means uncommon; the breath-sounds may be accentuated over the apex of the lung, both as the result of exaggerated breathing and of dense pleural adhesions connecting the parietal with the visceral pleura.

X-Ray Diagnosis.—A thickened pleura may be encountered both in this disease and in pulmonary tuberculosis, and is placarded by a uniform shadow of moderate density, which density is controlled entirely by the degree of pleural thickening, and an accurate interpretation of a plate made from a case of thickening of the pleura requires one thoroughly skilled, and, as put by Pfahler, "here experience alone will serve as a guide." Both an anterior and a posterior plate should be made see (*x*-ray diagnosis, p. 76).

Differential Diagnosis.—**Chronic adhesive pleurisy** with thickening of the pleura may be confused with serofibrinous pleurisy. The following table gives the prominent distinguishing features between the two conditions:

Thickened Pleura with Adhesions.	Pleural Effusion.
1. History of long standing.	1. Acute, of three to eight weeks' duration.
2. Interspaces depressed.	2. Interspaces bulging.
3. Dullness over base of pleura.	3. Flatness over base, with skodaic resonance immediately above the level of the fluid.
4. Area of dullness unaltered by posture.	4. Area of flat note changed by posture.
5. Measurements of affected side less than those of the opposite half of the chest.	5. Measurements increased on affected side.
6. Breath-sounds diminished over area where a dull note is obtained.	6. Breath-sounds absent over fluid, except when the quantity is large and the lung firmly compressed or when there are pleural adhesions.

Clinical Course and Duration.—The prognosis is favorable as to life, the majority of patients living for years. There is no known method by which the lung may be restored to its normal function.

Sequelæ.—Many of these cases terminate in cirrhosis of the lung and in cardiac disease resulting from increased pulmonary tension.

EMPYEMA (Purulent Pleuritis).

Pathologic Definition.—An acute or subacute purulent inflammation of the pleura. The pleura will be found to contain a variable quantity of purulent or seropurulent liquid. The degree of inflammation of the pleural surface is, as a rule, more extensive and more intense than in serofibrinous pleurisy. The pleura may be greatly thickened and the entire surface distinctly granular, whereas in selected cases the parietal pleura may show perforation.

Varieties.—(1) The ordinary type. (2) Traumatic empyema. (3) Empyema necessitatis (that form in which the pus escapes through the chest-wall and forms a tumor). (4) Pulsating empyema, characterized by distinct pulsation at the base of the affected side of the chest.

Predisposing and Exciting Factors.—**Bacterial Infection.**—A number of varieties of bacteria have been recovered from the purulent exudate obtained from the pleura; among these are: the pneumococcus (Micrococcus lanceolatus), Streptococcus pyogenes, Staphylococcus pyogenes albus, Staphylococcus pyogenes aureus, Bacillus coli communis, Bacillus typhosus, Bacillus of Friedländer, Bacillus aërogenes capsulatus, and Streptothrix pulmonalis. Fungi may also be present in the purulent pleural exudate, actinomyces having been found in quite a large number of cases.

Empyema frequently develops as a sequel of **acute serofibrinous pleurisy**, in which case the pleural fluid has become infected with pyogenic microorganisms.

In children, **pleurisy** is especially likely to terminate in empyema.

A pleural effusion frequently becomes purulent following **acute infectious diseases**—*e. g.*, miliary tuberculosis, pneumonia, typhoid fever, whooping-cough, scarlet fever, dysentery, and pyemia.

Malignant conditions of the lung may form a fistulous communication between a bronchus and the pleura, causing empyema, with or without pneumothorax. The extension of carcinoma from the esophagus, even if the pleura is not perforated, may be the exciting cause of empyema.

Tuberculosis of the spine or of the ribs is an occasional cause. **Traumatism** to the chest with fracture of the ribs is quite a common exciting factor among men. Rarely, empyema follows an **acute purulent endocarditis** and also **tuberculosis of the mediastinal glands.**

Principal Complaint.—As a rule, there is a history of acute pleurisy, followed by serofibrinous exudate, although such history may be obscure. The symptoms vary greatly according to the type of the case. In those cases in which empyema follows acute infections the *onset* may be sudden, beginning with a *chill*, after which the temperature rises abruptly and prostration becomes pronounced. *Pain* is nearly always a prominent symptom, and is aggravated by movements of the chest. In severe cases the typhoid state may be simulated, marked by continued fever, a rapid pulse, coated tongue, and delirium. Gangrenous changes in the pleura may take place, and when they occur, are always followed by the so-called typhoid state.

Chronic empyema is a type of this form of infection in which the symptoms develop insidiously, and the patient, in spite of the fact that he is much reduced in vitality and has a large purulent pleural exudate, still walks about. It is in these chronic cases that the diagnosis becomes especially difficult and aspiration of the pleura may be necessary to establish a diagnosis.

Complaint Referable to the Chest.—The *pain* is seldom severe in character, although more or less constant. The *cough*, which is more or less continuous, aggravates the pain, and is often accompanied by free expectoration, but neither the pain nor the cough is so distressing in empyema as in acute pleurisy. Again, there are certain cases in which both pain and cough are absent. When there is a virulent type of infection, *profuse sweating* is a prominent symptom, and, owing to the pressure of the exudate upon the sympathetics, there may be unilateral sweating.

Thermic Features.—In those cases ushered in by a *rigor* the *fever* rises rapidly to from 102° to 104° F., and may remain high, although an irregular temperature is the rule. In mild cases that have developed insidiously the temperature is irregular and may not exceed 102° F. After chronic pleurisy has existed for some weeks or even months fever may be absent.

Physical Signs.—Empyema displays all the physical signs detailed under Serofibrinous Pleurisy (p. 144), and to these are added certain signs distinctive only of this affection.

Inspection.—In addition to the bulging of the affected side of the chest, there are usually indentations and markings on the skin made by clothing, especially when the patient has been lying upon the affected side. Anemia and emaciation are apparent.

Palpation.—Upon making firm pressure on the chest overlying a purulent exudate pitting of the skin is common, and especially is this true in children. Long-standing empyema may perforate through the chest-wall, when a fluctuating mass will be displayed. By placing the hand over a large purulent effusion, the chest-wall may be felt to pulsate (pulsating empyema). The causes of this pulsation are doubtful, although the following factors are said to favor its production: (*a*) A copious effusion; (*b*) forcible

heart action; (*c*) relaxation of the chest-wall, with possible paresis of the intercostal muscles; and (*d*) possible association of a thoracic aneurism. Pulsating empyema differs in no way from the ordinary type described, except that pulsation is an added sign. Expansion is lessened at the base of the chest on the affected side.

Percussion.—The upper level of the area of flatness changes less readily with the position of the patient than it does in serofibrinous pleurisy (p. 145).

Auscultation.—Spoken-voice sounds are seldom heard over a large purulent exudate, and Baccelli's sign, transmission of the whispered voice sounds, is absent, although where there is a small collection of purulent exudate in the pleura, Baccelli's sign may be audible.

Laboratory Diagnosis.—Microscopically, fluid obtained from the pleura is seen to contain pus, and may show granules of blood-pigment. Stained specimens of this pus contain pyogenic bacteria. Cultures made from the pleural exudate invariably develop colonies of pyogenic organisms, cocci, and bacilli.

Blood.—Leukocytosis of 12,000 to 30,000 per c.mm. is present in all acute cases, but may be absent after the pus has been retained in the pleural sac for a long period and has become surrounded by a dense capsule of fibrous tissue. A differential leukocyte count shows the polymorphonuclear cells to be markedly increased—from 80 to 95 per cent. The number of red cells per c.mm. and the hemoglobin are reduced.

Urine.—The quantity of urine excreted during the twenty-four hours is approximately normal, unless there is an associated septic nephritis or a persistent high temperature, when the quantity will be diminished. The urine displays a high color, a high specific gravity, and is rich in peptone and indican. When nephritis occurs as a complication, a high grade of albuminuria and casts are present.

Summary of Diagnosis.—A history of preëxisting pleurisy or of traumatism to the chest, high irregular fever, marked prostration, emaciation, leukocytosis, and the recovery of pus from the pleural sac constitute the cardinal symptoms of this affection. An *x*-ray study may offer valuable assistance (see page 74).

Differential Diagnosis.—The only reliable method of distinguishing between a large *pleural effusion* and pus in the pleural cavity is by making an exploratory puncture into the pleura and recovering the fluid. This is best accomplished by employing a needle of unusually large caliber, which may be attached to an ordinary hypodermic syringe.

Aneurism.—Pulsating empyema may simulate thoracic aneurism, but a distinction is usually made from the fact that in aneurism pathologic changes (hardening) are present in the radials and other arteries. The radial pulses may be unequal, and the presence of bruit and thrill is characteristic of aneurism.

Clinical Course and Duration.—Empyema should be regarded as a serious disease, although the special etiologic factors present materially modify its clinical course. Rarely, spontaneous absorption of the pus takes place, but even in this event convalescence is protracted and the patient may never return to perfect health. Rupture into the bronchus, one of nature's methods of sending relief, may be followed by recovery, as may also those cases in which pus escapes through the chest-wall or burrows along the retroperitoneal tissue. In certain cases recovery follows aspiration and removal of the greater portion of the purulent material, whereas in others a purulent discharge continues for months or even years. Surgical inter-

vention becomes necessary in quite a large percentage of all cases, and more recoveries would doubtless follow if this condition were regarded as a surgical one, whenever the diagnosis is attained. Bilateral empyema has been reported, and is an extremely grave condition. In children the outlook is more favorable than in adults, though even here recovery is followed by, at least, partial obliteration of the pleural sac, with appreciable retraction of the thorax. The variety due to the pneumococcus often pursues a favorable course.

Complications.—Pneumothorax may result from perforation of the lung tissue, and perforation of the pericardium has been recorded. Pneumothorax may also follow infection of pus by gas-producing bacteria (colon bacillus, Bacillus aërogenes capsulatus).

PNEUMOTHORAX (SEROPNEUMOTHORAX; PYOPNEUMOTHORAX).

Pathologic Definition.—A secondary condition in which air escapes into one pleural cavity. Rarely, pneumothorax may follow infection of a pleural exudate with gas-producing bacteria. (See Special Varieties, below.)

Varieties.—(1) **Seropneumothorax,** a condition in which serum and air fill the pleura. (2) **Pyopneumothorax,** a variety in which the serous exudate has become infected with pyogenic bacteria. (3) **Traumatic pneumothorax,** a form resulting from stab wounds and fracture of the ribs, with rupture of the lung. (4) An additional variety is made up of those cases in which pulmonary cavity, pulmonary abscess, pulmonary gangrene, and pulmonary carcinoma have formed a fistulous communication between the lung and the pleura. (5) Carcinoma or abscess of the esophagus may extend to and perforate the pleura, allowing air to enter. (6) **Subdiaphragmatic pneumothorax** is a variety resulting from perforation of the diaphragm and pleura by gastric or duodenal ulcer, or from a subphrenic abscess rupturing into the pleura. (7) Pneumothorax may follow infection of a serous pleural exudate by gas-producing bacteria—*e. g.*, Bacillus aërogenes capsulatus and Bacillus coli communis. (8) Abscess of the liver that has ruptured into the pleura may give rise to pneumothorax.

Predisposing and Exciting Factors.—(1) **Age.**—Pneumothorax is extremely uncommon before the tenth year, and is most frequently seen during early adult and middle life.

(2) **Sex.**—The condition develops among males more often than among females, and is probably influenced by strenuous exercise.

(3) The commonest exciting cause (70 per cent. of all cases) appears to be pulmonary tuberculosis with cavity-formation, such cavity rupturing into the pleura. The left pleura seems to be affected in about 66⅔ per cent. of all cases. A cavity that has become partially encapsulated and undergone caseous change is also likely to rupture into the pleura. Among the other diseases of the lung that are to be considered in the etiology of pneumothorax are bronchopneumonia, pulmonary gangrene, pulmonary abscess, a suppurating echinococcus cyst, and abscess of a bronchial gland. Heavy lifting, and the like, has been known to result in rupture of the air-cells, with the production of pneumothorax; paroxysmal coughing, as in whooping-cough, and a bronchiectatic cavity, when situated near the periphery of the lung, may rupture into the pleural space and cause pneumothorax.

Thoracic aneurism, by pressure upon the root of the lung and upon the esophagus, may produce ulceration of the latter, which may communicate

with the pleura. Esophageal carcinoma and esophageal abscess are also uncommon causes. Empyema of long standing may produce an erosion, with subsequent perforation of the visceral pleura. (See Empyema, p. 161). Stab wounds of the chest, gunshot wounds, and fracture of the ribs, whenever these penetrate the pleural space from without, give rise to external pneumothorax; if the lung itself is ruptured sufficiently to communicate with the pleural space, the condition is known as internal pneumothorax.

The physical signs of pneumothorax may be *prominent* even when the pleura does not communicate either with the lung or with the external surface of the body; this is the result of infection of the pleural effusion with gas-producing bacteria. The gas recovered from such pleural sacs will be found to contain elements not present in the air—*e. g.*, hydrogen, hydrogen disulphid, or marsh-gas.

Subdiaphragmatic pneumothorax may follow a number of pathologic conditions, among which are hepatic abscess, infected echinococcus cyst of the liver, abscess between the layers of the diaphragm, ulcer or carcinoma of the stomach, ulcer or carcinoma of the duodenum, carcinoma of the pancreas, carcinoma of the liver, and, rarely, carcinoma of the colon, all of which may perforate the diaphragm.

Principal Complaint.—This will be found to vary greatly according to the exciting cause in the individual case. In those cases in which perforation of the pleura from the lung is the result of pulmonary disease or is due to traumatism from without, stab wounds, etc., the onset is sudden and *extreme pain* is one of the prominent symptoms. There is also a history of pronounced *dyspnea*, *weakness*, *nausea*, and a tendency to faint within the first few hours after air enters the pleura. The severity of these initial symptoms is in direct proportion to the volume of air that gains entrance to the pleura—the greater the volume, the more intense are the symptoms, and the more likely is collapse to ensue. The patient complains of weakness, is unable to move about the room, and has no desire for food. Within a few days symptoms referable to general sepsis arise—*e. g.*, intense heat during the afternoon hours, profuse sweating, headache, and other nervous manifestations. Constipation is likely to occur except in those cases in which some form of enteritis existed prior to the onset of pneumothorax.

Thermic Features.—Immediately following rupture of the pleura the temperature is found at normal, or even subnormal—97° or 96° F.; it is likely to remain below the normal for a period of from one-half to two hours. As a rule, within the course of from forty-eight to seventy-two hours the pleural fluid becomes infected, and the temperature rises to 100° to 101° F. As the condition progresses the fever becomes hectic in type, and an evening temperature of 103° or 104° F., with a morning decline to near the normal, is observed. The more profound the septic condition, the more continuous is the type of fever, and the patient may present the typhoid state. In that small proportion of cases in which recovery takes place the fever begins to decline in from the third to the sixth week, although convalescence is usually protracted.

Physical Signs.—Inspection.—When seen early, the face is dusky, the lips and extremities are cyanosed, the skin is covered with beads of perspiration, and the apex pulsation may be diffuse, displaced, and often absent. The neck appears to be unusually short and thick, and there is throbbing of the vessels. The patient inclines slightly toward the affected side, and the clavicle of this side is elevated; the respirations are rapid and shallow, and one side of the chest does not expand. The upper quadrant of the ab-

domen (Fig. 160 on p. 418), joining the affected pleura, is usually prominent. Inequality of the pupils, the result of undue pressure upon the spinal sympathetics, is quite common.

Palpation.—The skin at first is cold and clammy, but later it may be hot and at times dry; after sleep, however, as a rule, it is bathed in perspiration. Palpation further confirms inspection with reference to the movements of the two sides of the chest and displacement of cardiac pulsation. In right-sided pneumothorax the liver is readily felt below the costal margin,

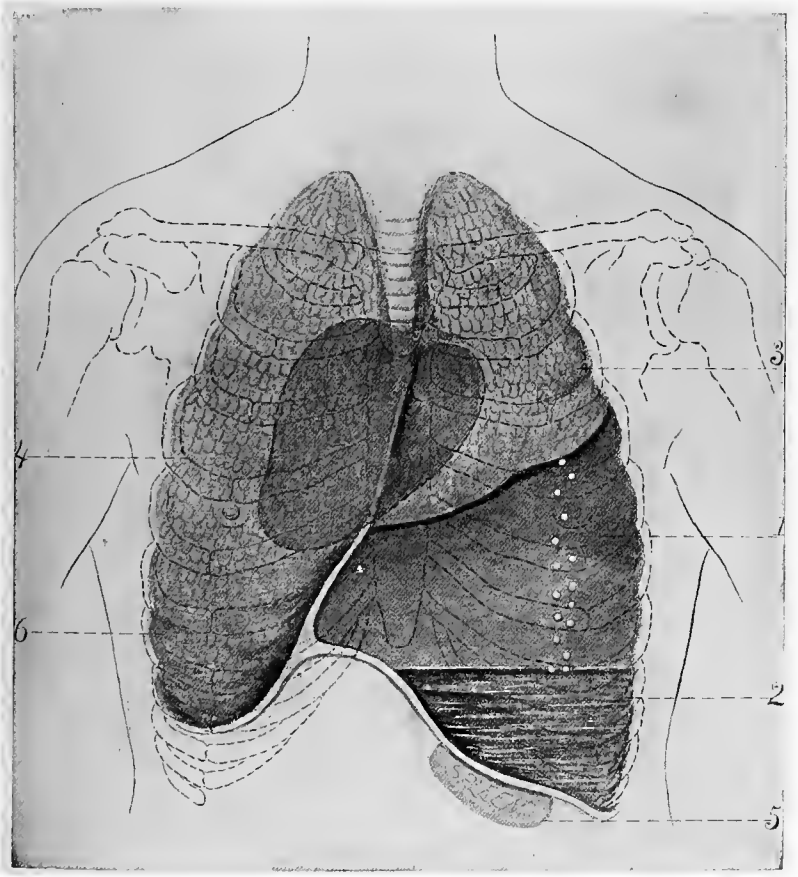


Fig. 53.—1, Air in the pleural sac; 2, fluid exudate at base of pleural sac; 3, compressed portion of lung; 4, displaced heart; 5, depressed spleen; 6, mediastinum pushed toward the right (Anders' Practice).

whereas when the left side is affected, the spleen is likewise pushed below the margin of the ribs (Fig. 53). Firm pressure over the affected side may elicit pain, although this is by no means constant.

Percussion.—Tympany is obtained over the affected side, except at the base of the chest, where, owing to the collection of fluid (pus) (Fig. 53), a flat note is elicited. At the apex of the lung skodaic resonance is elicited. As a result of the high tension under which air is held in the pleura a wooden or almost flat note is occasionally obtained between the lower border of the

lung and the upper margin of the fluid. The normal area of cardiac dullness is displaced regardless of which pleura is involved. Both hepatic and splenic dullness extend to a lower level when the pleura of their respective side of the body is involved (Fig. 53).

Owing to the fact that one lung is almost completely incapacitated, the opposite lung becomes hyperresonant. Occasionally a small area of cardiac dullness may be outlined, but it is commonly absent.

A point of much clinical importance is that the adjacent viscera are displaced to a greater degree by pneumothorax than by a large pleural effusion.

Wintrich's Change of Note.—If the air in the pleura communicates directly with a bronchus, the "cracked-pot" sound is elicited by firm percussion over the affected side of the chest, and Wintrich's sign—change of pitch in the percussion-note when the patient is directed to hold the mouth open and then to keep it closed—is also present.

Combined Percussion and Auscultation.—By placing a metallic substance, *e. g.*, a coin, over that portion of the pleura containing air, and the ear over a different area of the chest, a peculiar metallic, bell-like note is audible when the coin is tapped with a metal substance (Fig. 54). This is known as bell tympany, and is generally conceded to be one of the pathognomonic signs of pneumothorax; some writers, however, claim that it exists where there is a large superficial pulmonary cavity without the presence of free air in the pleura.

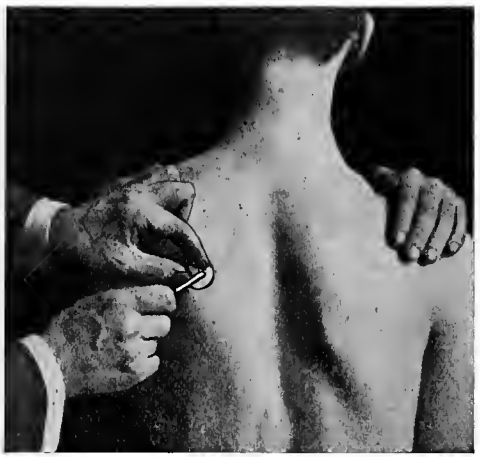


FIG. 54.—METHOD OF TAPPING TO ELICIT COIN-TEST IN CASE OF PNEUMOTHORAX.

The ear of the clinician is placed at a level corresponding to that of the coins, and on the anterior surface of the chest, while the assistant taps the coin.

Auscultatory percussion (p. 59) is a method of value in separating the lower level of the fluid from the superior surface of the liver. It is likewise serviceable in outlining the lower margin of the compressed lung, and in determining the exact outline of the heart.

Auscultation.—When heard over the affected side, the breath-sounds display a metallic quality. Bronchial breathing may be heard over the compressed lung, while over the fluid collected at the base of the pleura the breath-sounds are decidedly lessened or even absent. If the ear is placed at a level between the upper border of the fluid and the lower margin of the lung and the patient is shaken vigorously, the liquid is heard to splash against the pleura; this is known as the Hippocratic succussion splash (Fig. 55; also p. 72, Causes of Thoracic Splashing Sounds). If, immediately after obtaining the succussion splash, the ear is held against the chest-wall, a peculiar dropping sound (metallic tinkle) will be heard; this was formerly believed to be produced by the dropping of the liquid from the surface of the lung into the fluid below; a later theory, however, maintains that small bubbles are produced upon the surface of the fluid, and that the metallic tinkle is the sound generated by the bursting of each bubble. Another theory is that this sound may be the reëchoing of

vibrations of moist bronchial râles that are communicated to the free air in the pleura. If pleural exudate is placed in a bottle that is not tightly corked, it is possible to produce the metallic tinkle artificially.

The breath-sounds are exaggerated over the unaffected side, but may become weak, owing to extreme compensatory emphysema. The heart-sounds are rapid, and late in the disease they become weakened and irregular. Accentuation of the second pulmonic sound is present throughout the course of this disease.

X-Ray Diagnosis.—See *X*-Ray Diagnosis (p. 77).

Laboratory Diagnosis.—Except in those cases in which pneumothorax develops during the course of pulmonary tuberculosis, the laboratory findings are practically identical with those given under Empyema (p. 160). (See also the Laboratory Diagnosis of Pulmonary Tuberculosis, p. 806.)

Summary of Diagnosis.—The history of an acute onset, with severe pain in the chest, followed by marked prostration and a tendency toward collapse, should, at least, suggest the possible existence of pneumothorax. The diagnosis is confirmed by the evidence of certain physical signs—*e. g.*, bell tympany (the coin-test), the metallic tinkle, and the succussion splash.

Differential Diagnosis.—Pneumothorax may rarely be confounded with a large **pulmonary cavity** (Fig. 23). The following table points out the distinguishing features between these two conditions:

FIG. 55.—METHOD OF AUSCULTATING WHILE SHAKING PATIENT TO OBTAIN THE SUCCUSSION SPLASH.

PNEUMOTHORAX.	LARGE PULMONARY CAVITY.
1. Develops with acute pain in the chest.	1. Absent.
2. Absence of movements of chest on the affected side.	2. Restriction of respiratory movements over the apex only.
3. Entire half of chest (affected side) unusually prominent, with bulging of the interspaces.	3. Restriction with depression of the interspaces.
4. Measurements of the affected side unchanged by deep inspiration.	4. Expansion at those portions not overlying the cavity.
5. Tactile fremitus diminished or absent.	5. Increased over cavity.
6. Note flat at base of affected side; deep and full (modified tympany) above the fluid.	6. Note may be dull, or deep percussion may add a tympanitic element, but flatness is never present.
7. Cracked-pot sound unusual.	7. Quite common when the cavity is superficial.
8. Bell tympany (coin-test) an almost constant sign.	8. Rarely seen.
9. Succussion splash an almost constant sign.	9. Absent.
10. Metallic tinkle present.	10. Absent.

Subphrenic Abscess.—This form of abscess may be infected by gas-producing bacteria, and consequently be distended by gas, which may, in extreme cases, force the diaphragm above the level of the nipple, when the signs of pyopneumothorax—*e. g.*, bell tympany, succussion splash, and metallic tinkle—may be elicited. A valuable means of differentiating between pneumothorax and subphrenic abscess is by employing the coin-test, which will not show "bell tympany" above the upper level of the diaphragm in abscess, whereas in pneumothorax it is heard as well at the apex as at the base of the chest. The history of tuberculosis or of the other exciting causes of pneumothorax should be taken into consideration in differentiating the two conditions. A feature that renders the distinction between pneumothorax and subphrenic abscess difficult is that both are likely to follow disease of the stomach and of the liver.

Diaphragmatic Hernia.—This condition may be congenital, and there may be no history of the acute symptoms of pneumothorax. Again, diaphragmatic hernia may follow injury or heavy strain, in which case the history somewhat resembles that of pneumothorax. The existence of pulmonary tuberculosis or of gastric disease strongly favors a diagnosis of pneumothorax. Differentiation is made positive only by the evidence obtained by physical examination—*e. g.*, the tympanitic area is usually limited to the median line, and it seldom, if ever, corresponds to that of an expanded pleura. Bell tympany is not obtained at the apex of the chest, or at most, will not be detected when percussion is made over different portions of the affected side. In hernia a distinct gurgling sound is heard over the tympanitic area, a sign that is unknown to pneumothorax.

Distention of the Stomach.—When the stomach is well distended by gas, the left half of the diaphragm may be elevated well above the nipple. In gastric distention bell tympany and the metallic tinkle are absent over the apex of the left lung. By passing the stomach-tube or by evacuating the bowel the tympanitic area diminishes in size if gastric distention is present. The following table shows the differential points between pyopneumothorax, subphrenic abscess, diaphragmatic hernia, and gastric distention. (See Succussion splashes, pp. 72 and 506.)

Pyopneumothorax.	Subphrenic Abscess.	Diaphragmatic Hernia.	Dilated Stomach.
1. History of tuberculosis of the lung or of disease of the stomach, *e. g.*, gastric ulcer, duodenal ulcer, etc.	1. History of dysentery, disease of the stomach or liver, or of traumatism to the abdomen.	1. May be a history of acute epigastric pain following severe exertion and heavy lifting. The condition may be congenital.	1. History of dyspepsia of long standing.
2. Onset sudden, with acute pain in the chest or in the epigastrium, which pain may radiate to one or the other scapula.	2. Onset may be sudden or insidious, but is preceded by soreness and tenderness along the attachment of the diaphragm.	2. The patient may be unaware that there is any abnormality.	2. Negative.
3. Temperature subnormal at onset; later, 102°–104° F.	3. A temperature of 101°–105° F. the rule.	3. Temperature normal.	3. Normal.
4. Immobility of the affected side of the chest.	4. Restriction of movements at the base of the right or left chest.	4. Movements of chest normal.	4. Chest expansion normal.
5. Bulging of the affected side of the chest.	5. Bulging at the base of the affected side.	5. Negative.	5. Bulging at base of left chest and in superior left abdominal quadrant.

Pyopneumothorax.	Subphrenic Abscess.	Diaphragmatic Hernia.	Dilated Stomach.
6. Tactile fremitus absent except when pleural adhesions are present and at the apex of the affected side, when the fremitus may be normal or increased.	6. Fremitus is absent, and there is decided tenderness over the base of the chest on the affected side.	6. Fremitus absent over area of tympany.	6. Fremitus absent over area of tympany. May detect movements of the stomach in epigastric region.
7. Percussion elicits a tympanitic note over the upper portion of the affected side of the chest, while at the base there is a variable area of movable flatness, due to the presence of pleural fluid.	7. Tympany over the base of the affected side, which blends with stomach tympany. Seldom is there a variable area of dullness, which is below the normal location of the diaphragm.	7. Tympanitic note at base of chest in the median line, extending to one or the other side. Flatness absent.	7. Note of stomach tympany may rise to nearly a level with the nipple and extend well toward the scapula, or into the upper portion of the abdomen. Flatness absent.
8. Auscultation elicits bell tympany (coin-test), metallic tinkle, and succussion splash. Voice and breath-sounds are more or less constant and have a metallic element. Voice-sounds absent over the pleural fluid.	8. If the abscess wall is distended with gas, bell tympany and succussion splash are audible. Above the upper area of tympany normal breath-sounds are heard.	8. Normal breath-sounds around the area of tympany. Bell tympany imperfect where the hernia permits the stomach to rise into the thorax.	8. Absence of breath-sounds over tympanitic area. Normal breath-sounds immediately above this area. Succussion splash and imperfect bell tympany are present over stomach. Gurgling is common.
9. Aspiration over area of flatness recovers purulent fluid.	9. May detect pus by aspiration. The area of tympany diminished after aspiration.	9. Negative.	9. Negative usually.
10. Sputum usually profuse, and may contain tubercle bacilli. Leukocytosis—12,000 to 25,000 per c.mm.	10. Tubercle bacilli absent. Leukocytosis the rule.	10. Sputum and blood normal.	10. Negative.
11. Gastric contents contain normal amount of free hydrochloric acid.	11. When due to perforating gastric ulcer, free hydrochloric acid is in excess.	11. Normal.	11. Lactic and butyric acids present in large amounts. Vomiting of food eaten hours or days before.

Clinical Course and Duration.—Pneumothorax should be regarded as a serious condition, although those cases due to traumatism may terminate favorably. If pneumothorax complicates bronchopneumonia, pulmonary gangrene, or pulmonary abscess, or if it develops late during the course of pulmonary tuberculosis, the outlook is unfavorable, and, at best, the course is protracted. Those cases developing from pulmonary tuberculosis may in rare instances go on to recovery; two such cases have recently come under the care of one of us at the Philadelphia Hospital. At times the pulmonary condition is favorably influenced by the pneumothorax. When gastric or duodenal ulcer forms a fistulous communication with the pleura, surgical intervention is demanded.

DISEASES OF THE CIRCULATORY SYSTEM.

DISEASES OF THE PERICARDIUM, HEART, AND BLOOD-VESSELS.

METHODS OF EXAMINATION.

DATA TO BE OBTAINED BY QUESTIONING THE PATIENT.

General History.—Inquiry should be made into the previous existence of any conditions that influence cardiovascular activity and tension, and that influence the workings of the cardiovascular mechanism.

(*a*) **Conditions that alternately increase and diminish the heart's action,** and, at the same time, cause the peripheral vessels to dilate and contract and thereby induce increased wear. In this connection it becomes necessary to inquire into the use of such stimulants as tobacco, coffee, tea, alcohol, narcotics, and poisons; this same effect upon the circulatory system, although brought about in a somewhat different manner, is induced by extreme physical strain (athletics) and by mental anxiety.

(*b*) **Habit** materially controls the cardiovascular wear and tear, and, indeed, dissipation acts upon the circulatory system in a manner quite similar to that of narcotics and stimulants—*e. g.*, those who at any time of life worship excessively at the shrine of either Venus or Bacchus, or of both, are especially prone to acquire cardiovascular disease, and the circulatory system of sexual perverts is similarly affected.

(*c*) **Occupations** that necessitate sudden change from a cold to a warm room at a time when the skin is already bathed in perspiration encourage cardiac disease by the sudden and extreme change effected in the tension of the peripheral vessels. Alteration in the blood tension in the *liver* and in the *kidneys* likewise results in increasing the heart's work, therefore inquiry into these matters becomes of special importance.

Continued exposure to toxic substances, as lead, arsenic, mercury, and phosphorus, not only favors, but is likely to be the exciting cause of, chronic degenerative changes of both the endocardium and the arteries.

(*d*) **Age.**—The time of life at which cardiovascular disease is most likely to occur is dependent somewhat upon the type and character of the lesion or lesions present. Special mention, at least, must be made of congenital affections, but aside from these the acute inflammations are more frequently encountered during that period of life when acute infections are most common—*i. e.*, youth and early adult life. Degenerative lesions of both the heart and the blood-vessels are unusual before middle life, but exceptions to this rule may be seen in those who have suffered from repeated attacks of those acute infections known to favor the development of vascular degeneration. It is possible, therefore, to find a man of twenty-five whose

circulatory system is sufficiently degenerated to simulate that of a man of sixty or even eighty years, a condition that has given rise to the aphorism that "a man is no older than his arteries."

(*e*) **Sex** appears to exercise a decided influence on cardiovascular disease, but the fact that males are exposed more to the conditions that predispose to cardiac lesions than are females probably explains the great preponderance of cardiac maladies in the male sex. Females appear to be attacked more often by the so-called functional cardiac conditions, and those resulting from acute infections, than they are by those types of cardiac disease resulting from overstimulation of whatever nature.

Family History.—Information as to the general health and age and cause of death of the ancestors of the patient, at least, of parents and grandparents, should be obtained whenever possible, since cardiovascular disease not infrequently manifests itself in a second generation and in those whose father and mother may have been free from cardiovascular disease. Data regarding the number of brothers and sisters and the health of each not infrequently prove of value in formulating a diagnosis. A family tendency toward the so-called gouty and rheumatic diatheses is common in those suffering from cardiovascular maladies. Again, it may be found that certain members of a family have suffered from renal disease, whereas in other members of the same family the cardiovascular symptoms may have been more prominent, yet the intimate pathologic relation existing between these two conditions must not be overlooked.

Luetic infection of one or other parent or grandparent, although it may not actually influence the case under observation, should always be considered.

Clinical History.—Cardiovascular disease not infrequently develops as a complication or a sequel of certain of the acute infections,—*e. g.*, acute articular rheumatism, chorea, gonorrhea, scarlet fever, acute nephritis, pneumonia, typhoid fever, tonsillitis, epidemic meningitis,—and in such cutaneous conditions as erythema, eczema, and dermatitis.

Inquiry with Reference to Present Conditions.

Precordial Pain Not Due to Heart Disease.—Pain, more or less definitely localized to the precordia, may be present. Acute pain at the fifth and sixth interspaces may result from—(*a*) neuralgia; (*b*) myalgia; (*c*) circumscribed pleurisy; (*d*) pleurodynia; (*e*) diaphragmatic peritonitis; (*f*) periostitis; (*g*) localized abscess; and (*h*) gastric disturbances.

(*a*) **Pain that is neuralgic in character** is readily differentiated from other types of thoracic pain by the fact that there are localized areas of tenderness, and that such hypersensitive points correspond anatomically to the positions at which the nerve involved penetrates the fascia. It is possible, in this type of thoracic pain, to find areas of tenderness in the axilla, over the sternum, and even along the vertebral column.

Paroxysmal precordial pain is, at times, a symptom of neurasthenia, and deep-seated thoracic pain, which may radiate from the spine to the region of the heart, occasionally precedes the development of herpes zoster. Precordial pain is frequently a complaint of gouty and rheumatic subjects.

(*b*) **Myalgia.**—In this condition the pain is not distinctly circumscribed, prevents the patient from moving his chest freely, and is likely to be relieved by uniform pressure. Again, in myalgia of the thorax other regions of the body are likely to be attacked.

(*c*) **Circumscribed Pleurisy.**—The stabbing character of the pain,

together with the detection of a friction murmur that is synchronous with respiration, points to this affection. (See Acute Pleurisy, p. 139.)

(*d*) **Pleurodynia.**—As in myalgia, the thoracic pain is likely to be somewhat general, and when the tip of the finger is pressed firmly against the chest-wall, the pain is increased.

(*e*) **Diaphragmatic Peritonitis.**—In this condition the pain resembles that of pleurisy in character, is distinctly localized immediately beneath the apex of the heart, and, as a rule, develops in subjects that are suffering from some abdominal malady.

(*f*) **Periostitis.**—In diseases of that portion of the ribs overlying the heart precordial pain may be severe, but is distinguished from other types of thoracic pain by the facts that there are, in addition to tenderness, distinct swelling over the affected ribs, and that, by pressure upon the ribs at some distance from the site of the pain, tenderness is elicited.

(*g*) **Localized Abscess.**—Circumscribed abscess, either within the chest-wall or between the layers of the pleura or of the diaphragm, excite precordial pain.

(*h*) **Gastric Disturbances.**—If epigastric pain is severe, it is often quite difficult, at the height of the attack, to determine whether or not such pain is cardiac in origin. It has been stated that pain in the epigastrium may be the result of cardiac disease, but it is highly probable that, as a general rule, such pain follows symptoms referable to disease of the stomach. (See Gastralgia, p. 480.)

Pain Due to Pericardial Disease.—In mild cases of pericarditis mere discomfort or precordial distress may be experienced, but the rule is that during the first stage, or prior to the accumulation of an effusion into the pericardium, pain is severe. The pain of pericarditis may radiate to the left arm, less often to the spine and shoulder, and, indeed, it may radiate to the ensiform cartilage and to the abdomen. The presence of a friction murmur that is synchronous with the heart's action serves to differentiate this from other types of precordial pain.

Pain Due to Aortic Disease.—Cardiac pain may be present in acute inflammation of the aorta, and it is generally most intense along the course of the vessel, underneath the sternum, and at the spine. Both in aged and in gouty subjects this type of pain may be severe, and paroxysmal attacks are occasionally seen. It is quite difficult to designate correctly the etiologic factors in this form of pain, since in disease of the aorta morbid changes around the cardiac orifices, and particularly of the aortic leaflets, are commonly present. Substernal pain, practically indistinguishable from that just described, may be experienced during the course of syphilis, neuralgia, in alcoholics, and in those suffering from general atheroma.

Pain of Aneurism.—In thoracic aneurism pain is, as a rule, due to pressure exerted upon adjacent structures, and precordial pain is quite a common symptom. When the aneurism exerts sufficient pressure upon the bones of the thorax to cause an erosion, the pain is referred to as boring in character, and even before the bones have been seriously damaged, a dull, aching sensation is experienced. The pain of aneurism is usually increased by exercise, and diminishes when the patient is at rest.

Pain Due to Cardiac Disease.—Pericardial pain is dependent, at times, upon—(*a*) Alteration of rhythm; (*b*) paroxysmal attacks of palpitation, etc., although these may exist without producing even precordial discomfort. Pain is more commonly seen in the so-called reflex palpitation, due to diseases situated elsewhere in the body—*e. g.*, anemia, Graves' dis-

ease, and the like. Pain accompanied by disturbances of rhythm, due to organic cardiac disease, is excited and intensified by exertion.

Pain occurs in connection with disease of the aortic leaflets, and may be present during endocarditis affecting the mitral valves. (See Endocarditis, p. 252.)

Pain Due to Angina Pectoris.—This pain is excruciating and gripping in character, affecting the entire chest, and results in apparent arrest of the respiratory movements. It frequently radiates to the left, and at times to the right, shoulder, and to the back, neck, and may extend down the arms to the finger-tips.

Pseudo-angina pectoris develops in those who are either anemic or display an hysteric tendency. (See p. 305.)

Palpitation.—Etiology.—This condition may result from organic cardiac disease, but is more frequently due to extracardiac conditions.

(1) Persons displaying an increased excitability of the nervous system in general are likely to develop palpitation. Palpitation is quite common at puberty and at the menopause, and in hysteric and neurasthenic individuals it forms one of the prominent symptoms.

(2) Women are subject to repeated attacks more than are men, and the condition frequently follows emotional disturbances.

(3) The use of stimulants and narcotics, such as alcohol, tobacco, tea, and coffee, may be sufficient to excite an attack.

(4) Overexertion and prolonged muscular strain appear to figure prominently as an exciting cause in young men.

(5) During the course of organic heart disease, and especially after the development of myocardial changes, palpitation is one of the annoying symptoms. (See Myocarditis, p. 296.)

(6) In chlorosis or other of the essential anemias palpitation is induced by slight exertion. In secondary anemia from whatever cause palpitation may be a conspicuous symptom.

Characteristics.—In mild attacks the patient complains of a sense of fluttering or of sinking in the region of the heart, whereas in severe types the heart is felt to be throbbing violently against the chest. On careful inspection, during an attack of palpitation, the arteries will be seen to pulsate, and the area of cardiac impulse is unusually conspicuous. The pulse is markedly increased and may reach 120 to 150 beats a minute. In palpitation occurring in neurasthenic individuals there is flushing of the cheeks, and within the course of a few hours following the attack a large quantity of urine, usually pale and of low specific gravity, is voided. The latter form of palpitation is at times relieved by moderate exercise, a point that serves to distinguish it from palpitation resulting from cardiovascular disease.

In palpitation that is not the result of cardiovascular disease a physical examination of the heart shows the sounds are normal, although the second sound is decidedly accentuated during the attack. Again, in those suffering from anemia, the presence of hemic murmurs will be detected. (See Hemic Murmurs, p. 276.) When there is evidence of organic disease of either the heart or the aorta, the clinician must determine carefully whether or not he is dealing with palpitation the result of an associated neurasthenic condition, since palpitation of cardiac disease is of serious prognostic import. The attacks vary in duration from a few minutes to several hours.

Irregularity and **intermittency** of the heart's action may occur in

persons who are otherwise in perfect health, and, indeed, this condition may continue for an indefinite period without evincing any other manifestations of cardiovascular disease. (See Pulse, p. 193.) The patient not infrequently is conscious of alterations in rhythm, and such patients are usually neurasthenic; in organic heart disease, on the other hand, the irregularity is less likely to be appreciated by the patient.

In a general way the term arhythmia implies an intermittent pulse in which one or two beats are dropped at intervals of from one-half to one and one-half minutes or even longer, the beats being, however, equal in force and in volume. Among the causes of arhythmia are:

(*a*) A cerebral lesion situated in the medulla—*e. g.*, hemorrhage or even concussion—may cause alteration in the regularity of the pulse.

(*b*) Toxic influences, as overstimulation by tobacco, coffee, alcohol, and tea, and such drugs as digitalis, aconite, and belladonna may produce the same effect.

(*c*) Degenerative changes of the heart, with or without cardiac dilatation, atheroma of the coronary arteries, and pathologic changes in the cardiac ganglia are among other recognized causes of arhythmia.

(*d*) Lastly, reflex influences are recognized—*e. g.*, gastric dyspepsia and pulmonary, kidney, and hepatic disease.

Pulsation of the Vessels.—Abnormal arterial pulsation may be detected in the region of the carotids, brachials, femorals, and abdominal aorta during the course of either primary or secondary anemia, and it is also a feature of emotional disturbances. Pulsation in the right supraclavicular region may result from tricuspid regurgitation and from aneurism. Aneurism of the arch of the aorta or of its branches, when sufficiently developed, causes pulsation of the upper portion of the chest, and may at times be seen posteriorly in the region of the scapula. Pulsation over the abdominal aorta may either be due to dilatation of the aorta or, as is quite common, it may be transmitted by diseased abdominal viscera. Epigastric pulsation is occasionally a symptom of new-growths of the epigastrium, in which case the pulsation is transmitted from the abdominal aorta through the tumor mass.

Undue pulsation in the epigastrium in cardiac disease may depend upon a dilated right heart. In cardiac hypertrophy there may be pulsation over the entire organ, and an abnormal pulsation is the rule in the region of the apex-beat. Indeed, in cardiac disease the greater surface of the area of cardiac dullness may, at times, be seen to pulsate, and this pulsation must be distinguished from that of thoracic aneurism. (See Hypertrophy, p. 294, and Aneurism, p. 315.) Pulsation over the course of the arteries is a valuable sign in aortic regurgitation (Fig. 115).

Symptoms Suggestive of Cardiac Disease.

Thrombosis may develop during the course of heart disease, but is more commonly a condition resulting from disease of the blood-vessels. Cardiac dilatation also acts as a predisposing factor to thrombosis.

Pulmonary Phenomena Suggestive of Cardiac Disease.—When the circulation through the lung is materially embarrassed (passive congestion), *cough* becomes a symptom. It also occurs in pathologic conditions of the heart in which there may be pressure upon a bronchus or upon the pneumogastric nerve, or in pressure the result of a large pericardial

effusion. Hemorrhagic infarct and embolus of the lung (p. 117) occasionally complicate organic heart disease and give rise to cough. Following pulmonary infarction it is possible to have bronchopneumonia, but here again the cough is originally excited by the cardiac disease.

During organic disease of the heart cough may be accompanied by a bloody expectoration, a symptom most common in mitral disease. Pulmonary hemorrhage may, as previously stated, result from venous stasis in the lung, from hemorrhagic infarct, from actual rupture of the pulmonary vessels, or from localized cognestion produced by pressure of either an enlarged heart or an aneurism. (See Hemorrhage from the Lung, p. 116.)

In thoracic aneurism, whenever the recurrent laryngeal nerve is irritated, the cough is metallic (brassy) in character. (See Aneurism, p. 314.) In such cases the voice is commonly altered and the character of the cough paroxysmal.

Cyanosis may be either general or local. In advanced cases of cardiac disease it is customary to find the finger-tips and lips cyanosed, but after advanced myocardial change has taken place, a general lividity of variable degree is present.

Dropsy.—When failure of compensation, myocardial degeneration, and dilatation occur, edema of the extremities, and particularly of the feet, is present. In mitral regurgitation with secondary tricuspid regurgitation edema of the extremities is common, but it occurs less often in mitral obstruction and in disease of the aortic leaflets. Later there may be ascites and effusion into the pleural sacs.

Dyspnea.—Varieties.—(*a*) *Dyspnea Following Exercise.*—This is readily distinguished from other types of difficult breathing by the fact that the attack always follows exertion, although late in advanced cardiac disease but slight exertion is required to precipitate an attack.

(*b*) *Paroxysmal dyspnea* develops without apparent exciting cause, and may attack the patient during sleep, and, indeed, this type of dyspnea resembles somewhat that seen in uremia and in asthma, and is to be distinguished from these only by a careful study of the renal and pulmonary systems, and the exclusion of disease of both the lung and the kidney. In cardiac dyspnea the effort is made both at inspiration and at expiration, a feature that distinguishes it from asthma. Again, the remedies that relieve cardiac dyspnea are of but limited value in those cases in which the lung is diseased. (See Asthma, p. 96.)

(*c*) *Orthopnea* is a form of dyspnea in which, irrespective of the condition or conditions that have induced the symptom, the patient must of necessity remain in the erect posture.

(*d*) *Arhythmic dyspnea* (*Cheyne-Stokes respiration*) is that form of difficult breathing in which there is a respiratory pause of from one-half to three-quarters of a minute, which alternates with a period of increasing respiratory activity consisting in twenty or more respirations. The force and depth of the respirations also vary. (See Cheyne-Stokes Respiration, p. 51.) During the pause the patient may be unconscious and the pupils contracted, but with the beginning of an increase in the frequency of the respirations the pupils usually dilate. (See p. 51.)

Nervous Phenomena. — This class of symptoms is probably the result of altered cerebral circulation—(*a*) because of insufficient blood-supply to the brain; or (*b*) because the blood-supply to the brain is imperfectly

oxygenated (passive congestion; cyanosis). In either event it is customary for the patient to complain of repeated attacks of vertigo, languor, and faintness. In those cases in which the cerebral symptoms are but slight, the patient's mind is dull, and he may experience a variable degree of stupor; delirium may occur late during cardiovascular disease.

In cardiac disease it is possible to have true epilepsy or epileptiform seizures, and these are, as a rule, attributable to either embolism or thrombosis. Choreiform movements may be seen, and are possibly dependent upon pathologic conditions similar to those producing epilepsy.

In those patients who exhibit extensive atheroma of the cerebral vessels and hypertrophy of the left heart, hemorrhage into the brain is quite frequent, and is accompanied by paralysis and other somewhat characteristic symptoms. (See Cerebral Hemorrhage, p. 1050.) Cerebral hemorrahge may also occur during the course of valvular disease. (See Cerebral Embolism, p. 1051.)

Delirium.—Owing to causes probably similar to those previously outlined in connection with the nervous features of cardiac disease, there may be a more extensive atheroma of the cerebral arteries, in which case mental enfeeblement, loss of memory, and even maniacal delirium may be seen.

The Stokes-Adams Syndrome.—(See p. 190.)

Renal Symptoms.—Disease of the kidneys is by no means uncommon during the course of organic heart disease, and although the renal system is situated remotely, it must be considered in connection with every case suffering from cardiovascular changes. When compensation is lost, particularly in cases of tricuspid regurgitation, mitral regurgitation, or cardiac dilatation, the quantity of urine excreted is diminished, and the fluid is highly colored, rich in solids (urates), and is likely to contain albumin and casts. If the circulatory disturbance is of long duration, organic disease of the kidney will result. (See Nephritis, p. 661.)

In chronic cardiac disease in which a high grade of atheromatous change takes place in the arteries, together with sclerotic changes in the kidneys, the urine is often increased in quantity, pale in color, of low specific gravity, poor in solids, and will display a trace of albumin and a few casts. Hematuria results from renal embolism, although it may follow an acute exacerbation of chronic interstitial nephritis. Generally speaking, renal disease should be regarded as quite commonly associated with cardiovascular changes, and, indeed, it is at times quite difficult to separate the symptomatology of these two conditions. (See Nephritis, p. 674.)

Gastro-intestinal Phenomena.—Repeated attacks of **catarrhal gastritis** and of **enteritis** are commonly encountered, and the patient frequently complains of repeated attacks of indigestion, nausea, and vomiting. Owing to cyanotic congestion, hemorrhage into the stomach and vomiting of blood may take place. (See Hemorrhage from Stomach, p. 432.)

Flatulency is also attributable to cardiac disease, and when this symptom becomes especially annoying, it may induce palpitation and even cardiac pain. (See Palpitation, p. 171.) If gastro-intestinal symptoms become conspicuous, there is an associated passive congestion of other of the abdominal viscera, in consequence of which enlargement and pathologic changes take place in both the spleen and the liver. (See Cause for Enlargement of the Liver, p. 586.)

Throat Symptoms.—Pain in the region of the throat is an occasional complaint, and, as a rule, such pain is paroxysmal and is frequently associated with angina pectoris. Thoracic aneurism, by pressure upon certain of the structures at the upper portion of the chest, may give rise to intense pain in the throat, reflected over the region of the clavicles and scapulæ. In neurasthenic individuals distress in the throat frequently accompanies palpitation.

Alterations in the voice are the result of pressure either upon the recurrent laryngeal nerve or other vital structures.

Special Symptoms Referable to Arterial Disease.—It is practically impossible to isolate this group of symptoms from those referable also to disease of the heart, owing to the fact that cardiac and vascular diseases are, as a rule, associated. Nevertheless, a brief description of certain definite symptoms that result directly from degenerative changes of the peripheral vessels may prove useful. Headache, tinnitus aurium, vertigo, photophobia, impairment of vision, and paresthesia may result either from congestion or from anemia of the brain. The latter condition is probably the more common cause, since the degenerative changes present in the peripheral arteries prevent the normal distribution of blood in the extremities, and, as a consequence, we find the hands and feet cold as an expression of disease of the blood-vessels.

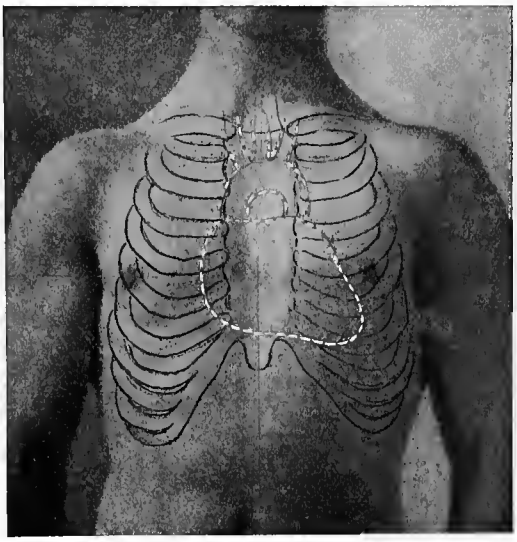


FIG. 56.—AREA OF NORMAL HEART, AORTA, AND ITS LARGER BRANCHES.

Pain is absent, irrespective of the degree of arterial disease present, unless there is an aneurismal expansion of some one of the vessels. The smaller vessels are likely to rupture, causing hemorrhages into various organs—*e. g.*, into the retina. Throbbing of the vessels, as previously mentioned (p. 172), is an almost positive sign of arterial degeneration, although it is encountered rarely in neurasthenics.

TOPOGRAPHY OF THE HEART.

In order to recognize the abnormalities of the heart in disease, a knowledge of its normal outline is necessary. The base of the heart, or that portion giving off the great vessels, corresponds above to a line drawn across the sternum, and continued a half-inch to the right and one inch, or possibly one and one-half inches, to the left of the sternum. (See Fig. 56.)

The base of the heart is represented by a line drawn from the upper border

of the third costal cartilage, one-half inch to the right of the sternum, to the lower border of the second costal cartilage, one inch to the left of the sternum. The right border of the heart is represented by a line that starts at the upper border of the third costal cartilage, one-half inch to the right of the sternum, and ends at the apex. At first this line follows the right border of the sternum until it reaches the level of the sixth costal cartilage; then it turns and passes transversely, with its convexity downward, just above the articulation between the second and third pieces of the sternum. The upper portion of this line corresponds to the right border of the right auricle; the lower portion, to the right border of the right ventricle (Fig. 57).

The left border of the heart is represented by a line that runs from the lower border of the second costal cartilage, one inch to the left of the sternum, with a slight curve to the left, to the apex (Figs. 56, 57).

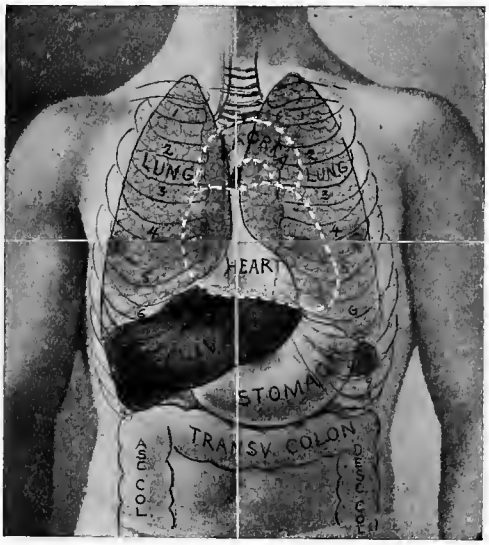


FIG. 57.—WHITE OUTLINE OF NORMAL HEART AND AORTA.
Shows also relation to other thoracic and abdominal viscera. Transverse line at nipples.

The apex of the heart is situated in the fifth interspace, three inches from the midsternal line.

VARIATIONS IN HEALTH.

In healthy persons the outline of the heart, as determined on the chest, may vary under certain physiologic conditions, *e. g.:* (*a*) As the organ swings from right to left, and from below upward, and from the sternum backward, the boundaries of the heart are temporarily changed, the outline as above (Fig. 57) being applicable when the patient is standing or sitting.

(*b*) The respiratory act, and particularly that of deep inspiration, may materially alter the left boundary of the heart, and may also depress the apex.

(*c*) If the patient lies upon his left side or upon his back, the relation of the heart to the external landmarks is likely to be altered.

(*d*) During respiration the movement of the ribs (upward and downward), as well as the rhythmic contraction of the diaphragm, influences slightly both the upper and lower boundaries of the heart, whereas in forced inspiration and expiration there may be an appreciable change in the position of the organ.

(*e*) Lastly, age is to be considered in connection with the relation of the heart to its external boundaries, and, generally speaking, during childhood the heart is situated approximately one rib higher in the chest than it is in adult life, and in most children the area of absolute dullness is proportionately greater than it is in older subjects (proportionately less of the organ is overlapped by lung). In aged subjects the heart is found at a lower level

than it is during early adult and middle life, the apex corresponding to the lower border of the sixth rib, and it may reach the sixth interspace; singularly, in this connection, the portion of the heart that is in direct contact with the chest-wall does not increase in size, and may be somewhat smaller than that found in young adults.

Landmarks.

As shown in the accompanying illustrations (Figs. 56, 58, and 59), the apex of the heart points downward and to the left, the base being directed slightly upward and toward the right; it is further seen that the greater portion of the organ rests in the left half of the chest, and that only a portion of the right side of the heart extends beyond the right border of the sternum.

(1) If a needle is introduced directly from before backward and in the middle of the third, fourth, or fifth intercostal space, at a point near the right margin of the sternum, it would pass first through the chest-wall, through the parietal and visceral layers of the pleura, through that portion of the lung overlapping the auricle, and thence directly enter the right auricle (Fig. 59).

(2) A needle passing through the center of the first intercostal space and at the right border of the sternum goes through a portion of lung and then enters the superior vena cava immediately above the pericardium.

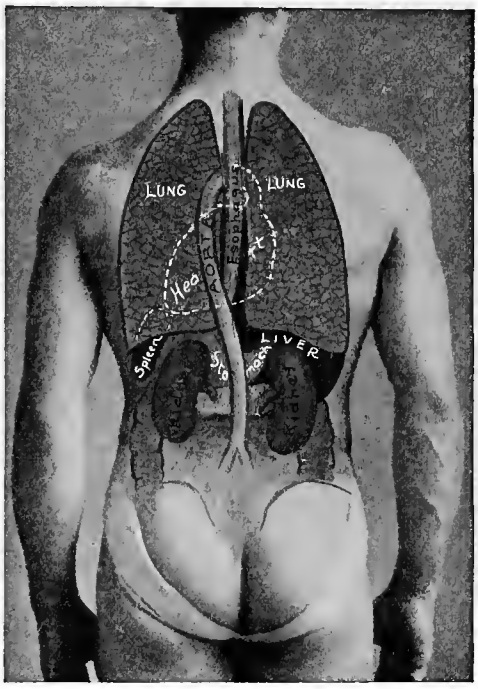


Fig. 58.—Posterior View, Showing Relation of Heart and Aorta to Lungs, Esophagus, and Viscera of Abdomen.

Topographic Relation of the Heart to the Lungs.

Anteriorly, the entire surface of the heart, except an irregular, imperfectly triangular or quadrilateral space that corresponds to the right ventricle, is overlapped by lung tissue. On account of the indentation at the anterior border of the left lung the quadrilateral area just mentioned is not covered by pulmonary tissue (Fig. 59).

The anterior borders of both lungs descend from the apices and approach each other in the median line, at a level with the second costal cartilage (Fig. 59), and from this point descend in almost direct apposition one with the other until they reach the fourth costal cartilage (Fig. 59). At this point the anterior borders of the lungs diverge, the right passing downward and outward to the fifth cartilage and the fifth interspace (Fig. 59), meeting the inferior border at the sixth rib in the midclavicular line.

At the fourth costal cartilage the anterior border of the left lung is reflected directly outward to the outer margin of the fourth costal cartilage, whence it passes obliquely to the fourth interspace at the parasternal line; from this point the lung is reflected inward and again downward and outward across the fifth rib to the fifth interspace (a course transcribing imperfectly the letter **S**), where it unites with the inferior border of the left lung at the sixth rib. (See Figs. 58 and 59.)

The boundaries of absolute cardiac dullness are as follows: The superior border corresponds to a line drawn along the lower edge of the left fourth rib, on the right, a line drawn from this level and the left sternal line, while the left boundary extends slightly outside of the left parasternal line, below the fifth interspace, and at a point where the heart overlaps the left lobe of the liver (Fig. 59). In those suffering from pulmonary disease, *e. g.*, emphysema, the area of absolute cardiac dullness may be gradually reduced, owing to overdistention of the lung tissue; again, in sclerotic changes in the lung there may be retraction, in which case the area of superficial dullness is increased. For practical purposes the area of absolute cardiac dullness is closely outlined in the following manner: (*a*) Draw a line to connect the lower border of the left sternoclavicular articulation with the left nipple, and divide this line into three equal parts. (*b*) Utilize the junction of the middle with the inferior third of the oblique line as the center of a circle that is two inches in diameter.

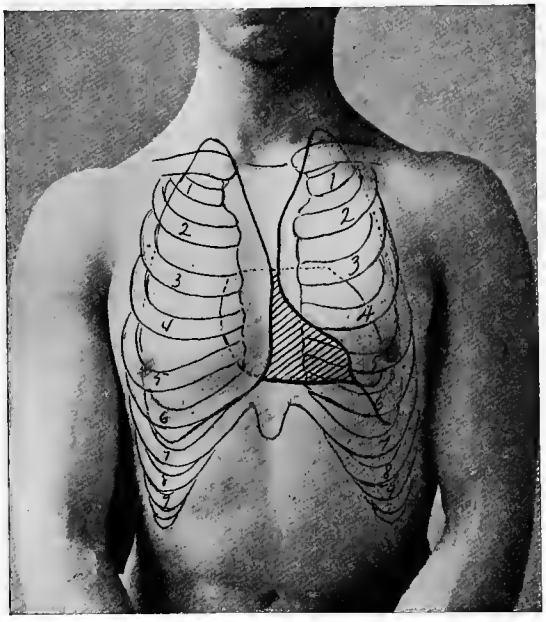


FIG. 59.—AREA OF ABSOLUTE CARDIAC DULLNESS DEFINED BY BORDER OF LUNG

POSITION OF CARDIAC VALVES.

The valves of the heart are situated fairly close together, in the neighborhood of the third and fourth chondrosternal joints. The pulmonary valve is the most superficial; the tricuspid is posterior to it, the aortic is next in point of distance from the anterior chest-wall, and the mitral valve is the deepest. The pulmonary valve is situated behind the third chondrosternal joint; the aortic valve is located behind the left edge of the sternum, at the third interspace; the mitral valve lies behind the fourth chondrosternal articulation, and the tricuspid valve is situated in the midsternal line, opposite to the fourth interspace (Fig. 60).

The anatomic location of these valves, however, must not be confused

with the points at which the clinician listens to determine the sounds made by them during a cardiac cycle (Fig. 61). These points for auscultation are determined by the nearest superficial point to which the flow of blood will conduct the sound made by the valve in question. The sounds made by the aortic valve are best heard at the second right interspace, because the aorta lies nearest the chest-wall at that point; the sounds made by the pulmonary valves are heard best at the second left interspace (Fig. 61), because the pulmonary artery lies nearest the chest-wall there; the sounds made by the tricuspid valve are heard best at the ensiform cartilage; and those made by the mitral valve are best heard at the apex (Fig. 62).

In children the heart and its valves are situated practically one rib higher, while in old age the entire organ is one rib lower than that herein given as normal.

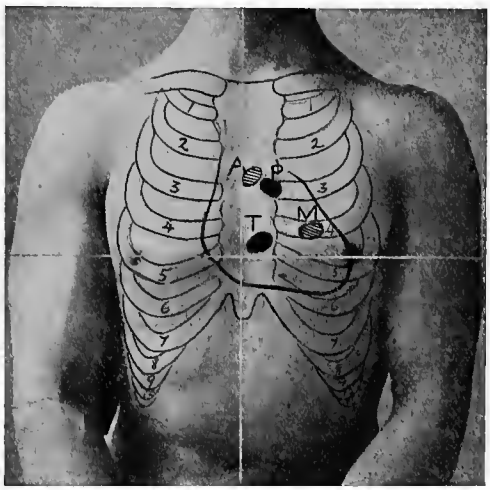


FIG. 60.—NORMAL RELATION OF CARDIAC VALVES TO HEART AND SURFACES OF CHEST.

THE PRECORDIUM.

From the foregoing remarks with reference to the topography of the heart and the relation of the organ and its valves to definite points upon the chest-wall it becomes immediately apparent that many definite physical signs of cardiac disease are to be found within this area. For practical purposes, the precordial space may be said to form a rectangular area located on the anterior surface of the chest. It is bounded above by a line drawn along the lower borders of the second ribs; below, by a line drawn along the upper borders of the sixth ribs; on the left, by the left midclavicular line; and on the right by the right parasternal line (Fig. 63).

INSPECTION OF THE HEART.

Here, as in examining the lungs, the anterior surface of the chest should be bared, and, still more satisfactory is it to remove all garments from the upper half of the body during inspection. The patient should be placed under a somewhat strong direct and oblique light, and the best results are to be obtained by inspection conducted by daylight. The patient does not need to assume any special position during inspiration, but when it is possible to change the position, it is most satisfactory to examine the patient first in a sitting or standing, and then in the recumbent, posture. Change of posture is of definite value not only in inspection, but also in performing palpation and auscultation; the variations resulting from the position of the patient, even though slight, always suggest a clinical factor that may be of importance in formulating a diagnosis. It should be the custom to examine not only the precordium and the surface of the chest in cardiac diseases, but also to inspect the chest carefully from apex to base, the

carotid regions, the temporal regions, and to follow the course of the great vessels—axillary, brachial, etc.

The condition of the fingers—whether clubbed, cyanotic, etc.—is also an important point to ascertain in connection with cardiac disease. The complexion, the degree of lividity, the presence or absence of cyanosis of the lips and buccal mucous membrane, and prominence of the eyes (exophthalmos) are important factors. The frequency and character of the respiration and the presence or absence of localized or general edema are points not to be neglected in inspecting those believed to be suffering from disease of the heart.

EXAMINATION OF THE PRECORDIUM (*Fig. 63*).

In making an examination of this particular area of the chest the following points are noted: (*a*) The degree of prominence; (*b*) the impulse and general character of pulsation present, and whether or not such pulsation is synchronous with that of the cardiac systole; (*c*) the degree of prominence or depression of the interspaces, and whether such alterations are general or local, and any other changes that may be apparent either at the time of systole or of diastole.

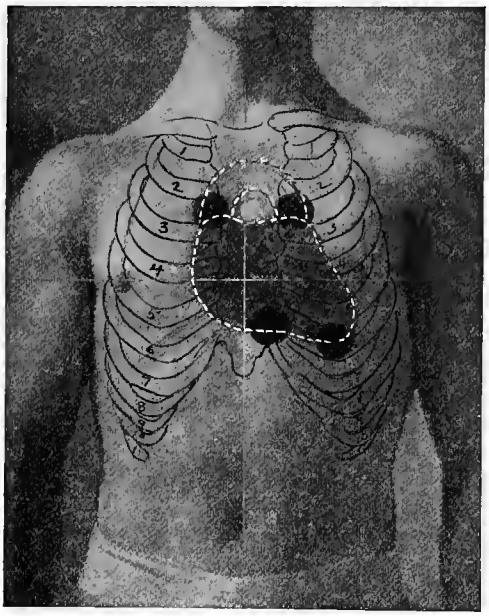


FIG. 61.—● AREAS WHERE DIFFERENT CARDIAC MURMURS ARE MOST CLEARLY AUDIBLE.

Age is a factor that bears upon bulging of the precordium, since this region is unduly prominent in children who may have suffered from only slight cardiac disease; in rachitic subjects, however, the prominence is especially conspicuous, its character varying greatly with the location of the initial bone disease. Both cardiac hypertrophy and dilatation give rise to undue prominence of the precordium, as does also a large pericardial effusion; in the latter condition, however, both the ribs and the interspaces are unusually prominent. Thoracic aneurism may also cause bulging over this area, but whenever it attains considerable size, bulging is likely to extend beyond the precordial region.

Prominence attributable directly to cardiac or pericardial disease is usually localized between the third and seventh ribs, to the left of the sternum, although in extreme cases there may be bulging extending from the right to the left nipple. Cardiac aneurism causes a bulging in the right nipple region, where the apparent apex-beat may be observed. An example of this condition recently came under the care of one of us at the Philadelphia Hospital, the diagnosis being confirmed at autopsy.

Extracardiac Conditions That Cause Undue Prominence.—Extracardiac conditions that give rise to undue prominence of the precordium

are: (1) Localized pleural effusion; (2) a pointing empyema; (3) thoracic aneurism; (4) enlarged bronchial glands; (5) any type of new-growths of the mediastinum.

Depression of the Precordium.—This may be either uniform or localized, and may result from some one of the following pathologic conditions: (*a*) Rachitic deformity of the thorax; (*b*) chronic pleural adhesions; (*c*) left-sided empyema, which may be followed by partial obliteration of the pleural sac and precordial depression; (*d*) chronic adhesive pericarditis; (*e*) fibroid tuberculosis of the left lung; (*f*) pulmonary tuberculosis with extensive cavity-formation in the left lung; (*g*) it may rarely be seen to follow traumatism of the left side of the thorax.

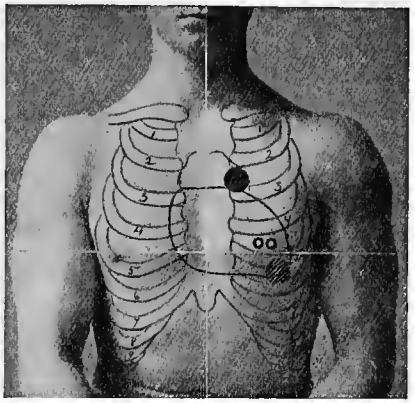


FIG. 62.—● WHERE PULMONIC SECOND ACCENTUATION IS BEST HEARD; ◍ WHERE, SYSTOLIC MITRAL IS BEST HEARD; oo AREAS WHERE PRESYSTOLIC MITRAL IS CLEARLY AUDIBLE.

Pulsation.—Apex-beat.—The impulse normally present in the precordium is that generally referred to as the apex-beat. During health this is felt in the fifth left interspace, just inside the midclavicular line; it is due to the contraction of the left ventricle at a point from three-quarters to one inch above the apex. This normal impulse of the precordium is synchronous with the first sound of the heart, and consequently is systolic; whenever, therefore, any other pulsation in the precordium is visible, it is fair to suspect, at least, that a pathologic condition of the heart, pericardium, great vessels, pleura, or lung is present or has previously existed. After repeated examinations one becomes familiar with the normal appearance of the impulse of the apex-beat, any deviation from this being regarded as abnormal.

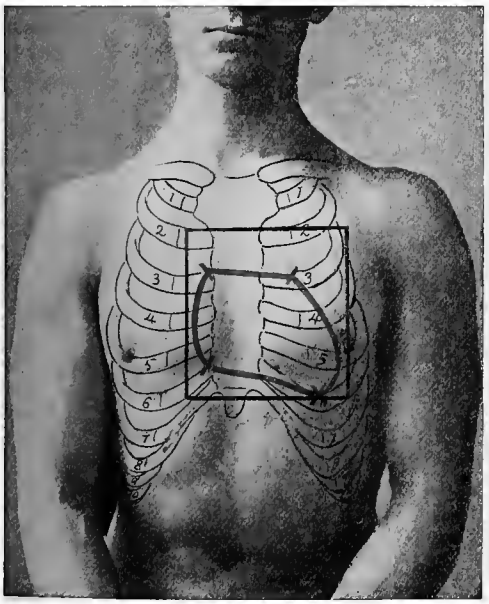


FIG. 63.—THE PRECORDIUM.
Relation of heart to bony structures forming the anterior chest-wall.

The Apex-beat in Disease.—The apex-beat may not be visible when extensive myocardial changes or a thick chest-wall, the result of obesity, are present. In pathologic conditions the apex-beat may be palpated and yet not be visible, and in certain cases the reverse obtains.

Displacement.—Displacement of the apex-beat is an extremely important finding in pathologic conditions, furnishing, as it does, a valuable guide to the location of the heart; when seen, it is of further clinical value, because it accords with the ventricular systole. The apex-beat may be displaced either as the result of actual change in the position of the heart from alterations the result of cardiac hypertrophy or of external pressure. Deformity of the chest is also a prominent factor in causing displacement of the apex-beat.

(1) *Displacement Upward and to the Left.*—This may result from—(*a*) A large pericardial effusion, and it is not uncommon to see a moderate amount of upward displacement in distention of the abdomen due to the presence of ascitic fluid, gas, or abdominal tumor.

(*b*) Fibroid changes of the lung and of the left pleura, such as are occasionally seen in tuberculosis and following interstitial pneumonia, may cause the organ to be displaced and the apex-beat to appear at the third interspace, or even higher upon the chest.

(*c*) If the left pleura is markedly distended by fluid or by fluid and air, the apex-beat is displaced to the right.

(*d*) Mediastinal tumor may give rise to cardiac displacement, and cases of this kind have come under our observation in which the apex-beat has been displaced to the left antero-axillary line.

Caution.—It is to be remembered that during infancy and in children under two years of age the normal position of the apex-beat is in the fourth interspace, and in or just outside the midclavicular line.

(2) *Displacement to the Left.*—Left displacement of the heart in a horizontal line is occasionally seen in those conditions specified under Displacement Upward and to the Left, but the chief causes of horizontal displacement are: (*a*) Cardiac hypertrophy; (*b*) dilatation of the right ventricle, in which case the impulse of the apex may be seen to the left of the midclavicular line.

(3) *Displacement Downward and to the Left.*—This is a characteristic feature of—(*a*) Hypertrophy of the left ventricle, and (*b*) dilatation of the left ventricle following hypertrophy, in either of which cases it is not unusual to see the apex impulse between the sixth and eighth interspaces and in the anteroaxillary or midaxillary lines.

Downward Displacement not Dependent upon Cardiac Disease.—(*a*) In well-advanced cases of emphysema the apex-beat may be seen at a low level, first, owing to the fact that the heart is depressed by the overdistended lung; secondly, on account of the inclination of the ribs in this disease; and, lastly, hypertrophy of the right ventricle appears to aid in lowering the apex-beat.

(*b*) Enlargement of the arch of the aorta, or even of the innominate artery, may, by pressure, depress the apex-beat.

(*c*) Mediastinal tumors of whatever nature, if occupying the upper portion of the mediastinum, displace the apex-beat below its normal site.

(*d*) Enlargement of the liver, as in leukemia, and septic conditions, as hypertrophic cirrhosis, may, by making constant traction upon the diaphragm, cause the apex-beat to be drawn downward on account of the pericardial attachment to the diaphragm.

Displacement of the Apex-beat to the Right.—This is a characteristic of—(*a*) Left pleural effusion; (*b*) left pyopneumothorax; (*c*) fibroid changes in the right lung and pleura, with adhesions to the pericardium; (*d*) tumor of the left lung.

Forcible Apex-beat.—The apex-beat may be forcible as the result of cardiac hypertrophy, but it is also a feature of pericarditis, pericardial adhesions, and acute endocarditis (early stage), and is naturally present in diseases that produce increased arterial tension—*e. g.*, chronic nephritis, atrophic cirrhosis, and pulmonary and arterial affections.

Rhythm of the Apex Impulse.—This point may be determined more satisfactorily by palpation than by inspection. Inequality of force, frequency, and intermittency all point somewhat directly toward disease of the heart muscle.

Double Apex Pulsation.—This phenomenon consists in two pulsations occurring at the apex to each pulsation of the carotid artery. One theory advanced for this unusual finding is that the ventricles do not contract synchronously; another, is that alternating weak and strong contractions of the heart occur, certain waves being too feeble to be transmitted as distinct pulse-waves.

Systolic Recession.—This condition is seldom limited to the area of the apex-beat alone, but a rhythmic depression of that portion of the chest overlying the lower portion of the heart is observed, and is a condition usually attributed to pericardial adhesions, to mediastinal tumors, and enlargement of the heart. Systolic recession may accompany *Broadbent's sign*, which consists in a similar rhythmic retraction of the eleventh and twelfth left interspaces posteriorly.

Pulsation at the Base.—There are a number of conditions capable of causing pulsation in the upper portion of the precordium. These, named in the order of their clinical importance, are: Aortic aneurism, enlargement of the heart, cardiac dilatation, pericardial effusion, retraction of the lung due to pulmonary disease, and adhesive pleurisy.

Pulsation seen at the right first or second intercostal space and near the sternal border is suggestive of the presence of aneurism. At the previously mentioned points it is seldom possible to detect true expansile pulsation, consequently other clinical signs must be searched for in order to make a diagnosis of aneurism. Expansion in this region may be due to the forcible beats of the heart, and, indeed, it not infrequently occurs as the result of the heart having been drawn out of place by pleural adhesions. These adhesions and pulmonary diseases that cause retraction of the right lung may also be the cause of precordial pulsation at the right second, third, or fourth interspaces, since in this region the right lung covers the right auricle, and whenever this portion of the heart is exposed, the pulsation becomes presystolic. Pulsation in the right third, fourth, and fifth interspaces, when it extends to the right parasternal line, is, as a rule, the result of displacement of the heart. The conditions that are capable of causing pulsation in this area are: (1) Left pleural effusion; (2) left pyopneumothorax; (3) left subdiaphragmatic abscess; (4) left empyema; (5) right pleurisy with adhesions to the pericardium.

Sternal Pulsation.—Pulsation over the sternum is invariably indicative of erosion of the bony structures, and is a common sign, occurring during the course of aneurism of the aortic arch and of the innominate artery.

Pulsation at the Left of the Sternum.—When pulsation is detected in the left second or third interspaces, near the margin of the sternum, it is usually arterial, and may result from retraction of the lung; after exposure of the pulmonary artery, however, it is systolic in nature.

Pulsation due to exposure of the left auricle precedes the apex-beat, whereas that due to aneurism is synchronous with or follows soon after

the apex impulse. A diastolic impulse may be detected in this region during the course of profound secondary and primary anemias, and, indeed, a systolic pulsation is not impossible in these pathologic conditions.

Pulsation at the upper portion of the chest and to the left of the sternum, while suggestive of cardiac or arterial disease, should also suggest the possibility of some existing pulmonary condition.

When pulsation is prominent at the region of the third, fourth, and fifth interspaces, between the left margin of the sternum and the left parasternal line, it is, as a rule, dependent upon hypertrophic dilatation of the right ventricle, although it is strongly suggestive of displacement of the heart; such displacement may result from right pleural effusion, right

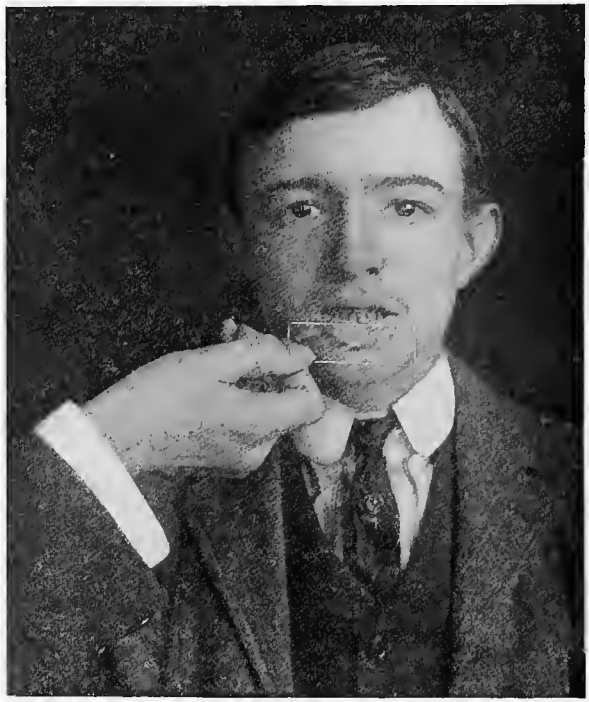


FIG. 64.—PRESSURE OF GLASS SLIDE OVER LOWER LIP TO DETECT CAPILLARY PULSATION.

pneumothorax, right subdiaphragmatic abscess, and left pleuritis with extensive adhesions to the pericardium.

Double Impulse.—An apparently double impulse, occurring more or less synchronously with each systole, is rarely observed in selected cases of mitral insufficiency.

Pulsation Outside the Precordium.—In the Supraclavicular and Carotid Regions.—Distinct pulsation in the right supraclavicular space is a feature of aneurism of the innominate and subclavian arteries, and is less often seen in aneurismal dilatation of the carotid artery. A distinct wave-like pulsation that may be prevented by placing the finger over the vein in this region is, as a rule, the result of tricuspid regurgitation. In the latter condition the pulsation may be systolic, and the wave be seen

well up in the carotid region. In aortic regurgitation there may be marked pulsation of the carotid vessels.

Pulsation over the right supraclavicular and carotid regions is usually the result of aneurism or of decided atheroma of the vessels occupying these sections. In cardiac hypertrophy there may be distinct pulsation in both carotid regions.

Epigastric Pulsation.—Pulsation in the epigastrium is most often the result of a dilated right heart, and is quite commonly seen in tricuspid regurgitation.

Pulsation in the scapular and axillary regions is practically always induced by aneurism.

Pulsation Over Remote Arteries.—In aortic regurgitation with well-marked atheromatous change of the arteries distinct arterial throbbing is to be seen over the axillary, brachial (Fig. 115), radial, femoral, and temporal vessels. Upon close inspection those cases showing marked arterial

Fig. 65.—Pressing Nail of Patient's Forefinger to Detect the Capillary Pulse.

changes generally display a capillary pulse of the lip and of the fingers (Quincke's capillary pulse).

Quincke's Capillary Pulse.—The patient's finger is grasped and his nail pressed gently by the thumb-nail of the examiner (Fig. 65); where, if held in a proper light, and if Quincke's capillary pulse is present, the pink line underneath the nail will be seen to advance and to recede with each pulsation of the heart.

Another expression of the capillary pulse is obtained by placing a glass slide over the lip, and exerting gentle pressure, when, if Quincke's pulse is present, it will readily be detected underneath the slide (Fig. 64). Another method of detecting the capillary pulse is performed as follows: By drawing the finger somewhat forcibly across the chest-wall it will be noticed that the skin along the track made by the finger first becomes pale, then flushed, then pale again, then slightly flushed, and finally pale, until it resumes the color of the surrounding skin. The capillary pulse is commonly seen in aortic regurgitation.

Distinct pulsation of the abdominal aorta may depend upon an aneu-

rismal dilatation of this vessel, or the pulsation may be transmitted through a solid viscus or growth.

Attitude of the Patient.—The position assumed by the patient is of but limited clinical importance in the majority of cardiac and vascular diseases. In acute plastic pericarditis, however, and in angina pectoris, the attitude is somewhat characteristic.

PALPATION.

Palpation serves, for the most part, to confirm the signs already revealed by inspection, and with reference to abnormal pulsation, detected either over the precordium or over other portions of the thorax or abdomen. In many instances it is quite essential that the signs obtained on inspection be confirmed by palpation.

So intimately connected are evidences revealed by inspection and by palpation that, clinically, both these physical methods should be employed at the same time. In examining the precordial area, palpation further acquaints us with the force of each pulsation analyzed, and determines whether or not such pulsation is expansile. Again, by palpation one is able to determine not only the degree of an impulse, but also its rhythm, and at the same time ascertain the resistance offered by the overlying tissue (the chest-wall). It is all important to detect, by palpation, whether abnormalities of the chest are the result of superficial edema or of deeper seated pathologic conditions.

In palpating to ascertain the presence of cardiac disease, probably second in importance should be mentioned the recognition of thrills, the clinical significance of which will be discussed at length under each particular cardiac and vascular condition wherein they form one of the physical signs. The friction fremitus, whether pericardial, pleural, or pleuro-pericardial, is elicited by means of palpation; its presence is an invaluable sign in both pericarditis and pleuritis.

Palpation over the great vessels may bring to light the most pronounced clinical evidence of the existence of both cardiac and vascular disease, and is important in estimating the force of the pulse, arterial tension, cardiac rhythm, and the influence of exercise and exertion upon the circulatory system.

PULSE.

Definition.—A sensation conveyed to the palpating finger as the result of the beating of the arteries, produced by the afflux of the blood propelled by the heart in its contractions.

In our opinion the tendency at present is to attach too limited an importance to a study of the pulse in disease, and to depend on other conclusive physical and laboratory methods to convey the facts elicited by the various characteristics of the pulse.

Physical Technic.—In making a study of the pulse the position of the patient must be considered, since both posture and mild exercise increase the pulse-rate and also influence, to a lesser degree, the other clinical characteristics of the pulse. The radial pulse is practically always palpated. The patient's arms should be allowed to rest upon the side of the bed or over his chest, or, if sitting, they should lie in the patient's lap. The hand of the patient may be held by the hand of the physician while the pulse is being examined. The finger of the examiner should be placed just above and inside the styloid process of the radius, and the index-finger should be

directed toward the heart. Clinicians differ as to the number of fingers that should be placed over the artery, some using only the index-finger, whereas others use the index-, second, and ring-fingers. Both radial pulses should be examined, since in certain maladies they differ in many respects. In palpating both the right and the left radial pulse at the same time it is impracticable to have the index-finger on each radial directed toward the heart, although this may be done if the physician assumes an awkward position. Palpating both radials at the same time is further necessary because of misplacement of the radial artery, or one pulse may be absent as the result of disease (aneurism).

The pressure exerted when palpating the pulse should at first be extremely light; when the pulse is full, strong, and bounding in character, pressure may be increased. (See Method of Compressing Pulse, p. 197.)

Evidence to be Obtained.—Physiologic Frequency.—Under normal conditions the pulse-rate will be found to vary within quite wide limits. The average number of beats for the male adult is 72 a minute, whereas in adult females it is slightly higher,—74 to 76 a minute,—although a pulse of 80 is not considered abnormal for certain females. The influence of temperament and certain personal idiosyncrasies may cause the pulse-rate to fluctuate between 60 and 80 beats a minute during early adult and middle life. In the new-born and during the first year of life the pulse-rate varies from 120 to 140 beats a minute; during the third year, under normal conditions, it drops to about 100, and by the fifth or sixth year a pulse of 90 to 95 beats is normal.

A normal pulse may be decidedly accelerated as the result of exercise, mental strain, anxiety, and excitement. Following a full meal, and during the later hours of the day, the pulse is somewhat more rapid than during the morning hours. Atmospheric temperature may exercise an appreciable influence upon the frequency of the pulse; consequently in extreme heat the number of beats a minute may be moderately accelerated. During health the attitude of the patient causes a variation of from two to four beats a minute.

Under normal conditions the pulse-rate may be unusually slow—the so-called "congenital slow pulse." In the aged a pulse of 60 is not uncommon.

Pathologic Frequency.—*Pulsus Frequens.*—The pulse-rate is increased, as a rule, in cardiac disease, with the exception of aortic stenosis. Cardiac neuroses are usually classed as nervous palpitation, and may result from the excessive use of stimulants and narcotics, as tea, coffee, alcohol, tobacco, and from venereal excesses. A similar condition occurs in exophthalmic goiter, as well as in paralysis of the vagus and irritation of certain portions of the sympathetic system. (See Fig. 66; also Sinus Irregularity, p. 194.)

Slow Pulse.—A pulse is said to be slow when the number of beats per minute is less than 70. A slow pulse is commonly seen in the aged afflicted with arterial sclerosis, chronic nephritis, and angina pectoris. The pulse is also slow in aortic stenosis, and the wave may rise slowly as the result of thoracic aneurism.

Tachycardia.—This is a condition in which there is a decided increase in the frequency of the pulse. The abnormality may be either continuous or paroxysmal in character, the number of beats in either form reaching 200 or even more a minute (Fig. 75) (extrasystole). It is ofttimes extremely difficult to learn the actual cause of such pulse frequency, but the majority of these cases are explained as belonging to the class of cardiac neuroses.

In extensive myocardial degeneration, as well as in angina pectoris, a decided increase in the pulse-rate may occasionally be seen. (See Fig. 67.)

Extracardiac conditions are also responsible for an increased pulse-rate, and among these should be mentioned diseases of the abdominal viscera,

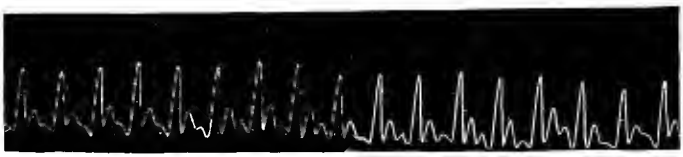


FIG. 66.—PULSUS FREQUENS (J. C. Da Costa, Jr.).

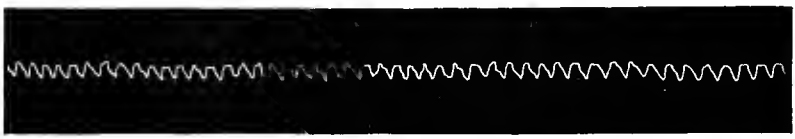


FIG. 67.—RADIAL PULSE DURING AN ATTACK OF PAROXYSMAL TACHYCARDIA (Anders).

FIG. 68.—PULSUS CELER IN AORTIC INSUFFICIENCY (Riegel).

FIG. 69.—PULSUS RARUS (J. C. Da Costa, Jr.).

FIG. 70.—PULSUS TARDUS IN AORTIC STENOSIS (Strümpell).

extreme anemia, either primary or secondary, and particularly any chronic condition that is characterized by marked weakness (exophthalmic goiter).

During the course of acute febrile maladies the frequency of the pulse serves as a valuable guide to the patient's general condition, and here we commonly find a pulse-rate of between 90 and 120 beats a minute.

Causes of Tachycardia.—The following table includes certain causes, of which one or more will be found to be an exciting factor in the majority of cases where tachycardia is a conspicuous feature.

PHYSIOLOGIC CAUSES:

Early during the period of gestation, During menstruation.

PATHOLOGIC CAUSES:

Exophthalmic goiter,
Chlorosis,
Secondary anemia,
Leukemia,
Mitral stenosis,
Myocarditis,
Cardiac dilatation,
Pericardial effusion,
Pernicious anemia.

Pneumogastric irritation by pressure from:
Enlarged mediastinal glands,
Dilatation of the esophagus,
Empyema,
Thoracic aneurysm,
Large pleural effusion,
Extensive mediastinal fibrosis.

FOLLOWING ACUTE INFECTIONS:

Diphtheria,
Influenza,
Acute articular rheumatism,
Chorea,
Scarlet fever.

Beverages when used to excess:
Tobacco,
Coffee,
Alcoholic stimulants.

The taking of certain foods may be responsible for attacks of tachycardia, as are also excitement, prolonged nervous strain, and fear.

Quick Pulse.—In this variety of pulse there is, as a rule, low arterial tension, and the pulse-wave makes a rapid ascent, but disappears almost immediately. (See Figs. 75 and 76, Extrasystole, p. 195.) A quick pulse is to be distinguished from a rapid pulse, the latter implying a high number of pulsations a minute. The so-called quick pulse is seen late during the course of acute febrile maladies, and is also present to a marked degree in aortic regurgitation—the so-called "water-hammer" or Corrigan pulse. (See Aortic Regurgitation, p. 269.)

Pathologic Infrequency.—*Pulsus Rarus.*—Bradycardia is the term used to describe a condition in which, owing to the slow action of the heart, 60 or fewer pulse-beats are recorded during the minute. In making a study of cases in which the pulse is between 40 and 60 beats a minute (Fig. 76, under Extrasystole), it becomes essential that the physician acquaint himself with the general temperament of the patient, and ascertain, also, whether or not an abnormally slow pulse is a family characteristic. As previously stated, an extremely slow pulse is found in diseases of the endocardium and of the myocardium, the former condition being best exemplified by the slow pulse of aortic stenosis, whereas the latter is seen when there is extensive myocardial degeneration (fatty degeneration), together with atheroma of the coronary arteries. It is not uncommon to find all these pathologic conditions of the heart present in the same individual, and such cases commonly display a pulse of between 50 and 30 beats a minute (Fig. 69).

Autointoxication may be responsible for an abnormally slow pulse, but in such cases there is, as a rule, increased arterial tension, which is best exemplified by the pulse of chronic nephritis (pulse of uremia), chronic indigestion, and jaundice. The pulse may also be slow in those suffering

from chronic poisoning by metallic substances, such as lead, and in those in whom there is undue irritation of certain of the nervous ganglia as the result of pressure from abdominal or thoracic tumors or effusions. Traumatism or other conditions that may effect moderate pressure upon the vagus may also be accountable for a slow pulse-rate. (See Fig. 76, p. 195.) There are many cases in which it is impossible to explain the cause of a slow pulse, which may persist for months or even years without any apparent inconvenience on the part of the patient, and many such cases come to autopsy in which atheroma of either the coronary arteries or of the arteries of the medulla is found. The heart's action must be learned by auscultation, and the rate compared with that of the peripheral pulse, since the systole may fail to emit a pulse-wave that is palpable at the wrist.

The *Stokes-Adams syndrome is characterized* (1) By slow pulse, indicating slow ventricular contractions or standstill of the ventricle; (2) by rapid pulsation in the veins of the neck, indicating auricular contractions that do not pass to the ventricles; (3) temporary dimness of vision; (4) temporary loss of consciousness; (5) vertigo, and (6) syncope and convulsive seizures. "The majority of the typical cases are unquestionably the result of heart-block" (Lewis). The slow action of the ventricle may be found apart from disturbed conduction, as well as from attacks of faintness, giddiness, and syncope. Well-marked slowing of the ventricle 30 to 50 beats per minute is rare in the absence of heart-block, but it is occasionally seen in selected instances, and is accompanied by nervous manifestations. There may also be seen cases where a decided slowing of the pulse is the result of early premature contractions, and these symptoms may also be combined with epileptic seizures.

Causes illustrative of the above statement have been reported by Lewis, James, Schmidt, and others. Lastly, it is to be borne in mind that both heart-block and epilepsy are not necessarily accompanied by a persistently slow pulse. It seems scarcely practicable that we should dwell at greater length in this volume upon the more refined clinical features that render a distinction between Stokes-Adams' syndrome and heart-block.

Heart-block.—Pathology and General Remarks.—The first record we find regarding this condition is that of Galibin,* who, in reporting a case where the pulse was 25 to 30 beats per minute, writes, "we have here a heart, the auricle of which sometimes contracted twice in the interval between two ventricular pulsations, and sometimes singly in the midst of a long pause instead of just before the systole of the ventricle." It was not until 1899 that Wenchebach and also His† both described heart-block in the human subject and pointed out its association with a lesion of the auriculoventricular bundle. The profession is indebted for our recent advance on this subject to the earlier writings of J. Mackenzie, which appeared between 1901 and 1906.

This combination of symptoms, known as heart-block, is seen in cases in which the bundle of His is involved in a disease process, and fails to conduct contractile impulses, which arise in the auricle, to the ventricle.

The extent of pathologic changes in the bundle are not always in direct relation to the grade of heart-block. Again lesions are, as a rule, not limited to the bundle, but on the contrary are part of an extensive process. Cases are also found where there are only slight degrees of apparent damage,

* Guy's Hospital Reports, 1875.

† Deutsches Archiv. f. klin. Med., lxiv.

yet in these dissociation has been complete and persistent. "Structural changes in the bundle are by no means infrequent in hearts in which the sequence of contraction is normal" (Lewis). This discordance in pathologic findings and symptoms doubtless arises from our inability to recognize the functional capacity of the remaining slightly damaged and undamaged fibers.

The nervous symptoms of heart-block unquestionably result from anemia of the brain, a fact substantiated by the experiments of Kussmaul and Tener, who found that by compressing the carotid arteries in normal male adults this act was soon followed by slow, deep, and sighing respirations, dilatation of the pupils, and moderate dizziness, to be later followed by unconsciousness. The influence of compression upon the carotid vessels has been further studied by Schiff and Hill, who produced unilateral nervous phenomena by pressing over one carotid artery.

It is of great clinical importance to remember that heart-block, whether partial or complete, is not of necessity the outcome of a destruction of tissue, since heart-block may result from stimulation of the vagus nerve. The experiments of Gaskell, which have been confirmed by Erlanger, show that by various degrees of compression of the bundle the clinical grades of the heart-block may be produced.

Heart-block is also brought about by the administration of certain toxic substances, *e. g.*, digitalis, adrenalin, physostigmin, muscurin, and aconite. It is less often seen as the result of asphyxia, and in selected cases of this class heart-block may not depend upon blood-pressure, vagal inhibition, cardiac dilatation, or an excess of CO_2. Mathison has shown that when heart-block occurs in this last variety of conditions it probably owes its onset to lack of oxygen, and perhaps to the accumulation of acid products in the cardiac musculature.

Rheumatism has been shown by Lewis to figure as a predisposing factor in over 15 per cent. of cases, and, indeed, it is rather common that a higher percentage of cases, displaying cardiac complications following rheumatic infection, are found to present evidences of impaired conduction.

Clinical Peculiarities.—Lewis found that an absence of the ventricular contraction for a period of from fifteen to twenty seconds to be a precursor of the epileptiform phenomena, and that the absence of the ventricular contraction for a period longer than ninety seconds was rarely followed by recovery.

Cessation of the ventricular action for a period of from two to two and a half seconds is usually followed by but slight disturbance, and when the absence of ventricular contraction continues over a period of from three to five seconds, momentary unconsciousness is customary. Following a pause of whatever length, the pulse-beats are detectable at the radial arteries before there is a return to consciousness.

Upon first appearance cases of heart-block display many, and at times practically all the features previously described under Stokes-Adams syndrome (p. 190). The evidence of clinical heart-block is definite in character, and especially is this true of observations recorded graphically when such records are compared with the records of experimental heart-block. In some cases displaying slow pulse, the fact that the auricle and ventricle are contracting at distinct rates may not only be observed, but definitely recorded. Rapid movements at the base of the heart over the auricle has been observed by many writers who have studied their cases radiographically.

The radial pulse is slow and the impression given by the palpating finger is in most cases a forcible one. The systolic blood-pressure is often high, and during the unusually long diastolic pause following each pulse-wave there is an appreciable drop in pressure. The characteristics of the radial pulse tracing when recorded by the Dudgeon instrument may display a rather complex curve (Fig. 77). By placing the stethoscope over the apex of the heart there are audible both dulled and distinct sounds. Curves obtained from the apex and the epigastrium also present evidences of auricular movements (Fig. 73).

Describing the venous curve it may be said that a definite group of three well-inscribed waves accompany each cardiac cycle, and whenever one of these lies directly in front of that point which marks the upstroke of c. this wave is recorded as a., and its upstroke represents the beginning of auricular systole (Fig. 93).

In heart-block a radiographic study of the heart's action doubtless approximates accuracy more closely than is found in any other clinical method now in use, and is discussed under Electrocardiogram. (See p. 217.)

Condition of the Vessel-walls.—By making firm pressure over the course of the radial artery it is possible to empty the vessel, and then, by careful manipulation, attempt to roll the artery under the finger-tips. If atheromatous changes are not present, it is difficult to palpate the radial artery distinctly, except in those in whom but a slight amount of extra tissue is present. If there is a moderate degree of atheroma, the artery has a somewhat cord-like or leathery feel. In marked atheroma both the radial and temporal arteries are extremely hard, and may even be pipe-stem-like in contour. There is probably no characteristic in the general clinical study of the pulse that is of greater prognostic value than an accurate determination of the degree of atheroma existing in each individual case. It may be well to add, in this connection, that where atheroma is extreme, the arteries assume a more or less tortuous course, and are seen to throb in the brachial and radial regions.

Size of the Pulse-wave and Artery.—While palpating to determine the degree of atheroma, gentle pressure is made by the index-finger as it is drawn along the course of the radial artery, and in this way an accurate determination of the actual size of the artery is readily apparent. With each pulse-beat one is at the same time forced to determine the size or volume of the pulse-wave, and since this factor depends entirely upon the myocardial systole and upon the tension of the arterial wall, it too furnishes valuable data as to the general condition of the heart.

Large Pulse.—In plethoric persons the pulse may be larger than normal, but hypertrophy of the left ventricle may give this same impression to the palpating finger. A large pulse, which, technically speaking, conveys only the size of the pulse to the palpating finger, may also result from an abnormally low arterial tension, as the result of which the radial artery is broader than normal. The best examples of large pulse are seen in cases of cardiac hypertrophy, in which there is little atheroma, and consequently the vessel-walls are flaccid and the pulse strikes a broad surface of the palpating finger. In regurgitation at the aortic valve the effect upon the artery may be similar to that just described. In acute febrile conditions, particularly during the early stage, the pulse may be bounding in character, and appears to be large to the palpating finger; atmospheric conditions—intense heat and humidity —are capable of producing, though to a less marked degree, this variety of

pulse. In chronic maladies in which there is general debility there may be an unusually large pulse as the result of general weakness, with a loss in the myocardial power.

Peculiarities.—During the course of mitral regurgitation with hypertrophy of the left heart the pulse may not be of abnormal size, and, indeed, the volume of the pulse may be smaller than normal, a condition that is amply explained when we consider that with each systole not all the blood in the left heart is forced into the arterial tree, but a portion of it, owing to the leakage present, is returned to the left auricle. Hypertrophy of the heart may be a feature of aortic stenosis, and yet, irrespective of such hypertrophy, it is impossible for a large volume of blood to enter the aorta.

FIG. 71.—PULSUS MAGNUS (J. C. Da Costa, Jr.).

FIG. 72.—PULSUS PARVUS (J. C. Da Costa, Jr.).

FIG. 73.—PULSUS PARADOXUS.
E, Beginniug of expiration; *J*, beginning of inspiration (Kussmaul).

Small Pulse (Pulsus Parvus).—Both disease of the heart with progressive weakness of the myocardium and disease of the arteries may produce an abnormally small pulse; by way of repetition, mitral stenosis exemplifies that character of small pulse resulting from the left heart being unable to propel the blood to the aorta. Again, in myocarditis there is not sufficient force exerted to propel the blood to the radials. During the course of chronic interstitial nephritis and in other chronic maladies characterized by prostration the pulse may be feeble and scarcely perceptible; in these cases it is often referred to as "*thready.*" A similar effect is produced upon the pulse of one side of the body by aneurism, and the so-called thready pulse is present toward the termination of such acute febrile conditions as peritonitis, pneumonia, and typhoid fever.

Clinical Significance.—The size of the pulse-wave may give but little,

if any, valuable data regarding the degree of ventricular force that is expended with each systole. It will be seen, when arterial tension is considered, that a strong pulse is not of necessity a large one, nor is a weak pulse necessarily a small one, since both of these conditions are influenced materially not only by the force of the heart, but also by the freedom of the blood-vessels and by arterial tension.

Rhythm.—Under normal conditions the pulse-waves succeed one another with regularity both as to time of contact with the palpating finger and as to the degree of force exerted by each impulse. In some apparently healthy persons the pulse-beats are slightly irregular, and a knowledge of such peculiarity is essential before any definite clinical significance can be attached to arhythmia. It is to be borne in mind that during old age, after exertion, and during profound mental strain both the force and the frequency of the pulse may be irregular, and the acts of respiration are at times capable of causing decided alterations in the pulse.

Arhythmia.—This condition is said to be dependent upon some disturbance of the nervous mechanism governing the contractions of the heart. It is also a feature of myocarditis.

Intermittent Pulse.—Almost every conceivable variety of irregular rhythm may be seen, but for present purposes only a few of these pulse peculiarities will be described. At times the heart-beats are intermittent, with a subsequent intermission in the radial pulses, and, on the other hand, the pulse may intermit without coincident intermission in the heart's action; this form of irregularity may be accounted for by the fact that the force of each heart-beat is not sufficiently strong to convey a perceptible wave to the radial artery with each contraction—a condition known as "false intermission."

True intermission of the pulse—that due to arrest of cardiac action—is always to be distinguished from the false intermission. Inequality of force exerted by each pulse-wave is important from a clinical standpoint. (See sphygmographic tracings, pp. 188, 193.)

Irregular Pulse.—This term signifies not only irregularity in the pulse-beats, but also inequality of the volume and pressure of each pulse-wave.

The equality of the pulse is by far the most important clinical feature, and will be considered under the following heads:

Sinus Irregularities.—"As the primitive cardiac tissue at the mouth of the great veins possess in a degree higher than any other part the power of rhythmically producing the stimulus for contraction, the rhythm of the whole heart follows normally the time set by this portion of the primitive tissue" (MacKenzie). Under normal conditions this rhythm is obtained, although in many patients a variation is observed during the course of the heart's cycle. The systolic period shows an appreciably greater constancy in its duration than does the diastolic period (see Fig. 74). "With the quickening of the pulse-rate, the shortening of the period of the cardiac cycle takes place about entirely at the expense of the diastolic portion" (MacKenzie).

Sinus irregularities become less conspicuous when the heart's action is rapid, and, on the other hand, when the heart is slow in its action, sinus irregularities are rendered more prominent. Deep inspiration tends to increase sinus irregularity, while swallowing causes its disappearance. Consequently, we find this type of irregularity best exemplified in young

subjects, and in selected cases of adults during convalescence from acute fevers, or at times when the respiratory movements are slow.

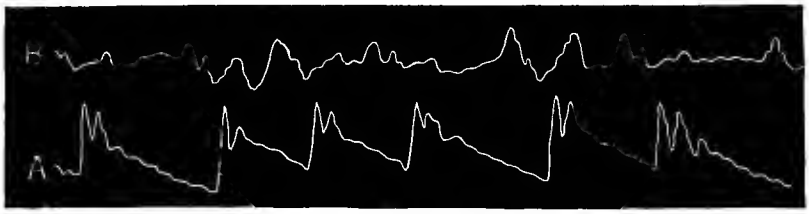


FIG. 74.—SINUS IRREGULARITY, AS SHOWN BY SIMULTANEOUS TRACING OF THE RADIAL (A) AND JUGULAR (B) PULSES.

Extrasystole.—This term should probably be limited to those extra contractions of the heart due to stimulus from some abnormal portion of the organ. Both ventricular and auricular extrasystole are recognized.

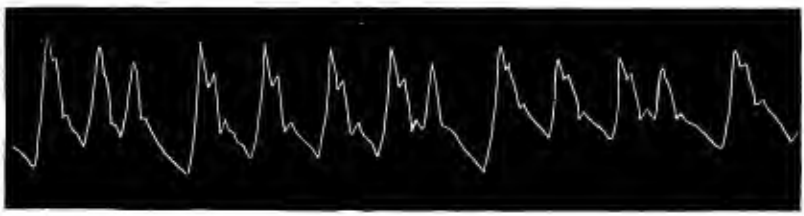


FIG. 75.—RADIAL PULSE, SHOWING EXTRASYSTOLES OCCURRING AT VARIOUS INTERVALS.

Characteristics of the Irregularities.—The most tangible evidence is the occurrence of an extra or premature beat when palpating the radial pulse; and this extra beat is followed by an abnormally long pause, when the small beats are due to extrasystoles.

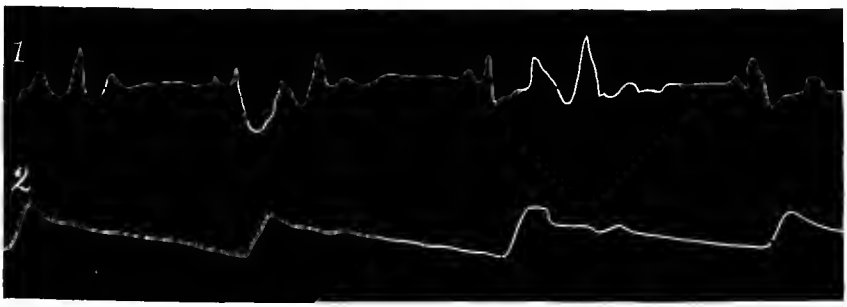


FIG. 76.—EXTRASYSTOLES, AS SHOWN BY SIMULTANEOUS TRACING OF THE RADIAL AND JUGULAR PULSE. (1) Jugular tracing; (2) radial tracing. Case of bradycardia. Radial pulse, 42.

Extrasystoles may occur after each normal impulse of the heart, or they may follow every second, every third, or every fourth beat, or, as is rather common, they may occur at irregular intervals. The sphygmographic tracing of the radial and jugular pulses may show the existence of extrasystoles which are too feeble to be detected by the pal-

pating finger (Fig. 75). The heart-sounds nearly always give evidence of irregularity, although it may at times be necessary to make a pulse tracing for the recognition of extrasystole. When extrasystoles are present, the pulse is commonly said to be intermittent, and many phases of such intermittency are detected in clinical work. Should the extrasystole follow each beat of the heart, the pulse taken at the wrist appears unusually slow, and the condition is regarded as bradycardia. Experience has shown that in all cases of bradycardia it is advisable to listen over the heart with a stethoscope while palpating the radial pulse. The accompanying illustration (Fig. 77) will serve to show the relation that exists

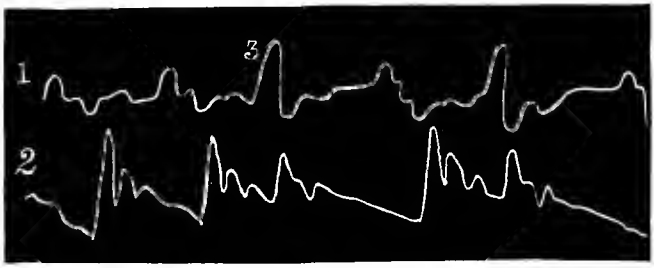


FIG. 77.—SCHEMATIC DIAGRAM, ILLUSTRATING SIMULTANEOUS TRACINGS OF (1) JUGULAR PULSE; (2) RADIAL PULSE; (3) NODAL EXTRASYSTOLE.

between ordinary ventricular contraction and extrasystoles, as shown at the radial and jugular pulses.

By auscultating over the body of the heart the recognition of extrasystole is rather easy, since the regular sequence of sound is abruptly interrupted by two short, sharp sounds. In those cases where the heart-sounds are unusually feeble but one sound may be heard, followed by an abnormally long pause.

Auricular Fibrillation.—*Definition.*—A condition dependent upon abnormal impulses arising in various auricular areas, and replacing the normal rhythmic stimulus produced by a single area. Most constant among the pathologic findings are a rather diffuse fibrosis together with atrophy of the cardiac muscle cells.

Remarks.—This condition is rather intimately connected with extrasystole (see p. 195), auricular flutter (see p. 197), and heart-block. The systoles of the auricle and ventricle do not follow each other with regularity. The ventricle probably receives a variety of impulses, and in its endeavor to respond to such impulses increases its rate of contraction, thus becoming decidedly arhythmic.

Predisposing Factors.—Lewis in his analysis of 126 cases gives a history of rheumatism or of chorea in 70 per cent. of them. He further adds that in non-rheumatic subjects auricular fibrillation is rather more frequent in males than in females. The condition is decidedly more prominent in non-rheumatic subjects after middle life. Auricular fibrillation is to be looked for in connection with mitral regurgitation, myocarditis, interstitial nephritis, and occasionally in aortic disease.

Physical Signs.—Inspection discloses the signs given under myocarditis (see p. 296). The ventricular rate of impulse will be found to vary between 100 and 200 per minute, but there may be radical variations from these figures, since in certain selected cases the ventricular rate may

fall to 60, to 40, or even less. The pulse is, as a rule, rapid, 120 to 140 per minute, and there is decided irregularity as to time, force, and volume. The pulse is compressible, and the clinical signs of aortic regurgitation, myocarditis, and tricuspid regurgitation are frequently present.

Auscultation.—Should the heart-sounds be sufficiently loud, the evidences of auricular fibrillation may be heard by placing the bell of the stethoscope at the apex of the heart.

Auricular flutter is an extremely rapid action of the auricle (200 to 320 per minute). This condition is rather intimately related to auricular fibrillation, and to other conditions that are characterized by rapidity of the heart. The action of the ventricle is usually one-half to one-fourth that of the auricle. Auricular flutter is at times paroxysmal.

Force.—The degree of force offered to the palpating finger with each impulse of the heart is a direct guide, first, as to the strength of the heart muscle, and, second, as to the condition of the arteries in rendering it possible for such forced impulse to be conveyed to the radial artery. The pulse is compressed in the following manner (Fig. 78): the index-finger is placed upon the radial artery, while firm pressure is made by another finger along

FIG. 78. —METHOD OF DETECTING INEQUALITY OF THE RADIAL PULSE.
Second finger raised to show the method of determining the degree of compressibility of the pulse.

the course of the artery between the index-finger and the heart; if the impulse is easily obliterated as the result of pressure, such pulse is referred to as compressible. The degree of pressure necessary to obliterate the pulse-wave is also of great clinical importance; consequently a pulse may be readily compressible or compressed with difficulty. The compressible pulse is a characteristic finding in myocardial disease, and is one of the earliest symptoms of fatty degeneration.

An abnormal increase in the force of the radial pulse may be either temporary or permanent, and in the vast majority of cases results from an

exaggerated ventricular systole—a feature of cardiac hypertrophy. Pulsation may also modify the force of the pulse-wave; thus in those cases exhibiting myocardial change the pulse-wave is decidedly weakened as the result of elevating the wrists well above the level of the head, while the patient is standing or sitting, but whatever means may be employed to determine the degree of force of the heart muscle, an accurate knowledge of such strength must be obtained.

Respiratory Arrythmia (Pulsus Paradoxus).—This variety of arrythmia is but feebly, if at all, noticeable in healthy adult subjects. In children respiratory arrythmia is the rule, and this type constitutes by far the greater proportion of all cases of arrythmia found in young subjects. Respiratory arrythmia is more conspicuous when approaching and at the time of puberty, after which period the pulse-rate diminishes, and the heart assumes the rhythm which it will, under normal conditions, maintain during adult life. Periodic respiratory arrythmia may be induced by deep breathing. It is well-nigh impossible to separate pulse irregularity from pulse tension, for in examining the pulse these two conditions are recorded simultaneously. An excellent example of this fact is shown by the so-called *"pulsus paradoxus"* which is one in which the radial pulse is obliterated during inspiration. Under normal conditions the pulse tension is slightly increased by inspiration, whereas in pulsus paradoxus the tension is low at the end of inspiration, and appreciably higher at the end of expiration. In this type of pulse there is, at the same time, a converse relation of the volume of the pulse to the respiratory act; in other words, during inspiration the volume of the pulse is lessened (the beats may be slightly more frequent and weaker than when the expiratory act is nearly completed), and during expiration it is increased.

Etiology.—This type of irregularity may be seen in the presence of a large pericardial effusion, extensive pericarditis, mediastinal tumors, and conditions that cause positive intrathoracic pressure, as well as those that constrict the great vessels near the heart and cause them to be drawn up or stretched during the act of inspiration. Venous engorgement commonly accompanies pulsus paradoxus.

Pulsus paradoxus may also be excited by conditions that increase the inspiratory pressure or prevent the air from entering the lungs—*e. g.*, stenosis of the large air-passages. Enfeebled heart action may also excite this phenomenon, and it is occasionally associated with a large pleural effusion. Irrespective of the nature of the condition that may excite this variety of pulse irregularity, the more vigorous the heart's systole, the less conspicuous is the pulsus paradoxus.

Alternating Pulse.—This type of pulse is best distinguished by pressing the brachial artery against the humerus at a point just above the elbow, and at the same time placing the index-finger of the operator's other hand over the radial artery. It will be found that when a certain amount of pressure is exerted upon the brachial artery, only alternate pulsations are felt at the radial pulse. This clinical feature is still more apparent by using the ordinary cuff, as is done in taking blood-pressure. After the systolic pressure is ascertained permit the degree of pressure to lessen, say five or ten points on the sphygmomanometer, and it will then be apparent that only certain of the pulse-waves are conducted beyond the cuff and felt at the radial. (See Blood-pressure.) This evidence is of great importance in estimating the contractile power of the myocardium. Where

the auscultatory method for estimating blood-pressure is employed (see p. 208), the alternating pulse is immediately apparent.

Dicrotic Pulse.—A dicrotic pulse is one in which the palpating finger feels two distinct impulses of the artery with each cardiac systole. The second impulse is due to an abnormally prominent recoil of blood after closure of the aortic valves, and occurs at the time indicated by the dicrotic notch on the sphygmograph. The phenomenon is observed when the arterial pressure is low.

Water-hammer Pulse; Corrigan Pulse.—In the water-hammer pulse the systolic impact against the finger is strong and the artery is full of blood; this impact immediately recedes, and the full vessel immediately empties itself. The phenomenon is characteristic of aortic regurgitation. The sudden impact is due to the force of the hypertrophied left ventricle; the volume of blood in the vessel is determined by the dilatation of the ventricle, and the sudden recession of both characters is due to the regurgitation of blood through the aortic orifice

Tension.—Definition.—The degree of pressure communicated to the finger by the blood through the wall of the artery. Tension includes the estimation of the pressure given by the artery when it pulsates as the result of systole and during diastole. The pulse-wave itself may display an unusually high tension, but the blood-pressure during the intervals between the beats may be unusually low—features of aortic regurgitation. Tension, therefore, collectively speaking, has to do with the prolonged high-tension pulse where the blood-pressure is high between the beats, and it is this type of high tension that is also of special clinical significance. The existence of continued high pressure is ascertained when one endeavors to palpate the artery between beats and further rolls the artery from side to side underneath the index-finger, when, if the artery remains firm and is not readily compressed, the tension is high between the beats; it is this type of high tension that is also of special clinical significance.

Caution.—In estimating the pulse tension extreme care must be exercised, with a view to discriminating between true increased tension and atheromatous change in the wall of the artery. There may be fatty atheromatous softening, on the one hand, or distinct atheromatous hardening, on the other, and should either one of these conditions be present in a given case, the influence of arterial disease upon the pulse tension must be taken into consideration. It is by an accurate appreciation of these pathologic conditions of the artery that our knowledge of the arterial tension becomes of great clinical importance.

A low-tension pulse is recognized by the fact that the pulse disappears when but slight pressure is exerted; and, likewise, a pulse of moderate tension requires moderate pressure by the index-finger to elicit the greatest impression. The high-tension pulse conveys the greatest impression to the palpating finger when firm pressure is made over the artery. The size of the artery is often of importance in connection with a pulse of high tension, but here there are many exceptions to the general rule that a small artery has a pulse of high tension, and that a large artery must display a pulse of low tension. Given a soft pulse, or one of quite low tension, it will be found readily compressible, and if a second much weaker rebound following each pulsation of the artery is also detected, the condition is known as a dicrotic regurgitant pulse. Tension is practically inseparable from blood-pressure herein considered.

BLOOD-PRESSURE.

General Considerations.—The pressure which the blood exerts upon the vessel-walls is known as blood-pressure. Physiologically considered, there are five (5) factors concerned in the maintenance and regulation of blood-pressure, viz.:

(1) The constant activity of the vasomotor system (resistance offered to the blood as it passes through the arterioles and capillaries).

(2) The elasticity of the arterial walls.

(3) The force and frequency of the heart's action (volume of blood forced into the arterial tree).

(4) Viscosity.

(5) The total quantity of blood in the body.

In the maintenance of continuous pressure the elasticity of the arterial wall plays an important rôle. If the vascular system were perfectly rigid, the pressure would rise to a very high point during systole and fall to zero during diastole. By virtue of the elasticity of their walls the arteries distend as the heart raises the pressure and forces the blood into the aorta. During diastole the distended arterial walls recoil, and so maintain with slight variations a continuous pressure, thus converting the rhythmic flow of blood in the arteries into a continuous flow in the capillaries and veins.

The entire energy exhibited in the vascular system arises primarily from the heart's action. An increase in the force and rate, other factors remaining the same, will cause an increase in both velocity and blood-pressure, and vice versâ.

The total quantity of blood in the body is to a certain extent of but limited importance in regard to blood-pressure. A loss of blood will cause a very transitory fall of blood-pressure if other conditions remain the same, as under normal conditions other factors, principally vasomotor tone, rapidly restore circulatory equilibrium, so that no permanent change occurs.

A clinically important factor in the maintenance and regulation of blood-pressure is the normal and constant action of the vasomotor system. We now know that the blood-vessels are supplied with motor fibers through whose activity the caliber of the vessel and, therefore, the capacity of the vascular bed is controlled.

Arterial Tonus.—There are two sets of fibers—the vasoconstrictors and the vasodilators. The vasoconstrictors are by far the most important. It is through these fibers that the vasomotor center in the medulla is continuously sending nerve impulses, by which the condition known as vascular tone or tonus is maintained.

Observations.—In the study of blood-pressure four estimations are recognized, viz.: Systolic pressure, diastolic pressure, pulse pressure, and mean pressure.

Systolic Pressure.—By systolic pressure is meant the maximum pressure by the systole of the heart.

Diastolic pressure is the minimum pressure in the artery. It corresponds in time to the diastolic phase in the cardiac cycle.

Pulse pressure is the difference between the systolic and diastolic pressures. The normal pulse pressure is said to range between 25 and 45 mm. Hg.

Mean pressure is fairly accurately estimated by taking the arithmetic mean of the systolic and diastolic pressures, *e. g.:*

Systolic pressure,	150 mm. Hg.
Diastolic pressure,	110 mm. Hg.
	2)260
Mean pressure,	130 mm. Hg.

It is essential that all these observations be taken into consideration, inasmuch as any alteration in one or more of them is of clinical value.

Standard for Blood-pressure.—Mention has already been made of the expression, millimeters of mercury, in connection with this discussion of blood-pressure.

In order to reduce the determination of blood-pressure to a definite and comparable basis, it has been found advisable to adopt as a *unit of measure the standard mercury column*, and to express all blood-pressure findings in figures comparable to the weight of the mercury column. The practical clinical study of blood-pressure has been made practical through the development of mechanical appliances known as sphygmomanometers, which are now quite generally employed in studying conditions of the circulation, and which, with the exception of the sphygmomanometer of Potain, all are graduated to express blood-pressure in mm. Hg.

Recent experimental work of Janeway, Bishop, and others conclusively show that the measure of pressure as determined by the sphygmomanometer closely represents the true pressure within the vessel, as shown by a cannula introduced directly into the vessel, and that this in the larger vessels at least closely approximates the pressure within the aorta.

Normal Blood-pressure.—Any basis for a study of blood-pressure must have its foundation on an established normal or normal limit of variation. This factor like the pulse is subject to variation within normal, as well as pathologic limits. An element of error may enter into these studies, until it is thoroughly understood at the outset, that figures employed at the present time are based almost entirely on examinations of the brachial artery, usually in the sitting posture, with instruments of recognized mechanical accuracy, employing a cuff of standard (5 inches) width, the operators following the visual or the palpatory methods of observation.

Earlier observations (before 1900) must be questioned regarding their accuracy, because many of the earlier instruments were inaccurate for mechanical reasons, and because the employment of the narrow ($2\frac{1}{2}$ inches) cuff of Riva Rocci gave records abnormally high.

The Factors which may Physiologically Modify the Blood-pressure.—From numerous observations by competent observers it has been found that records obtained by the usual means, visual or palpatory, give a systolic pressure in the normal individual in early adult life between 105 and 130. The best experimental work upon this subject has been done by Woley, who has prepared the subjoined table. From this it will be seen that pressure is modified to some extent by age and sex, and that Woley's constant factors can be used for comparison in all estimations of the normal pressure at any age.

In order to simplify the procedure of estimating the normal for any age, Faught has suggested the following formula, which can be employed

as a rough guide in determining the normal average systolic blood-pressure. "Consider the normal average systolic blood-pressure in a male aged twenty to be 120 mm., and that for every two years of life add 1 mm. to 120." To determine the pressure for women subtract 10 mm. This when worked out will be found to conform quite closely with the Woley chart (Fig. 79).

1. Sex. 2. Posture. 3. Age. 4. Time of day. 5. Digestion. 6. Altitude. 7. Exercise. 8. Emotion. 9. Obesity.

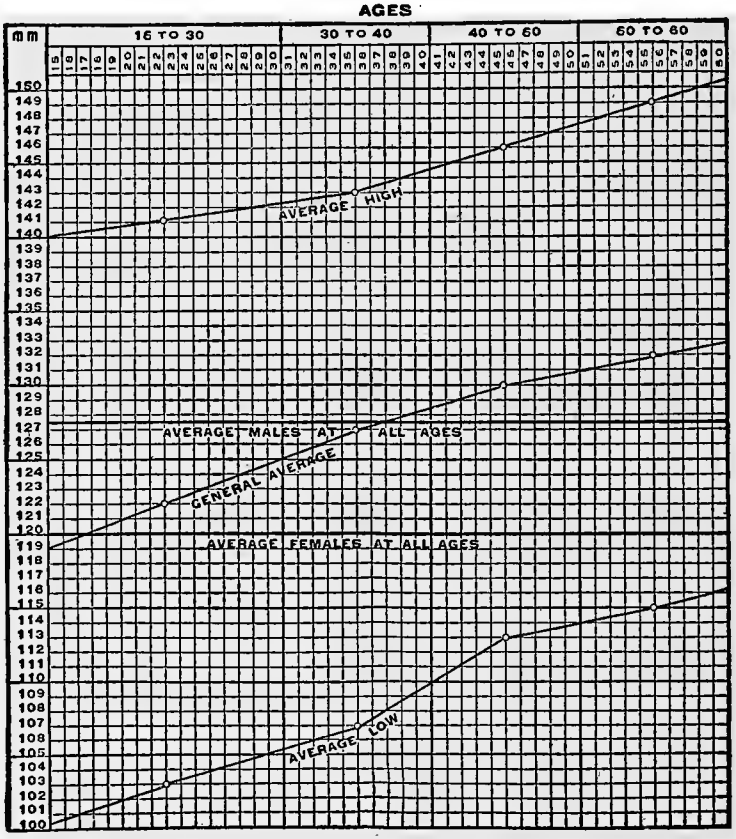


FIG. 79.—WOLEY'S CHART SHOWING EFFECT OF AGE ON BLOOD-PRESSURE, GIVING MEAN, HIGH, AND LOW AVERAGE. PALPATORY-ASCILLATORY METHOD (Jour. Amer. Med. Assoc.).

Sex.—Blood-pressure is said to be lower in women. Brunton estimates it to be from 10 to 15 mm. Hg. below that of men.

Posture is of some importance, since blood-pressure is lower when the patient is standing erect and highest when the head is lowered. Important in comparative readings.

Age has a decided effect on blood-pressure. It is lowest in childhood, and increases progressively with years.

Thayer, in a series of observations upon 276 healthy individuals, found the systolic blood-pressure average for the different decades to be·

1–10	years	104.6	mm.	average	pressure
10–20	"	128.7	"	"	"
20–30	"	136.9	"	"	"
30–40	"	140.8	"	"	"
40–50	"	142.2	"	"	"
50–60	"	154.8	"	"	"
60–70	"	180.0	"	"	"

Brunton found the systolic pressure for children eight to fourteen years of age to be about 90 mm. Hg., and subjects fifteen to twenty-one years were found to give a reading between 100 and 120 mm. Hg.

Time of day has a slight effect on blood-pressure, but this is of little clinical significance.

Digestion.—During digestion dilatation of the blood-vessels of the abdominal region takes place, which would naturally cause a fall in blood-pressure, but the heart increases the output to not only compensating for the fall, but also actually increasing arterial pressure.

Altitude.—Recent clinical studies would seem to show that change from low to high altitude causes a rise in blood-pressure in normal individuals, which becomes less marked as the subject becomes accustomed to the change, while in the tuberculous with a subnormal pressure high altitude affects a usually permanent rise.

Exercise.—Physical exertion in the normal healthy individual causes a sharp rise of from 5 to 30 mm., which persists for a while after the cessation of the exercise. This rise is less marked as the individual becomes accustomed to the exertion. This factor demonstrates the benefit on the circulation of graduated systematic exercises; extreme exertion, resulting in fatigue, is usually followed by a fall of pressure, which may be so marked, as after a boat race or marathon, as to be a sign of great danger, often of acute cardiac dilatation.

Emotion, Excitement, Etc.—The effect of these depends upon the temperament of the individual, and is an indication of the stability of the vasomotor system. The change in pressure may be extreme, and may in cases detract from the value of the observation.

Obesity.—Under ordinary circumstances this state does not materially affect the readings. The extremely obese may show subnormal pressures.

Pathologic Variations in Blood-pressure.—For convenience of clinical study we have considered pathologic blood-pressure under the following subheadings:

1. Those conditions showing high blood-pressure—hypertension.
2. Conditions accompanied by low blood-pressure—hypotension.
3. Those conditions displaying a primary rise and a secondary fall of blood-pressure.
4. Maladies in which there is an instability of blood-pressure.

Hypertension is frequently found in—

1. *Nephritis.*—In chronic interstitial nephritis the blood-pressure is always high except during the last few hours before death. Here a routine study of blood-pressure is of great clinical importance. An approaching uremic crisis can often be foretold before it becomes evident through any other clinical method. In the chronic parenchymatous form high tension may or may not be present. In amyloid and in the hemorrhagic forms of nephritis normal or subnormal blood-pressure is frequently encountered.

2. *Arteriosclerosis.*—Blood-pressure will be found high in proportion as the general arterial tree is involved, and will be highest in general

arteriosclerosis involving the splanchnic area. It should be remembered that local arteriosclerosis as in the radials may not be accompanied by high pressure.

3. *Cardiac Disease:* (*a*) Aortic regurgitation; (*b*) angina pectoris.

In aortic regurgitation, hypertension combined with a large pulse pressure (60 mm. or more) is a most characteristic sign of this disease. This reaction is purely physical, and is due to the fact that the heart at each systole is required to force into the aorta not only sufficient blood to maintain the circulation, but also an additional volume to allow for the regurgitation into the left ventricle. (See also p. 265.)

4. *Puerperal Eclampsia.*—The importance of a high and rising blood-pressure as an early diagnostic evidence of toxemia in the latter half of pregnancy can hardly be overestimated. For nearly two years the blood-pressure of every patient in the University of Pennsylvania Maternity, and also those cases in the private practice of Dr. John C. Hirst, has been taken from these observations. The following conclusions have been warranted:

(*a*) A high blood-pressure (average, about 190 mm. Hg., highest over 300 mm. Hg.) is noted in every case of actual eclampsia.

(*b*) A high blood-pressure, constantly rising, is noticed as an invariable, and very often the earliest, sign of toxemia in the latter half of pregnancy. In one private case it antedated albuminuria by three weeks, and in another by two weeks.

(*c*) As far as any diagnostic rule can be laid down, a blood-pressure of under 120 mm. can be disregarded; from 120 to 150 carefully watched, and over 150 usually means danger.

(*d*) When the membranes rupture in labor or are artificially ruptured in a patient who is toxemic, there is an immediate fall of blood-pressure of from 60 to 90 mm., followed by a prompt rebound to nearly the original height with, however, a marked amelioration in the subjective symptoms.

(*e*) There is a second fall of blood-pressure following the birth of the child, with a second rebound to near the original height, and then a gradual but steady fall until the normal is reached—three to seven days after delivery.

(*f*) Cases terminating fatally do not return to normal pressure.

5. *Toxic agents* (poisons from): (*a*) Lead; (*b*) nicotin.

6. *Metallic Toxemia.*—(*a*) Lithemia; (*b*) gout—here an unusually high pressure may persist for an indefinite period.

(7) *Cerebral and ocular hemorrhage* also causes a rise in blood-pressure.

B. **Hypotension** is a feature seen in:

(1) *Shock.*

(2) *Exhaustion.*

(3) *Cachexia and anemia.*

(4) *Certain infectious fevers, especially typhoid and tuberculosis.*

In typhoid fever observations of blood-pressure is of clinical value. The pressure falls gradually with each week of the disease, and this is very well shown in 115 cases studied by Crile, who found:

First	week	=	115 mm.
Second	week	=	106 "
Third	week	=	102 "
Fourth	week	=	96 "
Fifth	week	=	98 "

A rapid fall of blood-pressure should suggest hemorrhage. Crile and Briggs have found a sharp rise marking the onset of peritonitis following intestinal perforation.

All authorities agree that hypotension is the rule in tuberculosis, and this pressure falls correlatively with the loss in general bodily vigor.

(5) *Venesection, temporary.*

(6) *Excessive hemorrhage.*

(7) *Copious Diaphoresis.*—Both the hot-pack and the hot-air bath reduce the blood-pressure by free diaphoresis.

(8) *Diarrhea.*

(9) *Following aspiration of fluid from the pleural and abdominal cavities* the pressure is temporarily lowered.

(10) *Cholera asiatica.*

(11) *Cerebral embolism.*

(12) *Cardiac Conditions.*—(*a*) Myocarditis; (*b*) tachycardia.

C. A primary rise and a secondary fall of blood-pressure are seen in severe types of:

1. *Pneumonia.*
2. *Scarlet fever.*
3. *Peritonitis.*

D. Instability of blood-pressure is often seen in very nervous persons, in exophthalmic goiter, neurasthenia, and during the menopause.

Faught's Blood-pressure Indicator.—The Faught indicator, as shown by the accompanying illustration (Fig. 80), is unusually easy of operation, and does not require any special skill in order to obtain accurate, as well as constant, readings with this instrument. Another advantage worthy of special mention is that the Faught pocket indicator is light, and can, therefore, be readily brought to the bedside.

Application.—(1) Raise the lid, which carries the manometer until it locks in a vertical position. Apply the cuff to the arm just above the elbow (Fig. 80). The armlet and cuff are applied together, the ends of the rubber being smoothly overlapped.

(2) The straps are now buckled, when the cuff fits neatly, but without compression.

(3) Whether applied to the calf of the leg or to the forearm, if the limb is very conic, there will be a tendency to slip, and the rubber must be placed somewhat diagonally, otherwise the pressure will be exerted chiefly on the portion surrounding the larger portion of the limb, and the result will be the same as though a narrow cuff had been employed.

(4) If the mercury has become separated, gentle jarring up and down will cause it to reunite. Then set the scale (G), which is adjustable, so that zero is at the mercury level.

(5) Connections are made as follows: Attach the pump P by means of the expansile tubing to the nipple F, having the stopcock M. Connect the arm-band to the nipple D. Close the needle valve N and turn both mercury guard-cocks K and L in the vertical or open position (Fig. 80).

(6) Locate the pulse, with the left hand, on the same member to which the arm-band is attached, and do not change the position of the fingers during the test. Operate the pump with the right hand.

(7) Action of the pump will now force air into the closed system, distending the rubber armlet, and with the same degree of force driving the mercury up in one limb of the "U" tube H.

(8) When the pulse disappears, the valve M is closed by turning to right angle to the nipple F.

(9) Turning the needle valve N slightly in a contra-clockwise direction, or until the mercury gradually falls about 2 mm. per pulse-beat, and the pressure indicated on the scale when the pulse reappears at the wrist is the *systolic pressure;* disregard all motion of the mercury up to this time.

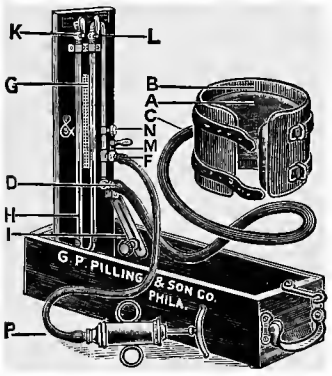


FIG. 80.—FAUGHT'S STANDARD OR MERCURY SPHYGMOMANOMETER.

(10) As the mercury column falls, oscillations of the mercury will follow the pulse, and will increase in magnitude until they reach a maximum and then quite suddenly decrease and finally disappear. The base line of the greatest oscillation (the line from which it starts) is the *diastolic pressure.*

(11) Patients presenting little or no oscillation may have the diastolic pressure measured by noting the mercury level on the scale, at the moment that the first full forcible impulse is felt at the wrist.

(12) After having determined the systolic and diastolic pressures disconnect one tube and allow all air to escape, then repeat the tests to verify the findings.

Cautions.—(1) *Uniform Method.*—It is wise to adopt a method by which all pressures are taken. This diminishes the time required and eliminates error. The muscles of the arm should always be relaxed; consequently if the patient is sitting the elbow and forearm should be supported, as muscular contractions show themselves on the mercury column. Preferably, the pressure should be taken with the patient in the recumbent posture. It is easier for most observers to take pressure from the left arm, as the necessary manipulations of the manometer are more easily performed with the right hand. In nearly all cases the first estimation will be found to be from 10 to 20 mm. higher than subsequent estimations; this is probably due to excitement. Several estimations should be made, until the level normal to the individual is obtained.

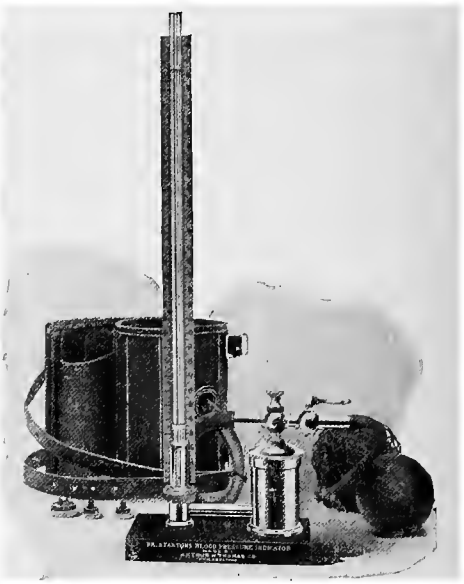


FIG. 81.—STANTON'S SPHYGMOMANOMETER.

(2) *Rapid Pulse.*—There is no pulse so rapid that the mercury instrument may not be used; actual experiment has shown that 200 beats per minute will be shown by synchronous oscillation of the mercury.

(3) *Slow Pulse.*—With a very slow, strong pulse the oscillations may be so large that it is difficult to record the largest ones. In these cases, by

leaving the valve A open, a part of the oscillation is absorbed by the elastic rubber bulb and the reading becomes easier.

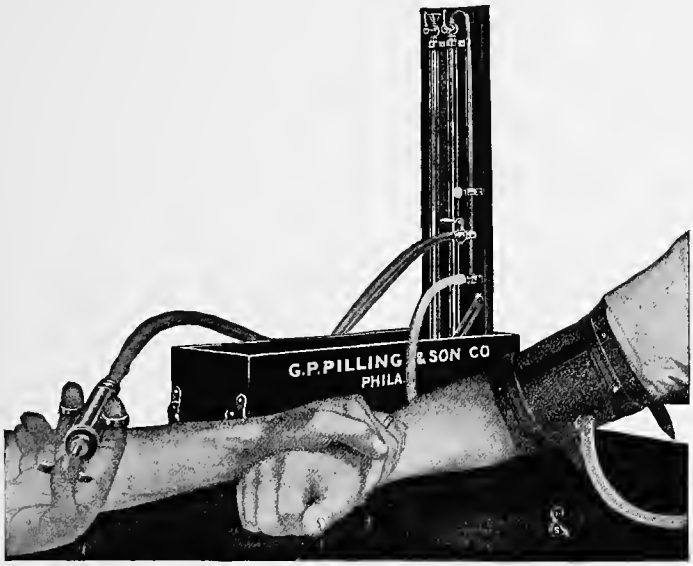


Fig. 82.—Faught's Mercury Apparatus in Use.

(4) *High Pressure.*—In those showing threatened circulatory failure, especially in case of high pressure, it will be found almost impossible to ascertain definitely either the high or the low pressure. Despite repeated estimations, the high pressure will vary from 5 to 15 mm. These cases may at times show a condition in which an occasional beat reaches a much higher level than that at which all the beats can be detected, a feature possibly influenced by respiration. In event of high pressure, the pocket instrument is most satisfactory.

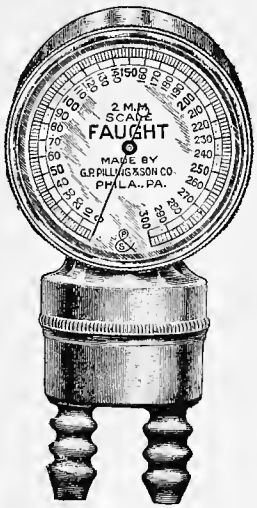


Fig. 83.—Faught's Pocket Indicator. Actual Size.

Pocket Sphygmomanometers. — The pocket, or so-called aneroid sphygmomanometers, have recently attracted great attention, and are rapidly being adopted for clinical purposes. Their chief advantage is their portability. A number of them are now made of such a size that they are all contained in a leather carrying case of pocket size.

The Faught pocket sphygmomanometer is made in two sizes, one (Fig. 83) has a dial of $3\frac{1}{2}$ inches in diameter and records pressure up to 300 in mm. Hg.

The method of use does not differ from the mercury instruments, excepting that if anything they are easier of application. The graduated standardized dial takes the place of the mercury column, while the cuff, to reduce its size, if of the bandage type, contains a compression bag of

standard size, 5 by 9 inches. This is retained on the arm by placing the rubber portion over the vessel and then wrapping the long fabric cuff over this until it is used up, finally tucking the end under the preceding turn, where it is held by the air pressure within the bag.

The rubber portion of the arm-band is easily removable from the fabric portion, which may then be easily washed or sterilized. The authors have grown to use this instrument exclusively.

Ausculatory method for determining the systolic and diastolic pressure is shown by the accompanying illustration (Fig. 84). Karotkow, of St. Petersburg, contributed the first paper on this method in 1905. Gittings, of Philadelphia, gave an admirable review of the subject, to-

FIG. 84.—AUSCULTATORY METHOD. FAUGHT'S POCKET INDICATOR IN USE.

gether with a report of his observations. The authors employ this method in all their clinical work.

The maximum and minimum pressure can be more readily and more accurately determined by the auscultatory method than by any other, and its use is attended with no difficulties.

(1) Before placing the cuff around the arm over the brachial artery the bowl of the stethoscope is placed immediately below the elbow and over the bifurcation of the artery. If it is possible to hear the sound of the artery, the auscultatory method cannot be employed in such a case. It is to be emphasized that in certain selected cases, especially those of aortic regurgitation, the so-called pistol-shot sounds are heard over the larger arteries, and in a case of this kind the auscultatory method is not applicable, except for the determination of high pressure. Again, it is essential that the radial artery be readily palpable.

(2) After placing the cuff around the arm, one must be especially careful that the second portion of the cuff surrounding the arm (that part of the canvas not containing the rubber bag) extends to both edges of the rubber bag and continues for one or two laps. The tip of the canvas is then

tucked underneath the bag. When the cuff is properly applied it should fit the arm sufficiently tightly, so that one can rotate the cuff without its slipping toward the elbow. It is well to place the center of the rubber bag across the brachial artery.

(3) Place the stethoscope over the artery below the elbow and, while listening, inflate the bag slowly, observing that no portion of the bag creeps from under the second lap of canvas while the inflation is taking place.

(4) Record the first sound that is heard over the artery (Fig. 85, 5). Continue to inflate the bag until the instrument registers ten to twenty points above where the first sound was obtained, and then permit the air to escape from the bag gradually until one ascertains at what point the sound disappears (Fig. 85, 5). This point must register identically with the reading at where the sound was first heard, and represents the (generally accepted) diastolic pressure.

In normal pressure the disappearance of sound is noted 2 to 4 mm. below the true diastolic pressure. In event of unusually high pressure, 160 to 220, add from 8 to 15 to this reading.

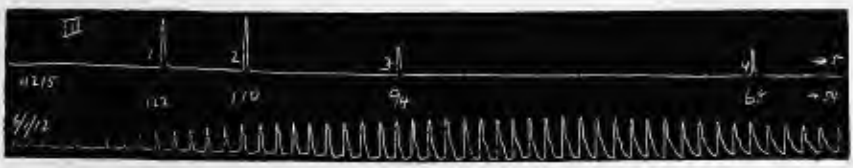


Fig. 85.—Fast Drum. Sudden Decrease in Size of Pulse-wave at 4, Marking the Change from Clear, Sharp Tone to Dull Tone (Warfield, in Jour. Amer. Med. Assoc., Oct. 4, 1913).

(5) Continue inflating the bag until a point is reached at which all sound over the artery disappears (Fig. 85, 1). Continue to inflate the bag until the instrument registers ten to twenty points above this reading, and then, as in the case of determining the diastolic pressure, permit the air to gradually escape from the bag until the first sound over the artery is heard. This reading and that for which the sound disappeared should be practically the same, and represent the systolic or high pressure (Fig. 85, 1).

Cautions.—(1) The cuff must be properly applied (see Fig. 84); (2) the muscles must be relaxed; (3) the patient dare not move the arm or hand during the procedure; (4) in aortic regurgitation the pistol-shot sound rarely present renders an accurate study difficult.

Character of Sounds Heard Over the Artery Below the Cuff (Fig. 84).—Ettinger, in a systematic study of 235 cases, found that the cycle began with the passage of the first waves of blood under the cuff. This usually produces a clear, tapping sound, and is designated the first phase. The second phase quickly follows the first, and is heard as a more or less distinct murmur, the duration of which is variable (this murmur may accompany the first sound, but rapidly replaces it). The third phase includes that period when the murmur is audible. The fourth phase occupies that portion of the cycle where as the mercury falls the clear sound becomes dull (this change is not distinct in all cases, although in health it is readily recognized). Ettinger holds that the beginning of this fourth phase (where the clear sound becomes dull) represents the true diastole. This

point, however, remains open for discussion. The fifth phase occupies the remaining portion until the disappearance of the dull sound.

Krylow claims that where there is extensive arteriosclerosis the auscultatory phenomena are apt to be unusually pronounced. Among the conditions to be taken into consideration are:

(*a*) Degree of narrowing of the vessel.

(*b*) The elasticity of the vessel.

(*c*) The size of the pulse-wave.

(*d*) The rapidity of transmission of pulse-wave. Ettinger has called special attention to the fact that the rapidity of the pulse increases the auscultatory phenomenon. In aortic regurgitation with marked dicrotism

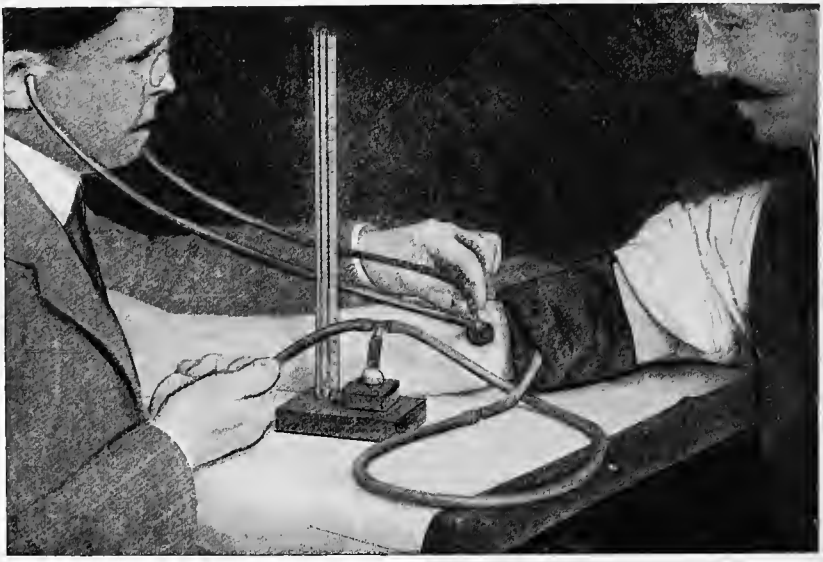


FIG. 86.—DETERMINING BLOOD-PRESSURE (AUSCULTATION METHOD). (KEARCHER'S BLOOD-PRESSURE APPARATUS IN USE.)

a double sound may be heard, and there may be a persistence of the second sound. Both Krylow and Gittings have suggested that alteration in auscultatory phenomenon points conclusively to insufficiency of heart muscle, and the latter observer has reported an interesting series of 63 cases where such alterations were conspicuous.

By employing the auscultatory method the maximum pressure is detected at a higher, and the minimum pressure at a lower, reading than are they when the previously described method (p. 205) is employed. It consequently follows that the mean and pulse pressure will be found higher where the auscultatory method is employed. Ettinger found this difference to be 13.2 mm. Personal experience has caused us to regard the systolic reading by this method approximately 5 mm. above that obtained by palpation.

THE SPHYGMOGRAPH.

The sphygmograph is an instrument for recording graphically the frequency, volume, force, and tension of the pulse, together with other general characteristics.

The *sphygmograph* was devised by Marey, and since its introduction many valuable modifications have been added to it, until, at present, Dudgeon has produced a clinical device that serves fairly well, although it is by no means without objectionable features. Irrespective of what instrument may be used, the actual value of the tracing or record depends greatly upon the personal skill of the operator; consequently the sphygmograph occupies a place widely different from that of many other clinical instruments, and records obtained through its use will, in the hands of skilled individuals, prove to be practically constant and reliable.

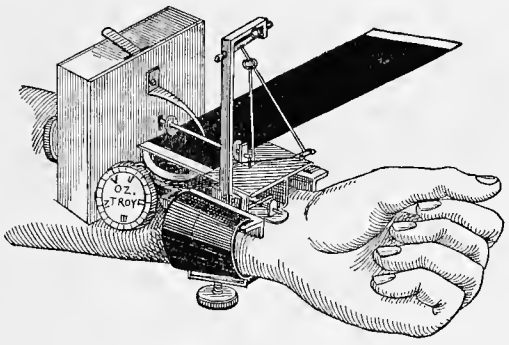


FIG. 87.—THE DUDGEON SPHYGMOGRAPH IN POSITION.

The clinician learns to recognize practically all the peculiarities of the human pulse by palpation of the radial artery, but despite this fact he is forced to be more careful by comparing the pulse with its written record. Again, the sphygmographic record may be preserved in conjunction with the clinical history, and in this way serve as a valuable datum.

Technic.—By careful observation of the sphygmograph we find that it consists, first, of clockwork, carrying a narrow strip of smoked paper; second, of a writing needle; and third, of a support or canvas by the use of which it is held in position over the artery.

(*a*) The clockwork is first wound by turning a special button.

(*b*) The patient is directed to place his forearm and hand in a comfortable position, expose the wrist at the site of the radial artery, flex the fingers gently, and allow the muscles of the forearm and hand to be relaxed.

(*c*) To apply the sphygmograph slip the band or support over the patient's wrist, when the free end of the band is then passed through a special retaining clamp (Fig. 87). The metallic box of the instrument should be directed away from the hand.

(*d*) The next step is to adjust the sphygmograph so that the bulging button, which connects the levers, is directly over the radial artery, and, while the instrument is now held in position by the operator's left hand, the band that holds it to the wrist is drawn through the clamp until the instrument is sufficiently tight so that the writing needle plays easily with each pulsation of the artery. Whenever the last-named result is obtained, fasten the clamp, and the instrument is ready for use.

(*e*) Place a strip of smoked paper (Fig. 87) between the rollers of the instrument and directly underneath the needle.

(*f*) There is a special thumb-screw for the purpose of gaging the pressure, and this must be adjusted to effect the best possible amplitude of vibration.

(*g*) Steady the patient's hand gently, start the clockwork, and permit

it to continue until the smoked paper has entirely passed through the rollers; then stop the instrument.

In Dudgeon's instrument the machinery is so regulated that a five-inch slip of smoked paper will pass in ten seconds, and by a simple matter of calculation the pulse-rate a minute is attained.

Preparation of Paper.—Smoked paper is usually employed in the making of sphygmographic tracings, and some writers recommend that this paper be glazed upon one side and rough upon the other. The paper must first be cut in strips, approximately $\frac{7}{8}$ of an inch in width, and six or seven inches in length.

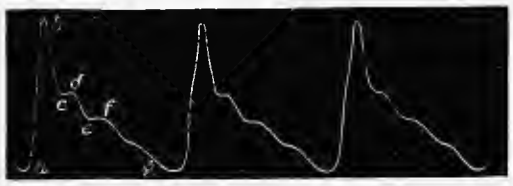


FIG. 88.—NORMAL PULSE TRACING.

ab, Up-stroke or percussion stroke; *bg*, descending or catacrotic limb; *abc*, percussion wave; *cde*, predicrotic or tidal wave; *efg*, dicrotic or recoil wave; *bcd*, protidal notch; *def*, aortic notch.

The glazed surface of the record paper is blackened by holding it over the flame produced by burning a small piece of gum camphor. Various devices may be employed for the purpose of keeping the paper exposed to the smoke, but the method most often employed is that of a strip of tin so bent upon itself as to catch and hold the narrow strip of paper at each end. Care should be taken not to blacken the paper too deeply, lest the lines of the tracing will be irregular and often indistinct.

Preservation of Record.—Write upon the record with a broken pen or a needle, or upon the unsmoked portion, with pen and ink, the patient's name, important features connected with the case, the date, and the name of the artery from which the tracing was made.

The record is preserved by dipping the smoked paper into a solution of shellac or into a solution of benzoin. The method is as follows: Grasp one end of the sphygmographic record by a pair of forceps and draw slowly, record side up, through the solution, and permit it to dry in the air. When records are likely to be handled or are to be preserved for a long period, it is well to give two or more coats of shellac.

Interpretation of Tracing.—*Normal Pulse Tracing.*—As the result of each contraction of the left ventricle a volume of blood is forced first into the aorta, which is appreciably distended, and the distended impulse is further transmitted by a characteristic wave-like motion to the remote portions of the arterial system.

The distending impulse elevates the button of the lever, causing the so-called percussion stroke (*a*, *b*) (up-stroke). The distending impulse has been exaggerated by the sphygmographic system of levers, and has been thrown too high; therefore, the lever falls by its own weight to a point too low; consequently it is again caught and elevated by the tidal blood to form the writing of the tidal wave (*c*, *d*, *e*). Further descent is again interrupted at *e*, forming the so-called dicrotic wave (*e*, *f*, *g*), which is dependent upon the recoil of blood from the closure of the aortic leaflets.

The normal pulse tracing (Fig. 88) will display the following characteristics:

(*a*) The percussion stroke, which is nearly vertical and of moderate amplitude.

(*b*) The apex or summit, which is fairly acute.
(*c*) Gradual descent.
(*d*) A small tidal wave.
(*e*) A distinct dicrotic wave (Fig. 88).

Features of the Sphygmographic Record to be Observed with Reference to its Clinical Significance.—(1) The characteristics of the percussion stroke, particularly its height, and whether or not it is vertical or inclined.

(2) Is the apex pointed, rounded, or unusually broad?

(3) Is the tidal wave conspicuous, scarcely perceptible, or absent? Also observe the same characteristics with reference to the dicrotic wave.

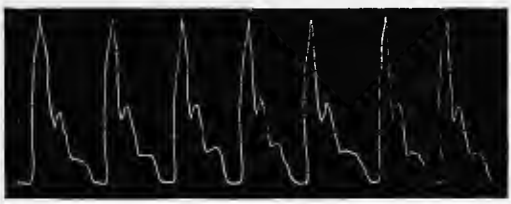


Fig. 89.—Pulse Tracing in a Case of Aortic Regurgitation (William Hoffman).

(4) Do the successive strokes occur regularly, irregularly, or do they intermit? Is the line of descent regular?

(5) Is the character of the base line or line connecting the bases of the different beats straight, curved, or irregular?

Clinical Significance of Variations in the Pulse Tracing.—Percussion (Up-stroke).—(*a*) *A long up-stroke* is observed when a large volume is present, and is also significant of a sudden quick systole or of a relaxed condition of the arteries. It is a feature of low tension and of aortic regurgitation. (See Fig. 89.)

(*b*) *A short up-stroke* corresponds to a small volume, and is indicative of those conditions in which but a small volume of blood is capable of escaping into the aorta—*e. g.*, aortic stenosis, mitral regurgitation, thoracic aneurism, and conditions causing obstruction to the peripheral circulation. If the up-stroke is vertical, it signifies a quick systole, regardless of whether or not the cardiac muscle is weak or strong or an abnormally large volume of blood is propelled into the aorta at such systole. A vertical up-stroke is frequently seen in association with the unusually low-tension pulse of aortic regurgitation.

(*c*) *An oblique up-stroke* may be obtained where the radial artery is covered with a thick layer of fat. It is also seen where the arterial system fills slowly—*e. g.*, in aortic stenosis, thoracic aneurism, marked arteriosclerosis with high tension, mitral regurgitation, and, rarely, it is a feature of myocardial change of the left ventricle.

The Apex.—(*a*) A pointed apex signifies that there is no obstruction to the peripheral circulation, that the tension is low, or that we are dealing with aortic regurgitation (Fig. 89).

(*b*) A broad apex signifies that the muscle action of the heart is forcible, but that high tension in the peripheral circulation, arteriosclerosis, aneurism, or aortic stenosis is present (Fig. 90).

A broad apex may also result where the sphygmograph is not correctly adjusted, or where the spring exerts too great pressure.

The Tidal Wave.—(*a*) If the tidal wave (Fig. 88) (*c*, *d*, *e*) is exaggerated, it indicates that there is high tension due to obstruction in the peripheral circulation (arteriosclerosis), or to aortic stenosis.

(*b*) If the tidal wave is feeble or absent, the heart muscle is usually weak, or possibly there is moderate or low arterial tension in conjunction with mitral or aortic regurgitation.

Dicrotic Wave.—(*a*) When the dicrotic wave is prolonged, the heart is weak or of but moderate strength, and there is low tension in the peripheral

FIG. 90.—PULSE IN ADVANCED ARTERIOSCLEROSIS.
Slow ascent and descent of the wave; anacrotism—only 32 beats to the minute.

circulation. A conspicuous dicrotic wave is less often seen in cases of high tension with cardiac failure.

(*b*) If the dicrotic wave is small or absent, obstruction to the peripheral circulation exists; but, despite the high tension, the heart is still strong—a feature of arteriosclerosis, aortic stenosis, aneurism of the great vessels, and, rarely, aortic incompetency.

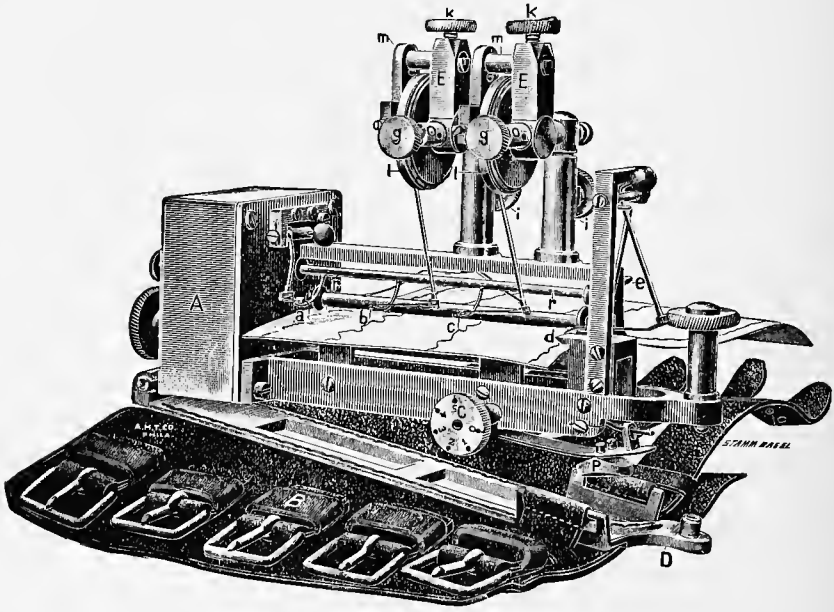


FIG. 91.—JAQUET SPHYGMOCARDIOGRAPH.

Line of Descent.—The line of descent in both mitral stenosis and regurgitation is made conspicuous by its irregularity.

Base Line.—The base line will be found to be irregular, and at times to

show some change, corresponding more or less closely to the acts of respiration in those suffering from cardiac disease who also display dyspnea. This last phenomenon may result from involvement of the cerebral center.

The degree of regularity or irregularity displayed by any given sphygmographic record is immediately apparent when other peculiarities that are present are analyzed.

Sphygmocardiograph.—The most reliable apparatus now to be had for obtaining tracings from the various parts of the circulatory system is

Fig. 92.—Tracing Produced by the Sphygmocardiograph.

that known as the sphygmocardiograph of Jaquet. With this apparatus (Fig. 91) it is possible to take tracings of the circulatory system from three points at the same time. It is provided with a time marker, which registers every two-fifths of a second. This method of clinical investigation was introduced by Mackenzie.

The advantages of this instrument are, first, the method by which the apparatus is fastened to the arm. This is effected by means of a metal

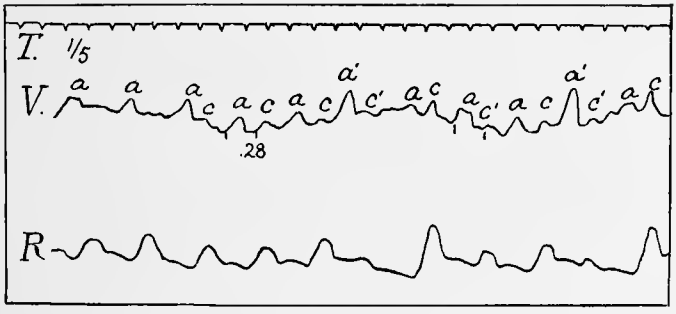


Fig. 93.—Polygraphic, Venous, and Radial Curves (Mackenzie).
V illustrates the auricular type of venous curve with prolongation of the *a–c* intervals; *R* displays the ventricular type of curve where great irregularity is present; *T*, time record, $\frac{1}{5}$ sec.

plate so perforated as to permit the spring to come in contact with the artery. It has the additional advantage of being easily adjusted to almost any wrist, and the width of the attachment is sufficiently great to support the instrument without the aid of the hand. The sphygmocardiograph is anchored and retained in place by a screw. The apparatus itself consists of a case containing the clockwork necessary to furnish the motive power of the machine, as well as the machinery that runs the time-marker. The power that drives the paper through the apparatus consists of a straight rod with four small wheels driven by clockwork. This may be run at either a slow or a fast rate of speed, the fast one being five times as quick as the slow one. The breadth of this driving surface is so great that the paper, once started, will pass through without catching or binding on the sides.

The writing apparatus consists of a spring and a lever of the type of the old Dudgeon instrument, and takes the tracing from the radial artery. The other two tracings are taken from the small air-capsules covered by rubber, which operate the two levers in the center. The pulsations are transmitted to these capsules by air through rubber tubes. The fourth writing point is the time-marker. The rubber capsules are so constructed that the rubber is very easily applied, and the adjustment of the levers is readily accomplished.

The tracing from the heart is made by means of a special apparatus called the *cardiograph*. It is adjustable to any shape or form of chest, and can readily be held in position. Pulsations from the jugular veins or from the liver are taken by means of small cone-shaped receivers, similar to those advocated by McKenzie. Although the apparatus is apparently complicated,

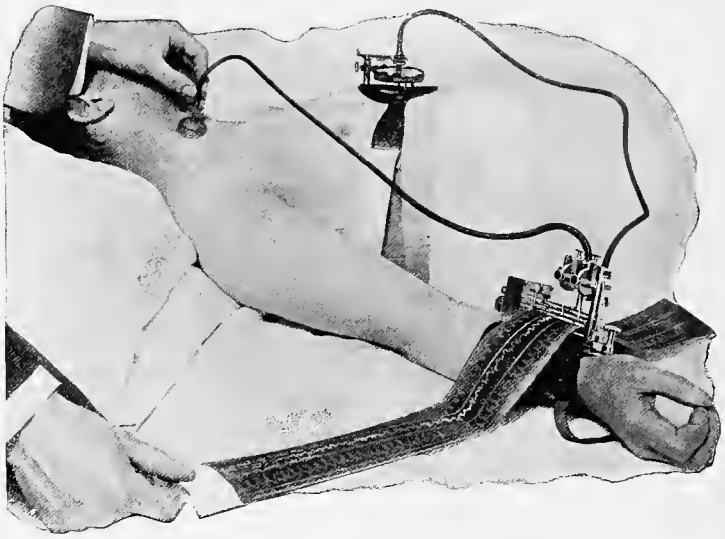


FIG. 94.—JAQUET'S INSTRUMENT IN OPERATION TO RECORD WRITING OF IMPULSE OVER HEART, RIGHT CAROTID, AND RIGHT RADIAL ARTERIES.

it may be thoroughly understood with but little study, and it can be operated by any one after a little practice. (See Fig. 94.)

The advantages of the sphygmocardiograph are:

(1) The size of the tracing, which is 70 mm. broad and about 30 inches long. (Fig. 92.)

(2) The ability to get the tracings from the various points of the circulation at the same time (Fig. 94) and on the same sheet of paper (Fig. 92).

(3) The mechanical advantage of a firm fixation to the arm, which permits of it being operated successively by one man.

(4) A two-speed drive, permitting the stretching out, as it were, of the pulse-curve, so that the individual characteristics can be studied.

(5) The time-marker, which enables one to figure out in time the various phases of the curves obtained.

The method of using the apparatus is well shown in the accompanying illustration (Fig. 94).

ELECTROCARDIOGRAM.

Through this system of study one is able to record changes in potential of the heart muscle that accompany its activity, thus giving a favorable view-point of the normal and abnormal heart action. It has been possible

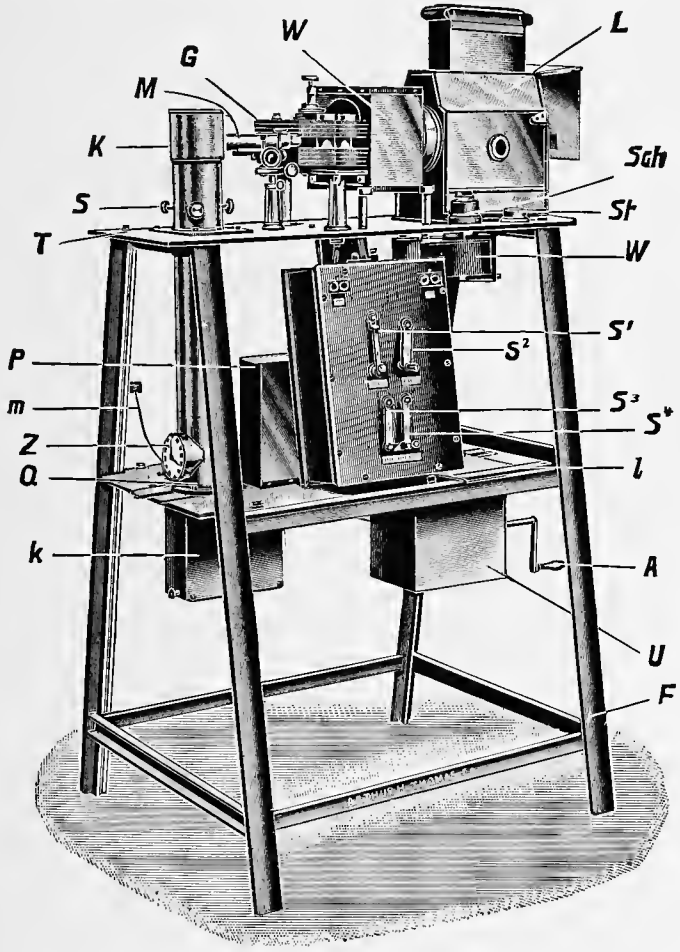


FIG. 95.—APPARATUS COMPLETE, CONTAINING EINTHOVEN THREAD GALVANOMETER AS EMPLOYED BY EDELMAN.

L, Arc lamp; *W*, cooling bath; *G*, Einthoven thread galvanometer; *M*, projection microscope; *K*, camera containing sensitized paper on which the cardiogram is made; S_1 S_2 S_3 S_4, switches controlling the normal element, resistance, and condensers for determining the sensibility of the galvanometer and for compensation of the skin currents.

through this course of study to obtain new light upon that complex problem presented by the pathologic changes of the heart.

Rapid strides in the development of electrocardiography have been observed during the past five years. In this volume it has been deemed advisable to consider only certain of the more practical phases in con-

nection with this clinical method. The authors are indebted to Dr. Thomas A. Cope for the illustrations in connection with this subject.

The electrocardiogram is a graphic record of the feeble action currents of the cardiac muscle (Fig. 96), and it is obtained by leading these currents to an hypersensitive galvanometer; the circuits of the moving parts of which are recorded by a shadow that is made permanently upon a moving strip of film (Figs. 101 and 102).

Certain established physiologic facts are essential to a full appreciation of electrocardiography. First, every muscle contraction has associated

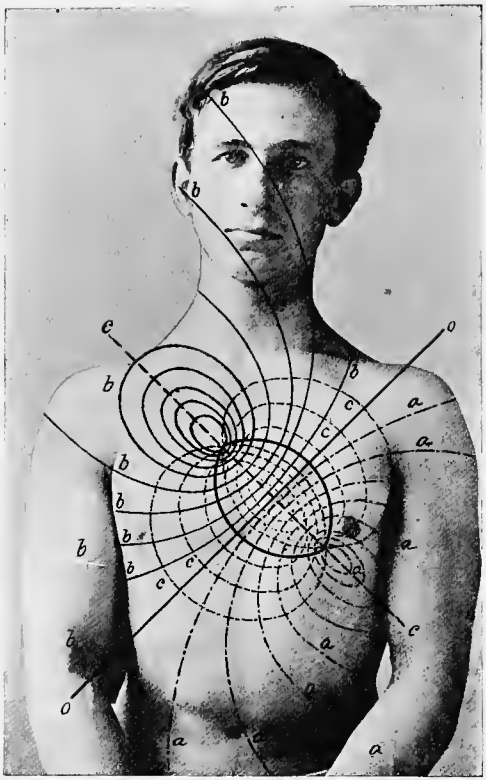


FIG. 96.—LINES OF DIFFUSION OF THE CARDIAC ACTION CURRENT (after Waller).

with it definite changes of electric potential; for example, a piece of muscle becomes in reality a feeble galvanic cell, in which the active portion of the muscle corresponds to the zinc plate and the passive to the copper, and when these two are connected the current which passes is designated the action current of the muscle. Again, the surface of that portion of muscle which is for the time active is negative to all the remaining muscle surface, which is at the same time positive (Fig. 97).

The electrocardiogram in health is a curve displaying general characteristics, although it is subject to certain variations. The cardiogram varies within rather wide limitations in pathologic conditions of the heart, and this is of great interest to the clinician when it placards definite anatomic changes and abnormalities in function. In referring to the

factors, which in turn cause changes in the form of the electric curve, it is to be remembered that this curve is solely the expression of changes in the electric conditions of the heart, and is due to muscular action. Movement of the blood-stream and mechanical disturbances of the cardiac

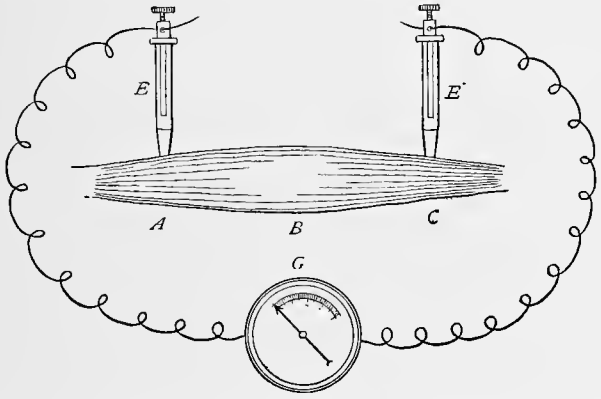


FIG. 97.—SCHEME TO ILLUSTRATE THE ACTION CURRENT OF A SIMPLE MUSCULAR CONTRACTION (James and Wilson, in Amer. Jour. Med. Sci., Sept., 1910).

valves do not exercise any direct effect upon this record. Another factor is that the character of the curve varies in accordance with the points of the body surface from which the current is led off. Likewise variations in the position of the heart will cause rather marked changes in the electrocardio-

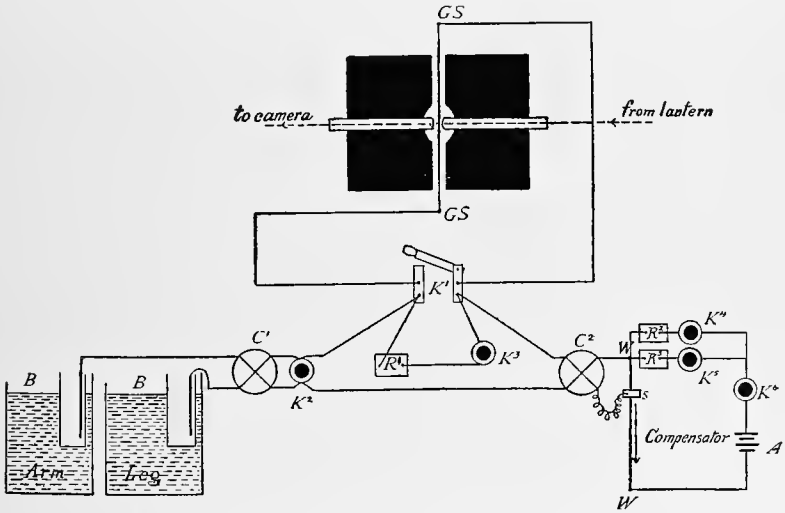


FIG. 98.—SCHEME OF CONNECTION EMPLOYED IN GALVANOMETRIC WORK FOR CLINICAL USE. *GS*, Galvanometer string; *K*, key; *R*, resistance; *C*, commutator; *A*, accumulator; *B*, bath; *W*, *W*, compensator high-resistance wire.

gram. Decided changes in the shape of the muscle mass also affect the curve, as do those contractions which originate in abnormal localities and pursue an unusual course through the muscle.

Leads.—The electrocardiographic curve is obtained by leading off from various points of the body, and for clinical purposes Einthoven recognizes three leads taken in pairs:

(1) Leading off from the right arm and left arm.
(2) Leading off from the right arm and left leg.
(3) Leading off from the left arm and left leg.

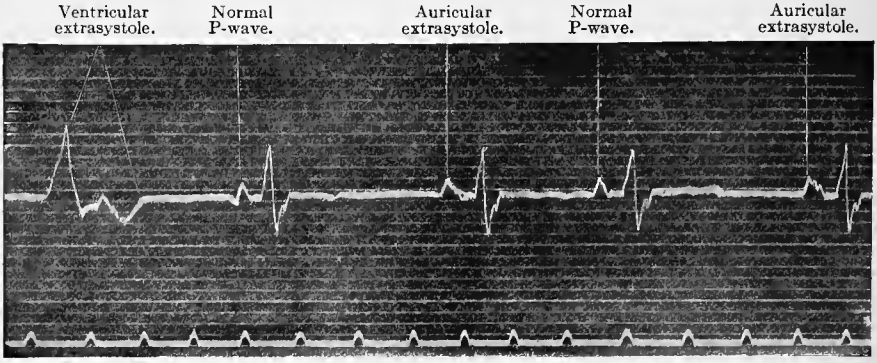


FIG. 99.—ELECTROCARDIOGRAM, SHOWING AURICULAR EXTRASYSTOLE. THE CURVE ALSO SHOWS VENTRICULAR EXTRASYSTOLE. Patient Philadelphia General Hospital (Cope).

Physiologic Mammalian Electrocardiogram.—The fact that no two individuals present during health exactly similar curves renders this problem one of considerable difficulty, but when we come to consider the lead 1 (from the right arm and left arm), certain features are common to the

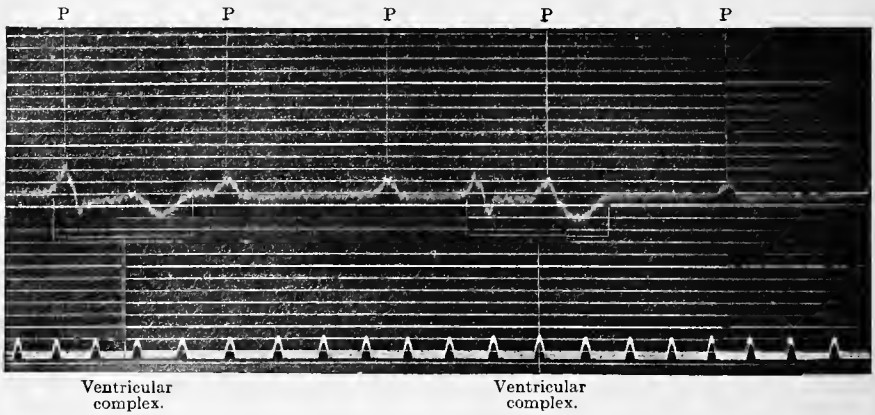


FIG. 100.—ELECTROCARDIOGRAM FROM A PATIENT SHOWING COMPLETE HEART-BLOCK (patient studied by Dr. T. A. Cope).

The P-wave, representing the auricular action, is independent of the ventricular contraction.

electrocardiogram. Again, the electrocardiogram of the normal subject consists of two parts: (*a*) auricular; (*b*) ventricular complex.

Auricular Complex.—The auricular complex is composed of a primary deviation in the "base negative" or upward direction. In the terminology of Einthoven, which is generally adopted, it is termed the summit P (Fig. 100). This summit P may be either rounded or pointed and is suc-

ceeded by a horizontal line (iso-electric period), or by a somewhat less conspicuous deviation in the opposite, "base positive," or downward direction.

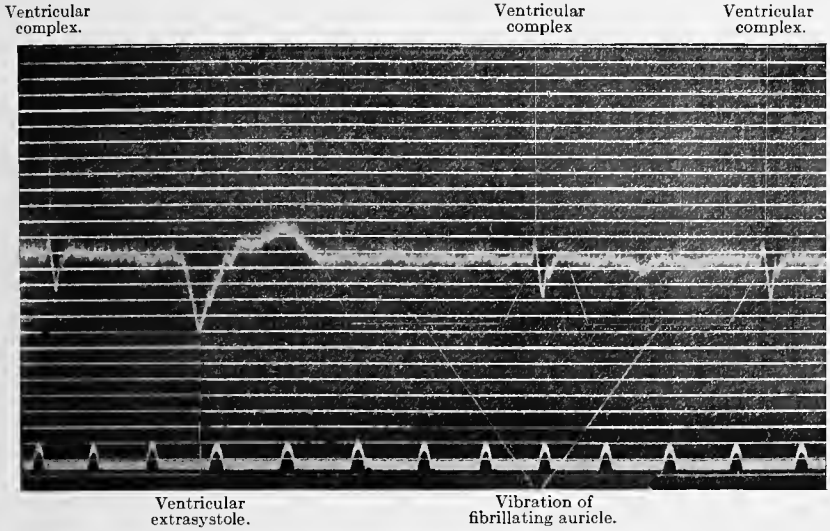


Fig. 101.—Electrocardiogram, Showing Auricular Fibrillation. Private patient) (Cope). Note entire absence of P-wave, but instead a continued slight vibration.

Ventricular Complex.—This is, as a rule, triphasic, and is composed of the variations R, S, and T (Fig. 101), of which the R and the T are deflected upward, while the S-curve is directed downward (Fig. 102). R is ordinarily

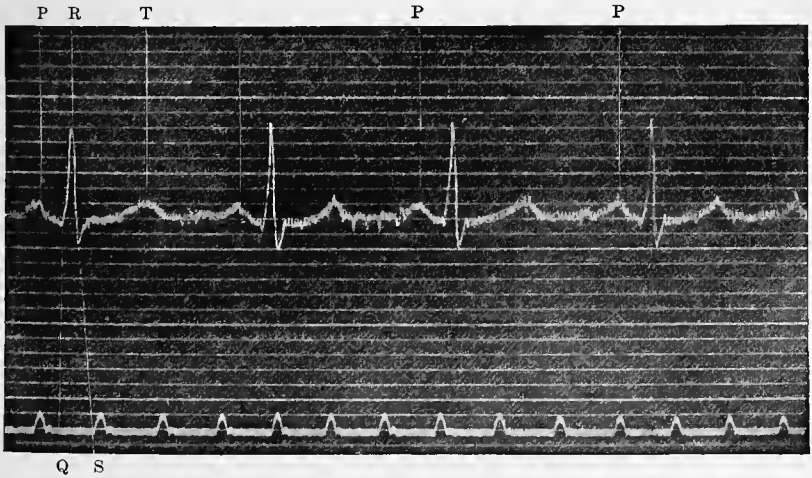


Fig. 102.—Electrocardiogram from a Case of Mitral Stenosis. University Hospital (Cope). Note unusually high P-wave (high as T-wave), denoting hypertrophic auricle.

found to be the most conspicuous summit in the curve; its duration is appreciably short (approximately .03 second). R may be preceded by a small and short deviation in the opposite direction, the summit Q (Fig. 102).

R is followed by a short downward deviation (S), which varies considerably in amplitude, often being scarcely perceptible, although it may be conspicuous.

It is immediately apparent that the opening phase of the electrocardiogram consists of summit P, which is associated with auricular contraction; summits Q, R, and S, which are associated with the initial events of ventricular systole. It is to be seen (Figs. 101 and 103) that the preceding group of variations is followed by a horizontal line of varied length, during which the contacts are iso-electric (contacts with the patient when no current is passing through the string), and the curve continues ending in broad and somewhat prolonged variation (T).

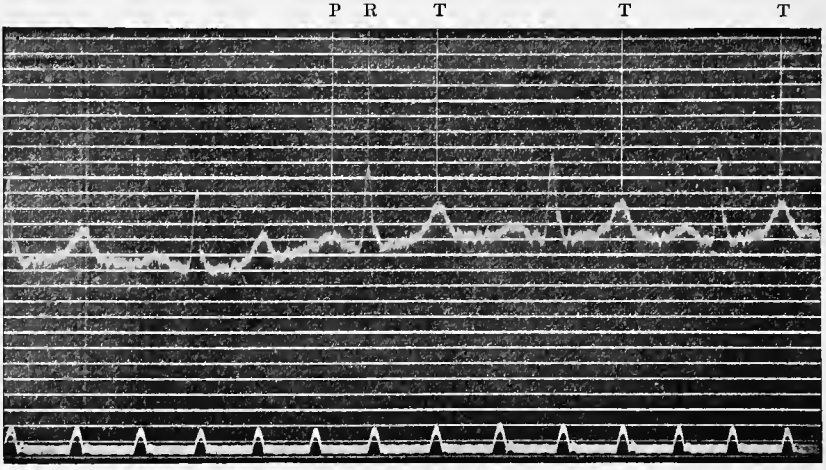


FIG. 103.—ELECTROCARDIOGRAM FROM A CASE OF HYPERTROPHIED LEFT VENTRICLE, ACCOMPANIED BY HIGH BLOOD-PRESSURE (Cope).
Note the high T-wave, denoting strong action current.

Time Relationship.—The electrocardiogram may be taken simultaneously with records from the auricular and ventricular musculature, as well as the sphygmographic curve, the intraventricular pressure curve, and the heart-sounds (Fig. 103). Through a correlative study of these records it has been possible to determine that P stands in direct relationship to the auricular systole, and that the upstroke P antedates by .012 to .017 second the stroke R (a phase known in the normal human electrocardiogram as the P–R interval). The upstroke R ordinarily precedes the beginning of ventricular contraction by .03 second, an interval referred to as "a measure of the latency of contraction" (Lewis). Summit T occurs during the systole of the ventricle and subsides abruptly .03 second before the occurrence of the second or the auricular sound.

Pathologic alterations in the electrocardiogram are seen when dissociation of the auricular and ventricular rhythm obtains, at which time summit P appears at uniform intervals in the curve (Figs. 101–103), but does not bear a constant relationship to the ventricular complex, which is also present.

Difficulty in interpreting the events upon which the physiologic type depends are very great, and at present we have but a superficial grasp of the factors determining its component parts. These difficulties are: (*a*)

the complexity of the path by which the contraction wave enters the ventricle, and (*b*) the variations in the course of fibers in the wall of the ventricle. Similar difficulties are likewise encountered in the study of electrocardiograms from patients presenting various types of arrythmia, yet definite changes in the electric record are rightly associated with certain pathologic entities, as is shown by the accompanying illustrations.

The reader is referred for complete technic, etc., to special works devoted to the subject.*

MENSURATION OF CHEST IN CARDIAC DISEASE.

By this clinical method it is possible to ascertain the circumference of the chest at different levels, *e. g.*, at the ensiform cartilage, the nipple, and the axilla. It is also possible to determine the measurements of but one side of the chest, as well as the amount of unilateral expansion during the act of respiration.

PERCUSSION.

Percussion serves, in great measure, to confirm the findings secured by inspection and palpation. With reference to cardiac conditions, percussion serves, first, to distinguish between cardiac enlargement, the result of either hypertrophy or dilatation, and pericardial effusion. In pericardial effusion the lower portion of the posterior lobe of the left lung becomes airless, as the result of pressure exerted by the effusion; hence dullness below the angle of the left scapula is a conspicuous sign of pericardial effusion. In performing percussion for the purpose of detecting diseases of the heart and pericardium, it is almost always necessary to distinguish between diseases of these organs and diseases of the lung and pleura. It shall, therefore, be our aim to emphasize, under each particular disease, the advantages of percussion; and here we wish again to call the reader's attention to the great advantages to be gained by the use of auscultatory percussion (p. 59) in differentiating between both diseased and healthy viscera that are in close proximity. (See Percussion of the Lung, p. 55.)

AUSCULTATION.

This method of physical diagnosis provides a means of obtaining the most valuable data with reference to diseases of the heart. In auscultating the clinician should first place his ear over the various areas at which the cardiac lesions are best heard—*e. g.*, the right and left second intercostal areas, the apex, and at the ensiform cartilage. The skilled observer can detect with the ear the character of both the first and second sounds of the heart, and determine accurately the condition of the cardiac muscle, weakness of which is also further placarded by arhythmia.

In order to recognize certain abnormal sounds heard over the precordium, or, as is often the case, exclude other sounds, the stethoscope serves as a valuable means of diagnosis. It is also possible, by the aid of the stethoscope (Figs. 104 and 105), to trace certain murmurs throughout their

* T. Lewis, "Mechanism of the Heart-Beat," 1911.

various areas of distribution, as well as to determine their points of greatest intensity. The stethoscope enables the clinician to obtain valuable data regarding endocardial, pericardial, and pleuropericardial murmurs. (See Auscultation of the Lung, p. 63.)

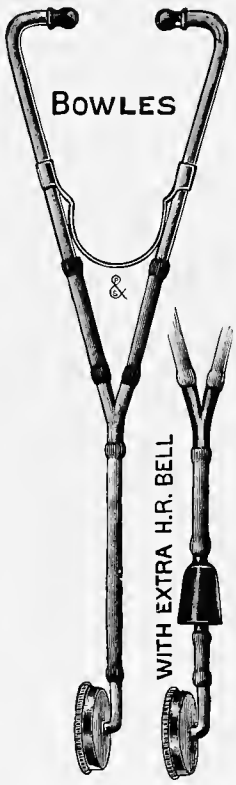


FIG. 104.—BOWLES' STETHOSCOPE.
Regular pattern.

FIG. 105.—BOWLES' STETHOSCOPE.
Flat iron pattern.

NORMAL HEART-SOUNDS.

When the stethoscope is applied over the heart in the third and fourth interspaces, within and in the left parasternal line, there is heard a rhythmic alternation of sounds and pauses. These sounds may be distinctly audible over all the precordium, and for some distance beyond it. The predominant sound is synchronous with the apex-beat and carotid pulse, and hence is called the *systolic* or *first* sound, because it coincides with the systole or the ventricular contraction of the heart. A short pause follows, which is in turn followed by a different sound—the second or diastolic sound. The second sound occurs at the beginning of a longer pause, corresponding to the diastole. The two sounds of the heart are often represented by the respective monosyllables, *lub-dub*. The first sound and short pause, together with the second sound and long pause, constitute the cycle. This rhythm of sounds and silences is not clear in infants.

First Sound.—Here the quality of the systolic sound is a dull, booming "lub," and its intensity is marked, while the pitch is relatively low and the duration long.

Second Sound.—The quality of the second sound is sharp and clicking. Its intensity is less loud than that of the first sound; while the pitch is distinctly higher. The duration is decidedly short.

The first and second heart-sounds may be heard over the whole precordium; their accent varies at different points, but the rhythm is maintained. The first sound corresponds to the ventricular systole, and its accentuation is heard at a point where the ventricular conduction of sound is clearest at or near the apex of the heart. The second sound is accentuated at the base of the heart.

Causes of Sounds.—The first sound is produced by the synchronous closure of both the mitral and tricuspid valves, and the synchronous contractions of the ventricles.

The *second sound* is undoubtedly caused by short closure of the pulmonary and aortic valves. Both sounds are caused practically by valvular action, although the character of the sound may be appreciably altered in affections influencing the vigor of the muscular contractions.

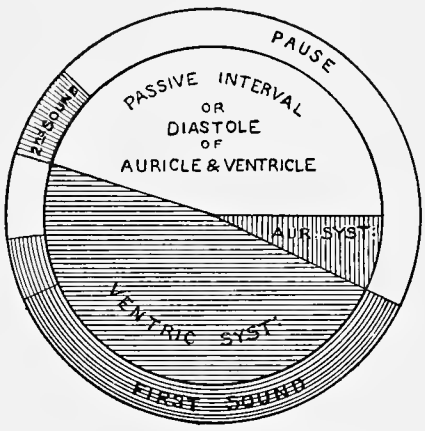


Fig. 106.—Diagrammatic Representation of the Movements and Sounds of the Heart—the Cardiac Cycle. (After Sharpey.)

The Cardiac Cycle.—The two heart-sounds and their production will be understood by reviewing the physiologic movement of the blood through the heart: "The blood flows from the body through the cavæ into the right auricle, whence, during the ventricular diastole, it passes through the right auriculoventricular opening, the tricuspid valve, into the right ventricle, being urged forward toward the end of the diastole by the weak muscular contraction of the right auricle. The systole which immediately follows drives the blood out of the ventricle, the tricuspid valve being at the same time closed, through the open pulmonary semilunar valve, into the pulmonary artery. The blood, prevented from flowing back into the ventricle during the diastole, which immediately follows, by the closure of the pulmonary semilunar valve, passes through the lungs, and from them flows through the left auriculoventricular opening, the mitral valve, into the left ventricle, whither it is again assisted at the end of the diastole by the contraction of the auricle. The left ventricle discharges its contents during the systole (mitral valve being closed) into the commencement of the aorta, through the open aortic semilunar valve, whence it is prevented from returning to the ventricle when the pressure from the ventricle ceases and the diastole begins, by the closure of the aortic semilunar valve. The blood then flows from the conus aortæ into the body" (Vierordt). (See also Fig. 106.)

In the second place the blood enters the aorta and pulmonary artery at the same time by the synchronous contractions of the two ventricles. With

the completion of systole relaxation of the ventricle begins, and at once the recoil of the arterial walls forces the columns of blood against the semilunar valves, which close with the snap of sudden tension at the commencement of the ventricular diastole. The four sounds that are created, one at each valve orifice, are normally audible as two sounds, because of the simultaneous closure of each homologous pair. The accompanying illustration, after Sharpey, serves to illustrate the cardiac cycle (Fig. 106).

The systolic sound, as stated, is partly valvular and partly muscular. It is to be remembered that the valves at the two orifices act simultaneously, consequently it becomes the part of physical diagnosis to determine whether the right or the left heart is the seat of any alteration in sound that may be audible. In health the condition of the cardiac muscle is not greatly concerned with reference to rhythm. (See Pathologic Conditions of Heart.)

Valve Areas.—A superficial area of half an inch square will include a portion of all four sets of cardiac valves (Fig. 60), so that stethoscopic examination here fails to detect the valve affected. Special valve areas are selected for auscultating the heart, and represent the points to which the vibrations from the corresponding valves are best conducted; consequently we have the mitral, the tricuspid, the aortic, and the pulmonary areas.

Maximum Intensity.—The areas of greatest intensity of the first, systolic or ventricular sound is near the apex; and the second diastolic sound is at the base of the heart.

(1) **Mitral Area.**—The sound produced by the closure of the mitral valve (at systole) is most clearly heard at the apex, within an area about 1 inch in diameter.

(2) **Aortic Area.**—The second right intercostal space near the border of the sternum is the point of maximum sound caused by the closure of the aortic leaflets.

(3) **Tricuspid Area.**—The point of election for auscultating the tricuspid element of the first sound of the heart is at the lower part of the sternum, especially near the left border, opposite the fourth and fifth interspaces.

(4) **Pulmonic Area.**—The closure of the pulmonary valve is heard best at the second left interspace, near the sternal border, or at the sternal end of the third left costal cartilage, a point directly over the valve.

The points of election just given are used to locate the seat of production of organic valvular murmurs, as well as for the purpose of differentiating the separate sounds themselves. The physiologic events causing the first and second heart-sounds as heard at the four valve areas have been summarized by Vierordt as follows:

"Apex of the heart (mitral orifice):

"First sound: Closure of the mitral valves and ventricular contraction.

"Second sound: Prolonged aortic second sound (closure of aortic valve).

"Under the sternum (tricuspid orifice):

"First sound: Closure of the tricuspid valves and ventricular contraction.

"Second sound: Prolonged pulmonary second sound.

"Second intercostal space, right or left (aorta, pulmonary artery):

"First sound: Sudden filling of the beginning of the aorta, of the pulmonary artery, and continuation of the first ventricular sound.

"Second sound: Closure of the semilunar valves of the aorta, or of the pulmonary artery."

Physiologic Variations.—The heart may vary in loudness, and both sounds together are relatively increased or diminished in intensity. The condition is often temporary, such increase of intensity depending upon

stimulation, diet, exertion, or mental excitement. More or less persistent loudness may depend upon thinness of the chest, as well as the flexibility and delicacy of the bony thorax in children. On the other hand, in those with thick chest-walls, especially women with large mammary glands, both sounds—the first usually more than the second—are relatively weakened. The heart-sounds also are less distinctly heard in an individual when he is lying on the back than when in upright posture, owing to the heart's swinging back from the precordial region.

"Thus it is apparent that the loudness of the cardiac sounds depends upon the nature and the thickness of the media through which they pass, the degree of blood-pressure within the heart and arteries, and the distance of the vibrating valve orifices and ventricular muscle from the front surface of the chest" (H. S. Anders).

The individual valve sounds vary with age, temperament, vigor, nervousness, and occupation. Variations in such normal qualities as pitch, duration, and rhythm are frequently observed.

In childhood the valvular element of the first sound predominates and has a high-pitched, shorter character. In the vigorous and robust the first sound is often of a loud, prolonged nature, whereas in the fat and indolent it is distant and indistinct.

The component elements of the second sound differ in relative intensity, and in childhood the pulmonic sound is the stronger of the two, while in middle life these sounds are about equal. In old age the aortic sound predominates over the pulmonic.

The rhythm of the first sound may be physiologically disturbed; to produce the so-called doubling, the sound is divided, but without any interval, such as exists between the first and the second sounds.

Reduplication of the second sound, while it may occur normally, is usually pathologic. It is rarely heard at the end of deep inspiration.

Heart-sounds of Pathologic Significance.

Murmurs.—These include all adventitious sounds heard over the precordium or *any* portion of the vascular system. The normal heart-sounds may be appreciably modified and at times replaced by these superadded sounds (murmurs). Murmurs are clinically considered as:

1. Endocardial. { *a.* Organic. / *b.* Hemic (functional).
2. Extracardial.
3. Vascular. { Arterial. / Venous.

Here may be the opportune place to call attention to alterations in the normal cardiac sounds the result of pathologic changes in the myocardium. Myocardial degeneration has been considered at length under Myocarditis (p. 297) and is given special mention here because cardiac murmurs are materially influenced by the force and tone of the cardiac muscle. (See Fatty Degeneration, p. 299.)

Organic endocardial murmurs result from structural defects in the cardiac orifices or their leaflets. Functional murmurs are believed to result from myocardial enfeeblement, together with alterations in the circulating blood.

Extracardial murmurs include the friction murmur of pericarditis; the splashing sound of pneumopericardium (p. 251); and the so-called "car-

diopulmonary murmur," or whiff. There are also fine râles audible synchronously with the heart's impulse (cardiopulmonic râles), a sign of questionable clinical importance. A pleuropericardial murmur is also audible in selected cases.

Vascular Murmurs.—The vascular murmurs heard over the arteries are best described by the bruit of aneurism, and the chucking and pistol-shot sounds, audible over the femoral artery in aortic regurgitation (p. 270).

A venous murmur, "venous hum," deserves mention, and organic heart murmurs may also be heard over the veins of the right side of the neck. (See Tricuspid Regurgitation, p. 289.)

Significance of Murmurs.—The chief physical alterations productive of organic endocardial murmurs are as follows:

(*a*) *Insufficiency*, or incompetency, causing regurgitation of blood when the valves fail to close at the appointed physiologic time.

(*b*) Stenosis, obstruction at the orifices, interfering with the free flow of blood at the time when the valve should normally be open.

(*c*) *Relative insufficiency* at a valve (especially the mitral or tricuspid) orifice, because of dilatation of the heart chamber (weakening of the myocardium) containing it, the simultaneous dilatation of the orifice causing incomplete closure of the leaflets.

Murmurs may occur at non-valvular orifices: *e. g.*, open foramen ovale or at a perforated ventricular septum.

A patulous ductus arteriosus may be responsible for a murmur.

"It will be seen that murmurs may be of several varieties: they may vary in causation, in combination, and in general and specific characteristics. There may be but one murmur present, with distinctive or obscure features, or two or three at different orifices, perhaps two at one orifice (double lesion)" (H. S. Anders).

Points to be Observed.—The observations to be taken regarding every murmur are:

1. Its location or area of greatest intensity.
2. Its time, place it occurs in the cardiac cycle.
3. Area of distribution and of transmission.
4. Acoustic attributes, volume, intensity, pitch, and duration.
5. Relation the murmur bears to the normal heart-sounds.

1. **Localization.**—*Point of Maximum Intensity.*—The first step in the diagnosis of a murmur is to localize it, and thus determine the valve or orifice affected (seat of the lesion). To recapitulate, the points of greatest loudness usually correspond to the areas where the respective valve sounds are best heard; *e. g.*, mitral valve murmurs are most distinctly audible at or near the apex; aortic murmurs, at the right second intercostal space, near the sternum; tricuspid murmurs, over the lower part of the sternum; and pulmonary valve murmurs, at the left second intercostal space, at the sternal border. Any murmur whose maximum intensity does not coincide with one of these areas is probably not valvular in origin.

2. **Rhythm (Time).**—The majority of organic valvular murmurs may be diagnosticated, that is, the lesions producing them may be rather positively inferred upon the basis of the facts of the area of greatest intensity and of the time. Determination of the area of maximum loudness determines the valve or orifice affected, and the time a murmur is heard during the heart's cycle indicates what the normal condition of function should be at that orifice at that given time, and dictates whether the lesion is obstructive or regurgitant.

Two subvarieties of organic murmurs must needs be studied correlatively:

1. Murmurs of regurgitation (insufficiency) are heard at that time during the heart's cycle when the affected valves ought normally to be closed, *e. g.*, they are heard at a systole when the auriculoventricular or venous (mitral and tricuspid) valves leak, and during diastole when the arterial (pulmonic and aortic) valves are diseased.

2. Murmurs of obstruction or stenosis occur at that time in the cardiac cycle when normally blood is passing through the orifices affected; consequently, they are heard during the systole, with disease at the arterial openings, and during diastole, when the auriculoventricular regions are affected.

Time of Murmurs.—The mitral regurgitant murmur is always systolic; the aortic regurgitant, diastolic; the aortic obstructive (stenotic) murmur, systolic; the mitral stenotic, diastolic (presystolic), because it is best heard near the end of diastole or just before systole.

With similar lesions on the right side of the heart, tricuspid and pulmonary valve murmurs have the same times.

The mode of reasoning in the timing of a murmur may be put forth as follows: a murmur that is best heard at or near the apex (the mitral area) and is systolic in rhythm, when normally the mitral valve should be closed. The valve must leak (insufficiency) in order to cause a murmur at this time.

Murmurs are timed by requesting the patient to hold the breath, so as to exclude the occasional intervention of the respiratory murmur. Placing a finger over the carotid or the subclavian arteries, which pulsate synchronous with the first or systolic sound of the heart.

The characteristic features of the murmurs present in aortic regurgitation, mitral regurgitation, and tricuspid regurgitation, as well as those of aortic and mitral stenosis, have been discussed at length in this chapter, and the reader is especially referred to the mechanism of the lesion under each respective heading. An endocardial murmur may be so loud as to obscure a portion of the normal heart-sound, and it is indeed common to meet with cases where these murmurs are so loud as to make it impossible to hear either the first or second sounds of the heart. In myocarditis accompanying endocardial lesions the cardiac rhythm may be so irregular as to cause great confusion with reference to the time and characteristics of a given murmur. The loudness of the heart's sound (muscular element) may also vary at different impulses, and this causes decided confusion in the study of organic murmurs.

Extracardial murmurs have been described in connection with pericarditis, aortic regurgitation (pistol-shot sounds), and aneurism.

X-RAY EVIDENCE IN DISEASES OF THE PERICARDIUM, HEART, AND BLOOD-VESSELS.

By George E. Pfahler, M.D.

THE PERICARDIUM.

Dry pericarditis gives no characteristic appearances by the *x*-ray, but may increase the heart's action, which can be observed fluoroscopically.

Exudative pericarditis gives a characteristic appearance, depending on the amount of exudate.

1. The cardiac shadow is much increased.

2. The outline of this shadow is less clearly marked than where the heart

muscle is observed. This is due to the lesser density of the fluid than of the heart muscle.

3. The complementary spaces are filled up. The cardiohepatic angle becomes a right or obtuse angle, instead of acute. The curve of the apex of the heart is lost (this is best observed when the tube is placed low or on a level with the lower border of the heart). The dome of the left side of the diaphragm is likely to be somewhat flattened (Brauer). This alters the shape of the usual cardiac shadow, and gives it more of a triangular appearance.

4. The movements differ from the usual cardiac pulsation, and this change may be recognized as an early sign, or in this early stage pulsation may be confined to the ventricle. In large effusions there may be only a general pulsatory movement, instead of a sectional wave of contraction, as is seen in the heart normally.

Obliterative pericarditis gives no characteristic appearance.

External mediastinal pericarditis gives, on the other hand, definite signs. If the patient holds his breath, distinct tugging movements of the surrounding tissues may be observed. This appearance is made more evident by deep inspiration or by bending sidewise, which will depend upon the location of the adhesions. If they are on the under surface, when the patient inspires deeply, the heart will be elongated more than usual, and the cardiodiaphragmatic angles will be modified. If they are anteriorly, adhesions to the sternum may at times be seen by oblique illumination (Brauer).

THE HEART.

General Remarks.—Exact measurements of the heart can be made orthodiagraphically (outlined by the central ray, which strikes the screen perpendicularly). This procedure, unfortunately, involves considerable exposure of the operator, and, like most fluoroscopic work, is extremely dangerous. An outline of the heart which is probably as accurate is obtained by long-distance roentgenography (plate 2 meters from the target of the tube—Köhler). The outline of the heart is modified normally by a number of factors, but probably most of all by the respiration. During deep inspiration it is decreased transversely and increased vertically. This is probably due to the rotation of the heart upon its axis. The shadow, as a whole, seems to take a more central position in the chest instead of extending greatly to the left. This modification in the general shape of the cardiac shadow is permitted because of an elongation of the chest cavity, and, therefore, will occur in any condition which permits of this elongation, such as visceroptosis, the phthisical chest, etc. As a result of this elongation and narrowing of the heart shadow, one would get the impression of a smaller heart. (Percussion would, of course, be affected by these same modifications.) During forced expiration, on the other hand, the heart is thrown more transversely, and, therefore, tends to give an impression of a wider heart shadow. Similarly, any condition which crowds the diaphragm upward will give a more transverse position to the heart, and therefore a false impression as to its size.

The size of the heart is affected by age, stature, weight, sex, and posture. These latter modifications have been carefully tabulated, and these tables of averages published by Dietlen and Groedel.* After an accurate record of the size of a heart has been made, it should be compared with carefully

* Groedel, Atlas und Grundriss der Röntgen-diagnostik in der inneren Medizin, Lehmann's Verlag, München, 1909.

prepared tables of this kind, or it is not of great value. Moritz has shown that in standing the cardiac shadow is narrower, while the length is not affected.

However, in the ordinary chest plate one can recognize gross enlargements and variations in shape. Such a record is of decided value in making comparisons in any particular case with subsequent plates made under like conditions, as well as in making an accurate diagnosis.

The *modifications* in the *shape* and *position* of the heart are of far greater diagnostic importance than the determination of its actual sizes. This information can be obtained by the ordinary methods of Roentgen examination of the chest. Stereoscopic methods are adding much to our accuracy.

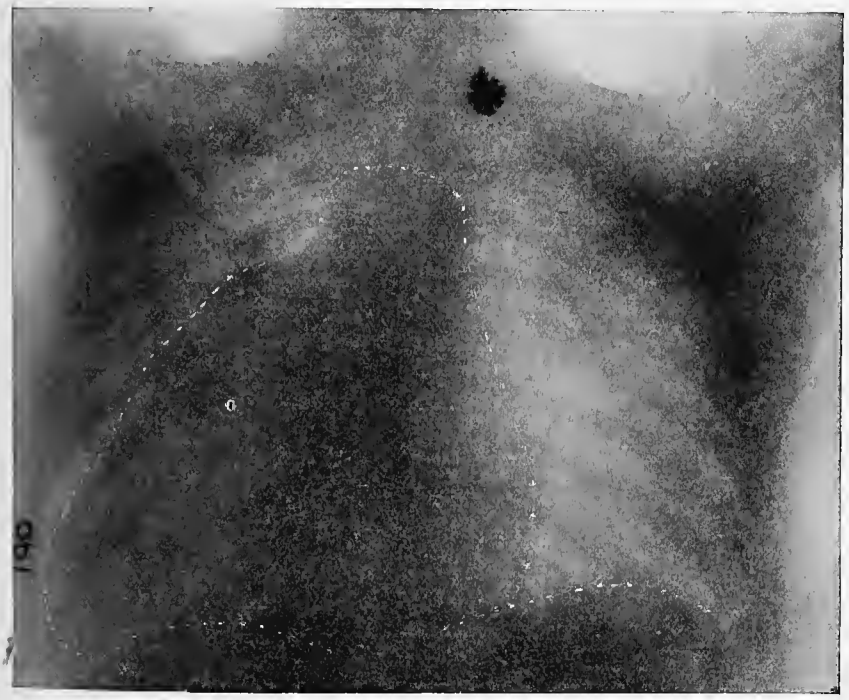


Fig 107.—Cardiac Dilatation (Pfahler).
Due to mitral and aortic disease. Notice the enlargement of the left ventricle, the left auricle, the pulmonary artery, and the right auricle.

Before studying the pathologic shapes of the heart, one must be familiar with the *normol curves*. On the right side, the lower curve indicates the outline of the right auricle, and above this is the curve of the ascending portion of the arch of the aorta. The right ventricle rests upon the diaphragm, and is only occasionally visible, when the upper part of the stomach is distended with gas.

The left border of the cardiac shadow is made up of the curve of the left ventricle below, and above this is the curve of the pulmonary artery. Between these two there is a lighter area of cardiac shadow, due to the left auricle. Normally, this does not stand out as a curve, but in dilatation of

this auricle it can be distinctly seen. Above the curve of the pulmonary artery we see the curve of the descending portion of the arch of the aorta. The latter shadow of the aorta can usually be traced from its origin, and its continuation can be followed posterior to the cardiac shadow.

The Pathologic Heart.—In the study of the pathologic heart one records its size, form, position, its mobility, and its peristaltic movements. Departures from the normal give valuable evidence in each of the various cardiac affections.

Displacements of the heart can be recognized, and their causes demonstrated. When the entire heart is displaced to the right, one must always think of a transposition of the viscera. If the displacement is due to this condition, one will find by the rays a corresponding reversal of the stomach, liver, and spleen.

If due to **adhesions,** these adhesive bands can often be seen, and when examined fluoroscopically, the displacement is sure to become more marked during inspiration. This is usually due to bands which are adherent to the pericardium.

Other displacements are due to abnormal conditions in the surrounding structures. The heart is supported by the great vessels above, and rests upon the diaphragm below. It is pressed by the lungs on each side, which form an elastic cushion. Any modification in any of these structures will tend to displace the heart. Therefore any condition which will raise the left side of the diaphragm (such as eventratia diaphragmatica, abnormal distention of the fundus of the stomach with gas, tumors, subdiaphragmatic abscess, etc.) will raise the heart, and if the right side of the diaphragm is not equally raised, the heart will be rotated to the right. Likewise if the right side of the diaphragm is elevated (enlarged liver, subdiaphragmatic abscess), the heart will be crowded to the left. With an elevation of both sides of the diaphragm (ascites, meteorism, pregnancy) the heart is seen to lie more transversely.

Emphysema causes a depression of the diaphragm, and therefore a more centrally located and a lower elongated cardiac shadow is obtained.

Tuberculosis.—Early apical tuberculosis on the left side may give a high position of the left side of the diaphragm, and, therefore, displacement of the heart to the right. If adhesions are present, it may be drawn to the affected side. With an atelectasis on one side, the heart will be crowded toward the affected side by the compensatory emphysema of the opposite side.

Pneumothorax, pyopneumothorax, and **pleural effusions** may be seen to crowd the heart toward the opposite side.

Aneurisms and **mediastinal new-growths** are variable in their effect upon the position of the heart, but usually there is a displacement downward.

Abnormal Mobility of the Heart.—Adhesions interfere with the normal downward movement of the heart if the attachments are from above, and increase the mobility if from below. All conditions in which one lung is contracted or compressed will be associated with movement of the heart toward the affected side during deep inspiration.

Abnormal Cardiac Pulsations.—Weak and wavy pulsations are seen in tachycardia, especially in myocarditis and in Basedow's disease.

Strong general contractions are seen in bradycardia and heart-block (Groedel), and in general cardiac hypertrophy.

The strongest pulsations of the left ventricle are seen in connection with aortic insufficiency. The shadow of the pulmonary artery may be seen to

pulsate in obstruction to the lesser circulation, especially in connection with mitral insufficiency. Strong pulsation is also seen in persistence of the ductus arteriosus.

In tricuspid insufficiency a strong pulsation of the right auricle may be seen.

Abnormal Size of the Heart.—Chronic nephritis gives a large heart of a globular form.

The small heart which is often found associated with *chronic tuberculosis* is probably, in the first place, a part of the general atrophy of the body, associated with the wasting disease (since it is found in other chronic wasting diseases), and, secondly, the smallness of the shadow is more apparent than real, because of a rotation of the heart upon its axis.

The *left ventricle* may become enlarged in any of the following conditions:

Aortic stenosis,
Aortic regurgitation,
Aneurism of the first part of the aorta.

MITRAL REGURGITATION.

Dilatation of the left ventricle.

OVEREXERTION AS SEEN IN:

Athletes,	Acrobats,
Pugilists,	Marathon runners.

Those following laborious occupations, *e. g.*, stokers, firemen, etc.:

Arteriosclerosis,	Nephritis,
Hepatic cirrhosis,	Alcoholism,
Exophthalmic goiter,	Congenital heart conditions.

Abnormal Form of the Heart.—*Persistence of the ductus arteriosus* gives an increase in the shadow of the arteriopulmonalis (de la Camp).

Aortic insufficiency gives a decided increase in the size of the left ventricle; the shadow of the whole heart is more horizontal, and the apex does not show through the diaphragm.

Aortic stenosis gives a very similar picture, but to a lesser degree, and the enlargement of the left ventricle is proportionately less.

Aortic sclerosis and dilatation give an increased shadow in the region of the ascending aorta.

Mitral stenosis gives a remarkably small heart and enlargement of the left auricle. This cannot always be seen.

Mitral insufficiency gives a general enlargement of the heart—only the shadow of the curves of the great vessels on the right side and the aorta on the left remain unchanged. The heart assumes a globular form. The right auricular shadow is increased, and the enlargement of the left ventricle is more upward, toward the axilla, than to the left.

Tricuspid insufficiency is usually associated with other lesions and, therefore, gives nothing characteristic unless there is a pronounced increase in the shadow of the right auricle. The right ventricle lies upon the diaphragm and cannot be definitely demonstrated.

THE MEDIASTINUM.

By good technic the entire mediastinum can be explored, and not only a positive, but often a negative, diagnosis can be made. Anteroposterior, postero-anterior, and oblique views, and then familiarity with the normal appearances, are necessary.

Mediastinal Tumors.—These are:

1. Tumors involving the mediastinal lymphatic glands, either primary or secondary, and due to tuberculosis, syphilis, leukemia, pseudoleukemia, carcinoma, and sarcoma.
2. Cystic tumors (simple dermoid or echinococcus).
3. Substernal struma and thymus tumors.

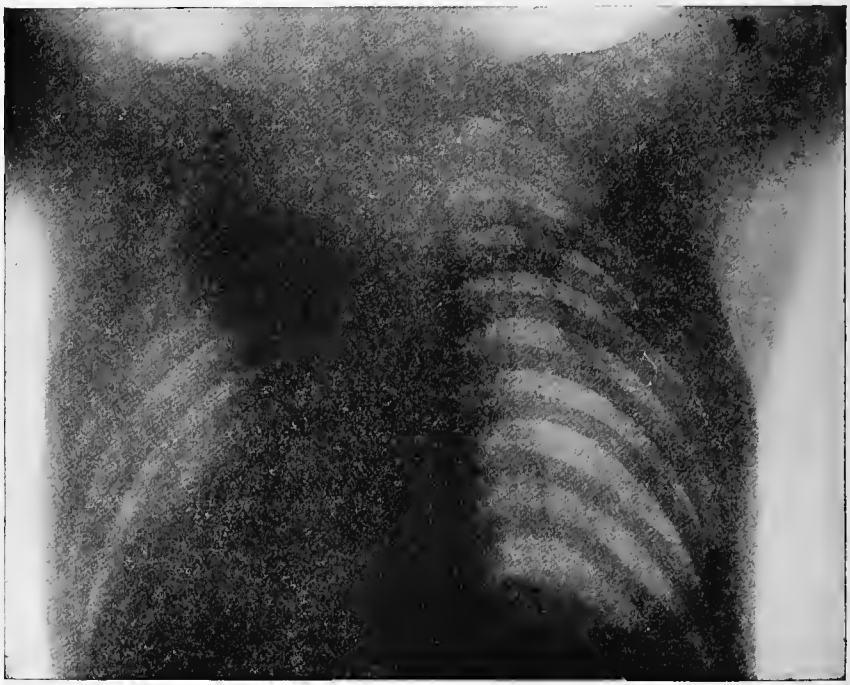


FIG. 108.—ANEURISM OF THE ARCH OF THE AORTA (Pfahler).
Note compression and congestion of the left lung.

In general, if the patient is examined before dyspnea and weakness become too marked, and before there are marked secondary changes in the surrounding lung tissue, one obtains rather definite outlines of the tumors. They should be examined both roentgenoscopically and roentgenographically.

The size, shape, extent, and definite location can be obtained in relation to other organs or tissues. The degree of density of the shadow, and whether multiple (nodular) or uniform, should be noted. One should decide upon the absence of expansile pulsation to eliminate aneurism. Pulsatory movements may be transmitted from the great vessels, but these are not expansile. In

this, as in all other affections, all the clinical evidence should be taken into consideration in making a diagnosis. In my observations in the study of metastatic mediastinal carcinoma there is usually a rather diffuse central shadow in the upper part of the mediastinum, and from this radiating shadows of small tumors can be seen extending into the lung area. The larger tumors of the mediastinum are often sharply outlined, and may be single or multiple. If single, the absence of expansile pulsation, the peculiar shape, and the location outside of the line of the aorta will eliminate aneurism.

ANEURISM.

In *aneurism* one finds an abnormal shadow in the course of the aorta. One should give attention to its size, form, location, degree of density, pul-

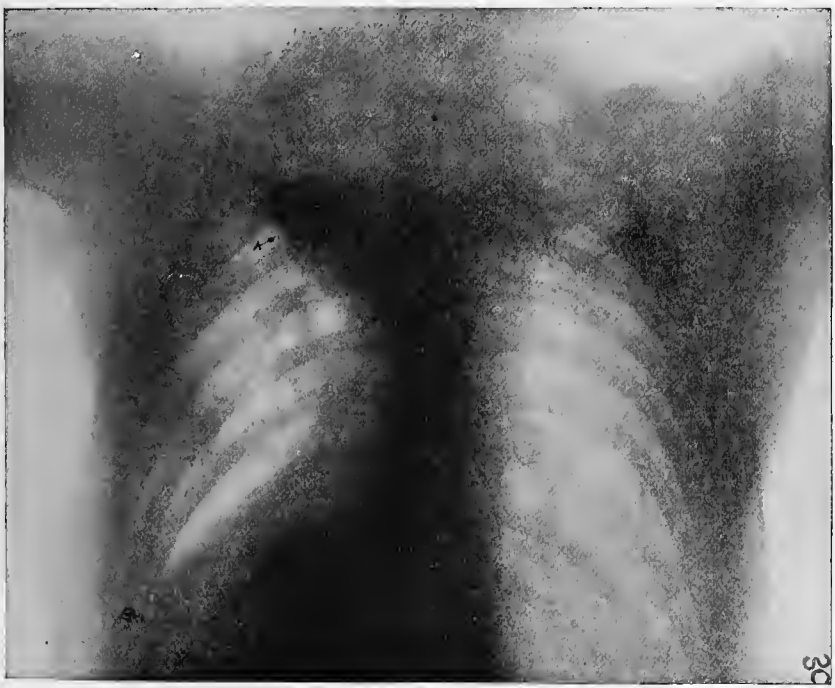


FIG. 109.—TORTUOSITY OF THE ARCH OF THE AORTA, SIMULATING ANEURISM (Pfahler). Notice the projection outward on the level with the second interspace. Autopsy in this case showed no aneurism. The left ventricle is hypertrophied.

sating appearances, the movement in swallowing, the delay in the passage of food through the esophagus (Lange), and any changes in the position of neighboring organs.

Examinations should be made both fluoroscopically and by plates. With the fluoroscope one can usually recognize the expansile pulsations, and with the plate a permanent record is made which enables one to recognize changes that may occur.

Pulsation.—Probably the most important point of investigation, after an abnormal shadow has been found, is to determine the presence or absence

of pulsation. This pulsation should be of an expansile character, and when obtained, it is pathognomonic. Pulsation may be transmitted to other mediastinal tumors, but this is not of an expansile character. On the other hand, an aneurism may not give pulsation if its walls are very thick, or if filled with an organized clot.

Its border is usually round and distinctly outlined by contrast with the transparent lung. This will not be true, however, if adhesions have formed or if there is accompanying atelectasis of the lung.

One should be very careful to make examinations in different positions. I have examined several patients who only complained of anginoid pain under the sternum, and in whom the usual physical signs were absent, but by the rays I was enabled to demonstrate a thin, flat, dissecting aneurism under the sternum, which gave an expansile pulsation, and which was visible only anteriorly. A plate made with the patients lying on the back in these instances would not have shown the aneurism.

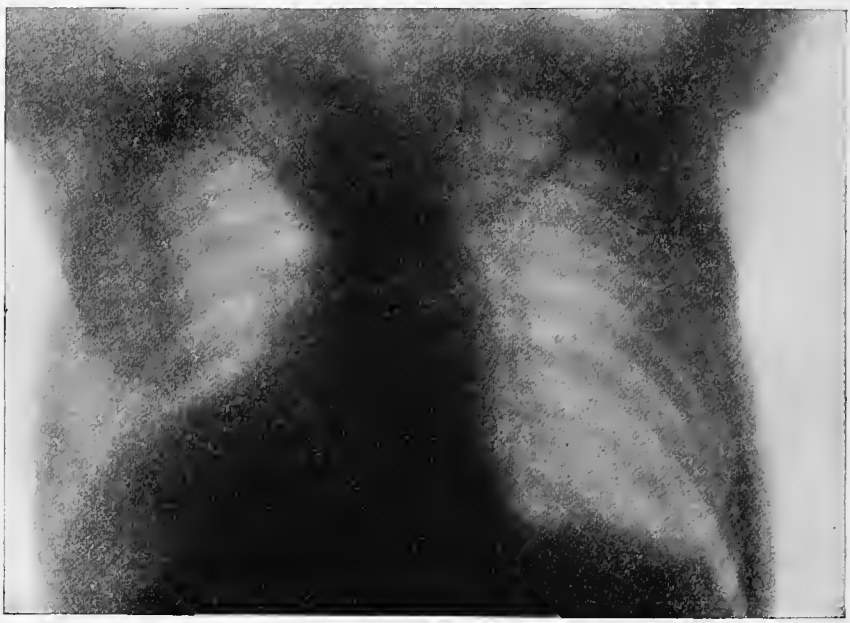


FIG. 110.—TORTUOSITY OF THE ARCH OF THE AORTA, WITH MARKED ENLARGEMENT OF THE LEFT VENTRICLE (Pfahler).

Tortuosity of the aorta gives appearances simulating a beginning aneurism in the descending portion of the arch of the aorta. There is a bulging in the shadow at this point, and often there are associated suggestive physical signs, such as diminution in the left radial pulse, episternal pulsation, and an abnormal area of dullness in the left second interspace, but fluoroscopically one does not get any expansile pulsation. With this condition one usually finds evidence of arteriosclerosis elsewhere in the body.

Arteriosclerosis in the extremities can often be demonstrated. This depends upon a deposit of lime salts in the vessel-walls. In the upper extremities the Roentgen examination will not often be necessary, but in the

lower extremities, where the arteries are not so easily palpated, the sclerosis can be shown. In *dysbasia arteriosclerotica* one may only find isolated plaques instead of the entire outline of the artery (Krause).

DISEASES OF THE PERICARDIUM.

PERICARDITIS.

Pathologic Definition.—An acute or subacute condition, characterized by inflammatory changes in the serous coverings of the heart; these changes are usually localized, although they may occasionally be general. At the onset the serous membrane is smooth, swollen, and congested, and punctate ecchymotic spots may be visible; with the progress of the disease, however, the affected serous surface becomes grayish in color and roughened, as the result of the deposit of a thin layer of fibrin. If the accumulation of fibrin upon the inflamed surface is profuse, the friction of the two surfaces of the pericardium gives it a honeycombed appearance. Following acute pericarditis there may be an accumulation of a serous exudate into the pericardial sac.

Varieties.—For convenience of study inflammatory changes of the pericardium may be considered under the following subheads: (1) Acute plastic or fibrinous; (2) subacute or serofibrinous; (3) purulent; (4) hemorrhagic; (5) chronic adhesive; and (6) tuberculosis of the pericardium as the result of direct extension from the lung.

Predisposing and Exciting Factors.—In cases representing the different varieties of pericarditis special contributing factors will be found that make classification of the conditions that predispose to this malady difficult. In acute plastic and in the serofibrinous variety of pericarditis the etiologic factors are practically the same, and the origin of the disease is bacterial. Acute plastic pericarditis frequently attacks males during early adult life, and, indeed, the disease not infrequently occurs as a complication of acute articular rheumatism, chronic nephritis, lobar pneumonia, and less often is it seen during the course of other acute infections. Barring the few cases in which pericarditis is either tuberculous or cancerous as the result of direct extension from adjacent viscera, we find that the infective agents are conveyed to the pericardium through the circulatory system. Too great importance cannot be attached to the development of a pericarditis as the result of direct extension from the lung, pleura, esophagus, or bronchial glands. Pericarditis may be seen following disease of the aortic valves, but this particular form of direct extension of disease to the pericardium is far less common than the other varieties mentioned. Acute pericarditis is prone to develop, without plausible explanation, during the course of acute articular rheumatism. Certain other acute maladies appear to show a predilection to attack the pericardium and other serous membranes; among these should be mentioned chorea, gonorrhea, scarlet fever, and epidemic meningitis.

Bacteriology.—When a pus-producing micro-organism gains access to the pericardium, it may set up an acute pericarditis. Among the bacteria recovered from the pericardial sac are the Staphylococcus aureus, the pneumococcus, the gonococcus, the tubercle bacillus, the streptococcus, Bacillus coli communis, and Bacillus pyocyaneus. The symptoms of pericarditis may be displayed, and yet no bacteria be found present in the pericardial fluid.

Acute Plastic Pericarditis.

Pathologic Definition.—The pathologic changes upon which the symptoms and signs are based are the presence of early localized areas of congestion and punctate ecchymotic spots, and, later, the same areas become roughened and covered with fibrin.

Principal Complaint.—Since, as previously stated, acute plastic pericarditis is seldom a primary malady, there may be but few, if any, symptoms pointing directly to this condition. As a rule, the symptoms are obscure. A history of acute articular rheumatism is common, although even in this class of cases subjective symptoms may be lacking.

In selected cases of a severe type the patient complains of *pain* in the region of the precordium, and of a feeling of *distress* or *constriction* about the chest. Actual pain may be absent. When pain is well established, with the accumulation of fluid in the pericardial sac, it diminishes gradually, but prior to this time the patient often complains of distress or pain that radiates from the heart to the left shoulder, the back, and the left arm. Pain in the region of the ensiform cartilage and over the upper portion of the abdomen may also be a marked or, at least, an annoying symptom. The pain of pericarditis is distinguished from similar thoracic pains by the fact that it is uninfluenced by pressure over the heart.

Palpitation may be experienced before there is distinct pain, and as the condition advances, this annoying symptom is likely to become more and more pronounced.

Dyspnea is a frequent, although by no means constant, complaint during the early stage of pericarditis.

Thermic Features.—The temperature will be found to rise one, two, or three degrees, depending upon the severity of the case in question. If pericarditis develops as a complication during the course of another febrile malady, rise in the temperature of one or two degrees is to be expected.

Physical Signs.—Inspection.—In those cases in which pericardial pain is severe the expression is anxious and the features are somewhat pinched. If dyspnea is present, rapid action of the chest and distention of the nostrils occurs. The impulse of the apex-beat is always vigorous and frequent.

Palpation confirms what has previously been detected by inspection, *i. e.*, the character of the respiration and of the apex-beat. In the early stage of pericarditis, and while the serous surfaces are comparatively dry, the hand over the heart will detect a distinct friction fremitus. This fremitus results from the rubbing of the congested or roughened pericardial layers one upon the other, and is, as a rule, most pronounced near the base of the heart and to the left of the sternal margin.

At the onset the *pulse* is increased in frequency, of good force, and the tension remains normal or increased until late in the disease, when it becomes appreciably weakened. If the cardiac muscle becomes involved later, the pulse is irregular.

Auscultation.—Among the signs of pericarditis the most valuable is the distinct friction sound, which is usually synchronous with the heart's action. The site of greatest intensity of the friction murmur is ordinarily at the junction of the fourth or fifth interspace with the sternum (Fig. 111), although it is commonly audible over the greater portion of the base of the heart. The friction murmur may, in selected cases, be distinctly localized to a small area, or it may be most intense over certain selected areas located near the

base of the heart, and, indeed, the friction murmur may be heard best at the original location of some one of the endocardial murmurs. If the friction murmur is most distinct over an area where endocardial disease of a certain valve would be audible, the distinction between these murmurs is made by exerting firm pressure upon the stethoscope, when, if the murmur has its origin in the endocardium, it is not affected by pressure; on the other hand, pericardial murmurs are often intensified as the result of bringing a greater area of the diseased pericardial layers into proximity. Pressure sufficiently strong to cause the layers of the pericardium to remain in contact would, on the other hand, prevent a pericardial murmur. Pleural and pleuropericardial murmurs may also be audible and vary with the acts of respiration.

Forced inspiration may possibly influence pericardial murmurs. There is no distinct quality that attaches itself to the pericardial friction-sound, yet in the majority of cases this sound is harsh, grating, or rubbing in character, and at times has a somewhat crackling quality. When the character

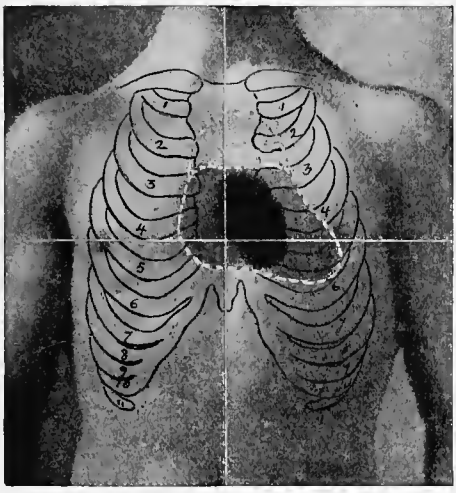


Fig. 111.—Shaded Area Shows Where Pericardial Friction Murmur is Commonly Heard and Where Friction Fremitus is to be Detected.

of the exudate is soft and the action of the heart unusually feeble, a soft murmur is heard, and, indeed, a characteristic murmur may be absent. A marked feature of the pericardial friction-sound is its superficial character.

Time.—The pericardial murmur may apparently be double, and seem to be produced by the movements of the heart, yet the murmurs in all cases are not synchronous with the heart-sounds. The murmur may or may not exceed the sound of the heart in volume and in duration.

Caution.—"A to-and-fro friction sound is, as a rule, indicative of plastic pericarditis, although it is an error to regard it as an infallible sign, since complete calcification of the coronary arteries, as well as excessive dryness of the pericardial surfaces, may rarely produce friction murmurs" (Anders).

Laboratory Diagnosis.—The character of the urine will be influenced largely by the preëxisting disease in which pericarditis developed as a complication. As a rule, it is diminished in quantity, of high color, and rich in solids. Fluid recovered from the pericardium may contain

pathogenic bacteria. A case now under the care of one of us at the Philadelphia Hospital was found to have tubercle bacilli in the pericardial fluid twelve days after onset of pericarditis.

Summary of Diagnosis.—The clinical history, and particularly the preëxistence of rheumatism, chorea, and gonorrhea, should not be neglected. Other diseases of the thorax, and particularly of the lung and pleura, may precede, and often have a direct bearing upon, acute plastic pericarditis. The character of the pericardial distress and possibly the existence of pain radiating to the arm and upper portion of the abdomen, occurring in those who have recently displayed an elevation of temperature of one or two degrees, should suggest pericardial involvement. The detection of a friction fremitus over the heart, and especially the presence of a friction murmur, are among the most important data in establishing a diagnosis.

Clinical Course and Duration.—Favorable cases of acute plastic pericarditis terminate in recovery within the course of a few weeks, although complete resolution may not follow; the pericardial exudate frequently continues to form fibrinous tissue, thus materially damaging the pericardium. Acute plastic pericarditis is often the first stage of serofibrinous pericarditis, to be described later. The clinical course is also modified by the presence or absence of complications—*e. g.*, pleurisy, pulmonary diseases, and nephritis, all of which materially retard, and often prevent, permanent recovery.

Complications.—Where the inflammatory process is sufficiently extensive to spread to the external surface of the pericardium and to the pleura, the case becomes one of pleurisy, or the so-called "pleuropericardial" type of this disease, known as mediastinopericarditis.

Subacute or Serofibrinous Pericarditis.

Pathologic Definition.—An accumulation of effused serum in the pericardium. The quantity of serum present varies in mild cases from two to ten ounces, but in the more severe grades of pericarditis one, two, or more pints may be contained within the pericardial sac. The lesions are similar in kind, but are more intense than those of acute plastic pericarditis, just described.

Predisposing and Exciting Factors.—In certain cases the exciting factor is doubtless acute plastic pericarditis, but in the vast majority of instances serofibrinous pericarditis develops during the course of acute articular rheumatism,—in from 30 to 50 per cent. of cases,—chronic nephritis, and chronic pulmonary tuberculosis. "I believe that, exceptionally, both serofibrinous and plastic pericarditis may occur in the course of rheumatic dyscrasia without the slightest evidence of arthritis" (Anders).

Serofibrinous pericarditis may occur as a complication during the course of certain of the acute eruptive fevers, and it may occur in acute lobar pneumonia. The extravasation of serum into the pericardial sac may result from bacterial infection—*e. g.*, by the tubercle bacillus. Inflammatory disease of other portions of the chest may, by direct extension to the pericardium, result in pericardial effusion.

Principal Complaint.—In a small percentage of cases there is doubtless a primary pericarditis, during which the patient complains of a *chill* or of *chilly sensations, anorexia, nausea, vomiting, prostration,* and *feverishness,* and at or about the same period there is experienced a peculiar

dull or *aching sensation* in the chest, although the patient's description of this discomfort may be quite indefinite.

Acute pain is an occasional complaint, and suggests an associated pleuritis. Secondary pericarditis may be well developed without causing any special discomfort or annoyance to the patient. Certain cases experience precordial oppression, with a variable degree of discomfort, pain, and soreness as an early symptom.

Dyspnea may be the initial complaint that points toward pericardial effusion, and in many cases shortness of breath develops simultaneously with the accumulation of fluid in the pericardium. *Orthopnea* is an occasional manifestation. The dyspnea in the case of a large effusion into the pericardium is due to two causes, since pressure is exerted both upon the heart and upon the lungs. Cardiac diastole may be materially interfered with, owing to pressure upon the right ventricle. When this occurs, the great veins fail to discharge their blood freely into the heart, and, as a consequence, the arterial system is imperfectly filled—a condition that must in time reduce the blood-pressure.

Nervous Manifestations.—Headache develops as an early symptom, and may be intense, particularly in those cases in which the circulation is feeble. Delirium may develop during the night, and in severe cases it is continuous, and may progress until stupor, and even coma, intervene. Maniacal delirium has been known to occur.

Thermic Features.—Typical cases display an irregular temperature, varying between 100° and 103° F. In those cases that terminate favorably the fever falls by lysis, whereas in those tending toward a fatal termination the fever is either continuous for an indefinite period, or suddenly rises to from 103° to 105° F., and with such hyperpyrexia the general clinical picture becomes less favorable. In those cases following acute articular rheumatism high fever is of grave prognostic import.

Physical Signs.—Inspection.—At first the skin appears unusually pale, but after a large effusion has accumulated there is cyanosis of both the skin and the mucous surfaces. Duskiness of the face has been a conspicuous feature in our experience. The veins of the neck are prominent, and in extreme cases they may display decided pulsation. The respiratory movements are hurried, labored, and may be irregular in rhythm. If the effusion is large, the expression is anxious, and the patient elects to rest in the recumbent posture, with his head and shoulders well elevated; as the condition advances from bad to worse it may be necessary for him to sit continuously. Should a large effusion be present, he reclines toward the left.

When there is but slight effusion, the apex-beat is exaggerated, but as the exudate increases in volume the heart is forced upward and backward, and the apex lies up and to the left of its normal site. In the presence of a large effusion the apex-beat is weak, but more diffuse than normal. If the pericardial sac is filled with fluid, the apex-beat may be imperceptible, since, owing to the heart being surrounded by liquid, the organ is not in contact with the chest-wall.

In adults who have previously suffered from pleurisy and in whom the lung tissue has shrunken away from the heart, a large pericardial effusion may produce bulging of the left side of the chest. The diaphragm may also be depressed, and the left superior abdominal quadrant rendered unduly prominent as the result of a large pericardial effusion. In children, whenever the pericardial sac becomes well filled with fluid, the interspaces are prominent, and there may be distinct bulging of the precordium. In young

children the respiratory movements of the left side are restricted as the result of a large pericardial effusion. Emaciation is rapid.

Palpation.—*Pulse.*—When the quantity of fluid in the pericardium is small, the pulse is full and strong; when, however, the effusion increases in amount, it may interfere with the heart's action, and the pulse is then apt to be small, feeble, and irregular. When, comparatively speaking, the pericardium is filled with fluid, the radial pulse is often absent during the act of inspiration—the so-called "pulsus paradoxus." (See Pulse, p. 198.)

Palpation confirms in many respects the data ascertained by inspection, and particularly is this true as regards the strength, location, and distribution of the apex-beat. The impulse of the apex-beat is elevated to the left, the degree of elevation depending upon the quantity of pericardial fluid present. Again, the position of the patient also alters the position of the apex-beat. When the pericardium is well filled, and the patient is in the sitting posture, the apex-beat may be imperceptible, but if he is directed to incline his body forward or to lie upon the left side, the apical impulse will be detected at some portion of the chest. When making a clinical analysis of the force of the apex impulse three factors are to be taken into consideration—(*a*) Whether or not the patient is at present suffering from myocardial changes; (*b*) the quantity of fluid contained in the pericardium; and (*c*) pericardial adhesions when associated with cardiac hypertrophy may be responsible for a forcible apex-beat when the pericardium is nearly filled with fluid.

The friction fremitus, which is a common sign in acute plastic pericarditis, may even be present during an effusion, and is oftenest felt over the base of the heart. This fremitus generally returns during the stage of absorption, and may then continue for an indefinite period.

Diminished expansion of the affected side of the chest is occasionally detected in children suffering from a large pericardial effusion, and fluctuation is rarely observed.

Owing to pressure exerted by a large pericardial effusion, the left lobe of the liver may be forced from one to one and one-half inches below its normal position, rendering it easily palpable in those cases in which the abdominal wall is relaxed.

Percussion.—In pericardial effusion the area of cardiac dullness is inverted, and also greatly increased, assuming a triangular outline with the base directed downward when the patient is sitting or standing. The apex of the triangle may be as high as the third or even the second interspace, and is usually most marked along the left sternal border. The lateral boundary lines of cardiac flatness diverge from the apex of the triangular area (Fig. 59), the right passing obliquely downward to the right edge of the sternum to the seventh rib, and the left passing in a similar manner to the anterior axillary line (Fig. 59). A flat note may be obtained well into the left axilla, and, if the effusion is large, in Traube's semilunar space.

In those cases in which the effusion is large and, owing to some anatomic or pathologic cause, occupies a position toward the back of the chest, there may be dullness over the lower lobe of the left lung posteriorly as the result of pressure from the pericardial fluid. In selected cases of pericardial effusion a small area of dullness may be found over the scapular region.

Auscultatory percussion serves to reveal, with considerable accuracy, the lower border of the pericardial fluid, as well as the beginning of liver dullness. Even though the quantity of fluid in the pericardium is moderate,

a flat note is obtained in the fifth interspace immediately to the right of the sternum (patient erect). If the pericardial effusion is large and a portion of the lung anteriorly is compressed or restricted, skodaic resonance is likely to be present around the area of flatness. Again, pleural adhesions may have bound the lung to the anterior wall of the chest, in which case the pericardial fluid is forced to the back of the chest, and the anterior area of cardiac flatness may be smaller than is to be expected from a given quantity of fluid.

Auscultation.—A friction murmur may be audible when the quantity of effusion is moderate during the stage of resorption. The characteristics of this murmur have been described under Plastic Pericarditis (p. 238). If the effusion is large, the sounds of the heart are distant, indistinct, or muffled. The second cardiac sound is less altered by a large pericardial effusion than is the first sound, consequently it may, at times, be heard clearly at the base of the heart throughout the entire course of serofibrinous pericarditis. The lower portion of the left lung may be compressed, in which case bronchial breathing would be audible at that portion of the chest overlying either congested or collapsed pulmonary tissue.

Laboratory Diagnosis.—During the febrile period the *urine* is highly colored, of high specific gravity, and may be rich in solids. The quantity of urine excreted is appreciably diminished in those cases that develop extensive edema.

Serum obtained by aspirating the pericardium will be found to simulate closely that recovered from the pleura in cases of subacute pleurisy. (See p. 150.) Pericardial serous fluid is, as a rule, free from bacteria.

Summary of Diagnosis.—The history of a preëxisting condition that markedly predisposes to pericarditis, such as rheumatism or nephritis, is of great value in formulating a diagnosis. A previous attack of acute plastic pericarditis always suggests the possibility of a serofibrinous type as a sequel. Doubtless, pericardial effusion often escapes notice, since it requires a more careful physical examination of the chest than is, as a rule, made in routine work. The physical signs possess the greatest value; thus the inverted triangular area of flatness and the friction sound, when both are present, make the diagnosis positive. The recovery of fluid from the pericardium by aspiration also furnishes positive evidence. Atypical cases of pericarditis are by no means uncommon, and in these the diagnosis is occasionally made only by exclusion. We recall studying several cases where, due to old pleural adhesions, aspiration alone made the diagnosis possible. (See also *x*-Ray Diagnosis, p. 229.)

Differential Diagnosis.—Cardiac Dilatation.—Unless a clear history of the case can be obtained, cardiac dilatation may be mistaken for pericardial effusion, and, indeed, in our hospital experience we have not infrequently encountered patients admitted to the medical wards in whom this mistake was made. The following table, modified from Anders, shows the points of differentiation between these two conditions:

Pericarditis with Effusion.	Cardiac Dilatation.
Clinical History.	
1. Recent history of gout, acute rheumatism, acute infectious or septic disease, scurvy, nephritis, or tuberculosis, chronic gonorrhea.	1. Usual history of chronic valvular disease of the heart.
2. Fever and slight pain often associated.	2. No fever or pain, as a rule.
3. Nervous symptoms are often present.	3. Absent or but slight.

Pericarditis with Effusion.	Cardiac Dilatation.
Physical Signs.	
4. Inspection often reveals bulging (more marked in the young). Apex-beat is elevated, feeble, and later absent.	4. Apex-beat usually visible, wavy, and diffuse.
5. Heart's impulse usually absent, or occupies center or upper border of dull area. Friction fremitus may be present.	5. Though feeble, the impulse is palpable.
6. Percussion shows a triangular flat area, and the boundary line above changes on altering the position of the patient. There is dull tympany (flatness in massive exudations) in the axillary region. Dullness over left lung below angle of scapula common.	6. Dull area varies with the chambers dilated; it is coexistent with a wavy impulse, does not extend so high (except in mitral stenosis), and does not vary with change of position. There is no dull tympany.
7. Auscultation shows the first sound distant and muffled; a friction-rub is often present.	7. First sound clear, short, and sharp, resembling the second sound (fetal heart). Friction murmur rare, but an endocardial murmur may appear later.
8. *x*-Ray shows triangular, movable shadow. (See page 230.)	8. Upper level of shadow (quadrangular) fixed.
9. Digitalis has little or no influence.	9. Cardiac stimulants cause marked improvement.

Pleurisy.—Serofibrinous pleurisy, in which a large effusion occupies the left pleura, is to be distinguished from pericardial effusion. The pre-existing maladies that predispose to the development of pericarditis also predispose to the development of pleurisy. A clinical feature of great distinctive value is that of pain, which is always acute early during the course of pleurisy—a condition rarely seen in pericarditis. In pleurisy the area of flatness occupies the entire base of the left thorax, and changes perceptibly with the position of the patient, whereas in pericarditis the area of flatness is always triangular when sitting and limited to the pericardial region. The adjacent viscera are displaced to a greater degree by pleural effusion than by fluid in the pericardium. In pericardial effusion the apex-beat is displaced upward and to the left, whereas in a left pleural effusion it is displaced to the right, or may be absent, on account of that portion of the heart being pushed behind the sternum. A pleural friction murmur is heard only with respiration, whereas the pericardial murmur is more or less closely synchronous with the heart's action. (See *x*-Ray Diagnosis of Pleurisy, p. 76.)

Encysted pleurisy with effusion, when occupying the anterolateral portion of the left chest, may give an area of flatness resembling closely that produced by pericardial effusion. During the course of encapsulated pleurisy the sounds of the heart are normal, and the apex-beat, if at all displaced, is pushed to the right. Again, the friction murmur is likely to be absent in encapsulated pleurisy, whereas the pericardial friction murmur (synchronous with the heart's action) is audible early and during the stage of resorption in pericardial effusion.

Clinical Course and Duration.—This will be found to vary considerably, owing to the individual peculiarities of the patient and the severity of the type of infection. In certain cases three distinct stages of pericarditis follow one another in rapid succession—*e. g.*, the dry or plastic stage, the stage of effusion or serofibrinous stage, and the stage of absorption. In another type of case the first stage may continue for one, two, or more

weeks, and the second and third stages be greatly prolonged. This last class of cases is often referred to as subacute or chronic pericarditis. The second stage of pericarditis may follow the initial stage within the course of a few days, and then the condition assumes a subacute or chronic form, absorption continuing for a period of several weeks. The effusion that collects in the pericardium following an attack of acute articular rheumatism often disappears within two or three weeks, absorption being quite rapid after the third stage is established.

One of the chief evidences that convalescence is established is the fall of temperature by lysis in favorable cases; as a consequence, with the absorption of the effusion the annoying clinical symptoms gradually subside. Thus, the appetite improves, and the renal, respiratory, and circulatory manifestations of the disease gradually approach the normal.

Complications.—(1) Acute pleurisy is rarely seen to complicate pericarditis, and when present, the likelihood of recovery is materially lessened. (2) Myocarditis is one of the most serious complications, and its onset is usually marked by attacks of syncope. (3) Acute endocarditis complicating disease of the pericardium renders the condition more serious and delays convalescence. If the effusion into the pericardium is large, it may exert pressure upon the esophagus, and in this way produce dysphagia. Pressure upon the recurrent laryngeal nerve is followed by paralysis of the vocal apparatus, as the result of which the voice is altered and husky, and there is a peculiar brassy cough. Empyema of the pericardium, while unusual, is a grave complication.

Purulent Pericarditis (Empyema of the Pericardium).

Pathologic Definition.—A condition characterized by an accumulation of pus within the pericardial sac. The membrane is appreciably thickened and presents a grayish, granular surface. Degenerative changes in the myocardium immediately beneath the serous covering are frequently seen.

Predisposing and Exciting Factors.—Empyema of the pericardium may follow serofibrinous pericarditis. The disease is occasionally encountered as a complication during the course of certain acute infections,—*e. g.*, pneumonia and scarlatina,—and, in our experience, pneumococci have been cultivated from the purulent pericardial fluid of persons dead of lobar pneumonia. Purulent pericarditis may follow infection of the pericardium with the tubercle bacillus, and in some cases of empyema other pyogenic organisms may figure as etiologic factors.

Clinical Picture.—The physical signs upon which emphasis was laid in connection with serofibrinous pericarditis (p. 241) are practically the same when the pericardium contains pus, although it is unusual to find the area of pericardial flatness of equal extent to that present in serofibrinous pericarditis. The temperature is usually high, and is often of the septic type.

Diagnosis.—The diagnosis is rendered positive by the recovery of purulent fluid from the pericardium by aspiration. The *x*-ray is of value. (See p. 230.)

Aspirating the Pericardium.

Different points of election and various methods for performing aspiration of the pericardium have been advocated from time to time, a few of which will be considered here.

By the Xiphocostal Route.—Although, as a rule aspiration of any one of the serous body cavities is condemned by many writers, it has been our custom, in private and in hospital practice, to perform aspiration on the pericardium, pleura, peritoneum, and spinal meninges. When, after a thorough physical examination, it has been determined that the pericardium contains an abnormal quantity of fluid, the next step is to outline accurately the lower border of dullness produced by the presence of such fluid. If it is found that the dullness extends well into the epigastrium, and that the diaphragm is appreciably depressed, the xiphocostal is the safest route by which to recover such pericardial exudate (Fig. 112).

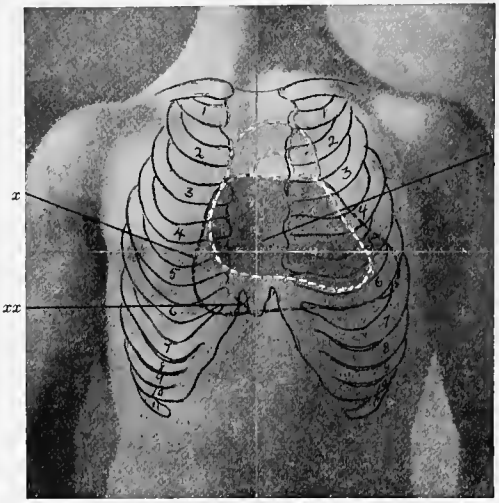


FIG. 112.—WHITE INDICATES NORMAL OUTLINE OF HEART AND AORTA.

x shows outline of pericardium when a large pericardial effusion is present; *xx*, costoxiphoid for aspiration of pericardium. Some prefer to insert the needle entering first at the costal margin of the left costoxiphoid angle.

The method of procedure is as follows: Place the patient in the erect posture, or possibly permit him to incline slightly forward, and direct the nurse or attendant to steady his shoulders firmly.

Next introduce the needle in the right xiphocostal angle, using local anesthesia and all necessary aseptic precautions. The operator should gage approximately the thickness of the body-wall, and guard the needle with his finger, so that he may not enter further than is necessary in order to reach the pericardium; when this is accomplished, the needle should be immediately withdrawn, and the handle of the trocar so elevated as to prevent the tip of the instrument from being directed toward the heart. Many prefer this route for the reason that there is but slight, if any, danger of wounding the heart, since as shown by the accompanying illustration (Fig. 112), during a large pericardial effusion, the body of the heart is elevated.

In entering the pericardium through the costoxiphoid angle it is well to have a knowledge of the attachments to the xiphoid cartilage. On the posterior surface, attachment is afforded to some of the fibers of the diaphragm and triangularis sterni muscles, hence by directing the needle slightly upward, the diaphragm may be avoided, since in a large pericardial effusion

the diaphragm itself is appreciably depressed. The aponeuroses of the abdominal muscles are attached to the lateral borders of the ensiform cartilage.

In removing fluid from the pericardium, the operator's hand should steady the instrument continuously, and whenever the heart is felt to come in contact with the tip of the instrument, the latter should be immediately with-

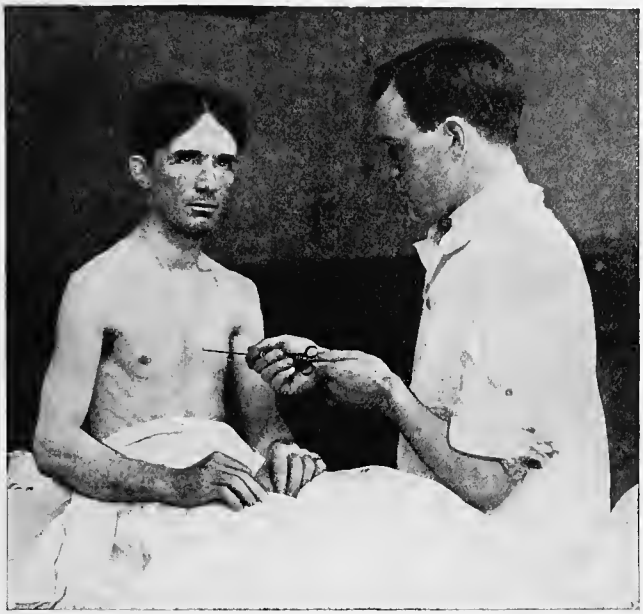


FIG. 113.—METHOD OF PERFORMING EXPLORATORY PUNCTURE OF THE PERICARDIUM, IN ORDER TO DETERMINE THE NATURE OF A PERICARDIAL EXUDATE (Eisendrath).

The patient can be thus explored either in a recumbent or upright position. The needle should be inserted in either the fourth or fifth interspace, close to the sternum, great care being taken not to insert it too deeply.

drawn, since the dangers of wounding a coronary artery or the heart muscle are extremely great.

Right Sternocostal Route.—If the pericardium is sufficiently distended to give flatness beyond the margin of the sternum, it is possible to reach the pericardium by inserting the needle in the fourth interspace at the right margin of the sternum. (See Topographic Anatomy of the Heart, p. 176.) This route is to be employed only when there is an unusually large effusion, but on account of the thin wall of the auricle, which normally rests near this situation, the danger of wounding the heart is greater than when the xiphocostal route is employed.

Eisendrath, in his "Surgical Diagnosis," calls special attention to the method of entering the pericardium through either the fourth or the fifth interspace, close to the left margin of the sternum (Fig. 113, the point of the needle being directed toward the median line. Despite the observance of all possible precautions, however, this route is less safe than those previously described.

Another method that is occasionally employed when the effusion is

large is to enter the chest just external to the outer margin of cardiac flatness, directing the needle obliquely downward and inward. The point of insertion is usually one to two inches external to the apex-beat, and near the level of the nipple. It has been our privilege to employ this method with perfect satisfaction where the effusions were large. It has no advantages over the xiphocostal route. Advocates of this method of puncturing the pericardium claim that, in a large effusion, the heart is elevated and the apex-beat is somewhat to the left of the nipple, consequently the danger of wounding the heart is extremely slight. We would, however, call special attention to the fact that the impulse, supposedly the apex-beat, may not be produced by the apex of the organ when the heart is displaced, and that for this reason there is no accurate way of determining the position of the heart in a large pericardial effusion.

Hemorrhagic Pericarditis.

Pathologic Definition and Remarks.—An accumulation of blood within the pericardium. As the result of local inflammation there may be an extravasation of blood into the pericardium during acute plastic and purulent pericarditis. The pericardium may also be the seat of inflammatory processes that have extended from the lung and adjacent viscera. Pathologic changes in the blood-vessels and in the chemic composition of the blood may also be present.

Etiologic Factors.—A pericardial effusion that is purulent in character may also display a bloody color. Tuberculosis of the pericardium is a common cause of hemorrhagic pericarditis, as is also chronic nephritis of the aged. Bloody fluid may accumulate in the pericardium as the result of infection with pathogenic bacteria, as has been demonstrated by the recovery of the pneumococcus from the pericardial fluid.

The **clinical picture** of purulent hemorrhagic pericarditis differs in no way from that given for empyema of the pericardium (see p. 245), and non-purulent hemorrhagic pericarditis displays both symptoms and signs quite analogous to those outlined under Serofibrinous Pericarditis (p. 240).

Adhesive Pericarditis (Chronic Pericarditis).

Pathologic Definition.—A condition characterized by the formation of dense pericardial and pleuropericardial adhesions. In some instances the opposed surfaces of the membrane are universally adherent, whereas in others the membranes are fairly adherent over a limited area. An appreciable thickening of the layers of the pericardium is observed, and such thickening will be found to vary greatly in different cases.

Predisposing and Exciting Factors.—Conditions known to predispose to other types of pericarditis are also concerned in the production of this form of the disease; special mention must be made, however, of tuberculosis of the pericardium.

Symptoms and Physical Signs.—These are in no way characteristic, and except that the heart is markedly displaced as the result of dense adhesive bands, this condition may pass unrecognized until the case comes to autopsy. In those cases in which an antemortem diagnosis was possible, the pulse was observed to be rapid, irregular, and of a low tension, whereas in our cases no pulse peculiarities were detected. There are dyspnea and the signs of cardiac enlargement at times sufficient to cause deformity of the chest. There is seen retraction of the chest overlying the heart. During ventricular systole there is often seen Broadbent's sign. The veins of

the neck may distend during inspiration, which veins also display a sudden collapse with the beginning of diastole. The pulsus paradoxus (see p. 198) may, however, be present.

Pericarditis Callosa.

General Remarks.—A type of chronic pericarditis developing during childhood, and characterized by prominence of the jugular veins, cyanosis, and moderate edema. In this type of pericarditis the entire circulation may become embarrassed, in which case there is effusion into the serous sacs.

Physical Signs.—**Inspection.**—The left side of the chest is usually seen to be somewhat retracted, and there may be unequal expansion of the two sides of the thorax. With each pulsation of the heart undue depression at certain of the intercostal spaces generally occurs, and such depression is, as a rule, synchronous with systole. In the region where the apex should normally be seen the entire chest-wall may be depressed with each systole, and in extreme cases the greater portion of the precordial space is thus affected by the heart's action. Respiration exerts some influence upon the degree of depression of the precordial area with systole.

Friedreich's sign may be observed, and consists in a sudden collapse of the jugulars during diastole. This sign is also seen in cardiac dilatation.

In those cases in which there is decided cardiac hypertrophy, the impulse of the heart is forcible, and the apex-beat is visible over an increased area of the chest. In many cases it is not until myocardial changes have taken place that the patient consults his physician, and there is often, at this time, a moderate amount of dilatation, with weakening of the impulse. Where there is adhesive pericarditis, change of position of the patient will cause the apex-beat to remain at one point—a valuable sign obtained by inspection.

Palpation confirms inspection as to the force of the apex-beat, and further detects any irregularity in the heart's action as the result of respiration. A diastolic shock, when present, is of great diagnostic value, and consists in the heart's forcible rebound during diastole. A diastolic shock is a prominent feature in adhesive pericarditis, while the heart's action is yet strong, but after dilatation has developed, it may be but feebly expressed.

Percussion.—The area of cardiac dullness is increased upward and to the left, owing to the presence of the following pathologic conditions: (*a*) Extensive pleuropericardial adhesions; (*b*) adhesions preventing the lung from overlapping the heart, as it does under normal conditions; consequently the upper border of the lung overlying the heart is retracted; (*c*) the area of cardiac dullness is not materially changed by change of position of the patient or by deep inspiration.

In those cases that have displayed myocardial and tricuspid regurgitation for an indefinite period the area of hepatic dullness will be found to be increased.

Auscultation.—A systolic murmur is frequently heard at the ensiform cartilage, and signifies that the right heart has become appreciably embarrassed, thus permitting of tricuspid regurgitation. Extensive pericardial adhesions may exist without evincing audible murmurs over any portion of the heart, whereas in other cases numerous murmurs, apparently endocardial in origin, are distinctly audible; yet it is with extreme difficulty that we are able to attach definite clinical significance to such murmurs.

Differential Diagnosis.—In those cases of chronic pericarditis in which there is also a moderate amount of effusion into the pericardium,

it may be necessary to distinguish between this condition and serofibrinous pericarditis. The clinical history, however, will usually serve to differentiate these two conditions, since serofibrinous pericarditis has, as a rule, been of short duration. In chronic pericarditis the apex-beat is forced upward, as is seen in pericardial effusion, but in the latter condition change of position of the patient will be found to alter the position of the apex-beat. Distinct bulging of the chest may result in adhesive pericarditis of the young, and here, again, it becomes necessary to differentiate this malady from a large pericardial effusion. The inverted triangular area of cardiac flatness, together with flatness extending well to the right of the sternum, will make the diagnosis of pericardial effusion positive.

HYDROPERICARDIUM (DROPSY OF THE PERICARDIUM).

Pathologic Definition.—A secondary condition characterized by distention of the pericardium by transuded fluid in the absence of inflammation of the pericardial surface.

The **symptoms** are usually those of the preëxisting malady, with a possible increase in the frequency of the pulse-rate and dyspnea.

The **physical signs** are practically those described for serofibrinous pericarditis (p. 241), except that the friction murmur is absent.

HEMOPERICARDIUM.

Remarks.—A rare condition in which pure blood escapes into the pericardium. The conditions that favor hemorrhage into the pericardial sac are: (*a*) Traumatism with rupture of the coronary artery; (*b*) rupture of the heart; (*c*) rupture of a thoracic aneurism; (*d*) stab wounds of the heart. If the condition results from the rupture of a thoracic aneurism, a large quantity of blood suddenly enters the pericardium and materially interferes with the heart's action. Following injury of the heart the blood may escape slowly into the pericardium.

The **physical signs** are difficult respiration, cyanosis, and the signs characteristic of serum in the pericardium. (See Pericardial Effusion.)

PNEUMOPERICARDIUM (AIR OR GAS IN THE PERICARDIUM; PYOPNEUMOPERICARDIUM).

Pathologic Definition.—A condition characterized by the accumulation of air (gas), pus, and frequently blood, in the pericardium.

Predisposing and Exciting Factors.—(1) Serofibrinous pericarditis in which the fluid becomes infected with gas-producing bacteria. (2) Stab and gun-shot wounds of the chest that have penetrated the pericardium. (3) Traumatism with fracture of the ribs and penetration of the pericardium. (4) The formation of a fistulous communication between a tuberculous cavity in the lung and the pericardium. (5) A fistulous communication between an empyema and the pericardium. (6) Subdiaphragmatic pneumopericardium, a condition in which gastric ulcer has perforated the diaphragm and communicated directly with the pericardial sac.

Principal Complaint.—This resembles closely what has been outlined under serofibrinous pericarditis, except that the condition in question usually develops somewhat abruptly, and the patient suffers more intensely from dyspnea than he would in the presence of a simple pericardial effusion.

Physical Signs.—**Palpation** may be negative, although the apex of the heart will usually be felt at some point over the precordium.

Percussion yields a tympanitic note over the greater portion of the

precordia, whenever the quantity of fluid present is large a variable degree of flatness is readily outlined. It is of great importance in the diagnosis that the position of the patient be changed; in this way the percussion-note will be materially modified—*e. g.*, dullness will be found to shift as the result of posture.

Upon **auscultation** the heart-sounds are usually intensified, and rasping friction murmurs, displaying a distinctly metallic quality, are audible. Besides pericardial murmurs, there is a loud, splashing sound with each impulse of the heart. In two cases in our practice the heart-sounds were feeble.

Differential Diagnosis.—Fluid and gas in the pericardium exhibit physical signs that closely resemble those resulting from fluid and gaseous substances in the pleura, the leading differential features of which are set forth in the accompanying table:

Pneumopericardium.	Pyopneumothorax (Left).
1. Patient has complained of a sense of discomfort in the pericardial region for several days, and possibly for weeks.	1. Sudden pain in the left side of chest.
2. Apex-beat displaced upward and to the left.	2. Apex-beat displaced to the right and may be as far as the right nipple.
3. Diaphragm but moderately depressed.	3. Diaphragm markedly depressed.
4. Heart-sounds clear, but confused by harsh, crackling splash.	4. Heart-sounds unaffected.
5. Vocal resonance unaltered.	5. Amphoric in quality and absent at base of chest over area occupied by fluid.
6. Small area of flatness at base of chest anteriorly.	6. The entire base of left chest is occupied by fluid that gives a flat note.
7. Vocal tactile fremitus normal.	7. Absent.
8. Bell tympany seldom present over the precordium.	8. Present over the entire left pleura.

Note.—In encysted pyopneumothorax (rare) the diagnosis may be difficult and even impossible.

Clinical Course.—The majority of cases run a rapid course, terminating fatally within from a few days to a week.

Diseases of the Endocardium.

ENDOCARDITIS.

Pathologic Definition.—A condition characterized by either an acute or a chronic inflammation of the lining membrane of the heart, which attacks most often the leaflets, but may involve any portion of the endocardium.

Varieties.—(1) Simple acute endocarditis; (2) ulcerative endocarditis; (3) chronic endocarditis.

Simple Acute Endocarditis.

General Remarks.—In this variety of endocardial inflammation there are slight vegetations upon the endocardial lining, these growths being most often situated near the base of the cardiac leaflets and on that surface opposed to the blood-current.

Exciting and Predisposing Factors.—Bacterial Infection.—The disease may result from infection of the endocardium with a variety of

pathogenic micro-organisms, and it may possibly be excited by the toxins of micro-organisms. Among the bacteria that have been isolated from the diseased endocardium are: The staphylococcus pyogenes aureus, which is conceded by some writers to be the chief agent in the production of this malady, the diplococcus of pneumonia, bacillus coli communis, streptococcus, gonococcus, bacillus of Eberth, the diphtheria bacillus, and the meningococcus.*

(1) "The most frequent cause of acute endocarditis is acute articular rheumatism, which induces the disease in not less than 40 per cent. of cases" (Anders). (2) Children and young adults suffering from articular rheumatism are more likely to develop endocardial disease than are older subjects. (3) It has been repeatedly shown that the severity of the attack of rheumatism has no influence on the likelihood of endocardial disease to develop as a complication. (4) Endocarditis may occasionally antedate articular rheumatism, although such instances are comparatively few in America. (5) Tonsillitis appears to be a predisposing factor in a certain percentage of all cases. (6) Children suffering from chorea may later develop acute endocarditis, and here the disease is likely to assume a chronic course. Chronic suppurative processes are not without influence in the production of simple endocarditis, and it is difficult to determine the effect of gonorrhea upon this type of the disease. (7) In the specific fevers simple endocarditis may develop as a complication, and although this is by no means common, it is encountered in diphtheria, measles, scarlatina, typhoid fever, small-pox, erysipelas, and, particularly, in pneumonia. (8) Simple endocarditis may also develop in those suffering from pulmonary tuberculosis, and from other maladies in which a large area of suppuration has existed. (9) Chronic disease of the kidneys and diabetes appear to predispose to the development of endocarditis. (10) Acute endocarditis may suddenly be attached to a chronic inflammatory process of the endocardium—the so-called recurrent endocarditis.

Principal Complaint.—The history usually shows that the patient has suffered from one or more attacks of rheumatism or other malady known to predispose to diseases of the endocardium. (See Predisposing and Exciting Factors, above.) The subjective symptoms of acute endocarditis are, as a rule, vague, and, indeed, may be absent. Precordial *pain* is an occasional complaint, and is sometimes described as extending to the left shoulder and down the left arm. *Dyspnea* is an early and annoying symptom, and is often the one for which the patient seeks relief. The heart *palpitates* violently upon the slightest exertion, and the patient may complain of throbbing at the temples and at the base of the brain.

Thermic Features.—In the majority of cases the temperature will be found to rise abruptly from 99° to 102° F., but the fever is often influenced by the preëxisting disease, so that the onset of endocarditis is in no way heralded by a special thermic phenomenon. In those cases in which one or more emboli are present, the symptoms of such involvement materially alter the general clinical picture.

Physical Signs.—The actual physical signs resulting from a simple endocarditis will be found to vary greatly, depending upon the valve that is involved and upon the extent of such involvement; for which reasons

* "Cerebrospinal Meningitis with Ulcerative Endocarditis and Abscess of Myocardium, Due to the Diplococcus Intracellularis of Weichselbaum," Med. Record, September 2 1899. (Boston.)

brief mention will be made of the types of murmurs produced by such lesion.

Inspection.—The area of visible cardiac impulse is increased, and in the majority of instances this increase is observed to extend downward and to the left. The impulse may be forcible and irregular as to time and strength.

Palpation.—In addition to confirming what has previously been detected by inspection, the force of the impulse will be found to vary greatly at different stages of the disease, and, indeed, there may be an appreciable difference in the volume of the apex-beat from day to day. As a rule, the force of the apical impulse lessens as the disease advances. After myocardial changes have developed, the apex-beat is feeble and may be almost imperceptible. When we are dealing with a recurrent endocarditis, a heaving impulse is to be expected, owing to a preëxisting hypertrophy of the heart the result of a previous endocardial disease. Rarely a systolic thrill is palpable over the area of the heart.

Percussion.—Early during the course of the disease the area of cardiac dullness is not altered, but as the disease advances enlargement in the transverse diameter is common, the area of cardiac dullness being appreciably increased to the left in well-marked cases—a condition that is believed to result from increased diastolic tension in the left ventricle. The right ventricle also meets with undue resistance and may, though rarely, be so seriously affected as to show an appreciable degree of dilatation. The more marked is the increase in cardiac dullness in simple endocarditis, the more extensive will be the area of cardiac impulse.

Auscultation.—Since the mitral leaflets are most often attacked, a soft, blowing murmur, systolic in time, is heard at the apex in the majority of cases. If the aortic leaflets are involved, the systolic murmur may be heard at the second right intercostal cartilage. Considering that the mitral valve is the site of the initial involvement, the area of maximum intensity of the systolic murmur, of which mention has just been made, is at the apex, or about one to one and one-half inches below the nipple and within the left midclavicular line. The apical systolic murmur is transmitted in the direction of the axilla to a variable degree. (See Mitral Regurgitation, Fig. 124.)

During the course of acute articular rheumatism, and when endocarditis is about to develop as a complication, a distinct prolongation of the first cardiac sound is heard if the stethoscope is placed near the apex of the organ. Careful examination will show that the second pulmonic sound is also accentuated at this time. In selected cases the first indication of cardiac involvement of the mitral valve is a muffled or "woolly" first sound, which, owing to its alteration, causes the second sound to be apparently intensified. The presence of a presystolic mitral murmur indicates that stenosis exists at the mitral ring, and may be detected early in certain cases of simple endocarditis.

It is possible that when a distinct systolic mitral murmur is audible, a questionable murmur may also be heard at the same time over the aortic area (second right costal cartilage). Early during the course of endocardial involvement one may detect an extremely soft, low-toned systolic murmur at the ensiform cartilage, and when present, this murmur has its origin at the tricuspid orifice, and suggests a probable relative incompetency. An acute endocarditis developing in an endocardium that has previously suffered one or more attacks of the disease may in no way alter the murmurs that were already present; consequently auscultation does not furnish a means for the recognition of an existing recurrent endocarditis.

Laboratory Diagnosis.—During the febrile period the urine is

slightly diminished in quantity, and its color is increased. Blood cultures are, as a rule, negative, since this form of the disease may develop without pathogenic bacteria circulating in the blood.

Summary of Diagnosis.—This is attained largely from a knowledge of one or more preëxisting conditions that tend to favor the development of simple endocarditis. Fever that has continued for several days, together with a moderate increase in the area of cardiac dullness, is strongly suggestive of this condition. Distinct cardiac murmurs, although always suggestive of a lesion of the endocardium, give us no information as to its extent and duration, and tend in themselves often to cause confusion regarding the nature of the endocardial disease in question. When the patient is seen sufficiently early, or, better, when the cardiac sounds have been analyzed from day to day during the course of an attack of rheumatism, considerable importance attaches itself to the presence of a slight prolongation or indistinctness (muffling) of the first sounds of the heart.

Differential Diagnosis.—The murmurs of acute endocarditis must be differentiated from **functional murmurs** heard during the course of acute fevers, and in those who are debilitated from any cause. In both conditions the murmurs are likely to be systolic in time. The murmur of endocarditis is heard most often at the apex of the heart, whereas functional heart murmurs are most clearly audible over the base of the organ, and frequently in the region of the pulmonary cartilage. Again, if a normal pulmonic second sound is present, and there is no appreciable increase in the area of cardiac dullness, the murmur in question is functional rather than organic.

Pericarditis.—The physical signs of acute simple endocarditis and those of pericarditis are widely different if either condition exists alone, but the fact that these two maladies may coexist in the same person and at the same time should always be borne in mind. If signs of endocarditis are present in a patient in whom both endocarditis and pericarditis exist, they will be obscured later by the development of a pericardial effusion.

Acute endocarditis may be distinguished from an old endocarditis by the fact that, in the former, but moderate cardiac hypertrophy exists. Endocardial lesions of long standing are likely to give rise to a distinct systolic murmur at the apex and a more marked accentuation of the pulmonic sound; at the same time there may be abnormalities as to the force and volume of the pulse, all of which features are less marked in the acute type of the disease. In well-marked cases of chronic endocarditis in which myocardial changes have taken place there is little or no difficulty in differentiating between these two conditions.

Clinical Course and Duration.—This is influenced largely by the character of the preëxisting disease and by the extent of involvement of the endocardium. Certain cases of acute simple endocarditis do not recover until the endocardium has been permanently damaged.

Complications.—Myocarditis may result from direct extension of the endocardial process. (See Myocarditis, p. 296.)

Ulcerative Endocarditis (Malignant Endocarditis; Infectious Endocarditis).

Pathologic Definition.—A disease characterized by ulceration of the endocardium, and possibly suppuration, or by both. Primarily, the leaflets are the seat of vegetations, such as are seen in simple acute endocarditis; these vegetations undergo necrotic changes and tend to increase in area, destroying a variable surface of the endocardium. Suppuration

may take place in the interior of the vegetations, and the resulting abscesses rupture, leaving an ulcerating surface. The vegetations, as a rule, become grayish or yellowish in color, and histologically they are composed of granulation tissue, fibrin, and micro-organisms. A distinct area of congestion may surround certain of the vegetations. Foci of suppuration generally develop in the viscera (brain, liver, kidney) as the result of particles of the sloughing ulcers and their bacteria being carried by the blood-current.

Varieties.—(*a*) Ulcerative endocarditis may, in rare instances, be a primary condition, but, as a rule, this particular type of endocarditis develops as a complication during the course of one of the acute infectious fevers—*e. g.*, pneumonia, sepsis, scarlet fever, etc.; (*b*) cerebral ulcerative endocarditis, characterized clinically by a predominance of nervous symptoms; (*c*) recurrent malignant endocarditis.

Predisposing and Exciting Factors.—(1) Ulcerative endocarditis with but few exceptions is probably a secondary condition, and develops in conjunction with the pathologic lesions of the endocardium characteristic of simple acute endocarditis; and, indeed, the simple type of the disease frequently precedes the ulcerative form.

(2) Endocarditis develops as a complication in about 10 per cent. of all cases of acute articular rheumatism.

(3) Ulcerative endocarditis is quite a frequent complication of lobar pneumonia, and occurs almost as often as the simple type of endocardial involvement.

(4) The endocardium may be attacked during the course of such acute infections as small-pox, erysipelas, scarlet fever, typhoid fever, epidemic meningitis, and tuberculosis.

(5) Ulcerative endocarditis sometimes develops during gonorrheal infection and in puerperal sepsis, and is less often encountered during the course of chronic nephritis.

Bacteriology.—The streptococcus pyogenes has frequently been isolated from the endocardial lesions, although the initial disease from which the patient was suffering may not have been excited by the streptococcus; the development of such malady, however, has furnished opportunity for the invasion of the endocardium by streptococci. Pyogenic staphylococci, bacillus coli communis, the diphtheria bacillus, and the anthrax bacillus have repeatedly been cultivated from the endocardium. The pneumococcus is present in a large percentage of cases complicating lobar pneumonia, and the gonococcus is a fairly common finding in those cases in which ulcerative endocarditis follows a virulent type of gonorrhea. One of us has isolated the bacillus of Friedländer from the endocardial ulcerations in a case dead of Friedländer's pneumonia, and the diplococcus of Weichselbaum has been found in endocarditis complicating epidemic meningitis.

Clinical Picture.—Ulcerative endocarditis frequently develops during the course of septic disease, and then the regular symptoms of the initial malady are intensified; it is important, in this connection, to bear in mind the fact that the endocarditis is secondary, and that its symptoms are, to a greater or lesser degree, masked by those known to accompany the primary disease. It is impossible to separate satisfactorily the symptoms resulting from the disease of the endocardium and those following general sepsis, which probably antedate the endocardial lesion, although it is understood that, after the endocardium has been attacked, sepsis may be disseminated, with consequent pollution of the blood. Ulcerative endocarditis may present a varied number of clinical pictures, many of which are in no

way distinctive; and, indeed, unless laboratory methods have been employed as a means of diagnosis, one often follows a case to autopsy before he is thoroughly convinced that endocarditis existed. We shall describe here the more common or typhoid form of ulcerative endocarditis.

The *onset* is somewhat abrupt, and is frequently heralded by a distinct *rigor* that may be repeated every twenty-four or forty-eight hours, or possibly every sixth to eighth day. The *pulse* and *fever* are also quite characteristic. (See Physical Signs and Thermic Features, pp. 256, 257.)

Contrary to what would ordinarily be expected in so virulent a disease as ulcerative endocarditis, local symptoms may be absent; when, however, the existing disease is one in which ulcerative endocarditis is a common complication, and when we are alert in watching for the development of endocarditis, the following symptoms may be recognized: Slight *oppression* in the region of the precordium, which may, at times, be described by the patient as a *faint pain*. *Extraprecordial distress* in the region of the different viscera, due to the irritation excited by emboli that occupy the special organs in question—*e. g.*, pain in the region of the spleen is quite common, and is ascribable to involvement of the peritoneum overlying the organ; pain in the region of the liver is probably due to a similar process involving the hepatic tissue and its capsule.

Multiple abscesses may occur either in the viscera or in practically any portion of the body as a result of septic emboli; these usually in turn excite not only pain, but the general symptoms and signs of a localized septic process. *Ecchymoses* and *multiple cutaneous hemorrhages* may also follow emboli of the skin.

Gastro-intestinal symptoms are, as a rule, well marked, and vomiting may develop early during the course of endocarditis; diarrhea is by no means an uncommon symptom.

Ocular Symptoms.—Dimness of vision and specks floating before the eyes may be due to retinal hemorrhage, septic emboli of the eye, or septic renal emboli that in turn excite acute nephritis.

Cutaneous Manifestations.—Profuse sweating develops as an early and annoying symptom, and it is not uncommon for the patient to experience one or more attacks of sweating daily. In well-marked cases it is frequently necessary to change the bed-linen after the patient has suffered from one of these drenching sweats, and in women the soaking of the hair by perspiration becomes a most annoying condition, it being often impossible to dry the hair between the attacks.

The patient rapidly emaciates, and will be found to lose several pounds a week while the disease continues.

Nervous Symptoms.—At first the patient may be unduly nervous and hypersensitive; the most annoying early symptom, however, is headache. As the disease advances mild delirium develops at night, and may progress until it becomes maniacal; later the patient becomes somnolent and, finally, comatose.

Thermic Features.—The fever is of the continued type, although it may be decidedly intermittent in some cases, whereas in others the remissions are but slightly manifest. The temperature may reach 105° or even 106° F. at various times during the day, while in other cases it continues quite steadily at from 102° to 103° F., seldom falling to the 100° mark. In one case coming under our care the temperature was of the continued type for from three to seven weeks, and in another a continued fever of 103° to 104° F. was displayed for forty-two days.

Physical Signs.—These may, in selected cases, be negative as regards the heart. In a case recently observed this characteristic was exemplified, since at no time during the attack was any definite information obtained by auscultation, yet laboratory methods showed that the patient was suffering from malignant endocarditis, a fact that was proved at autopsy.

Inspection.—Early during the attack the face may be flushed; the skin is bathed with perspiration, and respiration is rapid. As the disease advances emaciation becomes apparent; the lips become fissured; the tongue is heavily coated, the eyes are sunken, and the impulse of the heart is unusually conspicuous. The skin may show ecchymotic areas and even petechiæ. Small cutaneous abscesses are occasionally seen on various parts of the body.

Palpation.—At the onset the pulse is usually rapid—120 beats a minute—and irregular. As the disease advances it may, in certain cases, become more and more rapid, weak, irregular, dicrotic, and readily compressible, whereas in other cases the number of pulse-beats a minute will be found to diminish with the advance of the disease. The fact that an associated nephritis, with the consequent production of uremia, would tend to slow the pulse should be borne in mind in this connection. In those cases in which the pulse tends to become less frequent and the arterial tension to become increased during the course of ulcerative endocarditis, renal complications should be suspected.

Localized areas of tenderness will be found over septic emboli of the skin or of certain of the viscera—*e. g.*, splenic tenderness is not uncommon and, indeed, the spleen is, as a rule, enlarged and readily palpable. The liver is also felt to extend for some distance (two or more finger-breadths) below the costal margin, and may or may not be sensitive to firm pressure. Involvement of the bases of the lungs is generally followed by local tenderness when firm pressure is made upon the chest.

Percussion gives no definite information with reference to the heart. The areas of splenic and hepatic dullness are increased, and there may be areas of consolidation at the base of one or of both lungs as the result of a septic pulmonic process.

Auscultation.—In the majority of cases a systolic murmur is present, but this is of special value in diagnosis only when other symptoms and signs of malignant endocarditis are present. The disease may continue throughout its entire course without a distinct cardiac murmur being audible over any portion of the precordium. Auscultation determines the force and quality of the heart-sound, as well as the rapidity and regularity of its action. Late during the course of the disease, and when the patient shows much emaciation and prostration, the heart-sounds are not only weak and rapid, but the first sound is lacking in muscular quality. In these cases of recurrent malignant endocarditis there is distinct accentuation of the second sound, and a moderate degree of accentuation is heard during the initial stage of this malady. A distinct murmur may be audible over one of the larger arteries. The breath-sounds are usually increased over the base of the left lung, and in the event of an associated pneumonia or pulmonary embolism, the physical signs of lobar pneumonia may be present. The breath-sounds are increased in frequency in proportion to the degree of weakness of the patient, and as the heart becomes weak, numerous moist râles are audible over both lungs.

Laboratory Diagnosis.—Cultures made from the venous blood (see p. 255) will, in the majority of cases, be found to develop colonies of bacteria, and the reasons for failure to do this have been given (p. 255).

If the disease continues for a long period, well-marked secondary anemia develops, in which case both the red cells and hemoglobin are markedly decreased. Early during the course of malignant endocarditis the number of leukocytes in a cubic millimeter may be decidedly increased, but after a profound septic process has existed for some time the leukocyte count is of less clinical value.

Smears of blood, when fixed and stained, will be found to display marked degeneration of the red cells (poikilocytosis, alteration in the size of the cells, and the presence of numerous cracks, fissures, and inequality of staining with eosin). Microcytes and macrocytes are common, and nucleated red cells may be an occasional finding. A differential leukocyte count may show the polynuclear elements to be increased, a feature that is most constant early during the course of the disease.

Urine.—During the period when the fever is continuous the urine is high colored and contains albumin, but if septic nephritis develops, the urine is scanty, and anuria may obtain for several hours, or even until death. If the quantity of urine excreted is small during an acute septic nephritis, it will be found to be of high specific gravity, rich in albumin, and, in the majority of instances, to contain numerous bacteria, streptococci, and staphylococci. Cultures from such urine are, as a rule, positive. The colloidal albumin coefficient is increased. (See Gastric Cancer, p. 501.)

Microscopically, renal casts (granular, blood, and pus) may be present. Leukocytes and pus-cells are, as a rule, plentiful, and red corpuscles are a not uncommon finding. Pus obtained from the local abscesses of the skin will be found, both by direct staining and by cultural methods, to contain pathogenic bacteria.

The *feces* are at first liquid in character, but if septic diarrhea follows, they may contain an unusual quantity of serum; a microscopic study of such serum shows it to be rich in bacteria, and particularly in cocci. Cultural studies of the feces may be employed with positive results, but it is not recommended as a practical procedure, since it is possible to obtain positive data from other more available and more satisfactory sources.

Sputum.—If pulmonary infarction or pulmonary emboli develop, the sputum becomes bloody, and, indeed, quite characteristic: the patient expectorates with but little difficulty, and always in mouthfuls, so that a large, globular, bloody mass (size of a silver quarter or a half dollar) will float upon the surface of water.

Illustrative Case of Acute Ulcerative Endocarditis.—David C., male, aged forty-five; height, 5 feet 10½ inches; usual weight, 162 pounds; present weight, 155 pounds.

Family History.—Father died of pneumonia at the age of sixty-five; mother living at eighty. An older brother died of typhoid fever; a sister and two older brothers believed to be living and were in good health five years ago. There is no history of malignancy, diabetes, insanity, or tuberculosis in the family.

Previous History.—The patient believes he had the diseases of childhood, and recalls having had a severe attack of sore throat at the age of sixteen. At about thirty he developed typhoid fever, from which he made a perfect recovery. Four years ago he had an attack of acute articular rheumatism involving the left knee and right wrist, but he states that he was not confined to bed during this attack, although it incapacitated him for work for some weeks. Nine months ago he suffered from what his physician regarded as an attack of influenza. At this time he was confined to his bed for six weeks, and has never since felt perfectly well.

Social History.—Married, two daughters, aged sixteen and eighteen respectively, appear to be in good health. Patient is a teamster by occupation, and has not been compelled to lose a day's work. His occupation subjects him to exposure to cold and wet, although he claims that he was always well clothed and protected from cold and storms.

Present Illness.—Three weeks before coming to the hospital he developed an acute cold that terminated in a somewhat violent form of tonsillitis, although abscess forma-

tion did not occur. He states that swallowing was painful for several days, and that he felt feverish, had extreme aching in the back, muscles, arms, and lower extremities, was greatly prostrated, and had a distaste for all foods. Constipation was present, and the tongue was heavily coated. When admitted to the hospital he was extremely prostrated, being unable even to sit in bed. The most careful questioning failed to extract any definite information with reference to his disease other than that previously given.

There was slight cough, which was not accompanied by expectoration, but at no time was it especially annoying.

The patient grew rapidly worse, and in contrast to the condition that existed at the time of his admission to the hospital, he developed maniacal delirium during the night, and low muttering delirium was present during the day, with some picking at the bedclothes. Subsultus tendinum was present for a period of seven days, and there was a variable degree of delirium for three weeks following his admission to the hospital. The temperature was 102° F., and of a continuous type, with slight morning remissions, fluctuating, as it did, between 102° and 103° F. for a period of three weeks, when the remissions became more conspicuous; the temperature, however, did not become normal and remain at that point during his stay of six weeks in the hospital.

Physical Examination.—*General.*—The expression was anxious, the features pinched, and the skin wrinkled as the result of emaciation. The patient was almost continuously bathed in perspiration, and showed little or no tendency to move about the bed until delirium developed. (See Nervous Phenomena, p. 256.)

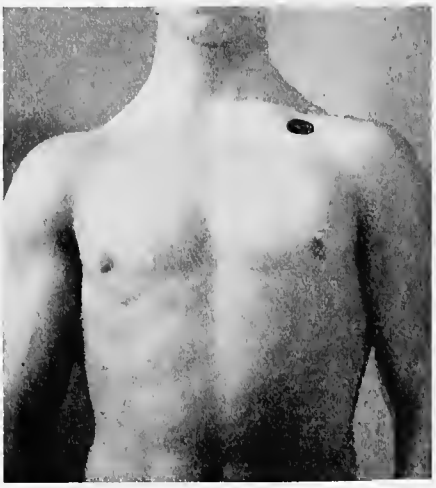


Fig. 114.—Black Spot Indicates where Thrill was Felt One Year Following an Attack of Ulcerative Endocarditis (Personal Observation at Philadelphia General Hospital).

Local Examination.—*Inspection.*—The cheeks were flushed at certain times of the day; the tongue was heavily furred, and after approximately two weeks of fever, it became deeply fissured, the base being brown in color. The lips became fissured and bleeding, and were covered with heavy, crust-like scabs. The impulse of the heart was unusually conspicuous, and there was some pulsation of the vessels of the neck.

Palpation.—The pulse-beats at first numbered 110 a minute, and were of good volume; as the disease advanced the pulse became more rapid,—from 130 to 160 beats a minute,—was weak, showed a tendency toward dicrotism, and was readily compressible; occasionally it was found to be intermittent. Early during his stay in the hospital the impulse of the apex-beat was forcible, but later it became feeble, and could scarcely be felt when the patient rested upon his back. The bony skeleton became unusually prominent, a condition that gave further evidence of rapid and progressive emaciation.

Auscultation.—The heart sounds were rapid and weak, and at the end of the third week the muscular quality of the first sound was wanting. Two weeks after entering the hospital a distinct diastolic murmur was present at the aortic cartilage, but this murmur was not at any time unusually harsh nor loud, and its area of transmission extended only for about 1½ inches down along the right border of the sternum. No murmur was audible over the mitral area, although there was an appreciable roughening of the first cardiac sound.

Laboratory Findings.—The so-called "febrile" urine was present at those times when the fever was high, containing as it did a trace of albumin, but renal casts were never detected. Upon admission the number of leukocytes was 26,800 per c.mm., and the red cells numbered 3,680,000 per c.mm. After five weeks' stay in the hospital the red cells numbered 2,100,000, and the color index was 0.54. A differential count of the leukocytes showed that 84 to 88 per cent. of them were of the polynuclear variety. A cultural study of the blood was made upon four occasions by obtaining blood directly from the veins of the arm, but all such studies gave negative results. Widal reaction negative.

Diagnosis by Induction from Clinical Data.—The history of the condition beginning as tonsillitis was not ignored, and the additional evidence that this attack was followed by a period when the patient displayed a continued type of temperature. The rapidity of the pulse and the high grade of prostration were considered as positive evidences of the gravity of his malady. In the absence of definite physical signs with reference to the heart the diagnosis still remained obscure, until laboratory methods were resorted to; the examination of the blood revealed a well-marked leukocytosis, and further showed that an abnormally large proportion of such leukocytes were of the polynuclear variety, a fact that strongly favored the existence of a septic process.

Differential Diagnosis.—The temperature, nervous symptoms, and prostration suggested the possibility of typhoid infection. His malady was differentiated from typhoid fever, however, by the following: (*a*) The absence of the Widal reaction; (*b*) the absence of well-marked abdominal signs; and (*c*) the freedom from diarrhea.

Course of the Disease.—For the first six weeks of his stay in the hospital the clinical course was practically typical of that described for acute ulcerative endocarditis. (See p. 255.) Emaciation progressed rapidly after his admission, and continued even for weeks after the temperature had reached the normal.

One year later we found that this patient had not recovered sufficiently to leave the hospital, although he is able to walk about the ward. He now displays the general signs and many of the symptoms of well-marked aortic regurgitation (see p. 267), and there is also quite a loud, though soft, systolic murmur heard at the apex; this murmur is sufficiently loud to be heard distinctly as far to the left as the midaxillary line. Since the patient left his bed he has gained thirty pounds, but is yet far below his normal weight.

Summary of Diagnosis.—It is important to consider the history and all the individual circumstances connected with the case in question, and this should be done particularly with reference to preëxisting disease. The symptoms of ulcerative endocarditis may be confounded with those of other *septic conditions, miliary tuberculosis,* and *typhoid fever* (see Differential Diagnosis, below), and these are best differentiated by laboratory methods. Considerable stress should be laid upon the early symptoms of this condition, and particularly upon the severe rigor, the character of the temperature, the presence of profuse sweating, and the occurrence of abscess as the result of septic emboli. Cultural studies of the blood serve as a positive means of diagnosis in the majority of instances. (See Cerebral Type, p. 262.)

With reference to the *recurrent endocarditis,* acute ulcerative endocarditis should be suspected, at least, whenever questionable symptoms arise in a patient who has previously suffered one or more attacks of endocarditis, and who has been known to have displayed an endocardial lesion prior to the present attack.

Differential Diagnosis.—The preceding remarks in reference to the clinical picture of acute ulcerative endocarditis will show conclusively that this disease must be distinguished from practically any condition that is characterized by continued fever. In our experience we frequently have to differentiate between *acute ulcerative endocarditis, typhoid fever,* and *miliary tuberculosis.* The subjoined table sets forth the distinctive differential features of these three conditions:

Ulcerative Endocarditis.	Typhoid Fever.	Acute General Miliary Tuberculosis.
1. Patient is at present suffering from, or has probably previously had, acute articular rheumatism, gonorrhea, puerperal sepsis, chorea, or simple endocarditis.	1. Previous history negative, as a rule. History of an epidemic or of association with other members of the same family who have recently suffered from typhoid.	1. Family history of tuberculosis, probably presence of an old tuberculous focus or of association with persons ill of tuberculosis.

Ulcerative Endocarditis.	Typhoid Fever.	Acute General Miliary Tuberculosis.
2. Disease not a primary condition.	2. Primary, with characteristic prodromes—headache, malaise, etc.	2. Develops more rapidly than typhoid.
3. Ushered in with a severe chill or a series of chills.	3. May be a recurrence of chilly sensations, but the disease develops rather insidiously.	3. Severe rigor unusual.
4. Fever rises abruptly following the chill, and is, as a rule, decidedly irregular.	4. Rises gradually, mounting higher day by day, until the tenth to the twelfth days.	4. Temperature rises rapidly and remains high, though decidedly irregular.
5. Unusual.	5. Epistaxis common during first week.	5. Absent.
6. Symptoms of emboli (hemiplegia, cutaneous abscesses, etc.) may be present.	6. Thrombosis of the femoral and spermatic veins may develop late during the disease.	6. Absent.
7. Respirations moderately increased in frequency.	7. Respirations but slightly increased in uncomplicated cases.	7. Respirations rapid—30 to 60 a minute.
8. The face is flushed early, but may later become cyanosed.	8. Cyanosis only when there are associated cardiac or pulmonary complications.	8. The face is dusky.
9. Diarrhea may develop at any time during the attack.	9. Diarrhea develops during the first week, and is characterized by pea-soup-like stools.	9. Constipation the rule. Feces show tubercle bacilli.
10. Absent.	10. Hemorrhage from the bowel quite common.	10. Extremely rare.
11. Widal reaction absent, leukocytosis the rule.	11. Widal reaction positive; leukopenia.	11. Absent.
12. Cultures made from the blood usually develop colonies of microörganisms other than the typhoid bacillus.	12. Colonies of typhoid bacilli are the only evidence of bacteriemia.	12. Tubercle bacilli may be present.
13. Sputum may be increased.	13. An associated bronchitis may give increase in sputum.	13. If there is chronic pulmonary tuberculosis, tubercle bacilli are present.
14. Ophthalmologic examination may rarely detect emboli.	14. Negative.	14. Miliary tubercles of the retina are somewhat common.

Recurrent Malignant Endocarditis.

This is a pathologic condition in which acute endocarditis develops during the course of chronic valvular heart disease. Recurrent attacks of simple acute endocarditis, to which reference has previously been made, are fairly common, although such recurrences may be so mild in character as to escape notice. Any type of endocarditis that predisposes markedly to infection of the endocardium with streptococci and other pathogenic bacteria favors the development of acute ulceration of the endocardium. The *onset* is abrupt, with a moderate *chill*, or possibly a distinct rigor. The *temperature* rises suddenly to 103° or 104° F., and within the course of one or two days it may become decidedly intermittent, although a continued type of fever is possible.

The **general symptomatology** of this form of endocarditis resem-

bles closely that described at length under acute ulcerative endocarditis, except that in the latter, in many instances, the recurrence of chills and the sweating are more pronounced than in the form now under consideration.

The engrafting of an acute ulcerative process upon a chronic endocardial lesion may, in selected cases, render the endocardial murmurs that have existed for an indefinite period more intense, but it is to be borne in mind that postmortem examination frequently discloses extensive acute ulceration of the endocardium where the physical signs of chronic endocarditis were not appreciably altered during the acute attack. The increase in the frequency of the pulse is quite a constant feature, and, indeed, acute endocardial ulceration may develop at a time when there is loss of compensation, in which case it simply aggravates the symptoms already present.

Recurrent malignant endocarditis may run a subacute course; the temperature may not exceed 100° to 102° F., and the general clinical picture is marked by mild symptoms.

Cerebral Type of Malignant Endocarditis.

In this form of endocarditis the clinical picture is that of acute purulent meningitis (see p. 1124), and on account of the predominance of meningeal symptoms, there is practically nothing to direct the attention to disease of the endocardium. A murmur may be present, although this is not a constant physical sign. In the so-called *cerebral type* of ulcerative endocarditis the general clinical picture also resembles closely that of *epidemic meningitis*, and the only clue to the diagnosis is obtained by lumbar puncture and by making cultural and other studies of the blood.

Clinical Course.—In recurrent malignant endocarditis the disease runs a rapid course, the patient's condition progressing from bad to worse for a period of from three to six weeks. This type of endocarditis is an exceedingly grave disease, and despite the early recognition of the disease and the application of treatment, recovery is doubtful. The cerebral type of endocarditis continues from a few days to possibly two or three weeks, terminating in coma. Recurrent malignant endocarditis may assume a subacute or chronic course, and in this variety of the disease the patient may suffer repeated attacks, lasting over a period of months or even years.

Chronic Endocarditis.

Pathologic Definition.—A disease characterized by a chronic inflammatory process of the endocardium, the most characteristic lesions consisting of infiltration and exudation, followed by cohesion of the segments with roughening of their surfaces, and a tendency to perforate the endocardium, with the development of slow infiltration, the formation of fibrous tissue, and consequent retraction of the leaflets.

Varieties.—(*a*) Chronic endocarditis developing as the result of an acute inflammatory process of the endocardium; (*b*) a second variety, in which the endocardial changes are sclerotic in nature from the time of their recognition, and progress steadily from bad to worse for a period of several years.

Predisposing and Exciting Factors.—(1) Chief among the conditions that predispose to the development of chronic endocarditis should be placed **acute rheumatic endocarditis**, a condition that is far more common in children than it is in the adult. (2) Endocarditis may be the only clinical expression of a rheumatic diathesis, and in selected cases of chronic endocarditis the endocardial condition appears to be extremely mild from

the onset. (3) "Not less than one-half of all cases of organic valvular disease are caused by rheumatism, and more than one-half occur between twenty and thirty years of age" (Anders). (4) Chronic endocarditis may frequently follow an acute attack that develops during the course of pneumonia, measles, chorea, or tonsillitis.

The **second variety** of chronic endocarditis, in which there are likely to be decided interstitial changes, is oftenest seen to follow: (1) Certain questionable **biologic irritants.** (2) Protracted malarial fever, chronic rheumatism, and neglected syphilis. (3) Persons suffering from the so-called *uric-acid diathesis* are especially prone to develop chronic endocardial changes, as are also those addicted to the *excessive use of alcoholic beverages; lead workers* likewise suffer from endocardial changes, as has been demonstrated by an examination of nearly 200 men employed for two or more years in the lead factories of Philadelphia, among whom over 80 per cent. gave evidence of chronic endocarditis and of hardening of the arteries. (4) *Undue muscular strain* must be regarded as a potent factor in the production of chronic endocardial disease; consequently those following certain occupations that necessitate heavy lifting, long-distance running, rapid marching, and athletic work are especially likely to develop the condition. (5) *Arteriosclerosis,* which is separated from endocarditis only with difficulty, is a decided predisposing factor, and is best exemplified by the condition of the heart in those cases of chronic Bright's disease and of lead workers in whom there are present extensive degenerative changes in the peripheral arteries. (6) *Increased arterial tension,* whether due to pathologic changes of the liver, lung, kidneys, or arterial system, should always be considered a potent factor in the production of chronic endocardial disease.

Among the other conditions that predispose to chronic endocarditis should be considered:

(*a*) **Heredity.** In this connection it may be stated that the parents of those suffering from chronic endocarditis may also have suffered from acute articular rheumatism, and the child has inherited a rheumatic tendency rather than a predisposition to simple endocardial disease.

(*b*) **Congenital deformity** of the cardiac leaflets, although it bears a close relation to heredity, must be considered as a predisposing factor of chronic endocardial disease.

(*c*) **Age** is not without influence, since during childhood and in young adults infectious diseases, including rheumatism, are frequent, and the mitral valves are most often attacked. After middle life and during old age the aortic valves are those most likely to be affected, although it is by no means uncommon to find aortic disease during early adult life, and, indeed, it is encountered during childhood by nearly every clinician.

(*d*) **Sex** exerts but moderate influence as a predisposing factor in this type of endocarditis. Chorea and acute rheumatism are found more commonly in females than in males, hence females are especially predisposed to chronic endocarditis; this predisposition, however, is probably to some degree overbalanced by the character of work (physical strain) to which males are subjected.

Illustrative Case of Chronic Endocarditis.—L. H., female, aged eleven; weight, 73 pounds.

Family History.—Father living at the age of thirty-three, mother at the age of twenty-nine, both in good health. One younger sister is also healthy. No history of heart disease, gout, or rheumatism in grandparents.

Previous History.—The patient had the diseases of childhood, including scarlet

fever at the age of seven years. At eight years of age she suffered from an attack of typhoid fever. When nine years of age she developed chorea, and the mother stated that the child's nervous condition did not completely subside for nearly one year, during which time several physicians were consulted, all of whom pronounced the case an unusually severe one.

Social History.—Since the attack of chorea she attended school until two months ago, when her physician advised that she discontinue school.

Present Illness.—This probably dates from the attack of chorea, for during that illness she frequently complained of shortness of breath, and now, one year after her well-marked nervous symptoms have subsided, she complains of the following: Shortness of breath upon slight exertion; cardiac palpitation, which may come on during sleep, and which is common after ascending a flight of stairs; a peculiar throbbing sensation in the head; ringing of the ears and lassitude. Walking or any form of exercise brings on violent coughing, which continues until the patient has rested for a few minutes. There is moderate constipation, and the appetite is somewhat disturbed, although she is able to eat one fairly heavy meal during the day. If an unusual amount of exercise is indulged in during the day, she may be annoyed by nausea and possibly by vomiting, and the following day she is unable to take food because of extreme nausea, which becomes more annoying when in the erect posture and when walking.

Headache is frequently present, although never severe. There is at times a sense of weight or of oppression over the precordium, but distinct pain in this region is not experienced. Cough, as previously mentioned, follows exertion, and after an unusual amount of exercise there may be considerable cough for two or more days. Violent paroxysmal cough has also occasionally been known to follow undue mental excitement.

The child is highly irritable, becomes startled upon hearing sounds, and at times displays more or less typical hysteric outbreaks.

Physical Examination.—*General.*—She appears fairly well nourished, although the skin and mucous membrane of the lips are pale. The general attitude is that suggestive of neurasthenia, and she persists in talking about her malady, always expressing considerable fear with reference to the outcome. Following exertion the finger-tips become somewhat cyanosed, and the hands and feet are cold. The muscles are fairly well nourished and firm, and the reflexes are normal. There is no evidence of choreiform movements.

Local Examination.—*Inspection.*—The impulse of the heart is forcible, and is seen approximately two inches below and just outside the left nipple. Upon exertion there is distinct pulsation over the greater part of the precordia, together with pulsation of the vessels of the right side of the neck. Following exertion epigastric pulsation is also conspicuous. When the patient is directed to sit with her arms partially elevated (see Fig. 115), there is visible pulsation of the axillary, brachial, and radial arteries. The femoral artery and the arteries in the superior surface of the feet are also seen to pulsate violently. Mucous membrane of the lips pale. Tongue coated.

Palpation confirms inspection with reference to cardiac pulsations. There is a peculiar impression offered to the palpating finger by the large arteries. Upon lifting the hand above the level of the body, the typical Corrigan pulse is detected at the radial artery. Following pressure upon the skin there is a peculiar flushing and paling of the forehead, back, and front of the chest. By compressing the nail of the finger gently, the red line (blood line) is seen to advance and recede with each impulse of the heart. (See Fig. 65.) By placing an ordinary glass slide, such as is used for microscopic work, upon the lip (see Fig. 64), a distinct pulsation of the vessels of the mucous surface is apparent.

Percussion.—The area of cardiac dullness is much increased, that of absolute dullness being about twice the size of the normal, whereas relative cardiac dullness extends to 2½ inches below and 2 inches to the left of the left nipple. The line of dullness extends from this point obliquely to the tip of the ensiform cartilage, and thence to a distance of approximately one inch to the right of the right border of the sternum. The superior boundary of the cardiac dullness is at or near to its normal level. The heart, therefore, is markedly enlarged. The area of liver dullness extends about 1¼ inches below the right costal border.

By *auscultatory percussion* the size of the spleen is found to be greater than normal for a child of her age.

Auscultation.—When ausculting at the apex there is some alteration in the first sound, but no distinct murmur is discernible. A pronounced diastolic murmur, moderately harsh in quality, is audible at the aortic cartilage, and this murmur is transmitted for a distance of 2½ inches obliquely downward and across the sternum. The murmur is not appreciably altered by exercise, and is sufficiently loud to be distinctly heard when the ear is placed directly on the chest-wall. Numerous râles are to be heard over the

bases of both lungs after undue exertion, being more conspicuous at such times as the patient suffers from paroxysmal attacks of coughing.

Laboratory Findings.—The hemic changes are those of secondary anemia, when the blood is drawn from the ear; but blood taken from the finger-tip after the hand has been allowed to hang below the level of the body shows the number of red cells to be above the normal (false polycythemia or polycythemia of cyanosis).

Course of the Disease.—Following rest and the administration of mild doses of cardiac stimulants, together with general tonics, the patient's condition improved until she was able to go about her usual duties without inconvenience. At the age of puberty the cardiac condition appeared to be somewhat aggravated for a few months, but after menstruation had been regularly established all the cardiac symptoms seemed to improve, although the physical signs were in no way changed. From the fourteenth to the sixteenth years hysteric outbreaks were somewhat more common than before puberty. At present, at nineteen years of age, the nervous condition, and also the general health, has greatly improved, yet at no time since her attack of chorea has she enjoyed perfect health.

VALVULAR DISEASE.

AORTIC REGURGITATION (AORTIC INCOMPETENCY; AORTIC INSUFFICIENCY).

Pathologic Definition.—A disease characterized by alterations (sclerosis and deformity) in the leaflets of the aortic valve, which prevent them from closing tightly after each systole, and permit a return flow of blood from the aorta into the left ventricle. Later microscopic changes with fatty degeneration of the heart muscle develop. Atheroma of the arteries, and especially of the coronary arteries, is also common.

Mechanic Influence of the Lesion.—The reflux current passes from the aorta backward through the imperfectly closed aortic valve into the left ventricle during the diastole of the heart, or while the left ventricle is being filled from the normal blood-flow from the auricle. Overdistention of the left ventricle results from two simultaneous influx currents of blood. To expel this increased cardiac power is required, and this overexertion causes a compensatory *hypertrophy*. Dilatation and hypertrophy of the left ventricle, therefore, develop *pari passu* until the left ventricle reaches enormous dimensions, a condition known as *cor bovinum* (Fig. 117).

Under existing pathologic conditions the arterial system is overcharged at each ventricular systole. Early in the disease the reflux of blood from the aorta into the left ventricle lessens the volume of blood distributed through the arterial tree, but this loss is counterbalanced by the large volume of blood expelled from the left ventricle with each systole; consequently the tissues are amply supplied with blood early during aortic regurgitation.

To meet the requirements an abnormal volume of blood is forced into the aorta with each systole, so that arterial tension is increased; this predisposes to arteriosclerosis, affecting more particularly the aorta at the point where the coronary arteries are given off, and, as a consequence, interference with the nutrition of the cardiac muscle follows. Fatty and fibroid changes in the cardiac muscle are attended by secondary dilatation, which in turn overcomes the original hypertrophy.

In consequence of the increased tension to which the mitral leaflets are constantly subjected, they may become the seat of sclerotic endocarditis, and later a variable grade of mitral incompetency may develop. Secondary dilatation, however, is the principal cause of insufficiency at the mitral orifice, and the blood-current through the mitral ring, before mitral regurgitation has developed, may be obstructed by the simultaneous influx into the left ventricle from the aorta, thus causing pulmonary congestion. Irrespective of the cause of mitral regurgitation or obstruction, the blood is

dammed back through the left auricle into the pulmonary tissue, producing obstruction to the current of blood coming to the lungs, and thereby increasing the work of the right ventricle. Here, again, the mechanism of the right side of the heart is quite like that previously outlined, hence tricuspid regurgitation eventually follows, with venous stasis, first, of the liver, and later, of the other abdominal structures and lower extremities (modified from Anders).

Special Exciting and Predisposing Factors.—**Incomplete recovery from acute endocarditis** is doubtless the most potent factor in the production of the chronic type of the disease, and in old subjects rheumatic endocarditis is to be considered.

Disease of the aortic leaflets may develop during the course of **chronic infectious processes**, as is exemplified in syphilis, yet it must be remembered that those suffering from luetic infection are not infrequently exposed to violent exercise, heavy lifting, and the like.

Certain **chemic irritants** appear to exert a selective action upon the endocardium, and consequently endocardial and particularly aortic disease is often encountered in gouty subjects, such patients also generally displaying general endarteritis. Rheumatic subjects, in whom the disease is due probably to a pathologic process, are also prone to develop endocardial (aortic) disease.

It is common to find degenerative changes in the endocardium among **lead workers** and those exposed to the inhalation of poisonous substances, although the condition is, as a rule, associated with pathologic changes in the kidney and more or less extensive atheroma throughout the general arterial tree. Those who imbibe too freely of **alcoholic stimulants** frequently develop chronic valvular disease.

When **endarteritis** involves the aorta, it may, and probably does in selected cases, extend to the aortic leaflets.

Increased Arterial Tension.—As previously stated, increased arterial tension, particularly when it is the result of heavy manual labor, stimulants, and narcotics, tends to increase the liability to the development of chronic endocarditis at the aortic ring, and it may be possible that increased arterial tension from whatever cause favors the development of chronic endocarditis. In this connection special attention is called to the fact that it is the more or less constant increase of tension that tends to produce disease of the endocardium.

Aneurism (a pathologic condition that is secondary to arteriosclerosis) is a potent factor in the production of aortic disease, since in this condition the increased work upon the part of the left ventricle is constant, even while the patient is at rest.

Age and **sex** are marked predisposing factors in chronic disease of the endocardium at the aortic orifice. Aortic disease is far more common in males than in females, a fact that possibly depends upon the variety of exercise and exertion to which males are subjected. The greatest number of cases are discovered late during middle life, although it is possible to meet with disease of the endocardium at the aortic orifice in early adult life and even during childhood.

Principal Complaint.—**Before Failure of Compensation.**—"So long as the hypertrophy of the left ventricle successfully overcomes the otherwise injurious consequences of the valvular defects, the harmonious balance of forces is maintained, and there is an almost entire absence of symptoms" (Anders). Compensation is, as a rule, lost later in young subjects than it is in older ones, consequently a decided aortic lesion may exist for a prolonged period without manifesting symptoms. After the heart has be-

come markedly hypertrophied, undue muscular effort and even emotional and mental strain produce overaction of the heart, and give rise to one or more of the following symptoms: pulsation and tension at the occiput, beating of the temples, a peculiar throbbing headache, tinnitus aurium, and attacks of vertigo. When, in addition to an aortic lesion permitting of regurgitation, extensive arteriosclerosis is also present, anemia of the brain follows, and, as a consequence, extreme pallor, headache, flashes of light, dizziness and even distinct vertigo occur, especially when the patient changes from the recumbent to the erect posture. Dyspnea may be an annoying symptom at any time during the course of aortic regurgitation, but early during the disease it is experienced only after undue exertion, and is, as a rule, the result of pulmonary congestion.

When the heart has become markedly hypertrophied, precordial oppression is quite common, but seldom gives rise to decided annoyance. A dull aching pain may be felt over the precordium, and will at times radiate to the shoulders and possibly to the left arm. In a small percentage of cases pain will be definitely localized to the left shoulder. Angina pectoris may develop during the course of aortic regurgitation. (See p. 302.)

After Failure of Compensation.—When failure of compensation takes place and the cardiopulmonary circulation becomes retarded, this unbalancing of the circulation through the lung produces dyspnea, which is increased upon even slight exertion. Among the symptoms now present are cough and the occasional expectoration of blood-streaked sputum. (See Mitral Disease, p. 277.) Sooner or later a moderate degree of general venous congestion occurs, and dyspnea becomes severe, compelling the patient to assume a recumbent or semirecumbent posture. The latter symptom is, as a rule, more marked during the night and early morning hours.

During the course of aortic disease emboli may be deposited in the various organs,—*e. g.*, in the brain, spleen, eye, kidney, and liver, at which time the symptoms referable to embolism of any particular viscus may arise. Plugging of the coronary arteries and of certain of the cerebral arteries, although more common in aortic regurgitation than in other forms of cardiac disease, is still somewhat rare. It is a cause of sudden death.

Nervous Manifestations.—At first there are decided irritability and peevishness after compensation has failed, and if the patient suffers from cerebral anemia, he may be unable to sleep. Melancholia may be a permanent feature late during the disease, although it is occasionally regarded as merely a coincident symptom.

Thermic Features.—Irregular fever, when present, is due either to the intercurrence of acute endocarditis or to some other inflammatory process.

Physical Signs.—Inspection (Local).—The area of cardiac impulse is greatly increased; the impulse of the apex is displaced downward and to the left, and may be seen as low as the sixth or seventh interspace, and external to the nipple. The precordial region may show some bulging, a feature more common in young subjects. Throbbing in the region of the apex-beat should always arouse suspicion, and suggests a forcible impulse. The arteries in the carotid region throb vigorously, and when the patient is directed to raise his hands (Fig. 115), the axillary, brachial, and arteries of the forearm will be seen to pulsate.

Pulsation of the temporal arteries is also common, and when the femoral region is exposed, throbbing of the femoral artery is observed; the arteries of the feet are also seen to pulsate.

When myocardial degeneration is present, the pulsation over the precordium and the throbbing of the arteries become less and less conspicuous,

owing to deficient cardiac muscular power. Later in the disease, and after tricuspid insufficiency has developed, there is decided pulsation over the veins of the neck. As a result of dilatation of the right heart, epigastric pulsation occurs. The fingers are cyanosed and often clubbed and the mucous membrane of the tongue is bluish.

Cutaneous Manifestations.—On drawing the finger firmly across the patient's chest or back, there is first seen a decided paling, which is soon followed by a marked reddening of the same area, which subsequently pales, flushes again, and then pales, until it is practically the same color as the surrounding skin. On holding the patient's finger and making general pressure

FIG. 115.—POSITION FOR INSPECTION OF CHEST AND ARTERIES.
Special position for making prominent the pulsation of the axillary, brachial, and arteries of the forearm in aortic regurgitation.

upon the tip of the nail (Fig. 65), the red or flushed line, which is well marked underneath the nail, will be seen to advance and recede with each cardiac pulsation—another evidence of the existence of Quincke's pulse. Rarely, the veins of the hands and those of the feet are seen to pulsate. Lastly, Quincke's pulse will be obtained by placing a glass slide over the lip and exerting moderate pressure, when, with each cardiac pulsation the mucous membrane underneath the slide will be seen to flush and pale (Fig. 64).

Ocular Phenomenon.—Upon *ophthalmoscopic examination* the retinal vessels are seen to pulsate in well-marked cases of aortic regurgitation, while cardiac hypertrophy is present.

Palpation.—A forcible heaving impulse is felt in the region of the apex of the heart before myocardial changes have appreciably weakened the cardiac muscle, but whenever dilatation predominates over hypertrophy, the impulse is proportionately weakened. A diastolic thrill may be felt over the base of the heart, although this is a somewhat uncommon sign. A presystolic thrill may rarely be present.

When aortic regurgitation has continued long enough for regurgitation at the tricuspid ring to result, the liver becomes increased in size as the result of venous congestion, and palpation may elicit the fact that the lower border

of this organ is well below the margin of the ribs, and in extreme cases it may extend to near the level of the umbilicus. Rarely, in these cases, the liver will be found to pulsate.

The Pulse.—The pulse in itself is characteristic of aortic regurgitation—*e.g.*, a quick, leaping, full pulse is the initial impression conveyed to the palpating finger, but as the pulse-wave strikes the finger, an abrupt recession is noted, giving a somewhat double sensation—the so-called "Corrigan" or "water-hammer" pulse. When the arm is lifted above the level of the head, a sudden collapse of the pulse is observed, and it is this method of examination that is usually employed for the demonstration of the Corrigan pulse. (See also Fig. 116.) After the heart has become greatly weakened and marked dilatation has occurred, the Corrigan pulse loses most of its characteristics, and it may then be possible to obtain the typical pulse only when the patient's arm is on a level with his body. When dilatation has become extreme, the water-hammer pulse is absent. (See Sphygmographic Tracing, p. 271.)

FIG. 116.—A SATISFACTORY METHOD FOR ELICITING THE TRIP-HAMMER PULSE SUGGESTED BY HAWKE.

Percussion.—The area of cardiac dullness is in direct correlation with the degree of hypertrophy or of dilatation, consequently cardiac dullness may be found to extend far to the left and even beyond the anterior axillary line, although it is also possible to find it extended downward to the border of the sixth, seventh, or even the eighth rib (Fig. 117). When secondary dilatation of the left auricle has developed, the area of dullness is increased upward and to the left. It is customary for dullness due to hypertrophy of the right ventricle to increase downward and to the right, extending well to the epigastrium. When dilatation exists, the area of cardiac dullness is seen to be greatest in its transverse diameter, although it extends slightly upward, and the apex of the organ is appreciably rounded, as compared with the normal (Fig. 117).

After the development of tricuspid regurgitation the area of hepatic dullness is increased, and as the disease advances, the liver note may be found to extend some distance below the costal margin, and will also be found to be lower than normal when percussion is made over the median line. The spleen may also become enlarged as the result of venous congestion, consequently the area of splenic dullness is perceptibly increased during the later stages of this type of valvular disease. Ascites may develop late,

and when it does, percussion will disclose the note characteristic of this condition. (See Ascites, p. 567.) A transudate may accumulate in the pleura, when a flat percussion-note will be obtained. (See Pleurisy, Hydrothorax, and also Physical Signs of Pericardial Effusion.)

Auscultatory percussion confirms both palpation and percussion with reference to the size of the heart, liver, and spleen.

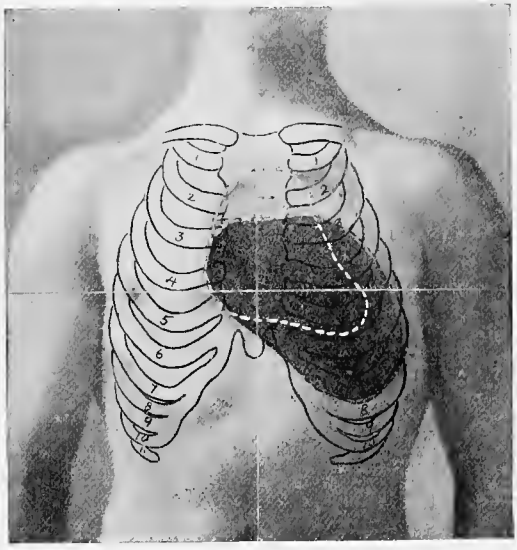


FIG. 117.—WHITE ILLUSTRATIVE OF NORMAL HEART. SHADED AREA SHOWS EXTREME HYPERTROPHY OF LEFT VENTRICLE AS SEEN IN AORTIC REGURGITATION WHILE COMPENSATION IS PERFECT.

Auscultation.—The characteristic murmur of aortic regurgitation is audible when hypertrophy of the left ventricle is in advance of dilatation; its distinctive features are as follows: A soft, blowing murmur—it may be loud in selected cases—heard at the second costal cartilage, to the right of the sternum, its area of greatest intensity being a trifle below and to the left of this point. This murmur is diastolic in time, and is transmitted along the left edge of the sternum as low as the ensiform. To the left, from the xiphoid cartilage, the murmur of aortic regurgitation may be heard as a diastolic whisper as far to the left as the axilla, and rarely close to the spine. This faint whispering sound is occasionally transmitted to the vessels of the neck, and is heard best in the right carotid region. Aortic regurgitant murmur may be best heard when the patient is in the recumbent posture. Occasionally no murmur is audible over the aortic cartilage, while a faint murmur is heard along the sternum or over the pulmonary area.

Quality of the Murmur.—Ordinarily, the murmur is soft and blowing, but when there is extensive atheroma at the aortic ring, it may be more or less musical, roughened, and at times loud.

Determination of Time.—In order to determine the rhythm of an aortic regurgitant murmur the stethoscope should be placed at the base of the heart and at the lower border of the second right costal cartilage. One or more fingers should then be placed either upon one of the carotids or upon the subclavian artery. The closer the palpating finger is to the heart, the more readily will the murmur be timed. It requires but a small amount of practice for the student to determine the murmur of aortic regurgitation, heard immediately following the impression conveyed by the artery to the palpating finger. The method here outlined for timing cardiac murmurs has been found to be most satisfactory.

Again, the rhythm of a murmur should be further studied with reference to the following characteristics: (*a*) Is the character of the murmur the same (as to length, loudness, etc.) during each pulsation of the heart or does it vary when the patient is at rest? (*b*) What influence, if any, has exercise

upon the murmur in question? (*c*) Is the murmur increased, lessened, or absent during forced inspiration, and how, if at all, is the murmur influenced by the ordinary respiratory act? (See Pulsus Paradoxus, p. 198.) A fact ever to be borne in mind is that the murmur of aortic incompetency is a prominent sign before dilatation has become pronounced, and that after extreme dilatation has occurred, it is materially modified and may, in many instances, be absent.

Associated Murmurs.—In the majority of cases in which aortic regurgitation has continued for a prolonged period other cardiac murmurs—*e. g.*, those of aortic stenosis, mitral regurgitation, and tricuspid regurgitation,—will be audible; the characteristics of each of these murmurs will be discussed at length under special headings.

Flint Murmur.—In the late stages of cases of aortic regurgitation, when the left ventricle is dilated to its maximum, a presystolic murmur, which is not transmitted, is often heard at the apex. This is the so-called Flint murmur. It is supposed to be due to the floating inward of the anterior leaflet of the mitral valve by the regurgitating blood from the aorta, so that the stream of blood coming into the left ventricle from the left auricle meets with an obstruction.

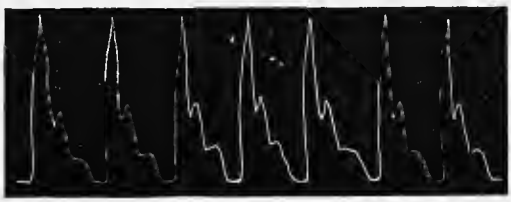


FIG. 118.—PULSE-TRACING IN A CASE OF AORTIC REGURGITATION (William Hoffman).

Sphygmographic Tracing.—This method of detecting aortic regurgitation is quite valuable, giving, as it does, evidence that is characteristic of the condition, more particularly since it discloses the presence of arhythmia and irregularity in the force of the heart's action. (See p. 213; also Fig. 118.)

Laboratory Diagnosis.—This is of no special value in determining the character of the cardiac lesion in question, but is of great importance in formulating a prognosis. Prolonged venous congestion of the kidneys may eventually lead to albuminuria and even to anuria. The characteristic evidences of acute nephritis (renal casts and albumin) may also be detected. Late during the course of aortic regurgitation there is a high-grade secondary anemia, the hemoglobin falling to 70 or even as low as 50 per cent., with a corresponding reduction in the red cells, although, in uncomplicated cases, the leukocytes are, as a rule, but slightly, if at all, disturbed. Staining methods reveal advanced degeneration of the red cells (irregularity in size, shape, and in the distribution of hemoglobin). (See Blood Changes of Secondary Anemia, p. 356.)

Summary of Diagnosis.—A history of previous attacks of acute endocarditis, rheumatism, or of one of the diseases known to predispose to degeneration of the endocardium should not be overlooked when formulating a diagnosis, and especially is this precaution necessary if the case is studied before a distinct diastolic murmur is heard at the aortic cartilage. Pronounced cardiac hypertrophy, as shown both by percussion and by the force of impulse at the apex, is strongly suggestive of disease at the aortic valve, and when the cutaneous manifestations of the disease—*e. g.*, throbbing of the arteries, Quincke's capillary pulse as shown on the skin, at the finger-nails, and on the mucous membrane of the lips—are also present, the evidence of aortic regurgitation is complete.

The detection of a soft, blowing diastolic murmur that is heard best at

the second costal cartilage, to the right of the sternum, and is transmitted along the left border of the sternum, is quite characteristic of the existence of aortic regurgitation, and upon the evidence of this finding the diagnosis may be established. In the early stages of aortic regurgitation the murmur may be feeble, and at this time it may be necessary to make the diagnosis from the other evidences previously detailed. (See Cutaneous Features, p. 268.)

Complications.—Among the more common of these are: Chronic gastritis, ascites, hydrothorax, myocarditis, and angina pectoris.

Clinical Course.—In young subjects aortic regurgitation may be present for a number of years without causing any discomfort as the result of cardiac diseases. As a rule, the patient survives the condition for a period of months and often several years after compensation has been broken. Sudden death occasionally occurs, although many patients are invalids after compensation has become ruptured.

AORTIC STENOSIS.

Pathologic Definition.—A disease of the endocardium characterized by a narrowing of the lumen of the aortic orifice, together with thickening and possibly the formation of adhesions of the aortic leaflets, and finally by the development of myocarditis.

General Remarks.—Simple aortic stenosis is a very rare condition. The development of stenosis of the aortic orifice is sooner or later followed by disease of the aortic valves, in consequence of which blood is regurgitated from the aorta into the left ventricle following each systole. Although it is common to find both aortic stenosis and aortic regurgitation in the same patient, in some cases aortic stenosis may appear to have been the primary lesion, and to have induced aortic regurgitation, whereas in a much smaller proportion of cases, aortic stenosis follows aortic regurgitation.

Exciting and Predisposing Factors.—(*a*) The exciting factor, as a rule, is a slowly progressing **sclerosis of the aortic leaflets,** together with the formation of calcareous deposits both in the leaflets and surrounding the aortic ring. Peter has called special attention to atheromatous changes at the root of the aorta, and, indeed, calcareous deposits are frequently found to extend into the coronary arteries. (*b*) Rarely, **rheumatic endocarditis** leads to the development of aortic stenosis. Seldom, indeed, this condition may be found to have followed other types of endocardial disease. (*c*) **Age** and **sex** are potent predisposing factors, since sclerotic changes at the root of the aorta, of the aortic leaflets, and of the endocardium at the aortic ring are common to males of advanced life.

Mechanic Influence of the Lesion.—The wall of the left ventricle hypertrophies as the result of having to force the blood from the ventricle through the small aortic opening into the aorta, and since the constriction of the aortic orifice has been gradually increasing, ventricular hypertrophy has likewise developed slowly; for these reasons but slight dilatation is present, unless a variable degree of regurgitation at the aortic ring also exists. Increased ventricular tension, which results from the obstruction encountered at the aortic orifice, is believed to favor the development of sclerotic changes at the mitral orifice. Hypertrophy of the left ventricle continues until it reaches a point when the cardiac muscle can no longer be nourished by the coronary arteries, when degenerative changes in the muscular tissue develop, soon to be followed by well-marked dilatation—dilatation in advance of hypertrophy. Following cardiac dilatation the result of a

primary aortic stenosis, mitral incompetency (relative) occurs, which permits the blood to be regurgitated into the left auricle and the pulmonary veins, thereby increasing pulmonary tension. Owing to the obstruction in the lung, the right heart first hypertrophies, later dilates, and then permits of tricuspid regurgitation, which is followed by general venous stasis.

Principal Complaint.—Aortic stenosis may be present for years without giving rise to any inconvenience, and the symptoms, as a rule, date from the first evidence of failure of compensation. At this time the patient complains of frequent attacks of slight dizziness, which may, however, be sufficiently severe to cause him to sit or cling to some object for fear of falling—a condition that usually follows exertion and is caused by an inadequate volume of blood entering the aorta. Attacks of syncope may be annoying, and headache is more or less constant. Owing to the roughening of the aortic leaflets, small clots are likely to form behind and about these atheromatous areas, and are frequently dislodged and escape into the circulation. As a consequence, embolism of the spleen, kidney, brain, lung, etc., tends to develop. The existence of an embolus will be manifested by characteristic symptoms and signs, depending upon the location in which the clot lodges. (See Pulmonary Embolus, p. 117.)

Physical Signs.—Inspection.—During attacks of vertigo there is extreme pallor, both of the face and of the extremities. Soon after compensation has been ruptured edema of the ankles and feet occurs, and gradually increases as the disease advances.

The apex-beat is seen one or more inches below its normal area, and in or to the left of the nipple-line, a feature that is explained by the high grade of hypertrophy of the left ventricle present. The cardiac impulse is, as a rule, slow, and may be of such force as to cause an appreciable heaving of the precordium, although in some cases the apparent impulse of the heart is not exaggerated. Depending upon the condition of the myocardium the area of the apex impulse may be diminished, and in emphysematous patients it may be absent.

Palpation.—After failure of compensation has occurred, the skin of the lower extremities pits upon pressure. As the result of an associated tricuspid regurgitation with venous congestion, the liver and spleen may be enlarged.

Ordinarily, the impulse of the heart is forcible, except when pulmonary emphysema or cardiac dilatation is present. On placing the hand over the base of the heart a well-marked systolic thrill is often detected, its area of greatest intensity being near the second right costal cartilage. In selected cases, in which an apical thrill is palpable, it may be felt in the region of the apex-beat. The same sensation is more pronounced near the base of the heart.

The *pulse* is quite characteristic, being small, regular, not readily compressed, and of slightly lessened frequency. (See Sphygmographic Tracing, p. 274, Fig. 120.)

Percussion.—Despite the high grade of cardiac hypertrophy, the area of cardiac dullness is not so decidedly increased in aortic stenosis as it is in aortic incompetency. In uncomplicated cases the area of cardiac dullness will be found to be increased downward and to the left.

Auscultation.—A harsh and sometimes rasping murmur is heard in the second right interspace; this murmur is systolic in time, and in typical cases is transmitted to the vessels of the neck. (See Fig. 119.) After compensation has been ruptured the murmur may be much softer and smoother in quality than it was during the early stage of the disease.

In our practice cases of aortic stenosis have frequently been followed over a prolonged period and it has been observed that within the course of one or more years a harsh, rasping murmur may entirely disappear, after which, instead of the booming quality of the heart that was originally present, the first sound has also lost its muscular element, and, indeed, the second cardiac sound, always diminished, may become practically inaudible. (See Differential Diagnosis, p. 275.)

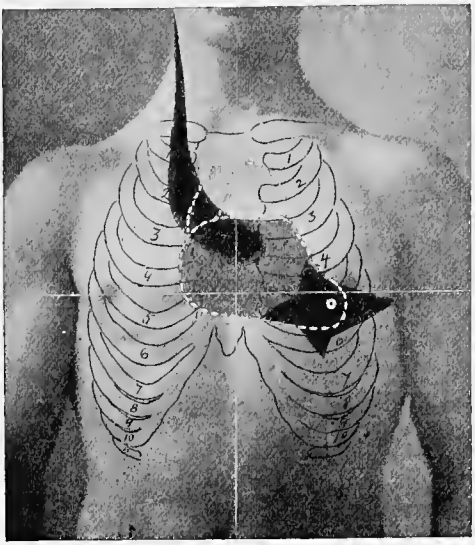


FIG. 119.—SUPERIOR AREA OF SHADING IS ILLUSTRATIVE OF AREA OF GREATEST INTENSITY AND DISTRIBUTION OF THE SYSTOLIC MURMUR OF AORTIC STENOSIS. SHADED PORTION BELOW SECOND CARTILAGE AND AS HIGH AS CLAVICLE IS WHERE AORTIC REGURGITATION IS HEARD.

a, Area where mitral systolic murmur is audible.

Caution.—Aortic regurgitation is so commonly associated with aortic stenosis that a murmur will probably be heard with both systole and diastole. (See Fig. 119.) Owing to the constriction at the aortic orifice and to the various blood-currents that are created as the result of roughening in the region of the aortic orifice, one sometimes detects an almost continuous, saw-like murmur. The so-called "double murmur" heard at the aortic cartilage refers to the distinct systolic (aortic stenotic) murmur and a diastolic (aortic regurgitant) murmur.

In those cases of cardiac disease in which arhythmia is a conspicuous symptom it is ofttimes practically impossible to time the murmur with any degree of satisfaction; the clinician must, therefore, depend upon the area of transmission of the murmur as the factor on which to base his diagnosis. The murmur of aortic stenosis is, with but few exceptions, transmitted to the vessels of the neck, and in well-marked cases it is heard as high as the angle of the jaw (Fig. 119). The murmur of aortic incompetency is seldom transmitted to the neck, but in nearly all instances it is heard along the sternum and, at times, from the aortic cartilage toward the apex of the heart. Every clinician has frequently found selected cases in which either a stenotic or a regurgitant aortic murmur may be heard over the entire precordial region, and at times well into the neck. In this last type of cases the diagnosis is attained not by auscultation alone, but by careful examination and judicious balancing of all the symptoms and signs displayed in the individual case.

Sphygmographic Tracing.—The sphygmographic tracing of aortic stenosis (Fig. 120) is in itself quite characteristic, showing, as it does, a marked slowness of the ascending curve and a gradual, step-like descent.

Summary of Diagnosis.—A most important factor in the diagnosis of aortic stenosis is the slowness of the heart's action, together with the small, soft, although not readily compressible, pulse. Repeated attacks of vertigo, associated with extreme pallor, and a tendency toward syncope are to be

considered in connection with this disease, although these symptoms may also be encountered during the course of other cardiac conditions. The detection, at the aortic cartilage, of a systolic murmur that is transmitted well above the clavicle and at times as far as the angle of the jaw (Fig. 120), coupled with a systolic thrill at the base and a small, tense pulse, makes the diagnosis of aortic stenosis positive.

Differential Diagnosis.—Calcareous deposits at the root of the aorta, especially when they involve the aortic segments, may create an

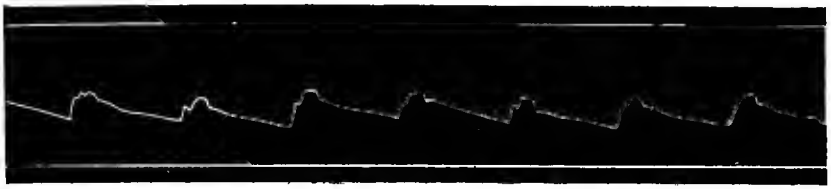


FIG. 120.—SPHYGMOGRAM OF AORTIC STENOSIS FROM A MAN AGED SIXTY YEARS (Anders).

adventitious sound that is systolic in time and resembles closely true aortic stenosis; this murmur is seldom, if ever, musical in character, a feature of aortic stenosis. In aortic stenosis the second sound is enfeebled or absent, whereas in those cases in which the murmur is due to sclerotic change in and about the aortic orifice distinct accentuation of the second sound is common.

During the course of **chronic Bright's disease** a high grade of aortic sclerosis is generally present, together with hypertrophy of the left ventricle, and, in consequence of such changes, a systolic murmur may be audible at the aortic cartilage. In these cases an analysis of the urine may give positive evidence of nephritis. A distinctly accentuated second sound favors a diagnosis of nephritic changes rather than one of aortic stenosis.

In **aortic regurgitation** a systolic murmur is not infrequently also present, but in such instances the condition should not be regarded as aortic stenosis unless the actual muscular quality and a systolic thrill are also present. Again, it is to be remembered that the pulse of aortic stenosis is not characteristic, when both stenosis and regurgitation are present at the aortic ring.

The accompanying differential table sets forth the distinctive features between aortic stenosis and aortic regurgitation:

AORTIC STENOSIS.	AORTIC REGURGITATION.
1. Absent.	1. Capillary pulse (Quincke's) is present over the skin, finger-nails, and mucous surfaces.
2. Arteries not well filled with blood at each systole.	2. Arteries well filled at each systole.
3. Arteries are not seen to pulsate.	3. Throbbing of the arteries of the arms and extremities.
4. Pulse slow and small, systolic thrill felt at base.	4. Characteristic "trip-hammer" pulse. Thrill uncommon, diastolic in time.
5. Cardiac hypertrophy moderate.	5. Hypertrophy of the left ventricle extreme.
6. Murmur rather harsh, systolic in time, and transmitted to the vessels of the neck.	6. Murmur less harsh, and usually soft in character, diastolic in time, transmitted downward along the sternum or toward the apex.

The so-called "**hemic murmurs,**" heard best over the base of the heart, are to be distinguished from the murmur of aortic stenosis. Hemic murmurs, while systolic in time, are soft and indistinct, and are not associated with hypertrophy of the left ventricle or a systolic thrill. Again, hemic murmurs are present only in those patients suffering from some pathologic condition in which profound primary or secondary anemia is a prominent feature.

Clinical Course.—Stenosis at the aortic ring develops somewhat gradually, and the patient may thus be afflicted for a number of years before any inconvenience is experienced. After the first symptoms, which become manifest at the time compensation becomes embarrassed, aortic regurgitation runs a chronic course, and may continue over a number of years. Sudden death, however, may result from the escape of certain calcareous particles into the blood-current, causing embolism of the brain or other organs.

MITRAL REGURGITATION (MITRAL INSUFFICIENCY; MITRAL INCOMPETENCY).

Pathologic Definition.—A disease of the endocardium, secondary to acute endocarditis, and characterized by sclerotic changes, which are followed by constriction or rupture of the mitral leaflets, which prevents the mitral orifice from closing completely during each systole; it is also caused by dilatation of the left ventricle, which, by increasing the lumen of the mitral orifice, makes it impossible for the mitral leaflets to come in direct apposition to each other during systole; the condition is also, though rarely, due to disease of the chordæ tendineæ. After regurgitation has existed for an indefinite period hypertrophy of the left ventricle occurs, and in typical cases hypertrophy and dilatation of the ventricle develop simultaneously. The wall of the ventricle becomes greatly thickened, and remains so until the myocardium is not perfectly nourished, when myocardial degeneration sets in and permits of dilatation.

Mechanic Influence of the Lesion.—Incomplete closure of the mitral leaflets permits a portion of the blood to return into the left auricle during systole. The regurgitated blood meets the normal current simultaneously, coming through the pulmonary veins to the left auricle, and offers an obstruction to the escape of venous blood from the lungs. A meeting of these two blood-currents in the left auricle—one of venous blood from the lung, the other of regurgitated blood from the left ventricle—must create an abnormal current in the left auricle, which probably gives rise to a murmur.

As a result of blood entering the left auricle from the lung and from the left ventricle at the time of each systole, the auricle becomes overloaded, and this leads to dilatation, although an attempt at compensatory hypertrophy of the auricular wall is also present. Now that the left auricle contains an abnormal volume of blood, an extra effort is made by this chamber of the heart to propel the increased volume of blood into the left ventricle; the result of such overaction eventually leads to overdistention of the left ventricle. Owing to the incomplete closure of the mitral orifice, all the blood now contained in the left ventricle is not expelled into the aorta with each systole, but, on the contrary, a portion of it again returns to the left auricle, which leakage demands extra work upon the part of the left ventricle, and, as a consequence, the walls of this chamber become hypertrophied—hypertrophy and dilatation develop simultaneously.

The hypertrophied left ventricle is capable, for an indefinite period, of forcing about the normal volume of blood into the aorta with each systole, and during this period the arterial tension approximates the normal. Sooner or later the cardiopulmonary circulation becomes impeded, and the blood-

current returning from the lung to the left auricle through the pulmonary veins is so decidedly obstructed as to increase the arterial tension in the lung. The damming back of blood in the lung obstructs the current of blood flowing through the pulmonary artery and capillaries, and increased tension here sooner or later brings about sclerotic changes in the pulmonary arterial system, which act as an additional source of interference with the circulation of blood propelled by the right ventricle to the lung. Increased circulatory tension in the lung in time causes hypertrophy and dilatation of the right ventricle. The presence of such increased tension is detected clinically by accentuation of the second pulmonic sound. So long as the right and the left ventricle are sufficiently hypertrophied to maintain the circulatory equilibrium through the lung and through the general arterial tree, serious symptoms do not arise, but whenever this equilibrium is disturbed, dilatation of the right ventricle in excess of hypertrophy and tricuspid regurgitation follows.

A regurgitant blood-current through the tricuspid orifice offers direct obstruction to the return of venous blood from both the ascending and descending vena cava, and as a result of this regurgitation the cardiac ventricles, particularly the left ventricle, are inadequately filled during each diastole, consequently the arterial tree does not receive the normal amount of blood. Again, on account of obstruction to the returning venous blood-supply, venous congestion of the viscera and of the extremities, together with the transudation of the fluid elements of the blood into the serous cavities, takes place.

Predisposing and Exciting Factors.—Among the predisposing factors should be considered those that favor acute endocarditis and, in addition, overwork, such as heavy lifting and the like. Acute endocarditis, whether rheumatic or simple in character, serves as the most potent factor in the production of mitral regurgitation. General arteriosclerosis also figures prominently in the causation, as do cirrhosis of the liver, chronic nephritis, and other forms of obstruction to the general circulation. Aortic stenosis occurring as a primary lesion may eventually produce mitral regurgitation. Moderate dilatation of the left ventricle, as is seen during the course of acute fevers and maladies characterized by profound anemia, may permit of a temporary regurgitation at the mitral orifice.

Principal Complaint.—While Compensation is Maintained.—During this period otherwise healthy persons do not complain of symptoms referable to disease of the heart. After the lesion has continued for some time,—probably several years,—slight embarrassment of the pulmonary circulation may be seen to follow excitement and overexertion. The symptoms now consist of temporary *dyspnea, cough,* and probably the expectoration of a small quantity of frothy, blood-streaked material. Many cases complain only of shortness of breath, and rarely of a dry, hacking cough which follows exertion. The physical signs present during this period are unusually interesting and of great diagnostic value. (See Physical Signs, below.)

After Compensation is Ruptured.—When compensation is lost, the right ventricle is no longer able to cope effectively with the existing circulatory tension (obstruction) offered to the return flow of venous blood from the general system, which obstruction in turn extends rapidly from the right heart both to the periphery of the body and to the viscera, and soon affects the general system. *Dyspnea, cough, expectoration,* and the symptoms common to the latter portion of the stage of compensation become intensified.

Cough is accompanied by a somewhat free expectoration, which is frequently bloody. The patient complains of *cardiac palpitation*, and this annoying symptom may follow even slight exertion. Vertigo and a tendency toward attacks of fainting may be experienced.

Pain is unusual, except in those cases in which a variable degree of stenosis is also present. *Gastro-intestinal catarrh, nausea*, the vomiting of mucus and at times of blood-streaked material, may be an early and annoying symptom. Following venous congestion of the liver *hemorrhoids* are likely to develop.

Physical Signs.—During Compensation.—*Inspection.*—Inspection may at first be negative, but as the disease progresses a distinct pallor is

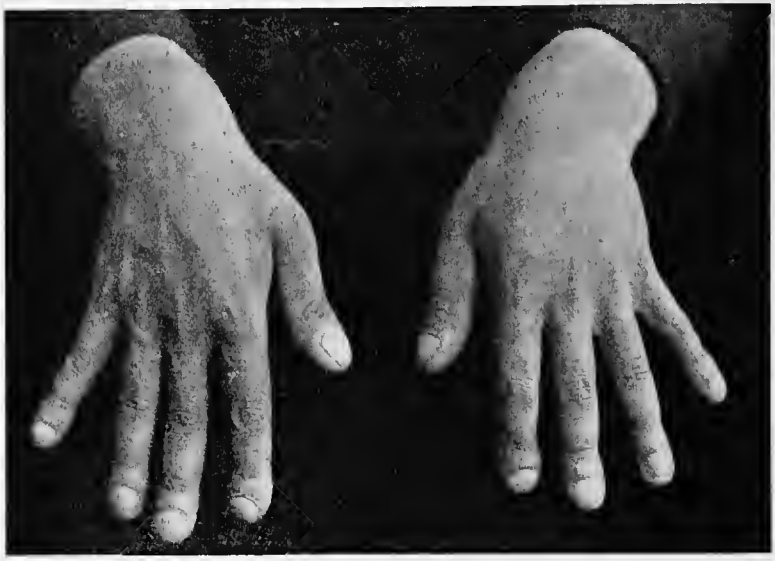


FIG. 121.—CLUBBING OF THE FINGERS DUE TO VALVULAR HEART DISEASE.
Child eight years of age. (Courtesy of Dr. J. A. McKenna.)

perceptible, the features are somewhat pinched, the lips and ears are moderately cyanosed, and the vessels of the neck are abnormally prominent. Clubbing of the finger-tips and nails is an almost constant feature in young subjects and is fairly common after middle life (Figs. 121 and 123).

After Rupture of Compensation.—*Inspection.*—The extremities are swollen, and in those cases in which compensation is fairly well maintained there is edema of the ankles. The abdomen may become pendulous as the result of an effusion into the peritoneum, and when ascites is present, the respirations are rapid and shallow (Fig. 122). Cyanosis becomes extreme, the lips, finger-tips, and nails displaying a variable degree of lividity. In well-marked cases the mucous membrane of the mouth becomes cyanosed and the face is dusky.

In the majority of cases the precordial region is prominent, a feature that is more conspicuous in children than in adults. The apical impulse is unusually large, and even late in the disease a diffuse wave is seen in the region of the left nipple, often extending downward to the sixth interspace, and well toward the anterior axillary line. Pulsation of the epigastrium may

also be present, and is a constant feature where there is associated dilatation of the right ventricle. After failure of the right heart has developed, a distinct wave is seen over the vessels of the neck.

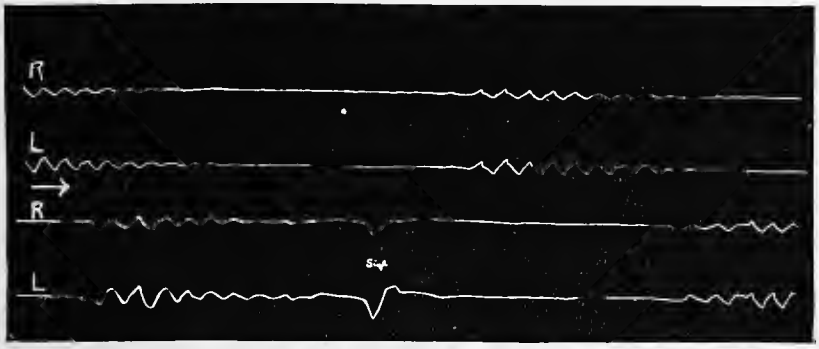


FIG. 122.—BILATERAL PNEUMOGRAM FROM A CASE OF CHEYNE-STOKES RESPIRATION, DEVELOPING DURING ADVANCED CARDIOVASCULAR DISEASE (Boston and Ulman).

Lower curves R and L were also made from the same patient. The marked downward summit of the curve, seen during the pause, resulted from the patient's sighing. (See Bilateral Movements of the Chest, p. 131.)

Palpation.—A systolic thrill is sometimes detected at the apex of the heart. During that period of the disease in which compensation is well maintained the apex-beat is forcible and heaving in character, but with beginning failure of compensation its force is proportionately weakened. Late in the disease, and after the left ventricle has become markedly

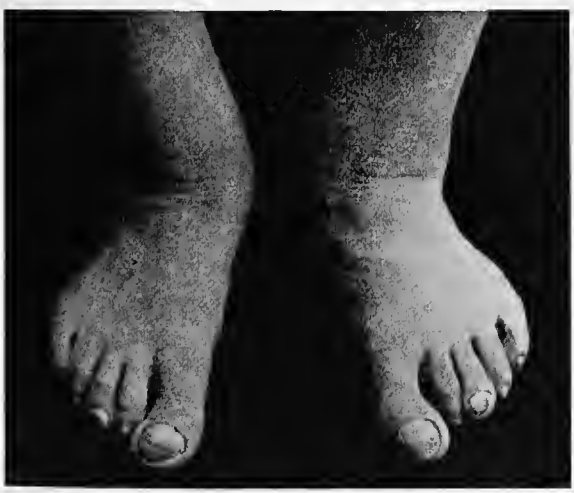


FIG. 123.—CLUBBING OF THE TOES DUE TO VALVULAR HEART DISEASE.
Child eight years of age. (Courtesy of Dr. J. A. McKenna.)

dilated and myocardial changes have taken place, the apex-beat is weak and often irregular. Throbbing of the veins of the neck, when due to an associated tricuspid regurgitation, is arrested by exerting mild pressure with the finger upon the vein immediately above the clavicle—a sign that serves to distinguish venous from arterial pulsation.

The Pulse.—The force and tension of the pulse are, as a rule, in direct ratio to the strength of the impulse of the heart, and while compensation is maintained the pulse is full, regular, and strong, although its tension may be slightly lower than normal. (See Blood-pressure, p. 204.) After compensation has become lost the tension, force, and regularity of the pulse are disturbed, and it is readily compressible. (See Fig. 78.) Arhythmia, however, while uncommon, may be present in selected cases during the stage of compensation. When the heart is markedly dilated, it may be possible to place the hand over the epigastrium, and, by gentle pressure, to feel the heart's impulse distinctly. After the application of judicious treatment it will frequently be seen that the dilated heart returns to nearly its normal size within the course of a few days, and that this sign—pulsation in the epigastrium—disappears. The lower border of the liver is often palpable and the spleen is also enlarged. (See Pulsation of the Liver, Tricuspid Regurgitation, p. 288.)

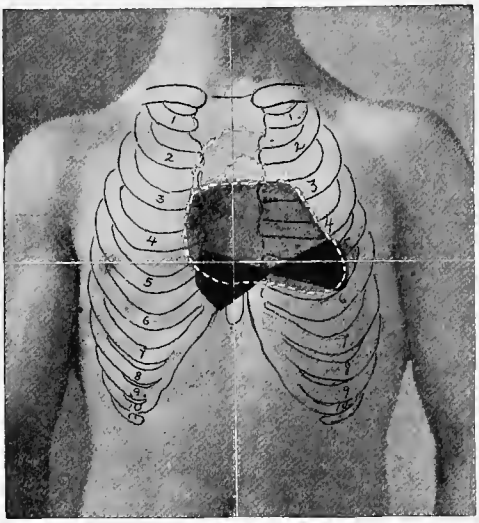


FIG. 124.—AREA WHERE TRICUSPID REGURGITATION IS HEARD.

Showing area where a loud systolic mitral murmur may be detected in addition to being transmitted toward the axilla.

Percussion.—The area of cardiac dullness is increased downward and to the left, and may extend to or beyond the anterior axillary line, this sign being present early, and often before the patient has experienced any symptoms referable to cardiac disease. The note of cardiac dullness extends to the right of that normally present, and in marked cases may be found one-half to one inch beyond the right border of the sternum. When cardiac dilatation has developed, the area of cardiac dullness is perceptibly widened, consequently the increase is more pronounced in the transverse than in the vertical diameter. Dullness may also extend well into the left half of the epigastrium.

Auscultation.—Preceding the development of failure of compensation a systolic murmur is audible, its seat of greatest intensity being at the apex of the heart Fig. 124). In selected cases this systolic murmur may be heard most distinctly in the third or fourth interspace, in the left midclavicular line. Rarely, as has been pointed out by various clinicians, the systolic murmur of mitral regurgitation is heard best over the base of the heart.

Posture may be an important factor in auscultating for the detection of mitral disease, since in some the murmur is audible only when the patient is in the recumbent posture, being absent when he is sitting or standing. A reverse condition was found in a man reported by C. J. Hoban, the murmur disappearing when the patient was lying upon either his side or his back. The systolic murmur of mitral regurgitation is transmitted from its area of greatest intensity near the apex toward the axilla, and is not infrequently audible as far to the left as the angle of the scapula. The mur-

mur, however, becomes less and less distinct as the stethoscope is carried toward the scapular region. In following the transmission of the mitral murmur it is well to keep the stethoscope directly over the rib or interspace where the murmur is found to be loudest at the apex, traveling on this line to the left.

When the stethoscope is placed over the third left costal cartilage, the pulmonic second sound of the heart is appreciably accentuated during the course of mitral regurgitation, a clinical feature explained in the mechanism by which the lesion of this disease is produced. (See p. 276.) The loudness of a mitral murmur may depend upon certain elements, the most prominent of which are: (*a*) Strength of the heart; and (*b*) degree of sclerosis in the region of the mitral orifice.

Other murmurs are frequently audible after mitral incompetency has continued for a prolonged period. After compensation is broken, a murmur is audible at the ensiform cartilage (Fig. 124), and signifies that tricuspid regurgitation exists.

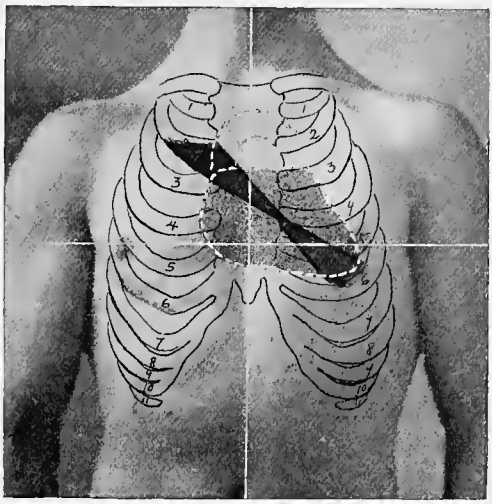


Fig. 125.—Shaded Areas Show Areas of Distribution of Aortic Regurgitation and Mitral Regurgitation when Latter Murmur is Unusually Loud.

Laboratory Diagnosis.—*Cough* is, as a rule, accompanied by quite free expectoration, which is found on microscopic examination to contain alveolar cells: within the body of many of these cells granules of a brownish pigment are to be seen. The sputum may be blood streaked, or the patient may expectorate nearly pure blood at intervals. Red blood-cells and leukocytes are, as a rule, present in the sputum, even though the macroscopic appearance does not suggest the presence of blood.

After tricuspid regurgitation and congestion of the abdominal viscera have developed, the quantity of urine voided during the twenty-four hours is greatly lessened, is of high specific gravity, and contains a trace of albumin. Casts and both red and white blood-cells are to be found late during the course of mitral regurgitation.

Aspiration of the serous sacs, *i. e.*, the pleura, peritoneum, and pericardium, may result in the recovery of a clear, slightly greenish, opalescent liquid, having a specific gravity of 1.002 to 1.005, or possibly 1.010, and giving a decided reaction for albumin.

Summary of Diagnosis.—The history of previous attacks of acute endocarditis, or of those affections known to favor the development of disease of the endocardium (see Endocarditis, p. 251), must be taken into consideration in formulating a diagnosis. A high degree of hypertrophy of the left ventricle, with an increase in the area of cardiac dullness to the left and right, is of great diagnostic value. The existence of mitral regurgitation is established by the detection of a systolic murmur, heard best at the apex, with its typical area of transmission. (See Fig. 126.) Marked accentua-

tion of the second pulmonic sound is additional evidence of the existence of this disease. After cardiac dilatation has taken place the diagnosis of mitral incompetency is made only with great difficulty, and, indeed, in many cases it is often impossible at this time to determine the nature of the original cardiac lesion.

Differential Diagnosis.—(*a*) The extensive transmission of the murmur of mitral insufficiency readily serves to distinguish this murmur from that of **mitral stenosis,** the latter being heard at the apex, and being but feebly, if at all, transmitted. (*b*) The time of these two murmurs also serves as a diagnostic factor, although in those cases in which arhythmia obtains, this clinical evidence may be uncertain. (*c*) The murmur of mitral regurgitation and that of aortic stenosis are both systolic in time, and the area of distribution serves as the most important factor for their differentiation—*e. g.*, the murmur of mitral incompetency is heard best at the apex of the heart, and is transmitted to the left, whereas that due to aortic stenosis is heard most distinctly at the second right intercostal space, and is transmitted to the vessels of the neck. (*d*) Again, in mitral regurgitation the pulmonary second sound is accentuated, a feature that is absent in aortic stenosis. (*e*) In mitral incompetency the thrill is most marked at the apex of the organ, in striking contrast to the thrill of aortic stenosis, which is rough and palpable at the base of the heart. (*f*) The pulse is likewise an important factor in differentiating these two cardiac lesions, the pulse of mitral regurgitation being large and moderately forcible, whereas that of aortic stenosis is always appreciably small.

Functional murmurs are to be distinguished from the murmur of mitral insufficiency. The accompanying table, from Anders, will assist in differentiating between them:

Mitral Incompetency.	Functional Murmurs.
History.	
1. Previous history of rheumatism or other disease causally related.	1. History of causative factors of one or other form of anemia and of debility.
2. Frequently there is definite knowledge of coincident rheumatism or organic heart disease in the same person.	2. Absent.
Physical Signs.	
3. *Inspection.*—Duskiness of mucous membranes and extremities; later, wavy pulsation in veins of neck.	3. Pallor of skin and mucous surfaces present.
4. *Palpation.*—Finger-tips placed over apex-beat are forcibly lifted. Pulse-tension somewhat lowered and not prolonged. Apex impulse displaced down and to the left.	4. Impulse of the heart feeble. Apex impulse not displaced.
5. *Percussion.*—Evidence of enlargement of the heart.	5. Dilatation of right auricle in approximately one-half of all cases, giving rise to dullness upward and to the right edge of the sternum.
6. *Auscultation.*—A systolic apex murmur (often musical), with characteristic area of transmission. This murmur is often heard posteriorly; pulmonary sound accentuated.	6. Soft systolic murmur over body of heart (variable in intensity, rarely transmitted to axilla); there may be a systolic murmur at apex, and a venous hum over the vessels of the neck.

Clinical Course.—Before Compensation is Lost.—This stage of the disease may vary from a few to several years, and during this time the

patient suffers little or no inconvenience, and, indeed, may be unconscious of the existence of cardiac disease.

After Compensation is Ruptured.—Here the clinical course depends largely upon the ability to carry out judicious treatment, and upon the presence or absence of complications. In those cases in which it is possible to take the patient from his work and to prevent all undue excitement and the use of stimulants, narcotics, and the like, life will be greatly prolonged. On the other hand, in those unfortunate individuals who must earn a livelihood and those who are addicted to the excessive use of stimulants the disease runs a rapid course, terminating fatally in from one to three years. Embolism and thrombosis of the lung are likely to occur at any time during the course of mitral regurgitation, and whenever this accident develops, the life of the patient is greatly endangered.

MITRAL STENOSIS.

Pathologic Definition.—A cardiac condition characterized by a narrowing of the lumen of the mitral orifice the result of calcareous deposits, thickening of the mitral leaflets, adhesions of the mitral valves, disease of the chordæ tendineæ, or extensive atheroma of the endocardium immediately surrounding the orifice.

Predisposing and Exciting Factors.—Practically all conditions that predispose to endocarditis (see p. 251) likewise predispose to mitral stenosis.

Age is an important factor, this cardiac lesion being more common in children after the fifth year and in young adults.

Sex exercises a moderate influence, females being attacked more often than males.

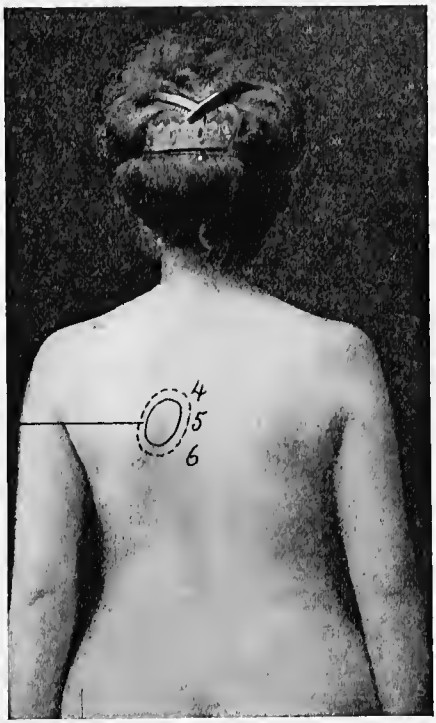


Fig. 126.—Rare Area of Transmission of a Loud Mitral Regurgitant Murmur.

Mitral stenosis may be seen to follow endocarditis complicating **scarlatina, measles,** and other of the **acute infections,** and many writers believe that the intense strain of **whooping-cough** favors its development. After middle life, in a large percentage of cases, mitral stenosis will be found to accompany **chronic nephritis** and general **atheroma of the arterial system.**

Mechanic Influence of the Lesion.—On account of the diminished size of the mitral orifice, undue force on the part of the left auricle is required to propel the blood from the auricle to the left ventricle, and in consequence of this overaction the wall of the left auricle hypertrophies and may attain a thickness twice that of the normal. Since the auricle is believed to accomplish much less by hypertrophy than does the left ventricle,

cardiac dilatation develops comparatively early, and at this time the auricular wall is unusually thin.

After dilatation has taken place the circulatory equilibrium is no longer maintained, and the blood occupying the overloaded left auricle offers serious obstruction to the current coming into the auricle through the pulmonary veins; as a consequence blood tension in the lungs and also in the right ventricle is increased. The right ventricle from the effort to overcome the obstruction offered in the pulmonary vessels, undergoes decided hypertrophy and eventually becomes dilated. Owing to dilatation of the right ventricle tricuspid regurgitation is permitted, and, as previously stated under Mitral Incompetency, interference with the venous blood from both the ascending and the descending vena cava follows, eventually causing edema of the viscera and superficial tissues.

Principal Complaint.—Before Failure of Compensation.—During the period when compensation is perfectly maintained, symptoms are practically absent, except when the patient takes unusual exercise, such as fast walking, running, heavy lifting, stair-climbing, mountain-climbing, and the like, when a variable degree of *dyspnea* occurs. During this stage fibrinous coagula may be dislodged from the region of the mitral orifice and escape into the general circulation, eventually reaching the brain and other viscera; consequently the development of *local paralyses*, the most common of which are hemiplegia and aphasia, is to be feared. At this time the patient is also subject to recurrent attacks of endocarditis, which, if severe, excite special symptoms. (See Endocarditis, p. 252.) Cardiac *palpitation* and dyspnea go hand in hand, and are frequently accompanied by a somewhat characteristic, stitch-like pain at the apex of the heart.

Following Failure of Compensation.—During this period the earliest symptoms are quite similar to those detailed under Mitral Regurgitation. (See p. 278.) *Dyspnea* is more or less constant, and is accompanied by serous and blood-stained *expectoration*, with periodic attacks of *hemoptysis*. *Pulmonary apoplexy* is to be feared late during the course of mitral stenosis, since the increased tension in the pulmonary vessels has given rise to atheromatous changes. A point of special importance is that during this stage (failure of compensation) general anasarca is unusual, although it may be present. Ascites is not uncommon.

Thermic Features.—Repeated attacks of fever occur during the course of mitral stenosis, and become more frequent as the disease advances. These febrile periods result from recurrent attacks of endocarditis. (See Recurrent Endocarditis, p. 261.)

Physical Signs.—Inspection.—The wave of the apex-beat is diffuse, although it is not displaced downward and to the left, unless hypertrophy of the left ventricle is also present. Pulsation is quite common at the second left intercostal space, and may frequently be seen in the third and fourth spaces, and there may also be well-marked pulsation along the right border of the sternum and in the epigastric region. In children the chest is abnormally prominent at the fifth and sixth costal cartilages, and the lower border of the sternum occasionally bulges.

After rupture of compensation the jugular regions become unduly prominent and pulsate, owing to engorgement of the venous system. The impulse excited by the apex-beat is feeble, and may be almost imperceptible. The lips are cyanotic, and the face at first presents a decided pallor, but later assumes a dusky appearance. Cyanosis of the finger-tips is also a late feature of mitral stenosis.

Palpation.—By placing the hand over the precordium so that the tips of the fingers cover the apex of the heart, a presystolic thrill will be detected, in a large proportion of all cases, at the third, fourth, or fifth interspace. The thrill of mitral stenosis is felt within the nipple-line and during the act of expiration. It may be absent when the patient is sitting or in the recumbent posture, and present after moderate exercise. In certain cases a thrill is not detected prior to the development of failure of compensation, but after this clinical stage of the disease is reached, it may be a prominent feature. The presystolic thrill is detected after the second sound, during diastole, when it appears as a soft, purring fremitus, which gradually increases in intensity, ending with a distinct shock with the cardiac impulse. "The fremitus and systolic shock are pathognomonic, and may be relied upon in the absence of the murmur" (Anders).

The impulse of the heart is frequently more forcible over the lower portion of the sternum than at the apex, a sign that results from hypertrophy of the right ventricle. In some cases, at least, a heaving impulse is detected to the left of the sternum, in the third, fourth, and fifth interspaces.

The pulse is small and quite readily compressible, and arhythmia is generally a conspicuous feature early during the disease.

Percussion.—In advanced cases the area of cardiac dullness will be found to extend from one-half to two inches to the right of the right border of the sternum, a sign that is explained by the extreme hypertrophy of the right ventricle. Cardiac dullness also extends upward along the borders of the sternum to the lower margin of the second rib. In selected cases it is possible to find the area of cardiac dullness increased to the left, but this condition, when present, is more likely to result from hypertrophy of the left than of the right ventricle, and is to be expected when there is an associated mitral insufficiency.

Auscultation.—The recognition of a rough, presystolic murmur, usually occupying only the latter half of the diastole, is a characteristic sign of mitral stenosis, and this murmur is synchronous with the thrill. A presystolic murmur is heard most distinctly one inch within the normal site of the systolic murmur of mitral regurgitation, and is not well transmitted (Fig. 62). In certain cases the murmur of mitral stenosis may be transmitted for a distance of one and one-half to two inches from its site of greatest intensity, a feature that, as previously stated, serves to distinguish this murmur from that produced by mitral regurgitation, since the latter is transmitted well into the axilla.

Characteristics of the Murmur.—In addition to being presystolic in time, the following characteristics are frequently observed: (*a*) The murmur is extremely brief; (*b*) it is not constant; and (*c*) it displays a low tone. Weakening of the right ventricle is conceded to be the cause for both temporary and permanent absence of the murmur during the course of mitral stenosis. Accentuation of the first sound is, as a rule, clear, whether or not the presystolic murmur is audible.

Rhythm.—The time of the murmur furnishes the most important diagnostic evidence; therefore, while auscultating in the region of the apex, the hand should always be placed over the precordium, since by these combined methods of physical examination both the ear and the finger are sensible of the shock that replaces the cardiac impulse. Again, a careful analysis of the sounds produced shows that the murmur terminates with the production of the shock. At times the cardiac impulse is not palpable, and in such cases the finger should be placed over one carotid or over the subclavian artery. A few cases have been seen in which the pulsation of these

arteries could not be detected; yet pulsation, when present, is practically synchronous with systole. The pulmonic second sound is greatly accentuated during the stage when hypertrophy of the right ventricle obtains, and the second aortic sound is appreciably lessened and often absent. Occasionally one encounters a case of well-marked mitral stenosis in which there is an apparent reduplication of the second sound.

Associated Murmurs.—Mitral stenosis is frequently an initial endocardial lesion, but it may also develop during the course of mitral regurgitation; consequently, the murmur of mitral regurgitation and of aortic disease, as well as that of tricuspid regurgitation, may also be present.

Laboratory Diagnosis.—*A hematologic* study made late during the disease shows that the patient is suffering from secondary anemia.

Owing to congestion and edema of the lungs, the sputum may be blood-streaked, and, indeed, periodic attacks of hemoptysis occur. *Microscopically*, many alveolar epithelial cells are seen, and certain of these display granules of yellowish-brown pigment. Both red and white blood-corpuscles are present, and the sputum may also contain various cocci and bacilli, but the latter are not believed to possess clinical importance.

Summary and Differential Diagnosis.—A diagnosis is based almost entirely upon the data obtained by physical examination; these include: (*a*) Increase in the area of cardiac dullness upward and downward and to the right; (*b*) a presystolic murmur with its seat of greatest intensity near the normal site of the apex-beat; (*c*) the murmur is distinctly localized, is not well transmitted, and terminates abruptly with a systolic shock; (*d*) there is marked accentuation of the second pulmonic sound, in striking contrast to the second aortic sound, which is usually feeble and at times absent.

Clinical Course.—In a fair percentage of cases mitral stenosis induces mitral regurgitation, hence the clinical course is that of the combined lesions (p. 282). There are exceptional instances, however, in which, after dilatation of the left ventricle has taken place, the calcareous degeneration at the margin of the mitral ring prevents dilatation of the orifice. The deformity of the mitral valve leaflets, however, permits regurgitation to continue.

TRICUSPID REGURGITATION.

Pathologic Definition.—A condition, rarely primary, characterized by—(*a*) Inflammatory changes, with shortening and coiling of the tricuspid leaflets; and (*b*) imperfect closure of the tricuspid valve (secondary), as the result of marked dilatation of the right ventricle, which is secondary to disease of the left heart or to obstruction offered to the arterial circulation through the lungs.

Mechanic Influence of the Lesion.—In a case of well-marked tricuspid regurgitation each systole is accompanied by a reflux of venous blood from the right ventricle through the tricuspid orifice into the right auricle, and if the power of the right ventricle is sufficient, this regurgitated blood is forced into both the ascending and the descending vena cava. As a result of the backward wave being carried into the venæ cavæ pulsation of the veins in the carotid region is seen and felt, and in cases of prolonged duration, the liver may pulsate with each cardiac systole—the so-called "pulsating liver of tricuspid regurgitation." (See Disease of the Liver.) On first consideration it appears clear that, owing to engorgement of the veins of the greater circulatory system and to leakage at the tricuspid orifice, the pulmonary circulation should receive less blood than under normal

conditions, but this does not exist for any length of time, hence the lesser (pulmonary) as well as the greater circulation soon becomes embarrassed.

Hypertrophy and dilatation of the right ventricle, with decided engorgement of this chamber, develop in the same way as was previously described for the left ventricle with mitral regurgitation (p. 276). It must be emphasized, however, that the right heart is not capable of hypertrophying to the same degree as is the left, consequently the area of cardiac dullness is not increased to the same extent from hypertrophy of the right ventricle, as it is when the left chamber is thus affected. A factor that induces dilatation as well as hypertrophy of the right heart is that during diastole, as the result of increased venous tension, an abnormal volume of blood is forced into the right ventricle. After the right heart is unable to maintain the pulmonary circulation, dilatation is further encouraged, and an extreme degree of thinning of its wall follows. Dilatation of the right ventricle with tricuspid regurgitation causes obstruction to the circulation through the liver, and, in consequence, effusion into the peritoneum and edema of the mucous membrane of the stomach and of the subcutaneous tissue of the lower extremities and body (general anasarca) follow.

Predisposing and Exciting Factors.—These are: (1) **Congenital deformity of the tricuspid orifice;** (2) **endocarditis** involving the leaflets or the endocardium at the tricuspid orifice; (3) **secondary regurgitation,** which includes by far the greater portion of all cases, is induced directly by increased tension and eventually by dilatation of the right heart. In this variety disease of the mitral, aortic, or pulmonary valves must in turn favor, and in the majority of cases induce, tricuspid regurgitation.

Principal Complaint.—Following the development of tricuspid regurgitation, those sections of the body that are drained by the portal circulation become the seat of passive congestion when the patient complains of the following: *anorexia, nausea, occasional attacks of vomiting,* and at times of the vomiting of blood. There is a sense of weight in the upper portion of the abdomen, and the patient may have observed that his abdomen is enlarging, and that his clothing is becoming extremely tight. *Hemorrhoids,* with the passage of blood and blood-stained stools, are likely to occur early. Swelling of the feet and intense itching in the region of the ankles are also experienced. *Dyspnea* is a prominent symptom throughout the entire period following the development of tricuspid incompetency, and may be so severe as to necessitate propping the patient up in bed or sleeping in the sitting posture.

Following the effusion of serum into the pleura and peritoneum, the symptoms just detailed are appreciably aggravated. In certain selected cases *ascites* does not develop until some time after rupture of compensation, owing to the fact that the veins of the liver become enormously distended, and in this way the liver forms a reservoir for the regurgitated blood and thereby protects the peritoneum.

Physical Signs.—Inspection.—Venous pulse in the neck is a characteristic sign of tricuspid regurgitation, and this throbbing is distinguished from pulsation of the artery in this region by placing the finger over the vein immediately above the clavicle, and making firm pressure, when venous pulsation disappears above the finger, to reappear after the pressure is removed. Pulsation coming from the artery, on the other hand, is but slightly, if at all, affected by mild pressure. Pulsation in the epigastrium may be pronounced when the patient is in the recumbent or semirecumbent posture. Leube records the time of the venous pulsation of the veins of the neck as presystolic.

Owing to increased venous tension, the blood is retained for an unusually long time in the capillaries, consequently *cyanosis* of the lips, face, finger-nails, and extremities occurs. The area of the apex impulse will be found to vary greatly, depending largely upon the initial cardiac lesion in question and likewise upon the degree of hypertrophy of the left ventricle. After dilatation has become well marked, and in case of mitral stenosis, no impulse may be visible in the region of the apex. Pulsation may be visible at the right of the sternum, and results from contraction of the right auricle and right ventricle, but such pulsation is in no way characteristic of tricuspid regurgitation, and has previously been described in connection with mitral stenosis.

In those cases developing ascites the abdomen is unduly prominent, and the upper portion may be distended in part as the result of enlargement of the liver and of the spleen. The lower extremities become swollen and pit upon pressure, as does also the tissue in the region of the loins late in the disease.

Edematous swelling of the genitalia, and particularly of the prepuce and scrotum, or the vulva, is an annoying late symptom.

Palpation.—A slightly heaving impulse is palpable in the epigastrium, and when the right ventricle is greatly dilated, it may be possible to force the fingers well underneath the left costal margin in the epigastric region, and there feel the heart throbbing distinctly.

When the patient is resting in the recumbent posture, he should be directed to flex the right thigh upon the abdomen, when the examiner, by pressing the finger forcibly against the abdominal muscles and immediately below the lower border of the liver, will find that this viscus pulsates with each cardiac systole—the so-called "pulsating liver." (See p. 286.) Rhythmic pulsation of the liver may also be detected by directing the patient to lie upon the back, with the arms raised; the examiner should then place the palm of his left hand over the right midaxillary region, and that of the right hand over the upper abdominal region. (See Timing of Murmurs, p. 229.)

Caution.—The liver may be depressed with each pulsation of the heart when the right ventricle is well dilated, but such hepatic movement is never expansile in character, a feature that distinguishes it from the so-called pulsating liver. Again, hypertrophy of the liver and new-growths situated between the liver and the abdominal aorta may transmit an impulse from the aorta through the liver to the palpating hand, but here again expanding pulsation is absent and the impulse is most marked over the left lobe, while in pulsating liver the impulse is found over the entire organ, and is always well marked at the junction of the midclavicular line with the costal margin. The tissue of the extremities and even of the trunk and loins may pit upon pressure. The pulse, as shown by the radial artery, is small, arhythmia is conspicuous, and the force of each pulsation will be found to vary greatly.

Percussion.—It is customary to find the area of cardiac dullness markedly increased, and such increase usually extends for an inch or two to the right of the sternum. In those cases in which tricuspid regurgitation follows either mitral or aortic regurgitation, the transverse diameter of the heart is also increased to the left (Fig. 117), and may extend from the left axilla to one or more inches beyond the right border of the sternum. The area of hepatic dullness generally extends below the costal margin, and the spleen is also enlarged. Late during the disease there is flatness at the base of both pleuræ, the result of the presence of transuded fluid in the serous sacs. If ascites is present, tympany will be found to surround the

umbilicus when the patient is in the recumbent posture (Fig. 229), and at this time dullness occupies the flanks, and change of posture changes the location of both the dullness and the tympany.

Auscultation.—The murmur of tricuspid regurgitation is, as a rule, soft and blowing in character, systolic in time, and heard in the region of the ensiform cartilage. (See Fig. 124.) This murmur is ordinarily audible until late during the course of the disease, and, in fact, may be present throughout that period after compensation has failed. The area over which the murmur is best heard varies greatly in different cases, and, of course, also depends upon the loudness of the murmur present. Ordinarily, the systolic murmur is heard for a distance of one to one and one-half inches to the left of the left border of the sternum, and for a somewhat greater distance to the right of the sternum. Cases are occasionally encountered in which the murmur of tricuspid regurgitation is heard two or more inches above the articulation of the ensiform cartilage with the upper two-thirds of the sternum. In those cases in which the heart is unusually weak the murmur is likely to be faint, and its disappearance is suggestive of further myocardial degeneration. Speaking collectively, with reference to cardiac murmurs, that of tricuspid regurgitation is softer and heard with more difficulty than is that resulting from any other cardiac lesion, with the possible exception in selected cases, of the presystolic mitral murmur. The influence of tricuspid incompetency upon the second pulmonic sound is by no means a constant one, consequently the degree of accentuation of this sound is of but limited clinical importance.

Laboratory Diagnosis.—Expectoration may be free, and, rarely, blood-stained sputum is ejected. The *sputum* varies somewhat with the initial cardiac lesion in question. Owing to cyanotic congestion of the kidney, the quantity of urine excreted becomes greatly diminished late in the disease, and the condition may approach anuria. At this time the specific gravity of the urine is high,—1.025 to 1.035,—the color is dark, and on standing it will be found to precipitate a heavy sediment. *Microscopically*, it is rich in leukocytes, granular débris, and, occasionally, red cells are to be seen.

When a *hematologic* study is made before the development of general edema of the viscera and subcutaneous tissue, together with effusion into the serous sacs, the hemic condition will be found to be that of secondary anemia, namely, there will be a proportionate reduction in the hemoglobin and red cells, with but limited changes in the leukocytes. After effusion into the serous sacs has taken place and edema of the subcutaneous tissue and cyanosis of the extremities have appeared, the number of red cells in a cubic millimeter will be found to equal, and often to exceed, that of the normal. We have seen cases of cyanosis in which the red cells have numbered from 5,000,000 to 8,000,000 per c.mm., with a hemoglobin percentage of from 90 to 100. This increase in the red cells and hemoglobin results from the blood condensation following the extravasation of its liquid elements into the intracellular tissue and serous cavities, and in such cases the actual number of red cells, as well as the amount of hemoglobin present in the body, may be lower than normal.

Summary and Differential Diagnosis.—Probably the most significant sign of tricuspid regurgitation is venous pulsation above the right clavicle and in the carotid regions, and pulsation of the liver is also a valuable sign. In the majority of cases a murmur is audible, and when present, is of great value as a diagnostic aid. The location of the murmur, its points of

greatest intensity, and its area of distribution serve to distinguish it from that of mitral regurgitation, which is heard best in the region of the apex, and is always transmitted to the left. (See Fig. 124.) It is to be borne in mind that frequently the murmur of mitral regurgitation and that of tricuspid regurgitation are present simultaneously, and in such cases the existence of these two systolic murmurs is quite easily determined by passing the stethoscope carefully first from the apex of the heart to the left, and in the event of a systolic murmur being audible at the anterior axillary line, mitral regurgitation exists. By passing the stethoscope gradually from the nipple obliquely toward the ensiform cartilage, the murmur of tricuspid regurgitation will become more and more distinct as we approach the ensiform, and will be heard for some distance to the right of the sternal border. (See Fig. 124.)

Clinical Course.—Tricuspid incompetency develops late during the course of other cardiac lesions, and in certain cases may exist for one or more years, although in the majority of instances, after the establishment of well-marked regurgitant currents through the tricuspid orifice at each systole, the clinical picture shows that the case progresses rapidly from bad to worse.

TRICUSPID STENOSIS.

Pathologic Definition.—A rare condition characterized by either a congenital or an acquired narrowing of the tricuspid orifice, venous stasis, and dropsy.

General Remarks.—Tricuspid stenosis is rarely encountered as a primary disease, and is usually seen in association with organic lesions of the mitral region,—a fact well substantiated by the statistics of Daland and McDaniel, who collected 186 cases of associated mitral and tricuspid stenosis, —although it may be found to coexist with aortic regurgitation. The influence of tricuspid stenosis upon the right side of the heart is identical with that previously described for mitral regurgitation and mitral stenosis (see mechanical influence of these murmurs, pp. 276, 283), in consequence of which the right auricle first becomes extremely dilated, to be followed finally by venous congestion. In an endeavor to compensate for the obstruction the right auricle hypertrophies.

Predisposing and Exciting Factors.—A **rheumatic history** is obtained in from 30 to 40 per cent. of all cases. Many authors regard an attempt at compensation for other cardiac lesions as a prominent predisposing factor, and certainly all forms of **endocarditis** favor the development of tricuspid stenosis.

Sex is a prominent factor, since mitral stenosis predisposes markedly to tricuspid stenosis, and compiled statistics of 160 cases of mitral stenosis show that lesion to develop in a ratio of five to one in favor of males.

Principal Complaint.—The symptoms complained of are the same as those previously given under Tricuspid Regurgitation (p. 287).

Physical Signs.—Inspection.—An indistinct venous pulsation of the carotid regions may be present, and a presystolic thrill may be detected over the right ventricle. Owing to undue engorgement of the right auricle there is an increase in the area of cardiac dullness. A presystolic rolling murmur is audible along the right border and over the lower portion of the sternum.

A feature of great clinical importance is that the physical signs of tricuspid stenosis are clear only in those cases devoid of complications and associated cardiac lesions—a class that constitutes a small percentage of all cases. Owing to the frequent coexistence of tricuspid stenosis with other well-marked cardiac lesions (mitral stenosis, aortic regurgitation), it is often

extremely difficult to recognize the stenotic condition with any degree of certainty, and, indeed, definite deductions are frequently impossible.

PULMONARY INCOMPETENCY.

Pathologic Definition.—A rare cardiac condition characterized by incompetency at the pulmonary orifice, the result of either malignant, simple, or chronic endocarditis, localized to the vicinity of the pulmonary orifice. The condition may also be due to congenital malformation.

Remarks.—Pulmonary incompetency exercises an influence upon the right ventricle, *e. g.*, it induces hypertrophy of the ventricular wall and finally dilatation.

Physical Signs.—A diastolic murmur may be audible at the second left interspace, and when well marked, is transmitted along the sternum, resembling in this respect the area of distribution for the murmur in aortic regurgitation. Practically speaking, the physical signs furnish but little, and ofttimes nothing, that is positively diagnostic of this condition.

Differential Diagnosis.—Pulmonary incompetency is to be distinguished from aortic regurgitation, and this distinction is based upon the following physical findings: (*a*) In pulmonary insufficiency hypertrophy and dilatation affect the right ventricle. (*b*) In aortic regurgitation hypertrophy and dilatation first involve the left ventricle. The so-called "water-hammer" pulse is also characteristic of aortic regurgitation. The various manifestations of Quincke's capillary pulse ascertained by inspection are significant features in connection with regurgitation at the aortic orifice. (See Aortic Regurgitation, p. 268.)

PULMONARY STENOSIS.

Pathologic Definition.—A condition characterized by narrowing of the pulmonary orifice, cyanosis, and distention of the systemic veins.

Remarks.—Pulmonary stenosis is not infrequently of congenital origin. Endocarditis may lead to stenosis at the pulmonary orifice. In a fair proportion of all cases of pulmonary stenosis developing after adolescence and in the aged the condition may be attributed to general atheroma. The mechanism of the lesion is such as to produce hypertrophy of the right ventricle, which may, in turn, be followed by dilatation, which permits tricuspid regurgitation to take place. (See Physical Signs and Symptoms of Tricuspid Regurgitation, p. 287.)

Principal Complaint.—The symptoms are not constant in character, and, indeed, may often be misleading until dilatation of the right ventricle occurs, after which the symptoms are similar to those due to tricuspid regurgitation. (See p. 287.)

Physical Signs.—Palpation.—In certain cases a systolic thrill is felt over the base of the heart.

Percussion.—The area of cardiac dullness is markedly increased to the right and upward, and after the right ventricle has become dilated, dullness may extend well into the epigastrium.

Auscultation.—A harsh, clear, systolic murmur is audible at the second intercostal space, although it is usually heard distinctly at the third left interspace near the margin of the sternum (Fig. 61). The systolic murmur of pulmonary stenosis is transmitted upward along the sternal border and for a short distance to the left.

Differential Diagnosis.—It is always necessary to distinguish between the murmur of pulmonary stenosis and that of aortic stenosis,

the latter being heard best at the aortic valve, and being always transmitted to the vessels of the neck, whereas the murmur of pulmonary stenosis, even though it be heard at the aortic cartilage, is not well transmitted to the aforesaid vessels. In pulmonary stenosis the second pulmonary sound is, as a rule, feeble, and there may be an associated diastolic murmur, which suggests pulmonary regurgitation. Generally, a diagnosis of pulmonary stenosis should not be made until the heart has been repeatedly examined and the patient's general condition carefully considered, since a systolic murmur heard at the base of the heart is common during the course of maladies in which secondary anemia is a prominent symptom.

COMBINED FORMS OF CARDIAC DISEASE.

The accompanying statistics are taken from a table compiled by J. F. Smith, and furnish the only means of determining the possible frequency with which certain combinations of valvular defects are to be found:

Aortic stenosis and mitral regurgitation	16.55 per cent.
Aortic stenosis and mitral stenosis	6.12 " "
Aortic regurgitation and mitral regurgitation (common in children)	5.21 " "

Aortic regurgitation lesions with reference to their relative frequency are as follows:

Aortic regurgitation and aortic stenosis and mitral regurgitation occupy first place in the category of combined lesions.

Mitral stenosis and mitral regurgitation have, in our experience, appeared to be extremely common.

Aortic stenosis and mitral stenosis, the one predisposing to the development of the other, come third in the list.

Tricuspid incompetency may be secondary to mitral disease, and, in fact, frequently develops late during the course of other cardiac lesions, this being especially true after cardiac dilatation has developed.

Physical Signs.—A systematic analysis of the different murmurs, their area of greatest intensity and of distribution, will in many instances enable one to determine with a fair degree of certainty the nature of the lesion or lesions actually present. In quite a large percentage of all cases in which arhythmia with decided irregularity in the force of the heart's action (myocarditis) is present, the evidence obtained by auscultation is confusing, rather than confirmatory.

COMPLICATIONS OF VALVULAR DISEASE.

In discussing the various forms of endocarditis and valvular disease, the accidents and complications most likely to occur have been considered. It may be well, however, again to call special attention to the following:

(*a*) Acute endocarditis, which frequently develops during the course of chronic cardiac lesions; (*b*) acute pericarditis, which is of much less common occurrence; (*c*) pleurisy the result of direct extension from the pericardium; (*d*) pulmonary complications, particularly pneumonia, pulmonary embolism, thrombosis, and atelectasis; (*e*) nephritis, which is often precipitated by prolonged cyanotic congestion of the kidneys; (*f*) emboli in various parts of the body; (*g*) epilepsy and insanity, conditions that develop in those cases in which there is associated extensive atheroma, involving particularly the cerebral arteries; (*h*) angina pectoris (see p. 302); and (*i*) erysipelatous processes, attacking most often the lower extremities after there has been extensive edema, with possible rupture of the skin.

CARDIAC THROMBOSIS.

Pathologic Definition.—A condition characterized by the formation of thrombi within the chambers of the heart, particularly in the right auricle, and less often in the right ventricle, such thrombi being secondary to both disease of the endocardium and to degenerative processes in the blood. Cardiac thrombosis may be suspected during life, but is seldom if ever diagnosed by clinical methods.

DISEASES OF THE MYOCARDIUM.

HYPERTROPHY OF THE HEART.

Pathologic Definition.—Increased thickening of the muscular portion of the whole or a part of the cardiac wall.

Remarks.—Cardiac hypertrophy may exist without any appreciable degree of dilatation of the chambers being present,—the so-called "simple hypertrophy,"—but it is far more common to find cardiac hypertrophy associated with a variable degree of enlargement (dilatation) of the chambers —the so-called "excentric hypertrophy." At autopsy the wall of the heart may appear to be decidedly thickened, whereas the chambers are appreciably diminished in size—a condition that develops after death, and is of no value from a diagnostic standpoint.

Varieties.—(1) Left ventricular hypertrophy; (2) right ventricular hypertrophy; (3) auricular hypertrophy.

HYPERTROPHY OF THE LEFT VENTRICLE.

Remarks.—A condition that results from mitral incompetency, aortic incompetency, and aortic stenosis, all of which conditions increase intraventricular pressure.

The work of the heart may be increased by any condition that offers obstruction to the circulation or interferes with the heart's action; consequently hypertrophy results from pericardial adhesions, general arteriosclerosis, such as is seen in gout, chronic rheumatism, lead-worker's disease, chronic syphilis, chronic nephritis (interstitial), congenital deformities of the aorta and of the heart, aneurism, and obstruction to the circulation through the liver—*e. g.*, atrophic cirrhosis. Again, ventricular hypertrophy frequently develops during the course of exophthalmic goiter, and doubtless results from persistent tachycardia.

Habit figures prominently as a predisposing factor, since those addicted to the excessive use of tea, coffee, and alcoholic spirits are especially likely to develop this condition. Overeating exercises an appreciable influence. Severe muscular strain, continued over several hours, or repeated daily for prolonged periods, is practically always followed by left ventricular hypertrophy, as is shown by the hearts of athletes and stevedores.

Principal Complaint.—Cardiac hypertrophy may have existed for many years without causing any inconvenience, and in many instances rupture of compensation gives rise to the first discomfort referable to cardiac disease. In typical cases, however, peculiar precordial symptoms, such as a "*fullness*," are experienced. Rarely, the patient complains of a *mild precordial pain* and of cardiac palpitation, except when hypertrophy develops in a person of neurasthenic temperament, or in one addicted to the excessive use of narcotics and stimulants. When hypertrophy of the left ventricle is due to the latter cause, headache, throbbing of the temporal

regions, periodic attacks of flushing of the face, vertigo, epistaxis, tinnitus, aurium, and flashes of light are generally present.

Among the accidents that may occur during the course of hypertrophy should be mentioned cerebral hemorrhage, a condition that develops only in those cases in which there is well-marked arteriosclerosis.

Physical Signs.—**Inspection.**—In children the precordium is unusually prominent, and in the adult, a mild grade of prominence is not uncommon. The area of cardiac impulse is extensive, and the impression is forcible. Cases have been seen in which the greater portion of the anterior surface of the left half of the chest displayed a heaving impulse with each systole. There may also be visible pulsation in the left supraclavicular space, and, rarely, the impulse is extended to the epigastric region.

Palpation.—A heaving impulse is felt in the region of the apex, and is always palpable unless dilatation has developed conjointly with hypertrophy, and exceeds the hypertrophy at the time of examination. The apex-beat is always felt below the fifth interspace and outside the midclavicular line. When the degree of hypertrophy is moderate, the apex impulse will be found in the sixth interspace, and one to two inches to the left of the midclavicular line.

During the time when the hypertrophy exceeds the dilatation the *pulse* is strong, large, regular, of increased tension and of about the normal frequency. When dilatation equals or exceeds hypertrophy, the pulse is markedly softened, compressible, especially when the hand is elevated above the body, irregular in force and volume, and more frequent than normal.

Percussion.—The area of cardiac dullness is increased downward and to the left. If the patient is seen after dilatation has taken place, the area of dullness would then be greatly increased in the transverse diameter.

Auscultation.—In a case of simple cardiac hypertrophy (hypertrophy without a valvular lesion) no murmurs are audible over the heart, but the first sound is unusually dull, appreciably prolonged, and is frequently referred to as booming in quality. The sound due to closure of the aortic valves is well accentuated, and has a clear, ringing quality. Reduplication of the second sound is often present. Late during the course of hypertrophy the quality of the heart's sound often changes and becomes somewhat clicking. After well-marked dilatation has taken place, the first sound of the heart is greatly shortened, and its original characteristics are lost—indeed, the two sounds of the organ become more and more alike.

HYPERTROPHY OF THE RIGHT VENTRICLE.

Remarks.—A condition often induced by mitral incompetency, chronic interstitial nephritis, pneumonia, emphysema, extensive pleural adhesions, and practically all pathologic conditions that increase circulatory resistance through the lung. Organic heart disease—*e. g.*, pulmonary stenosis—is also followed by right ventricular hypertrophy. Organic disease of the left side of the heart eventually increases the blood tension in the lung, and thereby encourages hypertrophy of the right ventricle. (See Mitral Regurgitation and Mitral Stenosis—Mechanisms of the Lesions, pp. 277, 283.)

Symptoms and Signs.—There are no symptoms during the stage of compensation, except possibly a moderate grade of *dyspnea*, which follows extreme muscular exercise and is usually accompanied by precordial discomfort and cough, the latter being more pronounced when ventricular hypertrophy is induced by emphysema or chronic interstitial changes in the lungs.

After dilatation of the right ventricle has occurred, tricuspid regurgitation develops, and is followed by *cough*, with the signs and symptoms of *pulmonary edema, hemoptysis, dyspnea, cyanosis*, and other symptoms referable to venous stasis. (See Tricuspid Regurgitation, p. 287.)

Physical Signs.—**Inspection.**—In a small percentage of all cases there is an appreciable bulging of the lower portion of the sternum at the sixth or seventh left costal cartilages. A diffuse impulse is seen over the lower portion of the sternum, and extends well over the epigastrium; there may also be pulsation as high as the third or fourth interspace, and along the right border of the sternum, the latter sign depending upon the degree of ventricular dilatation present. The apex-beat is seen to the left of the nipple-line, but is seldom displaced downward. The cardiac impulse in the region of the apex is forcible and may be somewhat diffuse.

Palpation.—Pulsation is detected over the same areas previously mentioned under Inspection. Prior to the development of dilatation the force of the apex-beat is greater than normal, but after dilatation has occurred it is weak, quite diffuse, and may be almost imperceptible. (See Cardiac Signs of Emphysema, p. 128.) During the time compensation is maintained the volume of the *pulse* is not above normal, and in many instances it is small. After dilatation has taken place arhythmia becomes a prominent feature, and, as a consequence, the pulse is irregular in volume, force, and frequency. (See Arhythmia, p. 194.)

AURICULAR HYPERTROPHY.

Remarks.—A condition that develops conjointly with dilatation. The left auricle is so affected in mitral regurgitation and in mitral stenosis. Right auricular hypertrophy is a feature of all pathologic conditions in which there is increased blood-pressure in the lungs. It also develops secondarily to incompetency or stenosis of either the tricuspid or the pulmonary orifice.

Physical Signs.—These are indefinite until dilatation occurs. Hypertrophy or dilatation of one or the other ventricle soon follows or develops prior to, or simultaneously with, auricular hypertrophy.

Inspection.—A presystolic impulse is seen in the second interspace. A presystolic wave of pulsation is seen over the base of the heart, and is particularly well marked along the sternal borders, at the third and fourth interspace. Jugular venous pulsation, systolic in time, may form one of the most conspicuous signs. (See Tricuspid Regurgitation, p. 287.)

Percussion.—Cardiac dullness may extend to the left of the sternum in the second or third interspace when the left auricle is hypertrophied.

Right auricular hypertrophy and *dilatation* coexist and cause extension of the area of cardiac dullness to the right of the sternum and in the third and fourth interspaces.

Differential Diagnosis.—Cardiac hypertrophy is to be distinguished from **thoracic aneurism,** although it is to be borne in mind that these two conditions are frequently present in the same individual, hypertrophy resulting from the conditions that permitted of an aneurismal expansion of the artery (arteriosclerosis). On examining the radial pulses, one pulse may be found of low tension—a sign that is absent in simple cardiac hypertrophy, the pulse here being full, bounding, and strong, the radial pulses being equal as to time and volume, with an increase in arterial tension. Pulsation of the precordium due to hypertrophy is limited to that portion lying between the apex of the heart and the third and fourth costal cartilage at their articulation with the sternum, pulsation being found to the right of

the sternum after cardiac dilatation ensues, whereas the pulsation due to aneurism is more expanding and less heaving in character, and seldom, if ever, extends outside the left midclavicular line, where the pulsation of hypertrophy is seen.

Cardiac hypertrophy may be confused with **cardiac dilatation,** and differentiation is especially difficult in those suffering from emphysema, owing to the fact that the area of cardiac dullness is not increased, because the heart is covered by emphysematous lung. The distinctive feature, however, that marks cardiac dilatation is a weak, rapid, irregular pulse that is readily compressible. In **dilatation** *dyspnea, cough,* and *cyanosis* of the face, mucous surfaces, and extremities are present. The muscular element of the first sound of the heart is absent in dilatation, whereas in hypertrophy it is normal or exaggerated.

MYOCARDITIS.

Acute Myocarditis.

Pathologic Varieties.—(*a*) Acute parenchymatous myocarditis, which is characterized by granular degeneration of the muscle-fibers of the heart, with numeric increase in their nuclei; later fatty degeneration may occur as the terminal stage of this condition.

(*b*) Acute diffuse interstitial myocarditis, in which the interstitial tissue of the myocardium is chiefly attacked, a variable degree of round-cell infiltration being present.

(*c*) Acute circumscribed myocarditis, characterized by degenerative processes that progress to form isolated areas of necrosis terminating in abscess formation.

Predisposing and Exciting Factors.—Sex.—Males are more often attacked than females.

Endocarditis and **pericarditis,** irrespective of the conditions that may have induced them, serve as potent factors in the production of pathologic changes of the myocardium. Simple **rheumatic myocarditis** is said by some writers to exist without the presence of either pericardial or endocardial disease. Myocardial degeneration may develop during the course of the acute specific fevers—*e. g.,* typhoid fever, diphtheria, etc. Septic processes in any portion of the body may furnish infectious emboli that plug the minute branches of the coronary arteries, and thereby produce acute circumscribed myocarditis, this pathologic change being most commonly seen in septicemia, pyemia, ulcerative endocarditis, and puerperal sepsis.

Principal Complaint and Symptoms.—These are, as a rule, negative with reference to disease of the myocardium, although symptoms referable to enfeeblement of the heart may be described, as is shown by attacks of *cardiac palpitation, syncope,* and *dyspnea* following slight exertion. Repeated attacks of vomiting with a tendency toward faintness should always arouse suspicion as to the existence of myocarditis.

Physical Signs.—Inspection.—Pallor is likely to be present, and upon slight exertion becomes extreme. Late in the disease the evidences of venous stasis are present—*e. g.,* cyanosis of the lips, finger-tips, nails, and extremities. There may be multiple abscesses of the skin, and venous pulsation of the carotid region is a late sign.

Palpation.—The action of the heart is always feeble, and frequently it is rapid and decidedly irregular. (See Extrasystole, p. 195.)

The *pulse* is weak, diminished in volume, readily compressible, and in well-marked cases the radial pulse may be imperceptible when the hands are

elevated high above the head. Irregularity as to time, force, and volume constitutes the characteristic feature of this disease.

Percussion.—Percussion is likely to reveal the evidences present in cardiac dilatation. (See p. 300.)

Auscultation.—During the course of acute myocarditis it is possible to obtain murmurs both over the body of the heart and at the various areas to which they are transmitted, but the character of the murmur or murmurs present is in no way diagnostic of myocardial change. The sounds of the heart show great irregularity as to both time and volume. The peculiar normal muscular element of the first sound of the heart is lost, and the first and second sounds approximate each other more closely than in health.

Chronic Myocarditis.

Pathologic Definition.—A condition characterized by either diffuse or localized areas of pathologic change (degeneration) in the cardiac muscle, affecting most often the wall of the left ventricle, the septum, and also the papillary muscles, although any portion of the heart muscle may be involved.

Exciting and Predisposing Factors.—**Age** and **sex** serve as prominent predisposing factors, chronic myocarditis being far more common after middle life and affecting males more often than females.

The **excessive use of stimulants,** such as alcohol and tobacco, serves as a marked predisposing factor. Those suffering from such diseases as **diabetes, nephritis, rheumatism, syphilis,** and **malaria** are also especially likely to develop this disease. Certain **toxic substances,** such as lead (lead-worker's disease—see p. 530) may induce myocarditis. The myocardial tissue may become involved by direct extension from the endocardium and from the pericardium.

Symptomatology.—Cases have frequently been seen at autopsy in which extensive myocardial changes were found, and yet these cases exhibited no symptoms referable to the condition during the course of the disease. "The symptoms, when present, are, almost without exception, untrustworthy for diagnostic purposes, since they bear a striking resemblance to those of the organic valvular diseases, minus their more characteristic physical signs. Among the earliest phenomena that point merely to failing heart power are *dyspnea,* and sometimes, also, on exertion, *palpitation* and a sense of *heaviness* or *constriction* in the precordium. The patient suffers from marked *general debility,* and becomes fatigued in consequence of the slightest physical exertion" (Anders).

When chronic myocarditis develops in the aged, one of the earliest symptoms is *extreme irritability;* as the disease advances mental enfeeblement is a frequent accompaniment, and chronic mania may develop. *Asthmatic attacks* are common, and extreme intestinal irritability, nausea, vomiting, and anorexia are generally present.

Pain in the region of the precordium is frequently experienced, and true angina pectoris may develop. (See p. 302.) *Palpitation* may be an annoying symptom, developing early and becoming more and more marked with the progress of the disease, until, finally, an attack follows the slightest exertion. *Vertigo, marked oppression,* and even *attacks* of *syncope* are often experienced, and the patient generally suffers more after the ingestion of a full meal than when the diet is carefully regulated. *Pseudo-apoplectic seizures* are common, and there may be *localized paralysis,* which disappears within the course of a few days or weeks, when other groups of muscles are likely to be attacked. Extensive cerebral hemorrhage is a disaster to be

feared during the entire course of chronic myocarditis, and when it occurs the signs and symptoms of apoplexy will be apparent.

Physical Signs.—Inspection.—Inspection is negative until late during the course of the disease, when, owing to cardiac dilatation, pulsation in the epigastrium and in the carotid region, and probably also in the large area of the precordium, may be evident. The cardiac pulsations are often greatly diminished in frequency, only 50, and possibly not more than 30, occurring in a minute. General cyanosis may develop, and swelling of the extremities due to edema is common.

Palpation.—The impulse of the apex-beat is feeble, though often quite diffuse, and the action of the heart is decidedly irregular. (See Sinus Irregularity, p. 194; also Extrasystole, p. 195.) In typical cases the pulse is slow—30 to 60 beats a minute—whereas during the early stage of the myocardial changes the pulse may be of normal frequency (Fig. 127). When fatty degeneration of the cardiac muscle is well marked, the number of pulse-beats may be normal or even in excess of the normal. A characteristic of the pulse is that it is always irregular as to both frequency and volume. By elevating the hands slightly and making moderate pressure

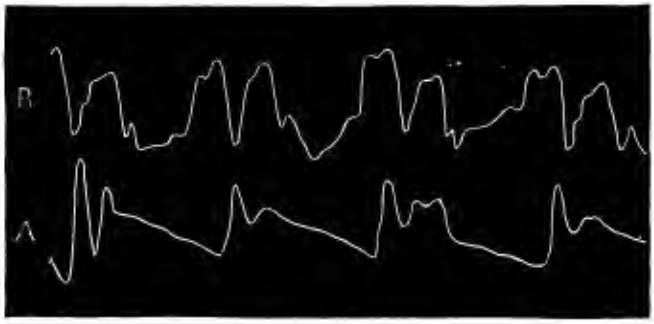


FIG. 127.—ILLUSTRATIVE OF RELATION EXISTING BETWEEN RADIAL PULSE (A) AND APEX BEAT (B), AS SHOWN BY SIMULTANEOUS TRACING.
Radial pulse, 48 per minute.

the pulse is obliterated. The pulse-wave is noticed to be lacking in force when it meets the palpating finger. The radial arteries are atheromatous.

Following cardiac dilatation the liver becomes enormously enlarged, and its lower border is felt well below the costal margin. In selected cases the spleen may be palpable.

Percussion.—Percussion elicits a moderate extension of dullness to the left. After cardiac dilatation develops, the area of cardiac dullness is increased to the right, and may extend for some distance beyond the right border of the sternum. The area of both liver and splenic dullness may also be increased. If a transudation of serum into the pleural sacs and into the peritoneum occurs, the physical signs are those of hydrothorax (see p. 135) and of ascites.

Auscultation.—During the early stage of chronic myocarditis the heart-sounds are fairly strong, and, comparatively speaking, clear, a condition that obtains as the result of hypertrophy of the remaining healthy cardiac muscle. Later during the disease, in those patients in whom the heart can be auscultated from time to time, an appreciable weakness in the heart-sounds will be found to progress with the disease, until later the sounds are those of cardiac dilatation. (See p. 300.) After dilatation takes place

cardiac murmurs may be present, and of these, a systolic murmur at the apex is most common; before the chambers of the heart are well dilated. Murmurs, however, are unusual. Owing to the high grade of arhythmia that obtains late during the course of chronic myocarditis, extreme irregularity as to force, loudness, and rhythm of the heart occurs, and we have the condition known to American writers as "the gallop rhythm," or the so-called "canter murmur."

Laboratory Diagnosis.—An associated *bronchitis* is somewhat common, and in consequence of this the expectoration may be free and occasionally stained with blood. In those suffering from chronic myocardial changes it is quite common to find an associated chronic nephritis, and in such cases the *urinary findings* are those of the type of nephritis in question. Irrespective of the existence of a true nephritis, late during the course of myocarditis the urine is likely to be scanty, of deep color, and of high specific gravity, and a feeble reaction for albumin is a fairly common finding.

Clinical Course.—Chronic myocarditis always progresses from bad to worse, and will be found to vary greatly as to the time of its duration. The conditions that largely govern the duration of this disease are associated renal or hepatic affections. Those cases due to luetic infection may, after the institution of appropriate treatment, terminate in recovery, but practically all cases in which the etiologic factors are uncertain show but moderate, if any, improvement as the result of treatment, but continue from bad to worse.

FATTY HEART.

Pathologic Definition.—This term is applied to three distinct pathologic changes affecting the heart: (*a*) *Fatty degeneration*, a condition characterized by fatty changes in the cardiac muscle-fibers; (*b*) *fatty overgrowth*, which is characterized by an abnormal quantity of fat deposited immediately about the heart; and (*c*) *fatty infiltration*, a condition in which fat-cells are deposited in the cardiac tissue and encroach upon the muscle-fiber.*

General Remarks.—Fatty overgrowth is detected in those suffering from obesity, in whom, also, the signs and symptoms of well-marked cardiac involvement are present. (See Chronic Myocarditis, p. 297.) In fatty degeneration both the symptoms and the signs are practically the same as those observed in chronic myocarditis, and it is only with extreme difficulty that this condition is distinguished from myocarditis.

Diagnosis.—In the majority of instances a positive diagnosis of fatty degeneration of the heart is not made antemortem, and the physical signs may be obscure.

DILATATION OF THE HEART.

General Remarks.—In cardiac dilatation the essential feature is enlargement of the chambers of the heart, although the thickness of the wall of each individual chamber may be greatly diminished. When both cardiac hypertrophy and dilatation develop simultaneously, the wall of the ventricle may be greatly thickened, and yet the cavity contain a larger volume of blood than under normal conditions; this constitutes the so-called "hypertrophy with dilatation."

Pathologic Varieties.—Dilatation with Thinning of the Wall of the Heart.—The wall of the heart is not greatly thinned, but the chamber

*See "A Contribution to the Study of Fatty Infiltration of the Heart Secondary to Subpericardial Overfatness," Amer. Jour. Med. Sciences, by J. M. Anders.

surrounded by such wall is markedly increased in capacity. This variety of dilatation is commonly encountered in the late stages of acute infectious fevers. If the size of the cavities of the heart becomes greatly augmented, the thinning process goes on until the cardiac wall may be only one-half the thickness normally seen.

Dilatation with but Slight Alteration in the Cardiac Wall.—Certain writers recognize this as a special type of cardiac dilatation. It occurs when the heart-wall is of normal thickness, but, owing to increased tension, a moderate amount of thinning follows.

Dilatation with Hypertrophy.—This occurs when there has been a progressive increase in the thickness of the heart muscle and in the capacity of the chambers, the two conditions developing simultaneously. After hypertrophy has reached a certain limit, the muscle fails to receive the proper nutriment demanded for further hypertrophy and needed to maintain a healthy condition in the already hypertrophied muscular tissue; consequently, at this time in the disease, a degenerative process begins. In the so-called excentric hypertrophy, the cardiac cavities contain a much larger volume of blood than under normal conditions, but embarrassment of the circulation is prevented by the increased action of the heart afforded by the associated cardiac hypertrophy. There is a difference between the type of dilatation previously described and the so-called dilatation with hypertrophy, yet the former condition usually merges into the latter, the size of the cavity now being out of proportion to the thickness of the heart's wall.

Varieties.—These are divided according to their etiology. There are two essential factors that figure in the production of cardiac dilatation: (*a*) Increased endocardial (pressure) tension; and (*b*) diminished resistance on the part of the cardiac wall. For convenience of study it is essential that we divide cardiac dilatation into two clinical forms,—primary and secondary,—and of these, the latter is the more important.

Primary dilatation may be expected to follow a recent obstruction to the circulation of sufficient magnitude to overpower the cardiac muscle, or at least to demand a rapid compensatory hypertrophy of such muscle. Acute transitory spasm of the blood-vessels is regarded by Jacob as another factor in the production of this type of dilatation. An increase in the endocardial pressure also figures prominently as a cause, and is best exemplified by the dilatation following the hypertrophy induced by lesions of the endocardium.

Secondary dilatation implies a condition in which cardiac dilatation develops as the result of some preëxisting pathologic condition, *e. g.*, organic heart, liver, lung, and kidney disease.

Acute Primary Dilatation.

Remarks.—This clinical phase of cardiac dilatation is, as a rule, brought about suddenly as the result of violent exertion, which may be coupled with sudden changes in altitude, as, for example, in mountain-climbing. Sudden emotion may produce primary dilatation as the result of contraction of the peripheral vessels, when, for the time, there appears to be a momentary arrest of the heart's action, soon to be followed by palpitation. In acute primary dilatation *cardiac palpitation* is a prominent symptom, and, indeed, the pulsation may often be extended well into the epigastrium, a sign that indicates that dilatation of the right ventricle is present.

This type is usually followed by prompt recovery, especially if the causal factors are removed and sufficient rest follows the initial attack.

Diminished Resistance Offered by the Heart-wall.—Both acute primary and chronic dilatation of the heart are likely to follow any condition that materially weakens the cardiac wall, and these are, indeed, numerous. Chronic myocarditis is a usual forerunner of the so-called chronic dilatation. Acute myocarditis may develop during the course of any of the acute fevers, and is especially common in typhoid fever, typhus fever, malaria, scarlatina, smallpox, diphtheria, rheumatic endocarditis, and pericarditis. In the last-named group of cases acute primary dilatation is believed to be dependent upon the toxic action of certain substances upon the heart muscle. In cardiac degeneration with either fatty or fibroid changes, the heart muscle is greatly impaired, and, as a result, dilatation is to be expected. Chronic gastritis and maladies in which malnutrition is present are potent etiologic factors in the production of chronic dilatation. The anemias, both essential and simple, exhibit cardiac dilatation as one of their clinical features, and, indeed, the area of cardiac dullness is frequently appreciably increased in subjects when the degree of anemia is not alarming.

Symptomatology.—If cardiac dilatation develops gradually, the symptoms are identical with those described for cardiac failure with loss of compensation. (See Aortic Regurgitation, p. 267.)

In acute dilatation the symptoms develop somewhat suddenly, and consist of dyspnea, palpitation, precordial oppression, and pain, and the patient may exhibit great anxiety and fear.

Physical Signs.—Inspection.—Sooner or later during the course of either chronic or acute dilatation a variable degree of cyanosis of the lips, ears, and extremities will be found to develop. Edema of the lower extremities often follows, especially in the chronic forms.

The pulsation in the region of the apex of the heart may be feeble or even absent, but if the patient is examined during an attack of palpitation, the precordial area, as well as the epigastrium, may pulsate violently. A characteristic feature of the impulse in dilatation is that it is extensive, although often indistinct.

Palpation confirms inspection with reference to cardiac pulsation, and shows the respiratory movements of the chest to be increased in frequency.

Percussion.—The area of cardiac dullness is appreciably increased. this increase being in direct relation to the degree of dilatation. In well-marked cases the heart may be found to extend from one to two inches to the right of the right border of the sternum and to the left axillary space. Cardiac dullness is increased more markedly in the transverse than in the vertical diameter, and patients have frequently been seen in whom the transverse area of the heart varied between eight and twelve inches.

Cyanotic congestion with enlargement of the liver is likely to follow cardiac dilatation, and in such cases the area of hepatic dullness is increased, and the signs of fluid in both the pleural and peritoneal cavities may be present. (See Ascites, p. 567; Pleurisy, p. 139.)

Auscultation.—The frequency of the heart-beats is usually increased, although there are instances in which, irrespective of the degree of dilatation present, the pulsations of the heart may not exceed 60 a minute. The first sound of the heart is devoid of its booming muscular element, and appears to approximate more nearly that of the normal second cardiac sound. In auscultating before dilatation has become well marked and before a high grade of thinning of the cardiac wall has occurred, murmurs may be audible, but after the wall has become greatly weakened and thin, distinct murmurs are rarely audible.

Ordinarily, the rhythm of the heart is markedly disturbed, and so pronounced is this feature that in many instances it is impossible to time a murmur, even though it is present. Owing to an associated venous congestion of the pulmonary tissues, numerous moist and bubbling râles are heard over the bases of the lungs, and if hypostatic congestion develops, the breath-sounds may be more or less bronchial over these areas. Because of the enormous size of the heart, the lower and posterior portion of the left lung is generally compressed, and in selected cases tubular breathing may be heard over this area.

CARDIAC ANEURISM.

Pathologic Definition.—A condition characterized by aneurismal involvement either of the wall of the heart or of the cardiac valves. Instances have been recorded in which aneurism of the wall of the heart has attained a size almost equal to that of the organ itself.

Predisposing Factors.—Pericarditis, ulcerative endocarditis, myocarditis, and cardiac gummata favor the development of aneurism.

Symptoms.—These are in no way characteristic of aneurism of the heart, and if any symptoms accompany this condition, they are similar to those of myocarditis.

Physical Signs.—These, too, are in no way distinctive of the disease in question, consequently aneurism of the heart is rarely diagnosed antemortem.

CARDIAC RUPTURE.

Pathologic Definition.—A rare accident that develops in the wake of fatty, fibroid or gummatous degeneration of the myocardium. It has also been reported as occurring secondary to cardiac embolism with softening of the heart muscle, as well as following cardiac abscess. In the majority of recorded instances rupture of the heart has followed undue exertion.

The **symptoms** are sudden intense pain in the precordium, precordial oppression, and the usual symptoms that accompany rupture of a viscus and internal hemorrhage, including shock.

Physical Signs.—The pericardium soon becomes filled with blood, when the signs of pericardial effusion (see Serofibrinous Pericarditis, p. 240) are in evidence. Soon after this accident has occurred the pulse is weak, rapid, thready, irregular, and compressible, and, as a rule, becomes imperceptible within a few hours.

Clinical Course.—The majority of cases terminate fatally within from a few minutes to several hours. Rarely, the patient displays grave symptoms for an indefinite period.

ANGINA PECTORIS.

(STENOCARDIA; BREAST-PANG.)

Definition.—Angina pectoris is a disease characterized clinically by paroxysmal attacks of severe pain in the precordium, which radiates to the left arm, shoulder, neck, and back, and which is accompanied by a sense of impending death.

Predisposing and Exciting Factors.—Angina usually attacks those suffering from **arteriosclerosis, cardiac hypertrophy, aortic regurgitation, adherent pericarditis,** or **myocarditis.** Extensive atheroma of the aorta and of the coronary arteries is the usual finding at autopsy, and is generally conceded to be a prominent factor in the production of angina.

Sex is a prominent predisposing factor, males being more often attacked than females. **Age** is not without influence, nearly all cases being seen in those beyond the fortieth year.

Angina may also develop as a sequel of certain of the **infectious fevers,** such as influenza, etc. The prolonged and extensive use of **stimulants,** particularly tobacco, appears to exercise some influence, as do also the habits and customs of the patient, angina frequently developing in those who are subject to undue **mental strain** and to **physical overexertion.**

Varieties.—True Angina.—*The Paroxysm.—Pain* develops abruptly, often following either excitement or exertion, although in certain cases anginoid attacks appear without cause. The pain is excruciating in character, and is usually described as a vise-like, gripping pain, involving more or less completely the entire chest, radiating to the left shoulder, and rendering the body motionless. At times the pain radiates to the fingers. A sense of *numbness* of the hands, fingers, and precordial region is often present. During the entire attack the patient suffers a fear of impending death.

Angina vasomotoria is a type of this condition in which there are extreme pallor of the face and coldness and stiffness of the limbs, owing to spasm of the peripheral vessels; cyanosis of a portion of the head, chest, and body may accompany the attack. This is a precursor of an attack of true angina.

Angina Without Intense Pain.—The so-called "angina (sine) dolore" is an occasional occurrence, and in this type of the affection the chief feature is a sensation of precordial oppression.

Pseudo-angina pectoris is a condition in which paroxysmal pain in the region of the precordium develops in anemic and neurasthenic individuals.

Physical Signs.—Inspection.—The attitude of the patient is distinctly characteristic. If standing or sitting, he inclines slightly forward, holds his chest as though it were fixed in a vise, and fears to move even after the pain has subsided. The face may be extremely pale or of a dusky hue, whereas the skin of the face and extremities is covered with beads of perspiration. The respiratory movements of the chest may be suspended for a short time, and as the attack subsides the respirations become shallow.

Palpation, in addition to confirming inspection with reference to the movements of the chest, elicits the fact that the skin is cold and clammy. The impulse of the heart's action is often regular and of fairly good force. The pulse is likely to be of high tension. After an attack of angina, physical examination may be negative.

Duration of the Attack.—This varies from a few seconds to two minutes, and there are exceptional cases in which the attack has persisted for hours. An attack of angina abates suddenly, and is often followed by vomiting, eructations of gas, and the voiding of an unusual quantity of urine.

Periodicity.—Angina may recur at intervals varying from a few days to several years. In cases of angina vasomotoria and those of angina without severe pain the attacks are rather frequent, following exertion. These forms of angina may antedate true angina by several years.

Illustrative Case of Angina Pectoris.—C. C., female, aged fifty-six years; height 5 feet 3½ inches; usual weight, 126; present weight, 114 pounds.

Family History.—Father died of heart disease at the age of forty-five and the mother of kidney disease at sixty. Three younger brothers are living, one of whom has been a victim of gout for several years, and another now suffers from some form of heart trouble. One sister died of pneumonia at the age of twenty-two. No history of malignancy or of tuberculosis in ancestry.

Previous History.—Patient suffered from the diseases of childhood, but does not recall having had rheumatism. For the past fifteen years she has been somewhat short of breath, although she was not compelled to consult her physician regarding her dysnpea until the present illness. Twelve years ago, when examined for life insurance, she was rejected because of a diseased heart.

Social History.—Married at the age of twenty years, since which time, and until within the past year, her duties have been that of housewife. Five children have been born to her, and all are living and apparently in good health. At the birth of the second child there was extensive laceration of the perineum, and since the birth of her youngest child, now twenty years of age, she has suffered from uterine prolapse. During the past five years the patient has displayed but slight inclination to go out of doors. The appetite was always voracious, and she displayed an unusual desire for meats and starches.

Present Illness.—Six months ago she consulted a physician for extreme dyspnea upon ascending a flight of stairs, and stated that she was unable to walk against a strong breeze without experiencing some discomfort in the precordium. She suffered more or less continuously from a sense of tightness of the head, which she described as resembling a band drawn tightly about the scalp; she further stated that this sensation became more and more annoying when she took an unusual amount of exercise. The appetite was fairly good, although at times she suffered from mild nausea. A peculiar sensation was experienced about the heart as the result of exercise, and frequently extended to the left shoulder, and occasionally to the arm and hand. Attacks of smothering developed more or less periodically, which occurred once or twice during the week. Constipation was frequently obstinate, demanding the administration of laxatives.

For the first ten weeks of her illness she complained merely of a sense of oppression in the region of the heart, and of numbness and tingling of the left arm and hand, which symptoms were excited by exercise or mental excitement. Approximately twelve weeks ago she was seized with a violent cramp-like pain in the region of the heart, which she described as radiating more or less over the entire chest and to the neck; the pain also extended down the left arm to the hand, and there was some numbness of the right hand. During this attack she forbid both the nurse and her friends from moving her, and she occupied a peculiar, though somewhat characteristic, position. (See General Examination.) Pain was severe for but a few minutes, after which it moderated, and one-half hour later, when seen by one of us, she still complained of pain and was in a state of great fear. There was more or less discomfort in the precordial region for a period of three or four days; at the end of twelve days the patient was permitted to leave her bed.

Slight cough was experienced late during the course of the illness, but at no time did it become an annoying feature.

She was unusually nervous, and explained that any strange sound startled her. She also noticed that her heart palpitated violently, even as the result of some slight family disturbance. She claimed that it was impossible for her to sleep upon the left side, because she was kept awake by the constant throbbing of her heart. At times, when palpitation was severe, the patient was unable to sleep during the entire night, and a peculiar feature of this case was that the loss of sleep did not appear to make her drowsy the following day. The third attack of violent pain resulted in a fatal termination.

Physical Examination.—*General.*—The patient's gait is somewhat feeble, and she breathes with the mouth open; the lips and finger-tips are slightly cyanosed, and there is moderate swelling of the ankles. The patient's general attitude is that of depression, and while she acknowledges that she is markedly concerned regarding her health, she also worries considerably with reference to the family and other affairs. During the attack she speaks but a few words, and these are an expression of great fear of impending death. For some minutes, and at times even for hours, after an attack of paroxysmal cramp in the precordium she is afraid to move the body, and particularly to change from the sitting to the recumbent posture.

Local Examination.—Inspection.—During the attack she sits in one position, inclines slightly forward, grasps her knees somewhat firmly with the hands, and refuses to move or permit any one to move her. The face is at first distinctly ashen gray, but in the final attack it was noticed to become cyanosed. The apex impulse of the heart-beat is forcible during the paroxysmal attack. During the intervals between the paroxysmal attacks of pain the apex impulse is feeble, although there is distinct throbbing of the vessels of the right side of the neck. By sitting and placing the arm in the position shown in Fig. 115 there is visible pulsation of the brachial, axillary, and radial arteries.

Palpation confirms inspection, and, in addition, shows the pulse to be quick, of fair volume, and collapsible; when the hand is elevated above the head, a typical Corrigan pulse (p. 269) is found at the radial artery.

Percussion.—The area of cardiac dullness is appreciably increased downward and to the left, while the right heart extends well to the epigastrium and to right of midsternum, signs that strongly suggest that hypertrophy and dilatation have been going on simultaneously.

Auscultation.—The first sound of the heart is lacking in muscular element, and more or less closely resembles the second sound. No distinct murmur is heard at the mitral area, while at the aortic area there is a soft, blowing diastolic murmur, transmitted down along the sternum. A few moist and crackling râles are heard over the base of the lungs.

Laboratory Findings.—The urine is clear, has a specific gravity of 1.015, and is free from albumin and sugar. The hemoglobin is 80 per cent.; red blood-cells, 3,500,000 per c.mm.; white cells, 8200; when stained, the blood shows the features characteristic of a mild secondary anemia. (The high reading of hemoglobin is probably due to cyanosis.)

Course of the Disease.—Within the course of approximately three months after her first visit she suffered from a typical attack of angina pectoris. Four weeks later she had a mild attack of pain in the precordium, and since that time she has had several more or less characteristic seizures, the pains radiating to the left shoulder and to the left arm. There was but moderate loss in weight, and the patient's general nervous symptoms, mental depression, etc., had greatly improved. About six months after the first attack of true angina, while preparing for bed, she was seized with another attack, and died five minutes later.

Summary of Diagnosis.—The age of the patient, together with a history of arteriosclerosis or of cardiac disease, with possible myocardial change, should always be taken into consideration. The sudden development of intense cardiac pain which radiates to the left shoulder, and in severe cases over the entire chest and to the arms, is of great diagnostic importance. In the majority of cases the physician is not present during the attack, hence the patient's description of a fear of impending death should not be overlooked. The duration of the attack is a prominent factor, since the majority of other conditions causing precordial pain are of longer duration than is angina. Attacks of precordial distress (mild pain) upon exertion are, as a rule, precursors of true angina. Vasomotor angina and angina without pain are early features of true angina and are of great diagnostic moment. Inability to walk toward a strong wind is suggestive of some form of angina,

Differential Diagnosis.—The distinctive features between angina pectoris and pseudo-angina pectoris are shown in the accompanying table, as modified from Anders:

Angina Pectoris.	Pseudo-angina Pectoris.
1. Etiology indeterminate, though generally associated with arteriosclerosis (including coronary disease) or aortic regurgitation.	1. The causes are hysteria, neurasthenia, toxic agents, and reflex irritations.
2. Occurs after the fortieth year, usually in males.	2. Occurs after puberty, and usually in hysteric women.
3. Paroxysms precipitated by undue exertion or mental emotion; they are rarely periodic.	3. Paroxysms arise spontaneously, are periodic, and often nocturnal.
4. Pain intensely severe and constricting, its chief center being to the back of the midsternum and toward the left.	4. Less intense pain, more diffuse over the thoracic region.
5. Duration of attack from a few seconds to one or two minutes.	5. From one-half to several hours.
6. Patient silent and body fixed.	6. Restlessness (patient tossing from side to side).
7. Arterial tension increased, as a rule.	7. Unusual.
8. Expression indicates fear.	8. Absent.

Locomotor Ataxia.—The girdle pain and gastric crises of this disease may, in certain respects, resemble angina pectoris. The following clinical features will be of value in the separation of these two conditions: In locomotor ataxia we find the Argyll-Robertson pupil, imperfect coördination, and absence of the patellar reflexes, all of which conditions are absent in angina pectoris. In the crises of ataxia the patient vomits during the pain, whereas in angina pectoris vomiting, when present, occurs after the pain has subsided.

Gastralgia may be of sufficient severity to lead to confusing it with angina pectoris. (See Gastralgia, p. 480.)

Clinical Course.—There is wide variation in the severity and course of true anginoid pain in different persons, and even in the same individual at different times. The milder cases present only precordial oppression and discomfort, with possibly slight precordial pain at intervals. All gradations between the mild type of angina and the severe grip-like, agonizing pain are to be seen. Rarely, the primary attack terminates in a fatal issue, although it is somewhat more common to find the seizures separated by a period of one or more years, and, indeed, a few cases have been studied in which a second attack did not occur, although a period of from three to seven years has elasped since the initial attack.

CONGENITAL AFFECTIONS OF THE HEART.

General Remarks.—Among these are anomalous conditions resulting from arrested development and from fetal endocarditis, although in certain cases it is highly probable that both conditions figure prominently as exciting factors. The variety of lesion most commonly encountered is best exemplified by Holt's analysis of 242 autopsies upon the bodies of children known to have suffered from anomalies of the heart:

Defect in the ventricular septum	149	cases;	only	lesion	in	5	cases
Defect in the auricular septum or patent foramen ovale	126	"	"	"	"	9	"
Pulmonic stenosis or atresia	108	"	"	"	"	6	"
Patent ductus arteriosus	68	"	"	"	"	3	"
Abnormalities in the origin of the great vessels	45	"	"	"	"	0	"
Pulmonic insufficiency	17	"	"	"	"	0	"
Tricuspid insufficiency	6	"	"	"	"	0	"
Tricuspid stenosis or atresia	3	"	"	"	"	0	"
Mitral insufficiency	1	case	"	"	"	0	"
Mitral stenosis or atresia	6	cases	"	"	"	0	"
Aortic insufficiency	1	case	"	"	"	0	"
Aortic stenosis or atresia	6	cases	"	"	"	0	"
Transposition of the heart	2	"	"	"	"	0	"
Ectocardia	1	case	"	"	"	1	case

THE MOST FREQUENTLY ASSOCIATED LESIONS.

Pulmonic stenosis with defect in the ventricular septum	92	cases;	only	lesion	in	20	cases
Pulmonic stenosis with defect in the auricular septum	52	"	"	"	"	8	"
Defects in both septa	82	"	"	"	"	17	"
Pulmonic stenosis and defects in both septa	36	"	"	"	"	21	"

Symptoms.—*Dyspnea* and *cyanosis* are constant and annoying symptoms and *cough* is frequently present. The symptoms are influenced somewhat by the character of the lesion present. It is usually observed that the child does not develop normally, is delicate, and usually suffers from gastro-intestinal disturbances.

Thermic Features.—The temperature is frequently subnormal in uncomplicated cases of congenital cardiac disease.

Physical Signs.—**Inspection.**—A constant and marked sign in congenital heart disease is *cyanosis*. The tint assumed by the skin is variable, it being at one time dusky, at another a deep violet, and, rarely, it is almost black. This discoloration is most noticeable about the lips and mucous membrane of the mouth, nostrils, conjunctivæ, fingers, toes, and lobules of the ears, and, as a rule, is general, though it may be a local condition. The tint may grow less distinct when the child is in perfect repose or sleeping; excitement or efforts at coughing, however, increase the intensity of the discoloration. The cyanotic hue comes on almost invariably during the first week of life. The fingers present a decidedly clubbed appearance (Fig. 121), and the nails are also clubbed and claw-like.

Palpation.—During infancy the impulse is feeble.

Percussion.—The area of cardiac dullness is increased, especially to the right. In older children the area of dulness is only slightly extended to the left.

Auscultation.—A loud systolic murmur is audible at the pulmonary orifice. When the auriculoventricular valves are the seat of endocarditis, the murmur may be apical. In pure pulmonary stenosis the second sound is feeble. In older children the murmurs heard are often loud.

Differential Diagnosis.—The accompanying table shows the chief differential points between congenital and acquired cardiac lesions:

Congenital Lesions.	Acquired Lesions.
1. History of almost constant cyanosis, beginning in the first week after birth.	1. History of endocarditis or of rheumatism or other complaints in which endocarditis occurs as a complication.
2. Slight enlargement of the right ventricle of the heart, chiefly non-progressive.	2. Enlargement marked, frequently involving the left ventricle, and progressive.
3. Loud and musical murmurs audible over upper third of sternum, with small area of transmission upward and to the left; second sound weak.	3. Audible over apex or base; definite large areas of transmission. Second sound frequently accentuated.
4. Deficient bodily development.	4. Good, as a rule.
5. Mental faculties in abeyance.	5. Mental faculties normal.

Clinical Course.—The majority of cases continue for but a few days after birth, and 75 per cent. terminate fatally before the third year. It is unusual for the patient to live until the sixth year, although, rarely, the condition may continue until puberty, and most of such cases display pulmonary stenosis with defective septa.

Complications.—Rarely cerebral abscess develops during the course of congenital malformation, and chronic bronchitis, pulmonary tuberculosis, epilepsy, and neurasthenia may occur as complications.

DISEASES OF THE BLOOD-VESSELS, MEDIASTINUM, AND THYMUS GLAND.

DISEASES OF THE ARTERIES.

ACUTE AORTITIS.

Pathologic Definition.—The morbid changes resemble those seen in acute endocarditis (p. 251), including the ulcerative variety (p. 254).

Predisposing and Exciting Factors.—The causes are not clearly defined, but the condition may follow the acute infections, *e. g.*, typhoid fever, pneumonia, scarlatina, and syphilis. Alcoholism and syphilis may be contributing causes. Various microörganisms have been discovered to be causal irritants. Boinet and Romary have recently shown that, in experimentally produced aortitis, a point of lessened resistance (either from traumatism or other previous arterial lesion) is necessary for the development of the disease.

Clinical Features.—These are both *local* and *general*. Of the former, *diffuse thoracic pain*, with substernal tenderness, following firm pressure and cardiac palpitation, is noted. The pain occasionally assumes the type of true angina pectoris.

Among the *general features* moderate fever is most constant. A positive Wassermann reaction is at times obtained.

Diagnosis.—The diagnosis in all cases is doubtful.

ARTERIAL SCLEROSIS (ARTERIOSCLEROSIS; ARTERIOCAPILLARY FIBROSIS; ENDARTERITIS CHRONICA DEFORMANS; ATHEROMA).

Pathologic Definition.—An overproduction of the connective tissue situated in the various coats of the arteries, but involving, for the most part, the media and adventitia.

Remarks.—Sclerotic changes may attack almost any portion of the arterial system, but it is more commonly seen to involve the aorta, coronary arteries, descending and ascending aorta, and the arteries at the base of the brain. The radials, temporals, iliacs, and femorals are also found to be the seat of atheromatous changes. It is not uncommon to find sclerotic changes in the hepatic, gastric, and mesenteric vessels, but irrespective of the portion of the arterial tree involved, the disease assumes two distinct types: (*a*) diffuse; and (*b*) circumscribed atheroma. There may also be sclerotic changes in the veins, but far more commonly the arteries are found to be thus involved.

Clinical Varieties and their Characteristics.—(1) **Cerebral.**—In mild grades of this type of atheroma, the patients complain chiefly of headache, vertigo, and tinnitus; attacks of syncope, and, at times, slight local paralyses are also present. In a case now under our care the patient has also had two attacks of subconjunctival hemorrhage. When the disease has progressed for some time, and especially in those over the age of sixty, vertigo and melancholia may be among the more prominent features. Temporary aphasia has also been observed.

(2) **Renal.**—Here the essential changes in the kidney are of an atrophic nature, and usually more or less general sclerotic change of the renal arteries occurs. Generally, however, atheromatous changes attack other portions of the circulatory system, but, owing to the change in the renal artery, its lumen

becomes narrowed and the symptoms displayed by the patient resemble closely those detailed under Chronic Interstitial Nephritis. (See p. 675.)

Coronary sclerosis is seen not only in cases of angina pectoris, but may be placarded by epigastric pains after eating, dyspnea, nausea, epigastric fulness, and discomfort.

(3) **Peripheral.**—Those arteries that may be readily palpated—*e. g.*, radial, ulnar, and temporal—display an unusual degree of hardening, and the terminal branches of the arteries likewise display degeneration. Sooner or later the peripheral arteries become incapable of nourishing the tissues, with resulting gangrene.

(4) **Pulmonary.**—Pulmonary atheroma has been referred to in connection with valvular heart disease (see p. 291), and altered blood tension in the lung is believed to induce such changes.

Predisposing and Exciting Factors.—The diffuse form of sclerosis of the arteries is rarely seen in young subjects, and more often attacks robust males during middle life, although the process is frequently continued after the age of fifty, when it commonly attacks both sexes. Luetic infection and the excessive use of alcohol are believed to be potent factors in the production of atheromatous changes of the blood-vessels, and atheroma is also common among lead-workers and those suffering from chronic nephritis, gout, and joint disease. Men who indulge in violent exercise, and particularly those whose work demands the lifting of heavy loads, are also likely to display more or less atheromatous changes in their arteries. Heredity appears to figure in certain selected cases.

Race.—The American Negro appears to be especially likely to suffer from atheromatous changes.

Specific microörganisms, *e. g.*, the malarial parasite, Treponema pallidum, and such **chemic irritants** as alcohol, lead, mercury, etc., doubtless figure prominently in the production of sclerotic changes in the arterial system. Arterial sclerosis and **chronic Bright's disease** often appear to develop simultaneously. **Conditions that materially alter the systemic blood-pressure** may be capable of contributing to the production of atheromatous changes in the general arterial tree, and **emphysematous changes in the lung** in turn favor the development of atheromatous changes in the pulmonary arteries.

Principal Complaint.—The disease may exist for a period of years without giving rise to any inconvenience, and, indeed, many cases go on to autopsy before the real nature of the condition is made apparent. In selected cases the earlier symptoms resemble those of neurasthenia, epigastric fulness, and distress after taking food, combined with progressive failure in health and malnutrition. Palpitation, dyspnea on exertion, precordial constriction, and pain over the heart are among the symptoms.

Thermic Features.—There may be attacks of mild fever.

Physical Signs.—Inspection.—In advanced cases, in which the peripheral arteries are chiefly involved, the course of the radial, brachial, and temporals is quite distinct, and usually tortuous, and these arteries are seen to pulsate. The apex-beat of the heart is forcible, and commonly displaced downward and to the left.

Palpation.—The arteries that can be palpated roll readily under the finger, and at times are wire-like to the touch. The pulse appears to be of high tension, and offers a distinct impression to the palpating finger, but this impression is frequently misleading, owing to the degree of atheromatous change of the arterial wall. The impression felt on taking the radial pulse is not infrequently found to be incorrect when compared with the degree of

blood-pressure. (See Blood-pressure, p. 203.) Certain cases will be found to display a low blood-pressure, whereas in others it is unusually high, the degree of such pressure being influenced, in part at least, by the muscular power of the heart. It is important for the clinician to keep in mind the fact that atheromatous change of the blood-vessels is commonly associated with valvular disease of the heart and with myocarditis; consequently the degree of blood-pressure may not be characteristic of any one of these three conditions. Again, sphygmographic tracings obtained at different times during the course of the disease will be found to differ widely from one another, and certain of these conditions may be due in part to changes in both the cardiac leaflets and the heart muscle.

Percussion.—Owing to the opposition offered to the circulating blood in the blood-vessels, the left ventricle becomes hypertrophied, and, as a consequence, the area of cardiac dullness is increased downward and to the left. Late during the disease myocardial changes may take place, to be followed by cardiac dilatation, in which event the physical signs are those described for this condition. (See p. 299.) The aorta may become markedly dilated during the course of atheroma, and in such cases there is an unusual area of dullness in the upper sternal region.

Auscultation.—The first sound of the heart is loud and somewhat booming in quality, and there is marked accentuation of the second sound. When myocardial changes predominate, the physical signs are those previously outlined under myocarditis. (See p. 296.)

Laboratory Diagnosis.—Sooner or later secondary anemia is likely to occur. Chronic interstitial change in the kidney is frequently associated with disease of the arteries, consequently a moderate amount of albumin and narrow hyaline casts are commonly found in the urine.

X-Ray Diagnosis.—The *x*-ray is of value. (See p. 236.)

Summary of Diagnosis.—Hardening of the arteries, enlargement of the left ventricle, increased arterial tension, and accentuation of the second aortic sound constitute the cardinal features of atheroma. In certain cases, however, the diagnosis may be further supported by the development of apoplexy and acute cardiac dilatation. Mild attacks of vertigo and tinnitus should always be regarded with suspicion.

Course.—The condition usually progresses from bad to worse, although treatment may exercise some influence. The duration of atheroma varies greatly in different individuals.

ANEURISM.

Pathologic Definition.—A circumscribed dilatation of an artery, the walls of which are formed by one or more of the vessel's coats. The aneurismal sac may have encroached upon other structures, and, in turn, have inflicted severe damage upon them.

1. *True aneurism* (aneurism verum; aneurism spontaneum), in which one or more of the coats of the artery form the walls of the tumor.

(*a*) *Dilatation Aneurism.*—1. Limited to a certain portion of a vessel—*fusiform aneurism; cylindroid aneurism.* 2. Extending over a whole artery and its branches—*cirsoid aneurism.*

(*b*) *Circumscribed Saccular Aneurism.*—The common form in the aorta in which there is distention of two or more of the coats, or distention of the adventitia after destruction of the intima and the media.

(*c*) *Dissecting aneurism*, with splitting of the coats to a greater or less extent, and occasionally with the formation of a new tube lined with intimal endothelium.

2. *False aneurism*, following a wound or a rupture of an artery, causing a diffuse or circumscribed hematoma.

3. *Arteriovenous aneurism*, a communication between an artery and a vein, either direct, *aneurismal varix*, or with the intervention of a sac, *varicose aneurism.*

4. *Special forms*, such as *traction aneurism*, *erosion aneurism*, and *parasitic aneurism.**

Miliary aneurisms occur along the course of the cerebral vessels. On the other hand, it is not uncommon to find an aneurism that has developed to enormous size from one of the greater vessels—aorta, innominate, popliteal.

Clinical Varieties.—The older writers called attention to two distinct clinical types of thoracic aneurism, but at the present time this classification has fallen largely into disuse, and the following subdivisions have been recognized:

(1) **Aneurism with symptoms,** in which there are displayed such characteristics as harsh paroxysmal cough, hoarseness, expectoration, dyspnea, pain, and unilateral sweating; in this class of cases definite physical signs are absent.

(2) **Aneurism with physical signs** is a type of aortic or innominate aneurism in which symptoms, if present, are slight, and such signs as tracheal tugging, bruit, expansile pulsation, thrill, and inequality of the pulses and of the pupils are present.

(3) **Thoracic aneurism** displaying certain definite symptoms, together with physical signs distinctive of this malady.

Predisposing and Exciting Factors.—Among the recognized causes are:

Arteriosclerosis.—The same conditions that give rise to arteriosclerosis (p. 309) also tend to bring about aneurisms.

Syphilis.—According to Rauch, syphilis was present in 56 per cent. of 25 aneurisms of the aorta. In Ansperger's series of 37 cases 48.6 per cent. gave a luetic history.

Sudden Strain.—This may be productive of aneurism, particularly in the early stage of arteriosclerosis. In no other manner can the fact that most cases of aneurism occur during the period of greatest bodily activity in men be satisfactorily explained.

Embolic plugging of a vessel, if complete, may cause aneurismal dilatation at the point of obstruction. Infectious emboli, in turn, produce inflammation and softening.

Mycotic Aneurism.—Osler has pointed out that the growth of microorganisms in the wall of the aorta may be responsible for the development of aneurism.

Trauma.—Aneurism may be produced experimentally by traumatism.

Age and Sex.—Aneurism is most frequently seen to occur between the thirtieth and fiftieth years—the period of greatest physical exertion. Males are more frequently affected than females, owing to differences in occupation.

Pupillary Reactions.—Woodroffe concludes that the normal size of the pupil results from a well-balanced action of the various forces, *e. g.*: (1) The third nerve in causing contraction of the sphincter; (2) the sympathetic in stimulating the dilator muscle; (3) the sympathetic inhibition of the sphincter, and at the same time controlling the amount of blood in the blood-vessels of the iris; and (4) the positive action of the elastic fibers of the iris.

* Osler, Modern Medicine, vol. iv, p. 450.

Among the conditions that appreciably alter the size of one or both pupils are the following: Light is the chief stimulus for contraction of the pupil. A number of conditions may be given as capable of causing the pupil to dilate, and among these the following are of clinical value: Sensitive stimuli as touching the skin on the side of the neck, or pulling the hair immediately in front of the ear; the sudden production of loud noises, and the irritation of the skin along the spine. Psychic stimuli frequently cause prompt dilatation of the pupil, the best examples of this being seen where the patient experiences sudden fear, extreme anger, and sentimental excitement. Lastly, the act of deep breathing may be followed by a moderate dilatation of the pupil.

Certain drugs and anesthetics are also capable of altering the size of the pupil, and among the diseases where inequality of the pupil is a conspicuous feature special mention should be made of thoracic aneurism and all other diseases causing pressure upon the thoracic sympathetics. Again in epidemic meningitis, apoplexy, and brain tumor, the size of the pupils is either altered or the pupillary reactions are disturbed. In certain continued fevers, and in maladies where prostration is extreme, the pupils may be unduly dilated. On the other hand, the pupils are unusually small in locomotor ataxia.

ANEURISM OF THE THORACIC AORTA (THORACIC ANEURISM; ANEURISMA AORTAE).

Remarks.—The thoracic portion of the aorta is involved in 75 per cent. of the cases (Anders), and the branches of the aorta and abdominal aorta are affected in about 25 per cent. of all cases. Lyman found that 60 per cent. of all aneurisms located in the thorax develop from the ascending portion of the aorta, and similar statistics have been offered by Hare and Holden, who analyzed 570 cases involving the ascending arch.

Predisposing and Exciting Factors.—See p. 311.

Clinical Remarks and Principal Complaint.—Intrathoracic aneurisms may exist, and, when small, give rise to no symptoms or noticeable physical signs; when, however, they attain any considerable size, they usually excite fairly characteristic physical signs and symptoms, the latter being the result of direct pressure, and hence varying with the seat and direction of the progressive enlargement. In a few instances diagnostic symptoms have been present even in the absence of a detectable tumor or physical signs. Finally, the more characteristic features—including the tumor—may be more or less intermittent.

It is important to note the condition of the neighboring organs upon which pressure is exerted by the aneurism, as well as the symptoms and signs thus produced. Aneurisms of the ascending portion of the arch may compress the vena cava, causing distention of the veins of the head and arms, although in a small number of cases the subclavian may be the only vein compressed, which condition is followed by edema of the right arm. A large aneurism may compress the inferior vena cava and cause edema of the lower extremities. Rarely, the tumor causes erosion of the ribs and sternum. One recurrent laryngeal nerve is quite commonly implicated, giving rise to dyspnea and aphonia.

Aneurisms of the transverse portion of the aorta, when they attain sufficient size, give rise to the most severe symptoms (*aneurism with symptoms*); these are due to the relatively shorter anteroposterior diameter of the chest at this point, in consequence of which great compression of the adjacent tissues takes place. By protruding backward they may exert pressure upon the

trachea, causing paroxysmal cough and dyspnea; or they may make pressure on the esophagus, causing dysphagia; both conditions are common. Pressure upon a bronchus may cause dyspnea, bronchorrhea, and sometimes circumscribed abscess.

The aneurism may grow forward, in which event it lies directly behind the manubrium, which beomes eroded from the pressure and may finally disappear in part or be perforated. In aneurism involving the transverse portion of the arch lateral pressure, toward both the right and the left, is also made, causing displacement of a portion of one or both lungs.

When the descending portion of the arch is affected, pressure is exerted upon the spinal column to the right, and upon the tissues as far as the shoulder-blades to the left. As a consequence of destruction and absorption of the vertebræ, compression of the spinal cord may ensue, in which event the patient suffers intense pain. Pressure may be exerted upon the left bronchus, causing bronchiectasis, with its sequelæ (bronchorrhea, fetid bronchitis, gangrene of the lung). Repeated small hemorrhages with the expectoration of blood may be a precursor of fatal rupture.

Important Diagnostic Symptoms.—*Pain* is constant, and is of two types: (1) That due to direct pressure upon and stretching of the nerves. When aneurism develops suddenly, a brief, excruciating pain is experienced in the upper sternal region, accompanied by a sense of something having given way. In consequence of the constant stretching of the nerves, continuous pain is experienced, and exacerbations occur when the intra-aneurismal pressure is increased. Pressure against the bony structures causes erosion, and gives rise to a continuous boring pain. In latent aneurism pain is absent. Where the aneurism is located near the heart, pain resembling cardiac angina may be experienced. It is possible, however, for an aneurism of fairly large size to exist without giving rise to severe pain, although such cases, in our experience, are uncommon.

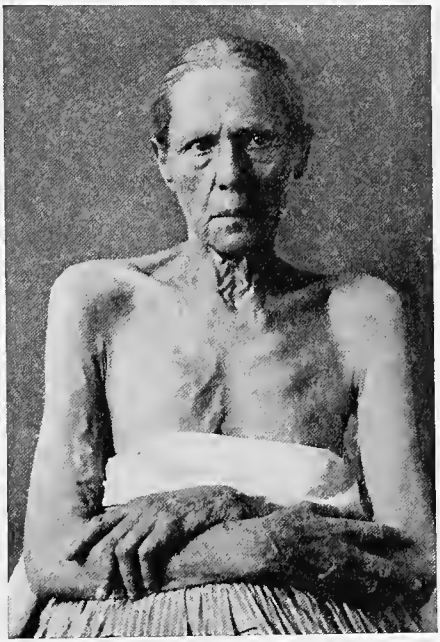


Fig. 128.—Thoracic Aneurism with Dilatation of Veins of Right Side of Chest, Neck, and Right Arm (observation at Philadelphia General Hospital).

(2) Reflected pains of a neuralgic character may be excited by aneurism, a feature that is particularly true of aneurisms situated in the transverse portion of the aorta, in which instances pain is commonly felt in the region of the neck and occiput, and down the left arm. When the growth is situated along the course of the descending aorta, intercostal neuralgia may be excited, and is due to pressure upon the intercostal nerves. In practically all cases in which there is destruction of bony tissue the pain is continuous for weeks or months, and is so severe as to prevent sleep.

Cough.—The cough is paroxysmal, and commonly displays a peculiar metallic, ringing character, that points to its laryngeal origin. Pressure upon the trachea is followed by a paroxysmal dry cough. Compression of a bronchus may lead to bronchiectasis, and the cough is then paroxysmal, recurring at intervals of hours or even days, and is accompanied by copious expectoration.

Dyspnea is a conspicuous symptom in aneurism of the transverse portion of the aorta, and results most frequently from —(1) Pressure by the aneurism upon the recurrent laryngeal nerve, or from entanglement of the nerve among the adhesions surrounding the aneurismal sac; (2) pressure upon the trachea; and (3) pressure on the left bronchus.

Paralysis of the vocal bands results from pressure of the aneurism upon the recurrent laryngeal nerve, the left being the more commonly compressed. Slight pressure or irritation of the recurrent laryngeal is followed by spasmodic contraction of the vocal membrane. Among the symptoms characteristic of involvement of the recurrent laryngeals are hoarseness, aphonia, and a harsh cough. It is essential to employ a laryngoscope in making the diagnosis, since paralysis of certain of the abductors may exist without giving rise to definite symptoms. Laryngoscopic examination serves further to distinguish between hoarseness and cough resulting from aneurism and that produced by tuberculosis of the larynx.

FIG. 129.—THORACIC ANEURISM CAUSING ANTERIOR DEFORMITY OF THE CHEST.

There was marked pulsation, and a bruit elicited over the most prominent portion of the sternum. (Patient studied during service, 1907, at Philadelphia General Hospital.)

Hemorrhage.—This may occur as a mere oozing from the aneurismal sac, and it may escape either into the trachea or through the esophagus. Owing to compression of the lung, a certain amount of bloody sputum may be expectorated. Profuse bleeding is commonly followed by a fatal termination, and in such instances rupture of the sac is found at autopsy. Aneurism may rupture into the mediastinum, pleural sacs, lung, trachea, or a bronchus.

Dysphagia.—In those cases in which sufficient pressure is exerted upon the esophagus there may be difficulty in swallowing. The symptoms of stricture of the esophagus (see p. 415) may also be present, and in such cases it is highly essential that the clinical features distinctive of both aneurism and esophageal stricture be considered before instituting treatment for the latter condition.

Physical Signs Resulting from Pressure upon the Spinal Sympathetic Nervous System.—(*a*) If the nerves of but one side of the spine are compressed, the pupil of that side will be either contracted or dilated, depending upon the degree of pressure exerted by the aneurismal sac. (*b*) Owing to the same cause, there may be unilateral pallor or flushing of the face; and (*c*) the same cause produces unilateral sweating of the

head and neck, and, in selected cases, there may be unilateral sweating of the upper half of the body.

Inspection.—*General.*—The facial expression is, as a rule, anxious and somewhat careworn; if observed closely, the head will be found to tremble with each pulsation of the heart if a large thoracic aneurism is present. The voice is drawn and often husky.

Local.—Visible pulsation is an early sign, and is most frequently observed at the right side of the sternum, above the level of the third rib; but it is also, although less frequently, seen on the left side over a corresponding area. Clubbing of the fingers, more or less incurvation of the nails, and, in advanced cases, a variable degree of cyanosis may also be present. In aneurism of the transverse portion pulsation may be seen at the episternal notch, although an impulse at this site is not pathognomonic of aneurism. The pulsation may occur in the absence of the slightest bulging, but when associated with deformity, its diagnostic importance is great (Figs. 129, 130).

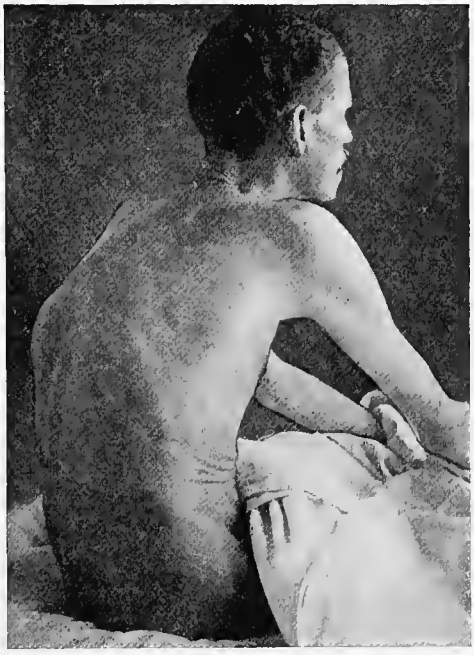


Fig. 130.—Large Thoracic Aneurism with Erosion of the Tenth Rib. Expansile Pulsation was Obtained over the Most Prominent Portion of the Tumor.

If the innominate artery is the site of the aneurism, there is pulsation in the neck above the sternoclavicular junction, and, less commonly, above the sternum; in most instances there is bulging corresponding to the seat of visible impulse. Slight bulging may be detected by making a comparative study of the two sides of the neck. Innominate aneurism may not cause deformity of the anterior surface of the chest, but when the examiner stands behind the patient, deformity may be apparent, particularly if one looks directly down upon the neck and shoulder. In inspecting the chest the patient should be moved so as to get different lights upon the inspected surface, and it is often well for the physician to stand or sit in order that his eye be on a level with the portion of the chest inspected.

In aneurism of the ascending portion of the arch the most frequent seat of the bulging and pulsation is over the first and second right interspaces, near the margin of the sternum, and, indeed, the adjacent portion of the sternum may also share in the deformity. If the aneurism is situated just above the aortic orifice, bulging and pulsation are prone to occur at the third interspace and along the left margin of the sternum. If the upper portion of the sternum is the site of bulging and pulsation, it is probable that the transverse portion of the aortic arch is occupied by the aneurism. When an aneurism develops from the descending portion of the aorta, bulging is seen

along the left margin of the sternum, at the second or third interspaces, and in the scapular region (Figs. 129 and 131).

The heart is ordinarily displaced downward and to the left, and the apex-beat is seen well below its normal situation, but this is dependent in part, at least, upon cardiac hypertrophy. When the patient is directed to elevate his arms distinct pulsation of the brachial and at times of the radial and ulnar arteries is observed.

Multiple Aneurism.—In a case recently under our care there was at autopsy disclosed an aneurism of the transverse portion of the aortic arch, and also another of the right carotid artery, the pulsation resulting from the second having created some doubt as to the actual conditions that existed. Another patient displayed well-marked pulsation in both the right and left carotid regions, and here again autopsy disclosed an aneurism of both carotids and a general dilatation of the aortic arch.

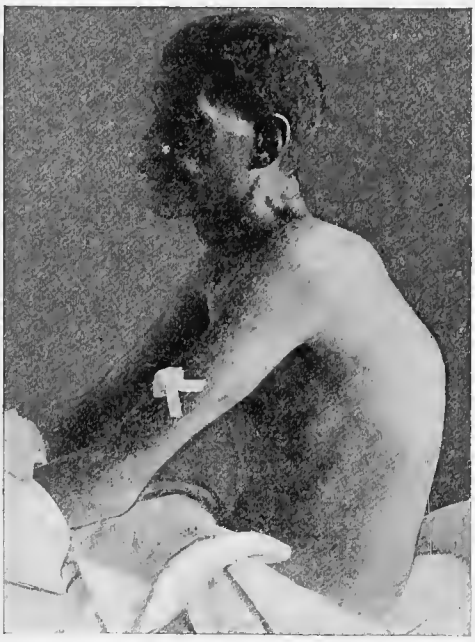


FIG. 131.—LATERAL VIEW OF PATIENT SHOWN IN FIG. 130 (studied during service, 1907, at Philadelphia General Hospital).

Palpation.—The protrusion consists of a more or less yielding and elastic mass, and when superficially seated, fluctuation is present. Expansile pulsation is to be classed among the characteristic signs of aneurism, and is best obtained by encircling a pulsating mass with the thumb and all the fingers of one hand; by this method alterations in the size of the pulsating mass are clearly discernible. The pulsation is also found to be synchronous with that of the systole. In deep-seated aneurism the pulsation may be indistinct, and is at times more forcibly elicited by placing the fingers of one hand over the anterior surface of the chest and the other hand at a corresponding point posteriorly. By placing the palm of the hand over the pulsating mass, a diastolic shock is occasionally detected. A systolic shock over the pulsating mass may be detected, and this is frequently associated with an extremely soft, purring fremitus.

Inequality of the radial pulses is an almost pathognomonic sign of thoracic aneurism, although aneurism may exist without causing any appreciable difference in the radial pulses. In aneurism of the innominate artery the right pulse only would be affected (delayed, softer, and more readily compressible than the left), but if the aneurism is situated in that portion of the aorta just beyond the point where the innominate artery is given off, the left pulse only would be affected. Lastly, if the aneurism was situated at a point beyond where the blood-supply to the left arm is given off, it would not influence either the right or the left radial pulse.

The condition of the radial pulses may, in certain cases, enable us to determine definitely the site of an aneurism.

Tracheal tugging is an extremely valuable sign of thoracic aneurism, although there are exceptional instances in which this sign is obtained without the existence of aneurism. Tracheal tugging is elicited by placing the index-fingers under the cricoid cartilages (see Fig. 132), directing the patient to elevate the chin, while the operator rests his fourth (little) fingers upon the clavicles. If this sign is present, there is a distinct tugging (pulling downward) of the trachea with each pulsation of the heart. The essential factors necessary to produce tracheal tugging are: (1) Elevation of the patient's chin; (2) proper grasp upon the trachea by the operator; (3) the aneurismal mass must be adherent to the trachea, and of sufficient size to cause an appreciable tugging with each pulsation of the heart, and at a time when the sac is distended by blood.

Percussion.—Percussion may give negative results, but if the tumor

Fig. 132.—Position of Both Patient and Operator to Elicit Tracheal Tugging.

causes bulging or comes in contact with the chest-wall, a proportionate area of flatness is detected. The abnormal field of impaired percussion-note may be the only clinical feature present. In aneurism of the ascending arch flatness is elicited to the right of the sternum; in those of the transverse arch, over the upper part of the sternum and to the left, whereas those of the descending portion are revealed by a flat area between the spine and the left scapula. A sense of increased resistance is offered to the finger that is in direct contact with the chest-wall. The area of cardiac dullness is ordinarily increased.

Auscultatory percussion is also of service in outlining the aneurismal mass and in separating it from the heart.

Auscultation.—Murmurs probably owe their origin, in part, to the presence of fibrin in the sac, yet they may be absent. When a murmur is present

over the aneurism, it is systolic in time, and heard with greatest intensity over the flat area or body of the tumor, and is transmitted in the direction of the blood-stream, being, therefore, distinctly audible along the course of the great vessels. The murmur has a booming quality.

Aortic regurgitation may be considered as associated with aneurism near the aortic ring when a double murmur is heard. In a few instances the diastolic murmur alone is detected; when, however, well-marked aortic regurgitation is present, an accentuated ringing second sound is likely to be heard.

The Peripheral Arteries.—See Inequality of the Radial Pulses under Palpation, p. 316.

Sphygmographic Tracing.—"The sphygmogram exhibits a slanting upstroke, with obliteration of the secondary wave though its characters are by no means constant" (Anders).

Laboratory Diagnosis.—If the aneurism rests directly upon a bronchus, cough is likely to be accompanied by free, and not uncommonly bloody, expectoration. (See Hemorrhage from Lung, p. 117.) If the bronchus becomes sufficiently compressed to give rise to bronchiectasis, the sputum is similar to that of the last-named affection. (See p. 101.)

X-ray Diagnosis.—This method of diagnosis is of unusual value in those cases in which the characteristic signs of aneurism are absent, and by its means deep-seated aneurisms of the aorta were detected weeks and even months before other definite symptoms or signs of this malady were apparent. In a large number of cases of aneurism the *x*-ray proved a positive means of diagnosis in every instance (See *x*-ray Diagnosis, p. 235.)

Illustrative Case of Thoracic Aneurism.—H. H. E., male, aged forty-nine years; height, 5 feet 9¼ inches; weight, 161 pounds.

Family History.—Father died of heart disease at the age of fifty; mother living at the age of seventy; a brother died of chronic nephritis at the age of forty-five; and another brother living in a foreign country is believed to be in perfect health. Sister died at the age of fourteen of scarlet fever. No history of either malignancy or tuberculosis in either paternal or maternal ancestors.

Previous History.—Had the diseases of childhood, including diphtheria at the age of nine years. Contracted syphilis at the age of twenty-seven and states that he took special treatment for a period of about one year, after which treatment was discontinued. Ten years ago he had an attack of stomach trouble and since that date has had what his physician pronounced catarrhal jaundice. Had influenza some years ago, but does not recall the exact date of this illness.

Social History.—Married at the age of twenty-three; one son living, born before patient contracted luetic disease. Following the contraction of syphilis three children were born, two of these being still born, and the third dying at the age of six months. Patient is a stevedore by occupation, consequently he is compelled to do heavy lifting. He has been addicted to a moderate use of tobacco, but uses alcohol to excess periodically.

Present Illness.—The first evidence of his present trouble was apparent about three months ago, when he observed that he became unusually short of breath following exertion; this dyspnea has increased until at present he is unable to follow his usual work. He is also conscious of an unusual throbbing in his chest, and hears the beating of his heart when resting quietly in bed. He complains of numbness and tingling of the fingers of the right hand, and states that the right side of his head and neck sweats profusely.

For the past few weeks pain has been more or less constant, and the patient describes it as boring in character, being most intense immediately beneath the sternum and between the shoulders, and at times radiating to the arms. The prone position appears to relieve the pain at certain times, although it is often sufficiently severe to prevent sleep.

A harsh, rasping, brazen cough has been an annoying symptom, and was present for two or more months before he experienced extreme dyspnea and at a time when he was yet able to work. The cough is more or less paroxysmal, and during such paroxysms he coughs for several minutes and expectorates copiously. The voice is husky, and a laryngeal examination shows atrophy of the right vocal cord.

Physical Examination.—*General.*—The patient appears to be well nourished, although his expression and general attitude are those of pain. When sitting, he inclines slightly forward and holds the chest in a more or less fixed position. There is sweating of the right half of the body, being more pronounced over the right half of the head and neck. The patient's mental condition is somewhat dull, the expression is anxious, and he is extremely fearful of pending danger. The finger-tips and mucous membrane of the lips show a mild grade of cyanosis. The muscles are soft and the skin is wrinkled.

Local Examination.—*Inspection.*—The right pupil is dilated, and there is flushing of the right cheek. There is distinct bulging, located at the junction of the second and third right costal cartilage with the ribs; there is also a distinct pulsation over this area and in the right supraclavicular region. The apex-beat is seen 2½ inches below and one inch external to the left nipple.

Palpation.—The apex-beat of the heart is unusually forcible, and there is an appreciable pulsation over the entire precordia; this is felt most decidedly over the area of bulging, and is somewhat expansile in character; at times a questionable thrill is detectable over this pulsating mass. The radial pulses are unequal, the right being softer than the left, more readily compressible, and slightly retarded. The right radial pulse is also imperceptible when the hand is elevated at arm's length above the head.

Percussion.—The area of cardiac dullness extends well down to the left, and appears to be continuous with that of the pulsating mass located at the junction of the second and third costal cartilages with the ribs. The area of hepatic dullness extends approximately one inch below the costal margin.

Auscultation.—The heart-sounds are unusually loud, and are heard over the entire precordium and as high as the clavicles. When the stethoscope is placed over the pulsating thoracic tumor, a systolic murmur (bruit) is audible, and a decided diastolic shock is also present.

X-ray Diagnosis.—A pulsating tumor the size of an orange was found to the right of the median line and in the anterior portion of the thoracic cavity, and the clinical evidences offered by the screen and those previously obtained by physical examination led to the diagnosis of aneurism of the innominate artery.

Diagnosis by Induction from Clinical Data.—A history of having contracted syphilis at the age of twenty-seven in itself suggested a possibility of disease of the greater blood-vessels. His occupation subjected him to undue strain, which again favored aneurism, and, indeed, his habits as to the use of tobacco and alcohol also favored the development of dilatation of the aorta.

Dyspnea upon exertion, harsh rasping or brassy cough, inequality of the pupils, unilateral sweating, and more or less constant pain in the thorax were considered sufficient evidence upon which to base a diagnosis.

Course of Illustrative Case.—The patient's condition continued to progress, the general symptoms, pain and dyspnea, becoming more distressing, until it was deemed advisable to wire the aneurismal sac, after which the patient was kept perfectly quiet for a period of four weeks, and later was allowed to leave his bed. As the result of wiring the right radial pulse became somewhat stronger, and the dyspnea less distressing, although the pain was not appreciably affected. One year following the introduction of wire into the aneurismal sac the patient came under our care, but death ensued before a careful physical examination could be made. Autopsy confirmed the diagnosis of aneurism of the innominate artery.

Summary of Diagnosis.—This is based upon the following symptoms and signs: Pain, paroxysmal cough, hoarseness, and unilateral sweating. Among the signs characteristic of aneurism are inequality of the radial pulses and of the pupils, deformity with bulging of the chest, the protrusion of a pulsating mass, and the presence of a distinct bruit over the site of the aneurism.

Differential Diagnosis.—Intrathoracic aneurism is to be distinguished from: (1) Pulmonary tuberculosis; (2) abnormal pulsation of the aorta; (3) pulsating empyema; (4) chronic adhesive pleurisy with displacement of the heart; and (5) new-growths of the mediastinum.

(1) **Pulmonary Tuberculosis.**—When the aneurism compresses a bronchus, bronchiectasis attended by cough, bronchorrhea, fever, and emaciation, may follow. In phthisis emaciation and fever are more pronounced, and

tubercle bacilli are present in the sputum. The cardiovascular signs of aneurism are absent in tuberculosis.

(2) **Abnormal pulsation of the aorta** is present in neurotic subjects, mostly in females, and in aortic regurgitation; occasionally it is associated with retraction of the right lung, with spinal deformity, and with displacement of the aorta. *Aortic regurgitation* is frequently associated with aneurism of the arch, and in its course a dilatation of the ascending portion of the aorta often develops. The diagnosis of aneurism of the arch should not be made, therefore, in these cases, unless the physical signs and symptoms are unmistakable.

(3) **Pulsating empyema** can be confounded only with large aneurismal growths, and does not bear the same definite relation to the central long axis of the body as do aneurisms. In empyema the abnormal field of flatness is situated at the base of the lung; moreover, the pulsation is not expansile. The physical signs of aneurism are absent in empyema.

(4) **Chronic Adhesive Pleurisy with Displacement of the Heart.**—In this condition and in fibroid tuberculosis there may be extensive retraction of the lung, or the heart may be displaced as the result of adhesive bands, in consequence of which localized areas of pulsation are detected over the anterior surface of the chest. In adhesive pleurisy or fibroid tuberculosis the anterior surface of the chest is markedly distorted, and the physical signs of aneurism (pulsation, bruit, and alteration in the radial pulses) are absent.

(5) **New-growths of the Mediastinum.**—Both cancerous and sarcomatous involvement of the mediastinal glands may give rise to signs and symptoms closely resembling those of aneurism. Syphilitic growths of the mediastinal glands are also likely to be mistaken for aneurism. In one case seen by us, that of a hospital patient suffering from locomotor ataxia, there was also present a large tumor in the mediastinum, as was shown by the *x*-ray; this patient presented most of the symptoms and many of the signs characteristic of aneurism, yet for three years of his stay in the hospital his physical condition remained practically the same, until finally when he showed well-marked pulsation over the upper portion of the sternum. This case exemplifies the great difficulty with which aneurism is distinguished from a solid tumor of the mediastinum, and, indeed, early examination made by means of the *x*-ray favored the existence of solid growth, rather than that of aneurism, although the existence of aneurism at present is unmistakable.

Clinical Course.—The clinical course may, in selected cases, be similar to that of chronic valvular disease, although in the majority of instances a fatal termination follows—(1) Rupture of the aneurism with extensive hemorrhage into the large vessels, esophagus, trachea, mediastinum, pleura, lung, spinal canal, and peritoneum; (2) General asthenia; (3) direct pressure; and (4) complicating diseases, among which are pulmonary abscess, gangrene, tuberculosis, and fibrinous pneumonia are among the usual modes of termination.

ANEURISM OF THE ABDOMINAL AORTA.

Remarks.—The favorite seat of abdominal aneurism is at or near the celiac axis. This form is less common than intrathoracic aneurism, although not rare. The aneurism may be of either the fusiform or the saccular variety.

Symptomatology.—The tumor may extend backward, but more frequently it comes forward. Projecting from the posterior wall, it usually erodes the vertebræ. Compression of the cord is likely to take place, produc-

ing paraplegia, which is preceded by tingling and numbness of the lower extremities.

Pain of a neuralgic or boring or gnawing character may be present, and is due to destruction of the bone. Rarely, the aneurism perforates the diaphragm, and finally ruptures into the pleura, lungs, or pericardium. When situated near the diaphragm, it may conceal itself until the sac has attained a comparatively large size. *Vomiting* and *gastric seizures* may be troublesome, and the fact that *embolism* of the superior mesenteric artery may occur and give rise to severe colicky pains must be remembered. *Jaundice* has been observed.

Physical Signs.—**Inspection.**—Epigastric pulsation and swelling may be visible.

Palpation.—There is a heaving, expansile pulsation that may be accompanied by a thrill. When the tumor hugs the diaphragm, the pulsation may be double in character. The femoral pulse is diminished and delayed.

Percussion shows an abnormal area of dullness when the tumor advances forward.

Auscultation.—A soft bruit is commonly elicited.

Differential Diagnosis.—In neurotic females, in advanced anemia, and in those having thin abdominal walls throbbing of the abdominal aorta may be unusually prominent. **New-growths** immediately overlying the aorta may also account for unusual pulsation of the abdomen, but such pulsation is not expansile in character. On placing the patient in the knee-chest position, if the abdominal pulsation is due to the new-growth, the mass tends to fall forward and the pulsation is less conspicuous.

Termination.—In rare instances nature effects a cure; as a rule, however, the case terminates by obliteration of the lumen by extensive clots, paraplegia from pressure of the cord, embolism of the mesenteric artery, and commonly in rupture into the abdomen or thorax.

ANEURISM OF THE PULMONARY ARTERY.

General Remarks.—When aneurism of this artery attains sufficient size, the physical signs and symptoms in a measure resemble those of aneurism of the arch of the aorta. It is with great difficulty and only in selected cases that aneurism of the pulmonary artery can be diagnosed during life.

Aneurism of the *hepatic*, *splenic*, *renal*, and *inferior mesenteric arteries* may occur, but these are indeed difficult of diagnosis, and commonly manifest the general features of aneurism of the abdominal aorta when such aneurism attains sufficient size to give definite signs.

ARTERIOVENOUS ANEURISM.

Pathologic Definition.—An artificial communication between an artery and a vein. An aneurismal sac may be located between the artery and the vein, or the fistulous communication may directly connect the artery and vein without any expanded portion separating the two vessels.

Diagnosis.—In common, the symptoms and certain signs are those of aneurism, previously outlined. Swelling develops, the veins become distended, the part becomes cyanotic, and edema is common. A thrill may be present, and is likely to be continuous. A continuous humming murmur, which is more pronounced during systole, is also an important sign.

Clinical Course.—Arteriovenous aneurism is, as a rule, a purely surgical condition.

DISEASES OF THE MEDIASTINUM.

The affections of the mediastinum are classified as—(1) Inflammations; (2) tumors; (3) diseases of the thymus gland; and (4) hemorrhage into the mediastinum.

INFLAMMATIONS.

Remarks.—Inflammation may be limited either to the glands or to the connective tissue, or, in selected cases, both may be involved. A moderate inflammatory process involving the glands (lymphadenitis) is commonly present in bronchitis and pneumonia, and is seen at times during the course of measles, influenza, and whooping-cough. Tuberculosis of the lymph-glands situated in the mediastinum may extend so as to produce definite physical signs, and inflammatory processes involving the tissues of this portion of the body may at times go on to the formation of abscess.

MEDIASTINAL ABSCESS.

Predisposing Factors.—Traumatism is an exciting cause, and the abscess may also develop as a complication or sequel of certain of the acute infections, *e. g.*, smallpox, erysipelas, and measles. Suppurative processes of the adjacent tissues may at times extend to the mediastinum, and tuberculosis of the mediastinal glands is generally conceded to be a potent factor in the production of abscess.

Principal Complaint.—Pain and tenderness over the sternum are the most prominent symptoms of abscess. At times the patient complains of a peculiar throbbing pain, which is, in a measure, characteristic of the accumulation of pus. Cough is, as a rule, present, and the patient frequently suffers from marked dyspnea. Chills, followed by profuse sweating and prostration, also occur. The *thermic features* are those characteristic of acute suppuration, *e. g.*, evening rise in temperature, with decided remissions during the morning hours. The abscess may rupture into the trachea, or the pus may descend to the layers of the diaphragm, and eventually escape into the abdominal cavity. Rupture into the pleura may also occur.

Physical Signs.—Probably the most important sign is obtained by **percussion.** By its means a variable area of dullness is discovered behind the sternum, this area increasing in size with the progress of the disease. In selected cases mediastinal abscess may perforate the chest-wall, when a distinct fluctuating mass is palpable. It should be remembered that abscess, when it extends outside the bony structure of the chest, may produce a pulsating mass that is to be distinguished from aneurism.

If the abscess is chronic in nature, both the symptoms and the signs may closely resemble those of a tumor of the mediastinum, and, indeed, the diagnosis is attained only with great difficulty.

Diagnosis.—The chief condition from which abscess is to be distinguished is aneurism of the arch of the aorta, and when the abscess develops slowly, this differentiation is made with great difficulty. There are certain features, however, that are, in a measure, characteristic of abscess; among these are chills, profuse sweating, prostration, and a hectic type of fever. Abscess ordinarily develops with great rapidity as compared to aneurism or solid growths of the mediastinum. The detection of a bruit serves to mark the existence of aneurism.

TUMORS OF THE MEDIASTINUM.

Remarks.—There are two varieties of tumors of the mediastinum that deserve special attention, although other types may also be encountered, as is shown by Hare's statistical analysis of 520 cases, which included: Carcinoma, 134; sarcoma, 98; lymphoma, 21; fibroma, 7; dermoid cyst, 11; hydatid cyst, 8; lipoma, gumma, and ecchondroma were also found. The same author further observed that in 48 of the cases of carcinoma and in 33 of sarcoma the tumor was situated in the anterior mediastinum. Sarcoma may have its origin in the thymus or the lymphatic glands or pleura, and less often in the lung. It is to be remembered that carcinoma is a somewhat common lesion of the esophagus, thymus gland, and lungs, and secondary carcinoma of the mediastinum is most likely to involve the lymph-glands.

Fig. 133.—Private Patient, Showing Marked Dilatation of the Veins of the Trunk, Extremities, and Head as a Result of Thoracic Tumor.

Predisposing Factors.—**Sex.**—Males are more often affected than females. **Age** figures prominently, the majority of all cases being seen between the thirtieth and fortieth years.

Principal Complaint.—Early during the course of tumor of the mediastinum the patient is likely to be languid, and suffers from a variable degree of dyspnea and from vague substernal pains. Pain is probably the most constant symptom, and while it may not be severe in all cases, it is usually accompanied by the general features of oppression. It is oftenest situated in the upper sternal region, and from this point it may radiate to the sides of the chest, and at times down the arms. Pressure upon the brachial plexus is invariably followed by pain in the arms.

Dyspnea may be an early and annoying symptom, and may result from pressure upon the trachea, bronchus, and at times from entanglement and pressure of the recurrent laryngeal nerve. Asthmatic seizures are by no means uncommon, and are always accompanied by severe cough and paroxysmal dyspnea. When there is involvement of the recurrent laryngeal nerve, the patient notices that his voice is altered.

Physical Signs.—Inspection.—Owing to irritation of the sympathetic nerves areas of hyperemia may be found over different portions of the chest and face, and unilateral sweating and inequality of the pupils are often present. The superior vena cava and other veins of the upper portion of the body may be compressed, in consequence of which there are edema and prominence of the veins of the head and neck, and at times

swelling of the arms and dilatation of the superficial veins (Figs. 128 and 133). Pressure from the growth may also cause distention of the veins of the anterior surface of the chest and abdomen, the latter resulting from the establishment of collateral circulation. The inferior vena cava may be compressed, but this occurs far less frequently than interference of the venous circulation of the upper portion of the body. There is likely to be bulging of

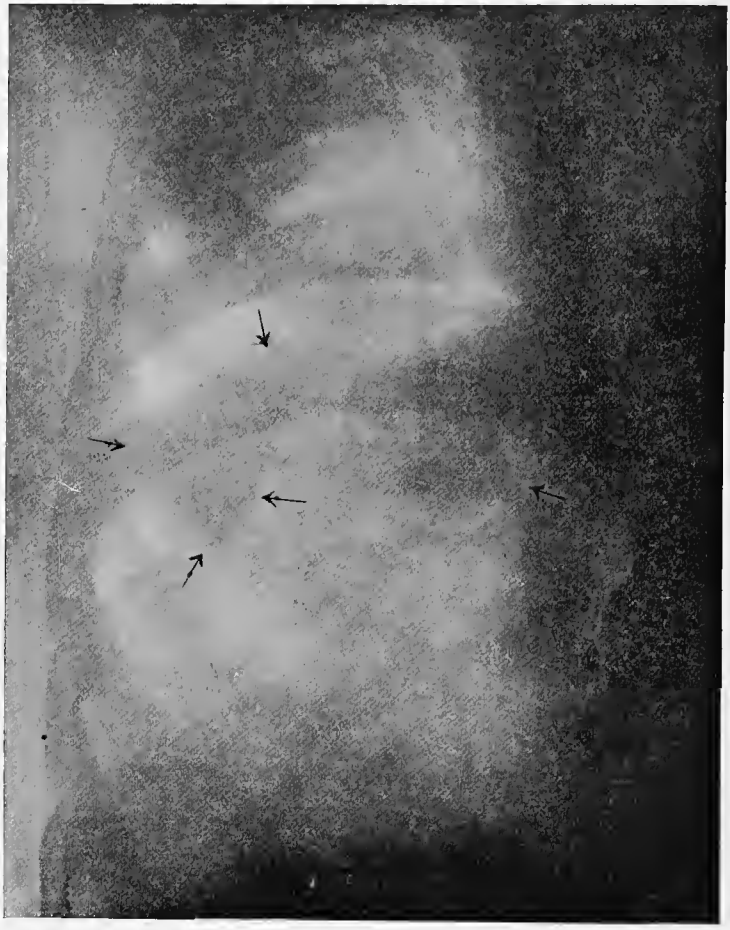


FIG. 134.—RIGHT SIDE OF CHEST.
Shadows of what may be a metastatic growth, and the radiations from the central mass. Private patient shown in Fig. 133.

the sternum, and in selected cases erosion of the sternum follows. Rapidly growing lymphoid tumors are those most likely to perforate the chest-wall.

Palpation.—The pulsation of the heart or of the aorta may be felt quite distinctly over a solid tumor, yet expansile pulsation is not observed. The tactile fremitus is decreased over the area occupied by the growth. In those cases in which there is irritation of the vagus or sympathetic nerves, the pulse will be either slowed or appreciably quickened, depending upon the degree of irritation offered by the growth.

Percussion.—A localized area of dullness is one of the most characteristic signs of mediastinal tumor, and may be detected even in the absence of other definite signs and symptoms.

Auscultation.—The absence of breath sounds over the dull area is highly suggestive of solid growth of the mediastinum, whereas in aneurism a distinct bruit is the rule. Both breath-sounds and voice-sounds are indistinct over the tumor, and in the majority of cases the heart-sounds are heard but feebly over this area.

***X*-Ray Diagnosis.**—This method of diagnosis is of unusual value for the detection of tumors of the mediastinum, and it is claimed that by its means it is possible, in practically all cases, to distinguish between aneurism and solid growths of the mediastinum. (See *x*-Ray Diagnosis, p. 234.)

Clinical Course.—In a large proportion of all cases this is of short duration, varying from six months to one year, although cases have been encountered in which mediastinal growths continued their course for from two to three years. There is at present under care of one of us the case of an engineer in whom tumor of the mediastinum was diagnosticated nearly six years ago.

MEDIASTINAL HEMORRHAGE.

Remarks.—There may be an effusion of blood into the mediastinal connective tissue following rupture of aneurism of the arch of the aorta. Hemorrhage, as a rule, follows pathologic conditions of the vessels of the thorax, and it rarely results from traumatism.

DISEASE OF THE THYMUS GLAND.

Remarks.—When tumors originate in the thymus gland, the organ becomes enlarged. Such conditions as hypertrophy of the gland and abscess are indistinguishable from mediastinal tumor and abscess previously discussed. The number of sudden deaths of young children reported in connection with various forms of enlargement of the thymus gland is steadily increasing. The thymus gland has been found enlarged in eighteen cases of epilepsy reported by Ohlmacher. Hemorrhage into the thymus gland is indistinguishable from hemorrhage of the mediastinum, just discussed.

THE BLOOD.

LABORATORY EXAMINATION OF THE BLOOD.

CLEANSING OF SLIDES AND COVER-GLASSES PREPARATORY TO MAKING A MICROSCOPIC STUDY OF THE BLOOD.

Both slides and cover-glasses should be cleansed for use in the following manner: Place a number of cover-glasses or slides in a glass containing warm water and soap: stir with a glass rod, then remove each cover-glass or slide separately, place upon a thin handkerchief, and rub gently between the thumb and forefinger. After it has been thoroughly dried, drop it into a second glass containing warm water. After all the slides have been transferred in this manner from the soap and water to the second glass,

stir again with the glass rod, and, after repeating the previous treatment, transfer them to a third glass, containing 70 per cent. alcohol. The treatment in the alcohol should be the same as that just outlined, after which the cover-glasses should be placed in a wide-mouthed bottle containing equal parts of alcohol and ether, in which they should be allowed to remain until required for use.

Caution.—All cover-glasses and slides should be removed from the solution in which they are kept by means of clean forceps. They should then be dried with a soft linen or silk handkerchief, and placed one at a time in a Petri dish, the bottom of which has been neatly covered with filter-paper. After the desired number of slides or covers have been removed, the dish should be covered and set on a warm stage or in an incubator, in order that the slides may be warm when the blood is applied, thus facilitating an equal distribution throughout the smear.

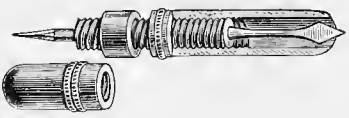


Fig. 135.— Daland's Blood-lancet.

Collection of Blood.

The points of election for the collection of blood are the lobe of the ear and the palmar surface of the finger, near its tip. The method to be described is employed in all clinical work except in bacteriologic study. It is performed as follows:

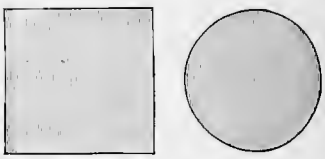


Fig. 136.—Styles of Cover-glasses.

If the finger is chosen as the site for puncture, the tip should be cleansed with water, rubbed gently with a soft cloth, and later moistened with ether. After the ether has evaporated the finger is grasped between the thumb and index-finger of the left hand, and the instrument with which the puncture is to be made, having first been cleansed with water and alcohol, is quickly plunged into the tip. The depth of this puncture should vary from one-eighth to one-quarter of an inch, depending on the thickness of the skin. Gentle pressure may be applied to start the blood, after which

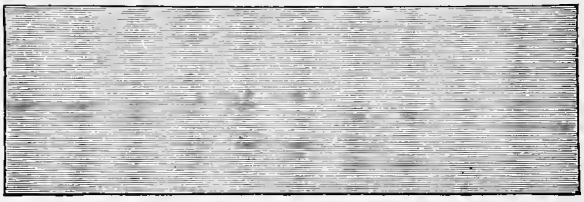


Fig. 137.—Glass Slides (Natural Size).

four or five drops of the first blood exuded should be removed with a soft handkerchief. When the ear-lobe is chosen, the same precautions just laid down are to be observed. The puncture may be made with Daland's blood-lancet (Fig. 135), or with one nib of a new steel pen.

Study of Fresh Blood.

After the first few drops have been removed by the handkerchief and a fairly large-sized drop exudes spontaneously, the cover-glass (Fig. 136),

which has been warmed, is held by its edge between the thumb and index-finger, and the summit of the drop of blood is allowed to touch the center of the cover-glass, care being taken that the surface of the glass does not come into contact with the skin. The cover-glass is allowed to fall gently upon the center of a slide (Fig. 137); the weight of the cover-glass causes the blood to spread between it and the slide.

Caution.—When pressure is applied to spread the blood, the cellular elements are likely to become distorted, hence reliable deductions cannot be drawn from the microscopic appearance of the specimens. The slide may now be carried to the microscope, and examined by any objective desired.

This examination is most essential in the study of malarial and other blood parasites. It acquaints us with the degree of leukocytosis; the character and the degree of viscosity, the presence of rouleaux formation; the size and conformation of the red cells.

Clinical Significance.—Extreme pallor and swelling of the red cells are conclusive evidence that the osmotic tension of the serum is low, and, as a consequence, the erythrocytes are swollen and have given up a certain percentage of their hemoglobin. Deformity of the erythrocytes may result from alterations in the viscosity and in the osmotic tension. The degree of viscosity depends upon the perfection of rouleaux formation, and where the cells are aggregated in dense masses, hyperviscosity exists; on the other hand, if the red cells are equally disseminated throughout the field, hypoviscosity obtains. The leukocytes are increased when more than two or three white blood-cells are to be seen in a single microscopic field of living blood ($\frac{1}{12}$ oil-immersion objective employed).

Smears.—When smears are to be made, the foregoing process is modified only in so far that the cover-glass, with its specimen of blood, is allowed to fall upon another cover-glass in such a manner that the edge of the one projects beyond that of the other; after the blood has spread, these overlapping margins are grasped between the thumb and index-finger of each hand and separated by sliding off horizontally. They are then placed upon a flat surface, specimen side up, and allowed to dry in the air, after which they may be placed together and kept for an indefinite period without further treatment.

A more satisfactory method for the beginner and for class work is to make the smears on slides. The slide upon which the smear is to be made is placed upon the table; a second slide is grasped between the thumb and index-finger, and the short edge of this slide is brought in contact with the summit of the drop of blood in such a manner that the blood collects on the under surface of the edge. The second slide is now placed at an angle of about 45 degrees to the surface of the first (which rests upon the table), and is then pushed evenly over the surface of the slide for its entire length. The object is to transfer the drop of blood collected on the edge of the second slide to the surface of the first slide by allowing it to smear. Far less skill is required to make good spreads by this method than by the use of cover-glasses.

ESTIMATION OF COAGULATION BY WRIGHT'S COAGULOMETER.

This instrument (Fig. 138) is composed of a central metal cylindric can, surrounding which is a closely fitting leather case containing nine small pockets, each lined with flannel. One of these pockets holds a thermometer graduated to 50° C. The remaining pockets serve as receptacles

for the capillary blood-pipets. Each pipet is composed of clear glass, is about 10 cm. in length, and has a lumen of 0.25 mm.

The method is conducted as follows: 1. Fill the cylinder with water at the body-temperature—37° C. (98.4° F.).

2. Label the tubes *a*, *b*, *c*, etc., and place them in the pockets, and when they are warmed, secure a fairly large drop of blood from the tip of the finger, and from it fill, by aspiration, tube *a* to one-half its capacity, and immediately place this tube in its pocket.

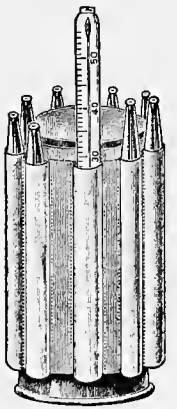


FIG. 138.—WRIGHT'S COAGULOMETER.

Caution.—Note the time of filling the tube labeled *a*, and also record the condition of its blood when examined three minutes later.

3. In a similar manner several of the tubes are filled with blood at intervals of one minute.

4. Remove the tubes from the instrument at various times after the blood has been withdrawn, and endeavor to force the blood from the tube by blowing. The time necessary for the blood to clot in the tube is regarded as the coagulation time.

Cleansing the tubes is accomplished by passing a fine wire into the tube and breaking up the clot, after which the tubes are washed with water, alcohol, and, finally, with ether.

Boggs' Coagulometer.—The most practical coagulometer is probably the Boggs modification of the Brodie-Russell instrument. The instrument consists of a round metal chamber with a glass bottom. A truncated cone of clear glass, the free surface of which is 4 millimeters in diameter, fits into this chamber from the top. A capillary metal tube projects into the chamber from the side in such a way that the point is just below the free surface of the truncated glass cone. This tube carries a small rubber bulb and tubing, of the kind used for operating the shutter of a camera, on its outer end. The chamber is placed on the stage of a microscope, the rubber bulb and tubing being in place. The free surface of the truncated glass cone is then touched to a drop of blood exuding from a puncture made in the usual manner, and is then placed in position in the chamber. The two-thirds lens of the microscope is then focused on the drop of blood, and gentle pressure is made on the rubber bulb, so that a fine jet of air plays on it. At first the corpuscles are seen moving separately parallel with the circumference of the drop, then in clumps parallel with the circumference of the drop, then with an elastic movement forward and backward parallel with the circumference of the drop, and finally radially inward and outward from the circumference toward the center of the drop. When the latter movement is obtained, coagulation is complete. The time at which the blood begins to flow from the puncture and the time at which the radial elastic movement above described should be noted, the difference between the two being the time necessary for coagulation. The average time neces-

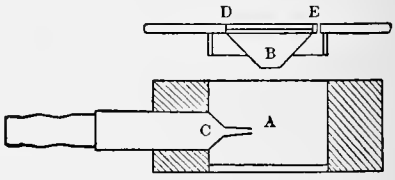


FIG. 139.—BOGGS' MODIFICATION OF THE COAGULOMETER OF RUSSELL AND BRODIE (Emerson).

sary for coagulation in normal individuals is about five minutes and six seconds (Hinman and Sladen).

SPECIFIC GRAVITY OF THE BLOOD.

The most available method for clinical use for ascertaining the specific gravity of the blood is that suggested by Hammerschlag,* which is a modification of Roy's † method.

Hammerschlag's Method.—Two solutions (chloroform and benzol) are mixed in a urinometer glass in such proportions that the specific gravity, taken by an ordinary urinometer, is 1.059, or that of normal blood; the first of these solutions is heavier than blood, whereas the second is lighter. The finger is punctured, a drop of blood is collected in a Thoma-Zeiss pipet, and a drop or two is blown into the chloroform-benzol solution. The blood shows no tendency to mix with this solution, but floats as a ruby bead. If the beads sink to the bottom, chloroform should be added, and if they rise to the top, benzol should be added drop by drop until the blood remains stationary in the body of the liquid. Since the specific gravity of the blood is that of the mixture surrounding it, the specific gravity of this liquid should be taken by means of an ordinary urinometer; the graduation figure obtained equals the specific gravity of the blood.

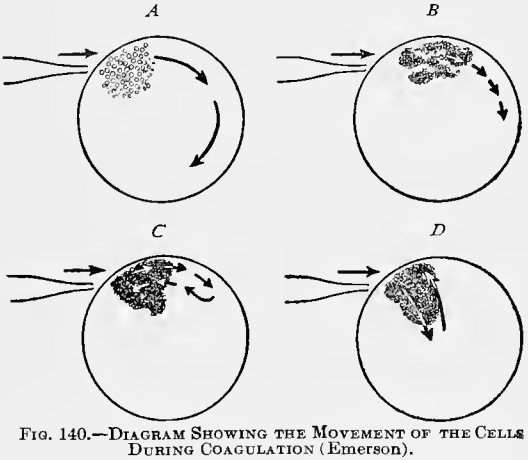


Fig. 140.—Diagram Showing the Movement of the Cells During Coagulation (Emerson).

Caution.—Add chloroform or benzol, a few drops at a time, stirring with a glass rod after each addition. Do not permit air to mix with the drop of blood. Rapidity is absolutely necessary. A urinometer with a scale graduated to 1.070 is best for this purpose.

HEMOGLOBIN.

This proteid contains nearly 96 per cent. of albumin and 4 per cent. of pigment (hemochromogen). In the red cells hemoglobin probably exists in combination with the nucleoproteid of the stroma. Its spectroscopic relations, however, are constant. While in the circulation it exists in the veins principally as reduced hemoglobin, and in the arteries, on account of molecular union with oxygen, it occurs as oxyhemoglobin. Each gram of saturated oxyhemoglobin contains 1.16 c.c. of oxygen, but this degree of saturation varies greatly. The percentage of hemoglobin in a healthy American varies from 85 to 95 during health; 75 to 85 is normal for the Chinese.

Oxyhemoglobin is non-diffusible, of a bright red color, and while it crystallizes with difficulty, it may form yellowish-red, rhombic plates that are readily soluble in water and in weak solutions of the alkaline carbonates. They are insoluble in strong alcohol, ether, carbon disulphid, benzol, and

* Wien. med. Woch., 1890, vol. iii, p. 1018.
† Proceedings Phys. Soc., 1894.

chloroform. Labbe found an increased quantity of oxyhemoglobin in the the newborn—ranging from 15 to 18 per cent. During the first ten days of extra-uterine life it falls to 14 per cent. Such bloods likewise contain a high percentage of reduced hemoglobin.

Reduced hemoglobin is of a dark, cherry-red color, but after high dilution it may display a greenish tint. It is not readily crystallized, but is more freely soluble than oxyhemoglobin. It is demonstrable in the blood of asphyxia and of the new-born.

Hemoglobinemia (**hemocytolysis**) is a condition characterized by a solution of the hemoglobin in the plasma. In man it is a pathologic condition that probably results from a lowered vitality of the erythrocytes, and also from abnormalities in the plasma. Diminished resistance of the red cells may accompany the hemoglobinemia following extensive burns, whereas the hemoglobinemia seen after poisoning may, in part at least, be due to changes in the serum.

Methemoglobin displays a brownish-red color, and crystallizes as brownish-red needles, prisms, and hexagonal plates. It is readily soluble in water, and contains about the same proportion of oxygen as oxyhemoglobin, but the oxygen appears to have formed a somewhat firm union. It is observed in poisoning.

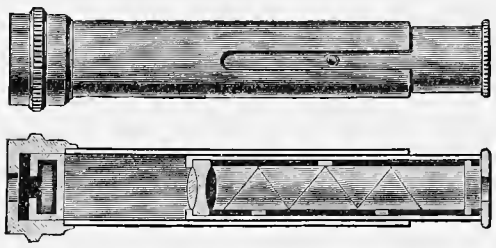


Fig. 141.—Browning's Spectroscope.

Carbonic-oxid hemoglobin is the name applied to hemoglobin containing CO, which gives the blood a rose-red or bluish color. Its crystals display a slightly bluish tint and are easily dissolved. It is present after poisoning by inhalation of illuminating gas, and may remain in the blood for a period of several days.

Hematin appears in the feces after gastro-intestinal hemorrhage, in bloody transudates and effusions, and in the urine after poisoning with arsenic.

Hematoidin is a derivative of hemoglobin, and appears either in the form of needles or as rhombic plates of a light- or dark-orange hue, soluble in ether, carbon disulphid, ammonium disulphid, and chloroform. It absorbs most of the violet end of the spectrum, but does not contain iron.

Hematoidin is present in bloody exudates of long standing, and in the urine after traumatism to the kidney (Yarrow).

Hemosiderin results from the destruction of hemoglobin; it is amorphous, and occurs in the viscera after extensive blood destruction.

Melanin is a yellowish-brown or black pigment. It is insoluble in water, alcohol, chloroform, ether, and weak acids, but is soluble in strong alkalis. It is destroyed by heat, and does not give the reaction for iron. It results from the action of the malarial parasites upon the hemoglobin, and should be distinguished from other pigments, which may or may not contain iron, and whose origins are unknown.

Spectroscopic Study of the Blood.

The spectroscopic examination of the blood serves as the most reliable test for the recognition of blood-pigments, and also for the determination of the particular form of pigment present. A 1 per cent. solution is found to

produce distinct absorption-bands, and when the blood has become dried, it is necessary to dissolve it by macerating in acetic acid. With such blood the spectrum of acid hematin is obtained. Blood from recent clots may be dissolved in water. When heat has been applied to the blood, it should be macerated in a solution of ammonia, when the spectrum of reduced or alkaline hematin appears. Browning's spectroscope (Fig. 141) is a satisfactory instrument for the purpose when strong daylight is employed. A collar serves to enlarge or diminish the aperture, and will be found necessary when different strengths of light and also when fluids of varying opacities are used. Fraunhofer's lines are brought into focus by careful adjustment of the tube. The fluid to be examined should be placed in

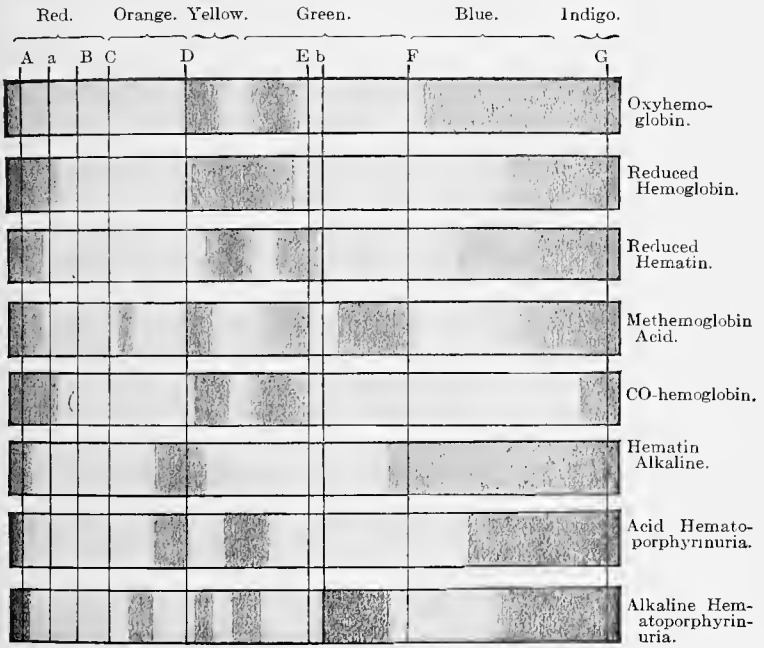


Fig. 142.—Diagram of the Spectra of Eight Substances Known to Concern us from a Diagnostic Standpoint (Boston).

small glass vials with flattened surfaces. The spectrum of fresh arterial blood is that of oxyhemoglobin, and shows two absorption-bands, between D and E (Fig. 142); one of these is sharp, dark, and well-defined near the orange, E. The indigo and most of the blue will be absorbed, and in strong solutions of oxyhemoglobin these two bands may unite.

If ammonium sulphid is added to such solutions, the color of the fluid becomes dark, and the spectrum changes to that of reduced hemoglobin, when one band of absorption occurs between D and E (Fig. 142). A positive indication of the presence of blood is evidenced by the fact that the spectrum may be transformed from that of oxyhemoglobin to that of reduced hemoglobin by the addition of reducing agents to the solution. Cochineal and ammoniated carmin give spectra simulating the spectrum of oxy-

hemoglobin. The addition of boric acid to a solution of these substances causes their spectra to be displayed by the blue, whereas the spectrum of the blood is unchanged. Other of the vegetable dyes have spectra simulating those of blood, but these become pale upon adding sodium bisulphite.

Methemoglobin.—Hematin is produced by adding acids or strong alkalis to blood. In acid solution the spectrum simulates that of acid methemoglobin, whereas in alkaline solution it gives a broad band at D (Fig. 142). An important clinical finding is the change of oxyhemoglobin into methemoglobin; this is revealed by the chocolate color of the blood, and by the fact that in acid or neutral solution it gives four absorption-bands: one quite distinct, between C and D; a second faint narrow band in the yellow, just to the right of D; a third broad, fairly distinct band, between the yellow and the green, just to the left of E; and a fourth broad band, to the left of F, sometimes extending beyond the line F into the blue.

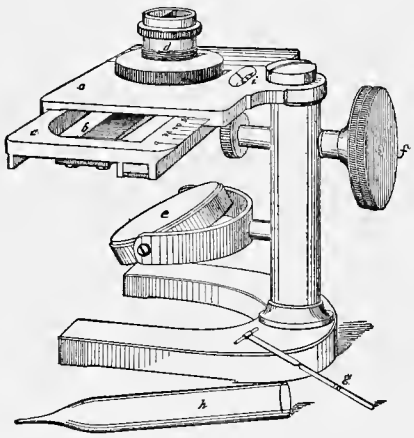


FIG. 143.—VON FLEISCHL'S HEMOGLOBINOMETER.

a, Stand; *b*, narrow wedge-shaped piece of colored glass fitted into a frame (*c*), which passes under the chamber; *d*, hollow metal cylinder, divided into two compartments, which holds the blood and water; *e*, plaster-of-Paris plate from which the light is reflected through the chamber; *f*, screw by which the frame containing the graduated colored glass is moved; *g*, capillary tube to collect the blood; *h*, pipet for adding the water; *i*, opening through which may be seen the scale indicating percentage of hemoglobin.

Carbonic-oxid hemoglobin is present in cases of poisoning by illuminating gas, and may be detected by the rose-red color it lends to both arterial and venous blood. A 0.5 per cent. dilution of such blood gives a spectrum that differs from that of oxyhemoglobin only in that its bands are broader and that the band at D is displaced to the right. The addition of ammonium sulphid causes the spectrum of oxyhemoglobin to be replaced by that of reduced hemoglobin, whereas that of carbonic-oxid hemoglobin is unchanged.

VON FLEISCHL'S HEMOGLOBINOMETER.

This instrument consists of a stand that somewhat resembles the base and table of a microscope; a cylindric diluting chamber with a glass bottom, divided into halves; a plaster-of-Paris reflector; a graduated color prism; a thumb-screw adjustment; and a capillary tube (Fig. 143).

Method of Application.—1. One compartment of the graduated cylinder is half filled with distilled water, care being previously exercised to cleanse the chambers of the cylinder.

2. The capillary tube, which has been cleansed and dried, is held horizontally, and brought into contact with the summit of a drop of blood, when it immediately fills by capillary attraction. All blood on the outer surface of the tube is removed with a soft handkerchief, and the tube is immersed in the distilled water contained in the cylinder. It is held horizontally, and shaken gently until apparently emptied, when a few drops of distilled water are forced from a dropper through the tube in order to wash every vestige of blood into the cylinder.

3. After the expulsion of the blood, the solution is gently stirred with the metal handle of the pipet, in order to effect a perfect mixture.

4. With a dropper both compartments of the cylinder are filled to the brim with distilled water. Moisten the ground cover-glass with the breath, and then allow the glass to fall gently upon the cylinder, when two small bubbles will appear one upon each side of the partition. If the bubble upon the side containing the blood is large, it is evident that sufficient diluting fluid has not been added and that the reading will be too high. When too large an amount of distilled water has been added, the solution containing the blood may be forced across the partition to mingle with the clear water on the other side, thus again defeating our purpose.

5. With the cover-glass properly adjusted, turn the cylinder so that the portion containing the blood will be toward the light; thus the portion containing only distilled water is over the graduated color prism.

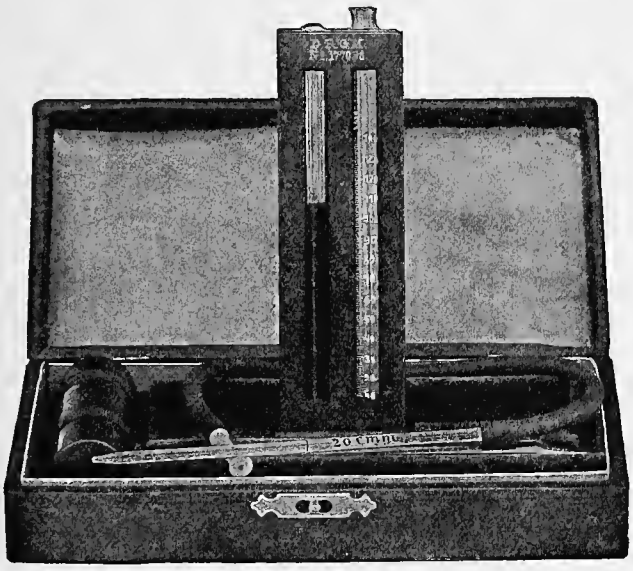


Fig. 144.—Sahli Hemometer.

6. A dark room is necessary for making this estimation, and the candle should be placed directly in front of the instrument, and the plaster-of-Paris reflecting surface so adjusted as to bring the column of light through the solution.

7. The operator should stand at the side of the instrument, bringing his face directly over it, and should read with the eye that is farthest from the light—all direct light from the candle should be excluded from the eye by holding some object on the anterior edge of the stage of the instrument. This may be accomplished by rolling a paper in the form of a tube.

8. The thumb-screw should be turned with short, quick turns, for when it is turned slowly, it is difficult to detect the changes in the color in the diluting chamber.

9. When the percentage of coloring-matter is believed to be extremely low, two or more pipetfuls of blood should be placed in the solution. When

the reading is below 40 per cent. the estimation cannot be made with any degree of accuracy.

Sahli Hemometer.

The Sahli hemometer is composed of a frame divided into two compartments, having a back made of ground glass. A tube containing a standard solution of acid hematin fits in one of the compartments, and the other is occupied by a tube of the same size graduated from 10 to 140. (Fig, 144).

There is a capillary pipet graduated at 20 cubic millimeters, and a larger pipet for adding the diluting fluid, which is decinormal solution of hydrochloric acid. In order to operate the instrument the graduated tube is filled to the 10 mark with the diluting fluid. Blood is then drawn into the capillary pipet up to the 20 cubic millimeter mark, and this is gently blown into the diluting fluid, care being taken to prevent the formation of bubbles. The capillary pipet is then washed out two or three times with the diluting fluid, the washings being added to the contents of the graduated tube. The mixture of blood and diluting fluid is now further diluted with the decinormal hydrochloric acid solution or with distilled water until its color matches the color of the standard tube of acid hematin. The percentage of hemoglobin is then read from the scale on the graduated tube. The advantages of the Sahli hemoglobinometer are, first, that acid hematin is compared with acid hematin; and, second, that the comparison can be made with any form of illumination—daylight, gaslight, electric light, candle light, or lamp light.

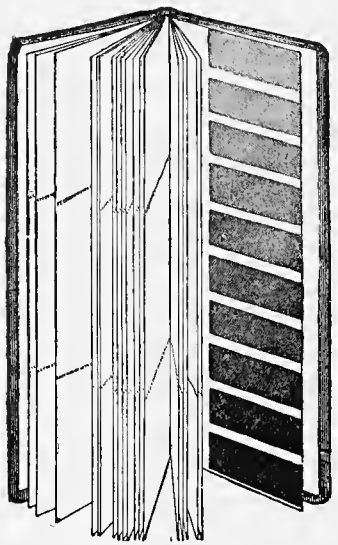


Fig. 145.—Tallqvist's Hemoglobin Scale.

The decinormal hydrochloric acid solution is prepared by adding 15 cubic centimeters of strong hydrochloric acid to enough distilled water to make 1000 cubic centimeters.

Blotting-paper Test for Hemoglobin.

Tallqvist has devised a color scale, which is accompanied by a booklet containing small sheets of a prepared paper. The color scale is graduated from 10 to 100. The summit of a rather large drop of blood is touched with the paper, and as soon as the blood has been distributed over that portion of the paper which it will occupy, the paper is laid beside the color scale, and moved until it matches one of the color blocks (Fig. 145). This method will prove satisfactory for average clinical work, and one with limited experience will obtain better results from this simple method than from the more elaborate methods previously described.

COUNTING OF THE BLOOD-CORPUSCLES.

Method of Thoma-Zeiss.—Among the many instruments devised for this purpose, that of Thoma-Zeiss, with Zappert's modification of the

ruling of the counting chamber, is doubtless the best. The method of using the Thoma-Zeiss instrument is as follows:

1. The part to be punctured is cleansed, and the drop of blood obtained in the manner previously described. In addition it is necessary that the blood be diluted for this purpose, and the following solution will be found satisfactory for the purpose:

Toisson's Mixture.

Methyl-violet	0.025 gm.
Sodium sulphate	8.000 "
Sodium chlorid	1.000 "
Pure glycerin	30.000 c.c.
Distilled water	160.000 "

This solution will preserve the red cells for twenty-four hours, and its specific gravity is such that the cells precipitate slowly. It stains the leukocytes a violet tint. This solution keeps well, but should be filtered

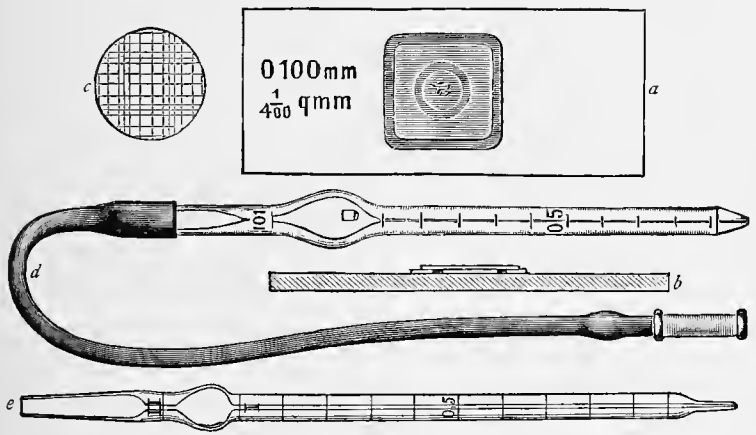


Fig. 146.—Thoma-Zeiss Hemocytometer.

a, Slide used in counting; *b*, sectional view; *c*, portion of ruled bottom of well; *d*, red pipet; *e*, white pipet.

whenever it displays the slightest cloudiness. A 0.5 per cent. solution of glacial acetic acid is used as a diluent when the white cells are to be counted. It should be remembered that acetic acid decolorizes the red cells and renders them transparent, so that, although the microscopic field is filled with red cells, they are not visible. Acetic acid also darkens the leukocytes and renders their protoplasm more granular, which makes these cells more conspicuous.

The pipet for the counting of the red cells consists of a glass tube with an expanded portion near one end, which contains a glass ball. Below this expansion it is graduated into tenths to 1, and above the expanded portion to 101 (Fig. 146). To one end of this tube a piece of rubber tubing is attached, which has a bone nipple at its other extremity.

The pipet for the estimation of the white cells differs only in that it is larger and is graduated in tenths to 1, and then above the expanded portion to 11. It will readily be seen that after the pipet for the red cells is filled with blood to 1, and then the solution is added until it reaches 101, the dilution

of the blood will be 1 part of blood and 99 parts of the diluent, since there are 100 parts of solution between 1 and 101. The rubber tubing is now removed, the tube is shaken for two minutes, and the 1 part in the graduated portion is blown out.

The white pipet, when filled to 1 with blood, and the solution added to reach 11, gives us a dilution of 1 part of blood and 9 parts of the diluent. A dilution of 1 to 20, or even 1 to 40, is more satisfactory when a high degree of leukocytosis exists.

The diluting fluids should be carefully watched, lest they become cloudy or contain any sediment that may obstruct the lumen of the capillary tube. The tip of the pipet is brought into contact with the summit of the drop of blood collected from either the ear or the finger, and gentle suction applied to the rubber tube, the nipple of which is placed between the lips.

When the blood is drawn to the desired graduation, the tongue is placed against the opening of the nipple, and the tube is withdrawn from the drop,

Fig. 147.—Method of Collecting Blood into the Graduated Pipet (Boston).

its tip cleansed with a soft towel, and the end of the tube now immersed in the diluting fluid. Suction is again made, and the pipet rotated rapidly between the thumb and index-finger as the diluting fluid enters—always holding the pipet in the vertical position. The glass ball in the expanded portion facilitates dissemination of the blood throughout the diluting fluid, and when sufficient fluid has been added to reach the 101 graduation, the tongue is again placed over the opening of the nipple, and the tube withdrawn from the solution. The rubber tube should be removed from the pipet, and after shaking for two minutes, the diluting fluid occupying the tube below its expanded portion should be blown out, since it plays no part in the dilution.

Method of Counting.—The next thing to be considered in the process for estimating the number of the blood-cells is the slide, which contains at its center a chamber divided into 400 squares, each of these squares being $\frac{1}{20}$ mm. square and $\frac{1}{10}$ mm. deep, and having a capacity of $\frac{1}{10}$ by $\frac{1}{20}$ by $\frac{1}{20} = \frac{1}{4000}$ c.mm. (Fig. 148). This chamber is surrounded by a narrow channel.

Every group of 16 of these small squares is surrounded by a double row of lines, and is known as a great square (Fig. 148).

There is a special ground cover-glass which must be brought in direct apposition with the slide in order that each of the squares be exactly $\frac{1}{10}$ mm. in depth. The tube containing the diluted blood is rotated rapidly between the thumb and finger for two minutes. From four to eight drops of blood are forced out of the pipet by blowing through the rubber tube. A single drop is made to collect at the tip of the pipet and transferred to the center of the slide. Place the special cover-glass upon the edge of the raised portion

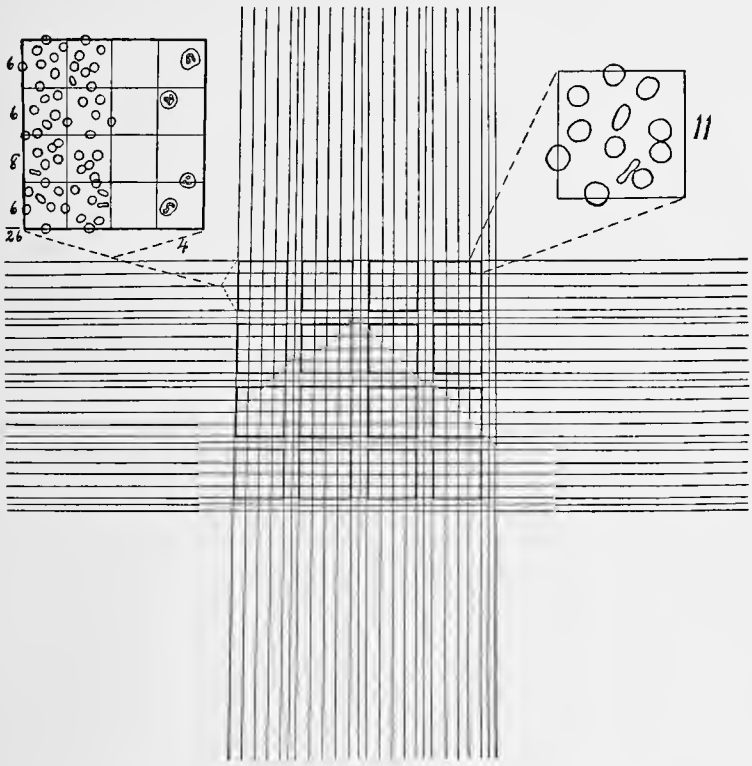


FIG. 148.—THOMA-ZEISS COUNTING CHAMBER.

Capacity, $\frac{1}{4000}$ c.mm. Sixteen great squares, heavily outlined, within the cross-lines, and bounded by double lines. Each great square contains 16 small squares. Each small square is $\frac{1}{10}$ mm. in depth by $\frac{1}{20}$ mm. square; $\frac{1}{10} \times \frac{1}{20} \times \frac{1}{20} = \frac{1}{4000}$ c.mm. Projected to upper left corner is one great square showing arrangement of red cells and number in each small square; also leukocytes in last column. Right hand shows small square containing 11 red cells. The floor of the chamber is ruled into 400 small squares (Boston).

of the slide. The forefingers are placed upon the cover-glass, while the second fingers and thumbs hold the slide at its corners. The forefingers are forced forward, using firm pressure until the cover-glass has passed beyond the opposite margin of the channel that surrounds the graduated chamber.

Counting the Erythrocytes.—In the estimation of erythrocytes all corpuscles touching upon the top and left-hand boundary lines are included in the square, whereas those resting upon the right and bottom lines are to be included in the count of the contiguous squares. (See Fig. 148.) In

this way the cells in the left-hand column can easily be counted, passing to the adjacent right-hand column until the four columns have been counted, which will give the total number of blood-cells for one great square—16 small squares. The slide is now moved, and 4 other great squares are counted in a similar manner. A mechanic stage greatly facilitates this process, yet it is not absolutely necessary.

Having found the number of cells in 5 great squares, we return to the degree of dilution and the capacity of the small squares as the other factors for the estimation of the number of red cells in a cubic millimeter. For example, if the dilution has been 1 in 200, and the number of red cells found in 5 great squares (80 small squares), 87, 95, 93, 86, 89 respectively, a total of 450, then—

450 = number of cells.
$\frac{1}{400}$ mm. = area of small square.
$\frac{1}{10}$ mm. = depth of small square.
$1:200$ = dilution.
80 = number of small squares counted.

Hence $450 \times 400 \times 10 \times 200 = 360{,}000{,}000 \div 80 = 4{,}500{,}000$, the number of cells in 1 c.mm. of undiluted blood. A rapid method of arriving at the number of cells in a cubic millimeter of undiluted blood (dilution 1 : 200) is to add 4 ciphers to the number of cells found in 80 small squares,—450, —which gives us 4,500,000.

Counting the Leukocytes.—The counting of the white cells differs from the method just described for the red cells only in that we are dealing with a much lower dilution of the blood, and that we count 400 instead of 80 small squares. The 400 small squares may often be brought into the field under a two-thirds lens, and when the number of leukocytes is not great, they may readily be counted. When the number of leukocytes is large, however, it is necessary to use a one-fifth lens, beginning at the upper left-hand corner of the slide, and moving the slide gently so as to move each column of great squares until the entire slide has been covered (Fig. 148). Here again the same precaution of counting all cells touching upon the top and left-hand lines, and of omitting those cells resting upon the right and bottom lines, must be observed.

After counting the cells in 400 small squares, diluted 1 : 20, and observing the other rules for estimating the red cells, the direct method for estimating the number of leukocytes in 1 c.mm. of undiluted blood is to multiply the number of cells found in the 400 squares by 200. Suppose the number found to be 35, and the dilution used $1:20: 35 \times 200 = 7000$.

ALKALINITY OF THE PERIPHERAL BLOOD.

The alkalinity of the blood is due to the presence of carbonates, bicarbonates, and albumins held in solution by the acid phosphates, hence it is difficult to estimate the changes in these several elements, and the variations in the reaction produced by the several processes in alkalimetry. When serum alone is used for the estimation (by titration), the alkaline principles of the clot are not included; and if laked blood is used, peculiar chemic changes are produced that depend upon the delicately balanced albumins and phosphates. It has been clearly shown that certain fairly constant alkaline principles exist in the blood and in the serum; these

may be sufficiently closely estimated by the processes about to be described to render this knowledge of clinical value. Again, it is evident that the degree of difference between the alkalinity of normal and that of diseased blood presents somewhat wide variation. During life the reaction of the blood is alkaline, owing to the presence of disodium phosphate and sodium carbonate. Under normal conditions the degree of alkalinity is estimated in terms of sodium hydroxid, and corresponds to 182 to 218 mgm. for every 100 c.c. of blood. Von Jaksch, however, makes a higher estimate of alkalinity —260 to 230 mgm.; whereas Canard places it at 203 to 276.

Estimation of Alkalinity by Dare's Hemoalkalimeter.—Dare's instrument (Fig. 149) consists of a glass tube (*a*) provided with a glass stopper (*b*), through which passes an automatic capillary pipet that tapers at its exposed point. This capillary pipet has a capacity of 20 c.mm. (15 mgm. by weight) of blood. The stopper and the capillary pipet are fitted tightly into the tube (*a*), which is graduated to 3 c.c., to present the equivalents in milligrams of NaOH to 100 c.c. of blood. The upper end of the tube is expanded, and upon this expansion is a minute opening (*c*) for the admission of air. A medicine-dropper graduated at 2 c.c. and provided with a piece of rubber tubing, which is applied to the exposed end of the capillary tube, and serves for the introduction of the test solution. A spectroscope is necessary, and for this purpose Browning's pocket instrument will be found satisfactory.

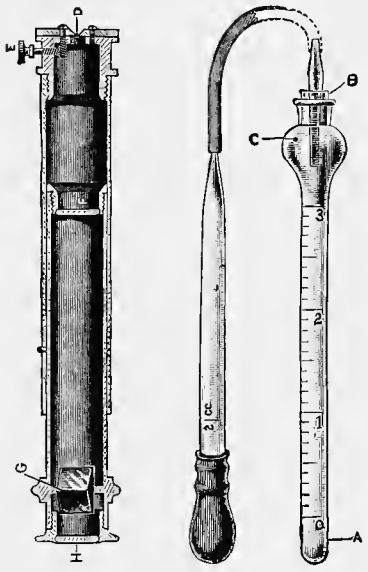


Fig. 149.—Dare's Hemoalkalimeter.

Test Solution.

Tartaric acid (Merck's reagent) 0.075 gm.
Alcohol (94 per cent.) ..... 20.000 c.c.
Distilled water enough to make 200.000 c.c.

Method of Application.—1. Obtain a drop of blood from the tip of the finger; hold the tube horizontally, and permit the exposed tip of the capillary pipet to touch the summit of a fairly large drop of blood, when it fills by capillary attraction.

2. Hold the tube vertically, and wash the contents of the pipet into the tube by forcing the distilled water from a medicine-dropper through the pipet. This washing is continued until the distilled water collected at the bottom of the tube reaches the graduated zero mark (0).

3. Close the opening of the tube by the finger, and invert the tube repeatedly until the blood and distilled water are thoroughly mixed.

4. A dropper is now filled with the acid reagent, and this solution is forced through the capillary pipet into the tube.

Caution.—Cover the opening in the expanded portion of the large tube before releasing the pressure from the rubber bulb of the dropper.

5. Without detaching the dropper, hold it in a vertical position and invert the tube several times.

6. Place that portion of the tube (*a*) below the graduation (0) in the cleft of the spectroscope, and examine for the presence of the absorption-bands of oxyhemoglobin. The acid solution should be added carefully after each examination until these bands disappear. They will be observed to fade gradually as the point of neutralization is approached. Invert the tube (*a*) after each additional drop of the acid solution.

Upon the disappearance of the bands of oxyhemoglobin the test is completed. Note the result from the scale on tube (*a*), which is graduated both in cubic centimeters and in the equivalents expressed in milligrams of sodium hydroxid to 100 c.c. of blood.

SCALE OF EQUIVALENTS COMPUTED FROM A BASIS OF 15 MGM. OF BLOOD TO 2 C.C. OF ACID SOLUTION (ONE TWO-HUNDREDTHS OF THE NORMAL).

Cubic Centimeters of Reagent.	Milligrams of NaOH to 100 c.c. of Blood.
2.6	345.0
2.4	319.0
2.2	292.0
2.0	266.0
1.8	239.0
1.6	212.0
1.4	176.0
1.2	169.0
1.0	133.0
0.8	96.0
0.6	79.0
0.4	53.0
0.2	26.6

Clinical Significance.—**Decrease.**—The alkalinity of the blood is low in women and children during the latter stage of digestion, when the hydrochloric acid and peptones are reabsorbed, and after violent exercise. Pathologically, a reduction occurs in several forms of anemia (primary or secondary or pernicious), leukemia, chronic hepatic disease, nephritis, diabetes, pseudoleukemia, and in the cachexia resulting from carcinoma. High fever, general toxemia, the prolonged use of acids, poisoning by acids or by carbon monoxid, acute mania (stage of excitement), and epilepsy are attended by a low degree of alkalinity. In the last-named disease the reduction begins just prior to a seizure, and continues to fall after the convulsion, varying according to the muscular contraction occurring during the convulsion. The normal alkalinity is restored in from five to six hours after a convulsion.

Increase.—An increased alkalinity is found early during the process of digestion and after a cold bath.

For every 2.2 c.c. of reagent employed, the equivalent of 292 mgm. of sodium hydroxid to 100 c.c. of blood is read from the graduation on tube *a*. Dare employed unshielded gas-light in making his observations.

Glycogen.—Blood smeared upon slides or cover-glasses in the usual manner and dried in the air is stained for from three to five minutes with the following: Iodin, pure, 1 part; potassium iodid, 3 parts; water, 100 parts; pulverized acacia, in excess. In the presence of glycogen a number of small granules of a mahogany-brown tint are seen in the leukocytes, and occasionally in the plasma.

Clinical Significance.—An increase in the number of granules,

as seen in disease, is shown by the presence of intracellular granules. Extracellular granules are possibly derived as the result of degeneration of leukocytes. Neutrophilic leukocytes have been known to contain these granules in cases of leukemia and diabetes, and they have been found in the plasma in other pathologic conditions; in fact, iodophilia may be present in any condition of which anemia is a symptom. The true clinical significance of these granules is not known.

Lipemia.—Normal blood contains between 0.75 and 0.85 per cent. of fat. The presence of fat in the blood may be demonstrated by fixing the films in a 1 per cent. solution of osmic acid for twenty-four hours, and staining for from one-half to one minute with a 0.5 per cent. aqueous solution of eosin. The particles of fat are stained black with the osmic acid, while the remainder of the field takes the eosin stain. Since all granules staining black may not be fat, a control method is necessary, and should be conducted as follows: Fix the film for twenty-four hours in alcohol and ether, and then in the osmic acid for twenty-four hours; counterstain with eosin and extract the fat by ether; the absence of black particles in the cells and plasma is evidence that the blackening displayed by the first specimen was due to fat. Free fat (palmitin, stearin, and olein) may be detected in the blood in health and in disease, but it is usually present in comparatively small amounts, and although recognized with some difficulty under an oil-immersion objective, the granules are at times conspicuous. The quantitative estimation of the blood-fats is not practical for clinical purposes.

Clinical Significance.—The quantity of fat in the blood is increased after a heavy meal and in acute alcoholism. An excess of 0.05 to 0.16 per cent. has been found in the blood of diabetes, 0.1 to 0.5 per cent. in nephritis, 0.15 per cent. in pneumonia, and 0.16 per cent. in typhoid fever. An increased quantity has been observed in starvation, phthisis, fatty embolism, carcinoma of the esophagus, and poisoning by carbonic oxid. A fat-splitting ferment has been detected in the blood. Von Jaksch has demonstrated the presence of fatty acids in the blood of diabetic coma, acute yellow atrophy (hepatic), acute infections, and leukemia.

Glucose.—Normal blood contains a trace of glucose, but the quantity may fluctuate, depending upon a diet rich in carbohydrates (increase); it is also influenced by muscular exercise and hunger (decrease).

The Freezing-point of Blood.—Cryoscopy as applied to the body fluids and secretions, especially to the blood, has as yet yielded but few definite results, and this is probably due to the fact that there are at present no practical methods for determining the freezing-point of blood and urine. Certain clinical changes are said to be accompanied by changes in the freezing-point of both the blood and the urine.

Clinical Significance.—When the metabolic products are retained in the blood as the result of renal insufficiency, its molecular concentration is increased, and consequently its freezing-point is lowered, as has been found in the blood of nephritis, hydronephrosis, pyonephrosis, and experimentally in animals after ligation of the ureters. The freezing-point of the blood is unchanged when, after unilateral nephrectomy, the remaining kidney compensates.

When the freezing-point of the blood is lowered to — 0.58° to — 0.61° C., and both kidneys are diseased, surgical operations of any kind should be deferred until the freezing-point is about — 0.56° C. When the freezing-point of the urine is less than — 0.9° C., this indicates kidney insufficiency, and when

one kidney alone is believed to be affected, the determination of the freezing-point of the urine obtained by catheterization of the ureters or by segregation may be of clinical value. Urine from the diseased kidney will congeal at a higher point than will that from its fellow; *e. g.*, urine from the diseased kidney freezes at $-0.50°$ C., whereas that from a healthy kidney freezes at $-1.75°$ C. Generally speaking, it is impossible to draw definite deductions from the estimation of the freezing-point of the urine, owing to the wide physiologic limits between which it may fluctuate—from $-0.1°$ to $-2.0°$ C. Tinker has recently asserted that cryoscopy is of value as an index to renal insufficiency. The method of Claude and Balthazard has lately appeared in a detailed translation by F. Burthe. Thus far cryoscopy is adaptable for use only by physiologists and is not readily employed by clinicians.

STUDY OF FIXED AND STAINED BLOOD.

Slides and Cover-glasses.

The first step in the preparation of smears or films upon either cover-glasses or slides has been previously described on p. 326. Such spreads are fixed by one of the following methods: The most satisfactory and available method is to use a strip of copper, about $\frac{1}{3}$ by 6 by 15 inches (Fig. 150), placing one end of this over the flame of a Bunsen burner. After the copper has been thoroughly heated, fill a pipet with cold water and drop it on the plate at different distances from the flame. When the water boils, but not sufficiently to cause bubbles that bounce off the stage, place the cover-glasses or slides at these points, specimen side down, and allow them to heat for from twenty minutes to several hours. After fixation the films should be stained at once.

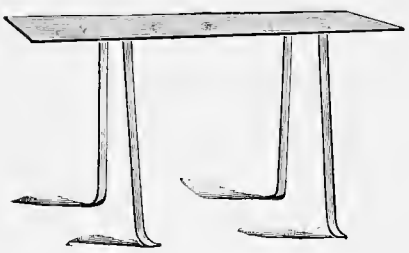


Fig. 150.—Stage for Fixing Blood.

A very satisfactory method of fixation of blood films is to expose them for about fifteen minutes to the action of pure methyl-alcohol. The slide containing the smear is immersed in the methyl-alcohol contained in a suitable vessel. Merck's or Kahlbaum's acetone-free methyl-alcohol should be used.

Staining.

One of the most essential facts in the staining of blood is that the results secured should be obtained by the simplest method possible. In 1891 Romanowsky detailed a method for the staining of malarial parasites by which the chromatin and the cytoplasm were stained differently. Since the appearance of Romanowsky's original paper, others have attempted to perfect similar staining methods. Thus Jenner detailed a most practical stain for blood, and Leishman simplified Jenner's method to some extent. It was not until 1902, however, that J. H. Wright perfected this method and made its practical application possible.

Eosin, Hematoxylin, and Methylene-blue.—The solutions necessary in this method of staining the blood are: 0.5 per cent. eosin in

70 per cent. alcohol; Delafield's hematoxylin; and 2 per cent. aqueous methylene-blue.

Application of Stain.—First stain the specimen with eosin for one-half minute and wash in water. Then, without drying, stain with hematoxylin for from one to three minutes, the time varying greatly with different stains, even though they be prepared in essentially the same manner. Wash and dry the specimen, and mount as previously described.

Wright's Staining Method.—The blood is smeared on slides or cover-glasses and allowed to dry in the air. The spread blood does not stain well after it has been exposed to the air for several months. Although the laboratory worker usually prepares his own stain, the inexperienced will find it better to procure the stain from dealers in laboratory supplies.

1. Add to the specimen enough of an alcoholic solution of the stain to cover the film, and allow it to stand for one minute, in order to fix the corpuscles.

2. To the alcoholic solution of the stain now on the specimen add water, drop by drop, until the stain becomes semitranslucent, and a yellowish, metallic scum forms on the surface. Allow this diluted stain to cover the specimen for two or three minutes.

3. Wash the heavily stained specimen in water until the film of blood presents a yellowish or pink tint to the naked eye.

4. When the desired tint is attained, dry immediately between blotting-paper, lest decolorization be carried too far. The specimen is now ready to be mounted in Canada balsam.

Stained Blood.—The red cells are orange or pink, the nuclei of the nucleated red cells are deep blue. By this stain granular basic degeneration of the red cells is made evident. Polymorphonuclear neutrophilic leukocytes show dark-blue or dark, lilac-colored nuclei, and the granules are of a reddish-lilac color. Lymphocytes have dark, purplish-blue nuclei; cytoplasm is robin's-egg blue, and in it are seen a few dark-blue or purplish granules. Eosinophiles display blue or dark, lilac-colored nuclei. The granules are stained red by the eosin, but the cytoplasm in which they are embedded is of a blue color. Large mononuclears present blue or dark, lilac-colored nuclei. Some of these cells show pale-blue or lilac cytoplasm, whereas others contain, in addition, dark-lilac or deep-purple granules. Mast-cells resemble the ordinary polymorphonuclear leukocytes, and in addition display coarse spheric granules which are stained dark blue, purple, or at times blackish. Myelocytes contain purplish or dark-lilac nuclei. In the cytoplasm numerous dark-lilac or reddish-lilac granules are seen. Blood-plates appear as small round or oval bodies, and are stained blue or purplish. These bodies show irregular margins, and their substance contains fine blue or purplish dots.

SERUM DIAGNOSIS.

Serum diagnosis is based upon a study of the action of blood-serum on pure cultures of known microörganisms. It has been found of the utmost importance in the recognition of disease, although it is only possible, at present, by its means to detect the presence of a limited number of affections. Specific serum reactions have been secured in Malta fever, dysentery, pneumonia, tuberculosis, plague, cholera, paratyphoid fever, and glanders, but as yet their application as a means of diagnosis is not practical in these infections.

The Widal Reaction.

The most characteristic and valuable among the reactions used in serum diagnosis is that known as the Widal reaction, which finds its chief field of usefulness in typhoid and paratyphoid fever.

Method of Collecting the Blood.—Two methods of collecting the blood for the Widal reaction are employed: (1) The dry method, in which a piece of glazed paper is allowed to touch the summit of a drop of blood, which is then dried in the air; (2) the method of collecting the blood in a capillary pipet and diluting it. Wright has suggested a special pipet for collecting the blood.

(1) Draw the blood into the pipet; heat the ends to seal them, after which the pipet and its contents may be transferred to the room in which the test is to be performed. (2) After sufficient time has elapsed to permit the serum to separate from the clot, break off the ends of the pipet and place the serum in a sterile watch-glass. (3) Transfer a portion of the serum to another watch-glass containing saline solution. In this manner dilutions of 1 : 20, 1 : 100, 1 : 500 or more are readily effected.

Culture.—A pure eighteen-hour-old bouillon culture of typhoid bacilli should be employed in the making of the Widal reaction.

1. To blood collected upon glazed paper add 20 to 40 times its quantity of sterile bouillon to the drop; then make gentle friction with a platinum loop over the surface of the drop at its margin, thus assisting in its solution and in mixing the serum with the bouillon. Place a drop of the mixture (blood and

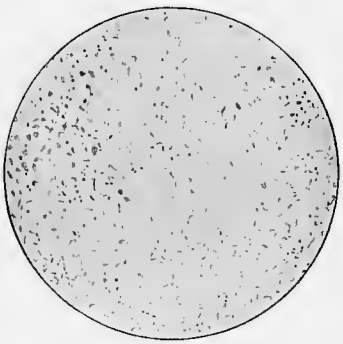


Fig. 151.—Typhoid Bacilli, Unagglutinated (Jordan).

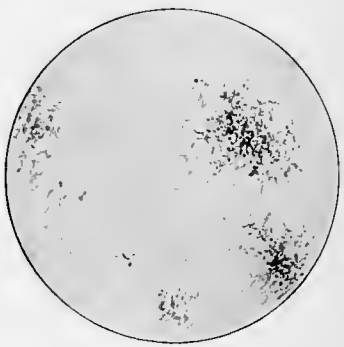


Fig. 152.—Typhoid Bacilli, showing Typical Clumping by Typhoid Serum (Jordan).

bouillon) upon the center of a cover-glass, and to it add a small drop of the eighteen-hour-old bouillon culture of typhoid bacilli.

2. A ring of vaselin is placed at the margin of the excavation on a hanging-drop slide, and then the slide is inverted over the cover-glass. Invert the slide quickly, and the specimen is ready for examination under the microscope.

Characteristics of the Reaction.—The various steps in the reaction are as follows: (*a*) Place the center of the drop of liquid under the objective, when actively motile bacilli will be seen throughout the entire field. (*b*) The first evidence that the reaction is taking place is detected by the

retarding movements of the bacilli. (*c*) A small bacillus attaches itself to the side of a larger organism,—the Y formation,—and later other bacilli are seen to come in contact with these, some of them remaining to form a clump, and others appearing free in the liquid. As the movements of the bacilli lessen, more and more bacilli become attached (Y formation), until there are decided aggregations of bacilli in certain areas throughout the entire drop of liquid. (*d*) The reaction is not complete until all motility of the organisms composing these various aggregations has ceased.

Clinical Significance.—A positive agglutination test, with a 1 to 40 dilution of blood-serum, indicates that the patient now has or has had typhoid fever. A negative agglutination test indicates that the disease from which the patient is suffering is probably caused by some other organism than bacillus typhosus. Pseudo-reactions should be considered negative. They are usually found in infections with other bacilli of the same (typhoid-colon) group—bacillus paratyphosus, bacillus coli communis, bacillus dysenteriæ, etc. A positive reaction may appear late in typhoid and during convalescence.

WASSERMANN AND NOGUCHI REACTIONS FOR SYPHILIS.

(See Syphilis of the Nervous System, p. 1121).

The principles of the serum diagnosis of syphilis will not be discussed and only the technic of the Wassermann and Noguchi reactions is given as briefly as possible. Inasmuch as these reactions differ in the hands of different workers, it is impossible to formulate absolute rules. The reader is referred to the more extensive writings on this subject by Wassermann, Noguchi and Kaplan.* The technic is copied from the latter.

WASSERMANN REACTION.

Equipment.—At least one dozen or more of Mohr's pipets, 1 c.c., graduated into 1-100. One dozen 10 c.c. pipets, graduated into 1-10. One gross of ordinary test-tubes. One gross of test-tubes 1 cm. in diameter, 12 cm. high. One-quarter dozen of graduated cylinders, 50 c.c.; ¼ dozen 100 c.c. Two 50 c.c. measuring flasks with glass stoppers. A few pounds of glass tubing, 5 mm. bore, to make capillary pipets. One-half dozen test-tube racks for Wassermann tubes; ½ dozen test-tube racks for Noguchi tubes. A piece of rubber tubing for tourniquet. One artery clamp for above. One dozen hypodermic needles, 19 bore. One thermostat regulated at 57° and one regulated at 37° C. One electric centrifuge. Labels and pencil for writing on glass. One tall glass for flushing through used pipets, height to be greater than any pipet used. One dozen petri dishes. One dozen beakers, 100 c.c. capacity. Two fine forceps and two Hagedorn needles. One package of quantitative filter-paper. One razor (for killing guinea-pigs). One 15 c.c. Luer syringe.

Technic.—Obtaining blood from patient: A fairly stout piece of rubber tubing is placed a little above the elbow and held in place by an artery clamp. Do not obliterate the pulse. This brings into prominence the veins at the bend of the elbow. To a stout hypodermic needle (Kaplan uses a 19 bore 1½-inch needle) attach a 2-inch piece of rubber tubing. Holding the free end of the rubber tubing in an ordinary sterile test-tube, quickly

* Kaplan, D. M.: American Journal of Medical Sciences, January and July, 1910.

plunge the needle into the most prominent vein; if expertly done, the patient will hardly feel it and the blood will immediately begin to flow. About 6 to 10 c.c. of blood is withdrawn and placed in an ice-box overnight to coagulate. The serum separates and may be pipeted off absolutely clear without cells. It is advisable to take the blood as far from a meal as possible, as proximity to a meal makes the blood lipemic, interfering with perfect working conditions.

Having obtained 1 or 2 c.c. of clear serum, it is placed in a test-tube in the thermostat at 56° for one hour. Care must be taken not to permit the heat to rise too high (over 58°).

After this, 0.2 c.c. is placed in each of two test-tubes, one the test, the other the control.

To each is now added 0.1 c.c. fresh complement.

To the test portion is added one unit of antigen. The control does not receive any antigen.

Each tube receives now 3 c.c. of a 0.95 per cent. NaCl solution. In order to be able to judge properly the correctness of the procedure, the more controls one has the better; it is therefore necessary to compare the serum to be tested with two sera from known positive and negative bloods.

Shake every tube well and place in incubator at 37° or 38° for one hour. During this time, if the serum is luetic the antibodies present will, together with the antigen, bind the complement and render it inactive for hemolysis.

After one hour incubation each tube receives two units of amboceptor and 1 c.c. of a 5 per cent. suspension of sheep cells in 0.95 NaCl. The tubes are again vigorously shaken and placed in the incubator at 37° and inspected after ten minutes. If the reagents are properly adjusted, hemolysis begins in the control tubes in fifteen to twenty minutes, and careful watching becomes a very essential point at this stage of the test. As soon as the control is completely hemolyzed the tubes are compared; only those should be pronounced negative that show a transparent fluid the same as the control.

Permitting the tubes to stand undisturbed in a cool place (15° to 17°) for twenty-four hours shows in the positive test a deposit of red cells, the size of the deposit depending upon the severity of the infection or proximity to the initial lesion as well as upon the degree of balance of the reagents used. Usually a markedly positive serum gives at the end of twenty-four hours a clear supernatant fluid of a light pink hue with a Bordeaux red accumulation on the bottom of the tube. The weaker the reaction, the redder the supernatant fluid and the scantier the deposit of cells. In testing more than one serum, the reaction in each individual test must be considered as finished as soon as the controls are completely hemolyzed, in which case the two tubes are immediately removed to a cool place.

NOGUCHI REACTION.

With a capillary pipet allow one drop of fresh serum to fall into a narrow (1 cm. lumen) test-tube. The pipet is not to be used for any other serum.

Add 0.05 c.c. fresh complement.

To the front row (rear row for control) add one piece (more or less, depending upon the titer) of antigen paper.

Prepare a suspension of human cells, 1 drop of blood to 4 c.c. NaCl 0.95 per cent. It is best to prepare about 60 c.c. of NaCl solution and allow 15

PLATE II

Wassermann reaction: *a*, Tube represents complete hemolysis—the end reaction of *negative* serum; *b*, represents partial hemolysis, some cells being hemolyzed, this representing a tube of a strongly positive "3 plus" (+++) serum after standing to sediment over night; *c*, represents inhibition of hemolysis—of a *very* strongly *positive* serum—"4 plus" (++++) after standing over night to sediment.

drops of blood to fall from the experimenter's finger into the solution. The human cell suspension is placed overnight in the ice-box. Next morning the supernatant clear salt solution is pipeted off and a fresh quantity of NaCl is added (about 55 c.c.) to the cells in the beaker. Of this cell suspension add 1 c.c. to every tube in the rack.

Incubate for three-quarters of one hour at 38° or 39°, preferably in a large dish of warm water. Occasionally shake the tubes, to insure proper solution of the biologic substance on the antigen paper.

Add to each tube (after incubation), front and back rows, one piece of amboceptor paper (more or less, the quantity depending upon the titer) and replace in the incubator, observing the result after ten minutes, and watching carefully the controls.

It will be noted in about fifteen minutes, more or less, that the rear row begins to get clear, and when complete transparency is obtained the test and control tubes are to be removed to a cool place and observed. If the reaction is positive, then the front tube (test) will be opaque, in marked contrast to the control, which is transparent. For convenience of observation, Kaplan makes use of a fine sealed tube (about 1 mm. in diameter) filled with black ink, which, when placed behind the control, will appear as a clear black line, whereas the positive tube will not show the black line, or it appears as a dim shadow—depending upon the strength of the reaction.

It has been stated that a positive Noguchi test and a negative Wassermann is often due to the presence in the patient's serum of antisheep amboceptors. It is not necessary to perform this test with every serum as a control. Only sera giving the above results need be subjected to a verification. To demonstrate the antisheep amboceptor, place 1 c.c. of a 5 per cent. suspension of sheep cells in a test-tube, add 0.2 c.c. of patient's serum and 0.1 c.c. complement, add 3 c.c. of NaCl solution, place in incubator, and observe. If the amboceptor is present, the cells will dissolve and the mixture become transparent. The time consumed depends upon the number of amboceptor units present. Kaplan observed sera capable of hemolyzing the cells completely in ten minutes.

Rationale of Controls.—In the Wassermann and Noguchi reactions it is of vital importance to have every possible error excluded. For this purpose the controls used will answer. The substances to be controlled are the antigen, the amboceptor, and each individual serum.

The Antigen Control.—This biologic reagent, as is known, can *per se* inhibit hemolysis. To measure the degree of such interference, a tube containing a well-known normal serum (or, as Kaplan found just as serviceable, no serum at all), plus antigen, plus complement, and antisheep amboceptor plus sheep cells ought to hemolyze in about twenty or thirty minutes. No reaction is to be considered as finished before the antigen control tube is completely hemolyzed.

The Amboceptor Control.—Upon the efficiency of the antisheep amboceptor depends the rapidity of hemolysis of the sheep cells. It is therefore necessary to establish the amboceptor efficiency in a separate tube containing sheep cells, plus complement, plus antisheep amboceptor. It is not essential to add normal serum. The tube containing the above ingredients is always the first to hemolyze, requiring about fifteen or twenty minutes for a complete hemolysis.

Control for Each Serum.—Every serum more or less has the power to interfere with hemolysis to a slight degree. In order to control the factor

of inhibition, every serum tested is placed in each of two tubes; the front tube contains the antigen and all other biologic reagents, the rear tube receives everything but the antigen. This shows the degree of individual inhibition as compared with the tube containing the amboceptor control.

Efficiency of the Entire System.—For this a well-known luetic serum is utilized. The reaction is to be positive, and hemolysis should not occur in the front tube, even if exposed to incubation temperature for hours after the controls hemolyzed.

Opsonins and the Opsonic Index.

Consideration.—This clinical method is based on the fact that certain substances present in the circulating blood or in the blood-serum render various bacteria subject to phagocytosis. Among the organisms that are affected are pathogenic streptococci, staphylococci, meningococci, gonococci, pneumococci, anthrax bacillus, tubercle bacillus, influenza bacillus, bacillus of diphtheria, bacillus pestis, the colon bacillus, typhoid bacillus, and numerous other organisms. The normal human blood-serum displays opsonic influence upon the bacteria above named, but it is worthy of special attention that in the case of certain bacteria phagocytosis is but feeble or does not occur, unless the bacteria present are not highly virulent. Again, phagocytosis may take place with certain bacteria (bacillus subtilis) in normal salt solution and in the absence of blood-serum.

It may be well to call the reader's attention to the fact that the chemical nature of the opsonins is unknown, and likewise the structure of the opsonins remains in question. Opsonins occur in practically all vertebrates, and actual experiments have shown that serum obtained from the guinea-pig, calf, rabbit, sheep, swine, dog, cat, chicken, frog, and turtle is capable of activating certain microörganisms for phagocytosis by leukocytes obtained from animals of a different species. In addition to bacteria, other cells are also capable of being opsonified; Hektoen having observed that the blastomycetes obtained from a human lesion became surrounded by leukocytes in the presence of human serum and of dog serum. The same author calls attention to the phagocytosis of trypanosomes, and Lavtchenko and Melkich observed pronounced phagocytosis of the spirochæta of relapsing fever while using the serum of convalescent patients. Barrath has observed the presence of opsonins for the red blood-cells.

Clinical Significance.—Wright and others have observed that during the course of certain bacterial infections the opsonins are at times diminished, and that by the use of bacterial vaccines it is possible to raise the opsonic value and thus to increase the patient's resisting power to the bacterium in question. From the foregoing statement it will be seen that a knowledge of the opsonic index gives an idea of the resisting power of the patient to the action of a specific organism, and may further indicate to the clinician whether treatment with a bacterial vaccine should be instituted. It has been satisfactorily demonstrated that bacterial vaccines represent a most important part as a therapeutic agent, and indeed it is often possible, in selected cases, to hasten improvement by the use of these vaccines. In chronic bacterial infections, both superficial and deep, vaccines may be found of inestimable value. The opsonic index serves as a guide to the administration of bacterial vaccines in these conditions. According to Wright, the injection of vaccines is likely to be followed immediately by a

decrease in phagocytosis—the so-called "negative phase"; and this feature, in addition to being variable as to degree, also depends, in part, at least, upon the amount of vaccine injected. Next in order follows an increase in phagocytosis, the so-called "positive phase," and coincidentally with this increase a corresponding improvement in the patient's condition is expected.

A low opsonic index is to be expected in cases of chronic infection, and especially is this true when such an infection is more or less completely localized. A high opsonic value is also to be expected in connection with acute cases that display well-marked systemic manifestations, but such high indices frequently alternate with comparatively low ones. Truly diagnostic values are possible when there is a distinct deviation from the normal value in relation to a special microörganism. Wright has written extensively concerning the diagnostic value of the opsonic index. The following summary of Wright's conclusions concerning the significance of the opsonic index in tuberculosis is taken from Simon:

(*a*) Deductions to be made when one has at his disposal records of a series of blood examinations made from the patient in question:

"(1) When a series of measurements of the opsonic power of the blood reveals a persistently low opsonic power with respect to the tubercle bacillus, it may be inferred, in the cases where there is evidence of a localized bacterial infection which suggests tuberculosis, that the infection in question is tuberculous in character.

"(2) When repeated examination reveals a persistently normal opsonic power with respect to the tubercle bacillus, the diagnosis of tuberculosis may with probability be excluded.

"(3) When there is revealed by a series of blood examinations a constantly fluctuating opsonic index, the presence of active tuberculosis may be inferred.

(*b*) Conclusions to be drawn from a single blood examination:

"(1) When an isolated blood examination reveals that the tuberculo-opsonic power of the blood is low, we may—according as we have evidence of a localized bacterial infection or of constitutional disturbance—infer with probability that we are dealing with tuberculosis—in the former case with a localized tuberculous infection, and in the latter with an active systemic infection.

"(2) When an isolated blood examination reveals that the tuberculo-opsonic power of the blood is high, we may infer that we have to deal with a systemic tuberculous infection which is active, or has recently been active.

"(3) When the tuberculo-opsonic power is found normal or nearly normal, while there are symptoms which suggest tuberculosis, we are not warranted, apart from the further test described below, in arriving at a positive or a negative diagnosis."

Whenever a certain serum is found to retain its power of inciting phagocytosis after it has been heated to 60° C. for ten minutes, it is reasonably fair to conclude that "incitor elements" ("immune opsonins") have been produced in the organism either as the result of resistance to autointoxication which took place spontaneously during the course of tuberculosis, or followed the artificial stimulus given through the inoculation of tubercle vaccines. It is the consensus of opinion among workers with this clinical method that diagnostic importance attaches itself only to the results obtained through repeated examinations, and only when there is especially pronounced deviation from the normal. It would appear to us from the literature at hand that a single observation is of comparatively trivial importance in the diagnosis of either acute or chronic infections.

At present there appears to be considerable variation in the results attained through the work of different investigators, consequently we find it impracticable to draw deductions as to the actual clinical assistance a knowledge of the opsonins contributes toward the diagnosis of both acute and chronic maladies.

Technic.—The materials necessary for the determination of the opsonic index are as follows: (1) Patient's serum; (2) normal control serum; (3) washed corpuscles; (4) bacterial emulsion.

The Patient's Serum.—To obtain the patient's serum a few drops of blood (6 to 8) from the peripheral circulation are collected by means of a pipet and transferred to a small glass tube, which is then sealed in the flame of a Bunsen burner. After coagulation has taken place the coagulum is separated from the walls of the tube, and by means of the centrifuge the serum is separated from the corpuscles. The serum should not be more than twenty-four hours old when used.

The Normal Control Serum.—The normal control serum is obtained from a normal individual in the same manner as the patient's serum. It is, however, advantageous to mix equal quantities of sera from three or four persons. There are certain factors which have some influence on the opsonic content of the blood. For this reason the sera of women during menstruation, of hard smokers, and of those who are pale and under weight should not be taken as control. It is preferable to take the blood of the patient and the normal at approximately the same time of day.

The Washed Corpuscles.—For this purpose a small amount of blood is collected in a tube containing some substance which will prevent coagulation. The fluid is then centrifugalized. The supernatant fluid which collects is next removed, and an equal quantity of 1.2 per cent. saline solution replaced. This is repeated, and then the fluid is again removed. The superficial layer of corpuscles is opaque and consists principally of leukocytes (leukocytic cream). Wright uses only this layer of corpuscles. Others, however, shake until the cells are thoroughly mixed.

Bacterial Emulsions.—Perfectly uniform bacterial emulsions cannot, as a rule, be secured. Wright recommends that the emulsion should be of such thickness that one or two organisms are found on an average for each cell. Emulsions made from the different bacteria require special bacteriologic technic. (See special works on laboratory methods.)

Having obtained the four requisites, we may proceed to determine the opsonic index. For this purpose equal quantities of the patient's serum, washed corpuscles, and bacterial emulsion are mixed together. The control test differs in no way from that just described, except that normal serum instead of the patient's serum is employed. It is then incubated at 37° C. for about fifteen minutes. A drop is next mounted upon a clean slide and stained without previous fixation (except tubercle specimens), preferably with a 1 per cent. aqueous solution of methylene-blue. Tubercle specimens require a different technic.

Determining the Opsonic Index.—Before determining the opsonic index we obtain: (1) The bacillary index; (2) the percentage index.

To obtain the bacillary index it is only necessary to determine the average number of organisms seen within a series of 50 or more polymorphonuclear leukocytes.

The percentage index is obtained by merely noting the percentage number of phagocyting cells.

In obtaining the opsonic index two methods are in use. Wright's method is based upon a comparison of the bacillary index of the patient with that of the normal control serum, the latter taken as one. It may be expressed by the following equation:

$$NBi : 1 :: PBi : x,$$

in which NBi represents the bacillary index of the normal control serum, PBi the bacillary index of the patient, and x the opsonic index. For example, if the bacillary index of the normal control serum was 10 and that of the patient was 5, then—

$$10 : 1 :: 5 : x = 0.5 \text{ opsonic index.}$$

On account of certain fallacies to which Wright's method is open a second method has been brought forth. The opsonic index is obtained by comparing the percentage index of the patient with that of the normal control serum, the latter being also taken as one. For example, if the percentage index of the normal control serum was 50 and that of the patient was 70, then—

$$50 : 1 :: 70 : x = 1.4 \text{ opsonic index.}$$

DISEASES OF THE BLOOD.

ANEMIA.

A condition in which there is a deficiency in the red cells or the hemoglobin, with or without change in the number of leukocytes. Clinically, anemia may be divided into two great classes:

Primary anemia, in which the exciting cause is believed to affect the blood-making organs primarily.

Secondary anemia, in which the abnormalities of the blood are attributable to some previously existing disease, such as chronic suppuration, heart, renal, and gastric disorders.

The secondary anemias are of two types:

(1) The chloro-anemias, in which there is a moderate reduction of red cells, a greater reduction in hemoglobin, with a low color-index, and little or no change in the leukocyte formula, except a possible increase in the polymorphonuclear neutrophile cells. This variety of anemia is the usual one found in cases of prolonged discharge of pus, cardiac disease, renal disease, malignant disease, etc.

(2) The secondary anemias of the pernicious type, in which there is a great reduction in the number of red cells, a less reduction in the hemoglobin, with a high color-index, little or no change in the leukocyte formula, and the presence of nucleated red cells in the peripheral blood. This type is sometimes seen accompanying carcinoma, sarcoma, after severe malarial infection, after hemorrhage, and in cases of infection with dibothriocephalus latus.

Oligocythemia is a term applied to a diminution of the number of erythrocytes in a cubic millimeter of blood. It may be moderate, when the cells number from 4,500,000 to 3,000,000; marked, when they are between 3,000,000 and 1,500,000; and excessive, when they are below 1,500,000. Oligochromemia is a term applied to a diminution in the hemoglobin percentage. This may also be moderate, marked, or excessive.

The histologic study of the stained blood will give important data for diagnostic purposes.

The normal erythrocyte is spherical. It varies from 7.2 to 7.8 microns in diameter; stains pale pink with eosin, and presents a pale area in its center, due to the normal concavity of the cell.

If the cells are deficient in hemoglobin, this central pale-staining area is larger than normal, and it may be eccentric in position and somewhat distorted in shape.

If the cells vary markedly in size, we have a condition known as **anisocytosis.** A red cell which is smaller than normal is called a **microcyte;** one which is larger than normal is called a **macrocyte.**

If the erythrocytes are altered in form so that oval, elliptic, pear-shaped, club-shaped, and other irregular forms are seen, we have a condition known as **poikilocytosis.** The deformed cell is called a **poikilocyte.**

If the erythrocyte, instead of staining pink with eosin, stains purple, on account of taking the methylene-blue stain as well as the eosin, the condition is known as **polychromatophilia.**

Many erythrocytes show small blue dots in their cytoplasm when stained with eosin-methylene-blue combinations. These dots may be uniformly distributed throughout the cell; they may form a ring around its circumference; they may be collected into two or three groups, or they may be irregularly distributed throughout the cell. This condition is known as **basophilic degeneration,** or **granular degeneration.**

Nucleated red cells are often found in the peripheral blood in pathologic conditions. A nucleated red cell which is normal in size; has a normal staining cytoplasm; and a single, double, or triple nucleus, is called a **normoblast.**

A nucleated red cell, which is larger than a normal erythrocyte; has a single large vesicular nucleus; and a polychromatophilic cytoplasm, is called a **megaloblast.** These cells sometimes show karyokinetic figures.

A nucleated red cell which does not present all the histologic characteristics of a normoblast, on the one hand, or a megaloblast, on the other hand, is called an **intermediate.** Microblasts are small nucleated red blood-cells.

Clinical Significance.—Anisocytosis, poikilocytosis, polychromatophilia, and basophilic degeneration of the erythrocytes are seen in all of the severer forms of anemia, whether primary or secondary. The most noticeable degrees of poikilocytosis and anisocytosis, however, are found in progressive pernicious anemia. Basophilic degeneration of the erythrocyte is found earliest and in most noticeable amount in the anemia of lead-workers. It has been shown to be present before symptoms are manifested.

Normoblasts are found in all severe anemias, most commonly in the post-hemorrhagic cases and in progressive pernicious anemia. They are likely to appear in the peripheral blood suddenly and in large numbers, and to disappear suddenly; such a phenomenon is called a **normoblastic shower.** Normoblasts are considered by many writers to indicate an attempt at regeneration on the part of the bone-marrow.

Megaloblasts may be found in all the severest anemias in small numbers—one or two to 500 leukocytes. Their constant presence in large numbers is seen in progressive pernicious anemia only. The absence of megaloblasts is not conclusive evidence of the absence of progressive pernicious anemia; particularly when all the other features of the blood-picture point to that disease, the failure to find megaloblasts may be disregarded.

POLYCYTHEMIA.

Pathologic Definition.—A condition characterized by the presence, in the circulating blood, of an excessive number of red corpuscles.

Varieties.—In the majority of instances polycythemia is **physiologic,** *e. g.*, it follows physical exercise with profuse perspiration, and hot baths; it occurs during pregnancy, and at altitudes above 4000 feet.

Pathologic polycythemia is associated with bronchial asthma, emphysema, cardiac insufficiency, pneumonia, pleural effusion, ascites, and conditions causing obstruction to the return circulation.

Again, polycythemia may be either **general** or **local.** General polycythemia of obscure origin is quite common. Anesthesia (chloroform and ether) and the prolonged use of such drugs as antikamnia, phenacetin, acetanilid, "headache powders," etc., are followed by general polycythemia.

The exact physiologic effect of acetanilid in the production of this cyanosis has not been thoroughly proved, but certain conditions probably take place: first, the heart muscle may be involved, and consequently the organ will have inadequate power to force the blood through the capillaries; second, the capillary system may be relaxed, and the polycythemia at first involve the capillaries, while later, from obstruction offered to the circulation, general polycythemia may follow. There are many other theories that might be advocated, among which should be mentioned circulation through the lungs and through the kidneys, either or both of which may be materially interfered with by the use of acetanilid or similar preparations. That the capillary circulation of the brain is materially interfered with, and that there is also a storing-up of some deleterious substance in the brain tissue, is evident from the fact that mental dullness is a common symptom in both chronic and repeated acute intoxications with acetanilid.

Infants present normally a polycythemia of from 5,444,000 (Stengel and White) to 7,000,000 (Emerson).

Blood taken from a portion of the body which shows cyanosis will contain more than 5,000,000 corpuscles per cubic millimeter, a condition which may be termed **local polycythemia.**

Physical Signs.—On **inspection** the complexion is often florid, the cheeks are of a bright reddish hue, and the skin in general appears to be too ruddy. In a few instances persons of normal complexion are found to have too many red cells, but such individuals are, as a rule, fat and muscular. When cyanosis is general, the skin and mucous surfaces assume a peculiar blueness that is slightly changed by pressure, and which disappears gradually when the obstruction to the circulation is removed.

In cyanosis the patient's attitude is that of exhaustion; the respirations are shallow and rapid, the lips are separated, and he is often found sitting up or propped up in bed. Epigastric pulsation, due to dilatation of the right heart, and pulsation at the third rib on the left, due to dilatation of the left auricle, are to be seen. The nails of the fingers and toes show a peculiar reddish and at times present a bluish-black hue. Cyanosis often involves but one or two fingers of the one hand, or it may affect but a single extremity. In the majority of instances, however, it becomes general sooner or later, and in such maladies as asthma and valvular heart disease it becomes chronic.

Upon palpation the skin is smooth to the feel, and there may be some evidence of edema of the extremities, lips, and eyelids.

Laboratory Diagnosis.—The laboratory findings in physiologic

polycythemia are quite uniform, and consist chiefly in an increase in the number of red cells. The hemoglobin is likewise increased. While the leukocytes may show moderate increase, in many instances they remain normal in number, and, in fact, cases have been cited in which the number of white cells was subnormal (leukopenia).

In the polycythemia of obesity and of plethora the individual red cells are found to contain an excess of hemoglobin, yet the size and form of such cells remain normal. The specific gravity of the blood is increased. Vaquez has found that the total nitrogen content of the blood-serum is increased, a fact probably dependent upon the presence of hemoglobin in solution. The whole blood is also richer in nitrogen than normal, but the red cells when separated from the entire blood display a deficiency in nitrogen.

In most forms of polycythemia the urine is of a high color and of high specific gravity, with an excess of uric acid and oxalates.

Summary of Diagnosis.—Chronic polycythemia is oftenest encountered in those cases in which there is some defect in the circulation, and it develops after the extraction of a large quantity of liquid from the body, as is seen in cholera, diabetes, etc. Polycythemia, the result of disturbances in the arterial tension, is also to be seen after extensive burns of the skin; thus Locke reports an instance in which the red cells increased 4,000,000 in a cubic millimeter following an extensive burn. An increase of 2,000,000 in a cubic millimeter is to be expected in moderate burns.

Anesthesia also causes polycythemia, which is, as a rule, local, although it may be general; an increase of 1,000,000 or more red cells in a cubic millimeter is often seen after the administration of either chloroform or ether, but at this time the percentage of hemoglobin is reduced.

Pathologic polycythemia is to be found after the ingestion of poisons, *e. g.*, phosphorus, carbonic oxid, and during diseases displaying chronic cyanosis.

Altitude exercises a decided influence upon the number of red cells in a cubic millimeter, and it is the rule to find the number of red cells above the normal in individuals residing at an altitude above 1000 feet. This increase is proportionate to the rise in altitude, until, at 14,000 feet, the normal number of red cells in a cubic millimeter has been found to be 8,000,000.

Certain drugs taken for prolonged periods give a peculiar dull or dusky color to the skin, which is not infrequently accompanied by polycythemia; in a number of cases observed by us there were neurasthenia, anorexia, and insomnia, with a moderate enlargement of the liver and the spleen. In these cases it appeared that the polycythemia and associated conditions resulted from the use of some one, or in many instances of several, of the coal-tar products, particularly from the continued use of headache powders.

Polycythemia resulting from the prolonged or excessive use of acetanilid may show peculiarities in the hemoglobin, yet the evidence furnished by the literature appears to be conflicting. Blood smeared upon slides and permitted to dry in the air develops a peculiar dull, dusky, lusterless color, which is best demonstrated by preparing a similar film of normal blood and subjecting it to the same treatment.

SECONDARY ANEMIA.

Definition.—Secondary anemia is a condition that may result from a variety of maladies, which, after a variable period of existence, produce

moderate alterations, and at times destructive changes, in any one or in all the elements of the blood.

Varieties and Causes.—For convenience of study, secondary anemias are classified according to their etiologic factors, and they will be here considered in the order of their importance and frequency of occurrence.

Hemorrhage is by far the commonest cause of secondary anemia, and since it occurs under a great variety of circumstances, this factor must be considered in conjunction with both physiologic and pathologic processes. It would at first appear that hemorrhage the result of surgical operation would constitute the commonest cause of secondary anemia; this, however, is not the case, but, on the contrary, the vast majority of conditions associated with secondary anemia are in no way dependent upon hemorrhages resulting from surgical intervention. Menorrhagia, metrorrhagia, postpartum hemorrhage, hemoptysis, bleeding from the mucous surfaces, as is seen in gastric and duodenal ulcer, hemorrhoids, and intestinal parasites, *e. g.*, necator americanus, constitute the common sources of hemorrhage. The repeated small bleedings that occur in these conditions eventually so impoverish the blood that hemorrhages from the mucous surfaces become more and more common.

Inanition.—In all instances in which inanition figures prominently there is a decided secondary anemia, which may be due to food which is either insufficient in quantity or poor in quality. Again, an abundance of nutritious food may be taken, and yet, owing either to defective digestion or to incomplete assimilation or both, the individual may derive but little nourishment therefrom.

Grave secondary anemia occurs during the course of chronic gastritis, gastric and esophageal carcinoma, and other conditions that interfere with peptic digestion.

Elimination of Albumins.—The discharge of a large amount of albumin from the system, as occurs in both acute and chronic nephritis, prolonged lactation, dysentery, chronic suppuration, etc., causes anemia through constant depletion.

Toxic Agents.—Poisons, either organic or inorganic, when taken in sufficient amounts, give rise to secondary anemia. The inorganic substances that commonly excite secondary anemia are phosphorus, lead, arsenic, and mercury. (See Chronic Plumbism.) Anemia is also caused by the toxins of both acute and chronic infectious diseases, and is seen to follow scarlet fever, diphtheria, typhoid fever, acute articular rheumatism, and such chronic maladies as tuberculosis, syphilis, etc.

Parasitic Anemia.—When the human economy becomes infected with animal parasites, a variable degree of anemia follows. An extreme type of secondary anemia occurs after infection with the hook-worm (necator), and, in fact, it is this variety that simulates essential or idiopathic anemia most closely. Infection with the tape-worm, particularly the dibothriocephalus latus, tænia solium, and tænia mediocanellata, are also followed by serious blood changes. In children the round-worm and the pinworm often induce anemia, and the condition also results from infection with flagellates and with the Amœba coli. Protozoa in the blood, as in malaria and trypanosomiasis, are frequently a cause of extreme anemia.

Chief Complaints.—Most prominent are dyspnea, cardiac palpitation upon slight exertion, headache, progressive weakness, anorexia,

indigestion, and mental fatigue. The patient does not feel rested after a night's sleep, and in many instances insomnia forms a conspicuous and troublesome symptom.

Hemoglobinemia is occasionally observed in secondary anemia, and is a condition in which the hemoglobin of the red cells is dissolved and escapes into the serum. In hemoglobinemia the percentage of hemoglobin, as estimated by the hemoglobinometer, is normal, or may, as seen in one of our cases, reach 100 per cent.

When the red cells are studied individually, they are found to be deficient in hemoglobin, and occasionally erythrocytes that are practically devoid of coloring-matter in their protoplasm are to be seen. The serum into which the hemoglobin has been given off by the red cells is highly stained with blood-pigment, and lends a characteristic appearance to the stained blood.

Physical Signs.—On **inspection** the body usually appears emaciated, although it may be well nourished, and the lips and conjunctivæ are pale. The skin of the extremities is also pale, but when the anemia is extreme, it may be cyanosed. In secondary anemia in which there is disease of the abdominal viscera or of the suprarenal body, the skin is dark and brownish, but in such cases the conjunctiva serves as a true guide to the degree of pallor. The respirations are rapid on exertion.

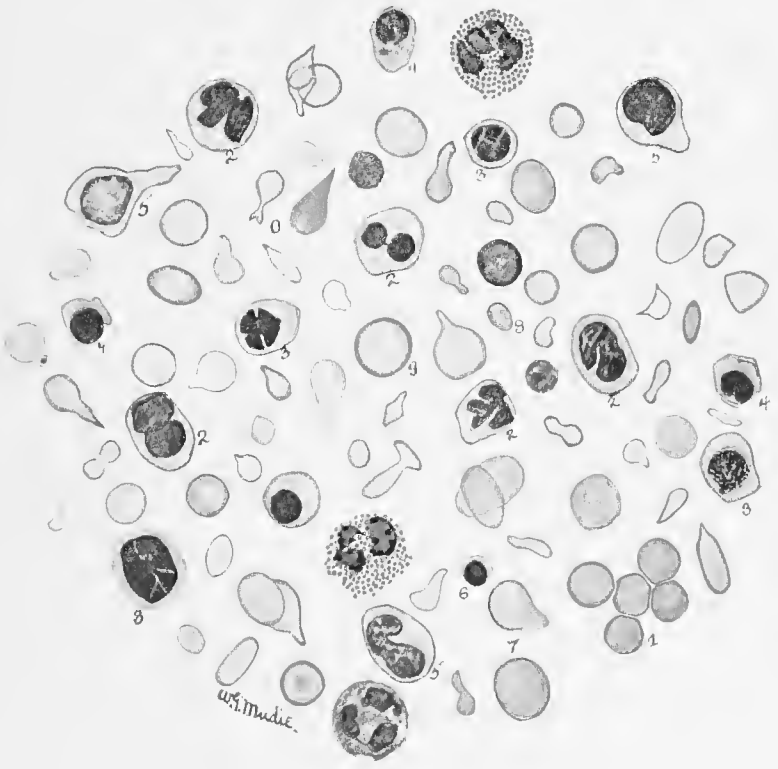
Auscultation discloses the presence of a soft systolic murmur over the base of the heart. The murmur over the precordium is regarded as hemic, since it is not transmitted, and also disappears as the blood condition improves.

Laboratory Diagnosis.—Upon pricking the finger the drop of blood that exudes is more or less pale and watery, and when smeared between cover-glasses, it does not display the normal adhesiveness (viscosity).

Hemoglobin.—There is a corresponding reduction in both the hemoglobin percentage and in the number of red cells, the fall in hemoglobin preceding the reduction in the red cells. In average cases of anemia the hemoglobin may vary between 75 and 40 per cent., but we have found it to be much lower in the anemia following hemorrhage and in that due to animal parasites. Hemoglobinemia, in which the hemoglobin has been dissolved from the red cells by the plasma, is to be seen in certain secondary anemias, and particularly in those due to poisoning by gases. It follows the introduction of snake venom into the system.

A drop of fresh blood, when placed under a $\frac{1}{12}$ oil-immersion objective, will show lowered viscosity, a fact that is evidenced by failure of red corpuscles to appear in coil-like rolls or piles—the so-called rouleaux formation; on the contrary, however, the erythrocytes are disseminated equally throughout the field. When the anemia is due to the ingestion of mineral poisons, the viscosity may be increased, and instead of appearing in rouleaux, the red cells are seen to form densely aggregated masses (hyperviscosity). It is the abnormal degree of viscosity and the specific gravity of the serum in which the corpuscles are suspended that cause the red cells to give up their hemoglobin; hence the earliest changes characteristic of anemia are: Pallor, uneven distribution of the hemoglobin areas, a basic degeneration, and swelling and distortion of the erythrocytes. There is also irregularity in the form and size of the red cells, many abnormally large cells (macrocytes) and also extremely small cells (microcytes) being present. The large red cells are greatly distorted and assume various forms and shapes (poikilocytes).

PLATE III



Various Forms of Erythrocytes (Boston): 1. Normal erythrocytes; 2, karyokinetic changes in the nuclei of erythrocytes; 3, pigmentation of the nuclei of erythrocytes; 4, polychromatophilia in nucleated erythrocytes; 5, megaloblasts; 6, microblast; 7, polychromatophilia of macrocytes; 8, microcytes; 9, macrocytes; 10, poikilocytes. At the lower margin of the picture is seen a basophilic leukocyte. (From blood of a child studied at Pennsylvania Hospital. Obj. B. and L. one-twelfth oil-immersion.)

Erythrocytes.—The train of events in a secondary anemia is possibly something like the following theoretic outline: As a result of the action of a toxic substance which circulates in the blood from the seat of the disease, or as a result of a continued drain on the body-fluids, the hemoglobin and the number of erythrocytes in the peripheral blood are depressed. In order to compensate this loss the bone-marrow puts out into the peripheral circulation a larger number of erythrocytes, each one of which carries a somewhat smaller load of hemoglobin. This would explain those cases in which the erythrocytes are above 5,000,000 with a low color-index. As the deleterious action continues the depression of both hemoglobin and number of erythrocytes progresses until, finally, the very low cell-counts and hemoglobin percentages are reached. The change in size (anisocytosis), in shape (poikilocytosis), and in tinctorial characters (polychromatophilia) go on with the increase in the action of the pathologic substance. In the severe cases, or in cases of hemorrhage in which considerable blood is lost, nucleated red cells (normoblasts) appear in the peripheral blood, and are looked upon as evidence of an attempt on the part of the red marrow to replace the lost elements. In the severest cases megaloblasts appear in the peripheral streams, and in some instances this is to be looked upon as evidence of reversion to the fetal type of erythrocyte production, a true degeneration of the bone-marrow.

Leukocytes.—In secondary anemia the number of leukocytes may be either normal or greatly increased, and, rarely, they are diminished. An increase in the number of leukocytes (leukocytosis) of from 10,000 to 20,000 is the rule in the secondary anemias dependent upon acute inflammatory processes.

Stained Blood.—Some of the large red cells are but feebly stained and may appear as mere shadows. Again, an occasional cell will be stained a purplish hue, taking the eosin red and the methylene-blue stains. This phenomenon results after a portion of the protoplasm of the red cells has given up a liberal percentage of its hemoglobin, but yet retains sufficient to take some of the eosin stain, while the portion from which the hemoglobin has been extracted is stained by the hematoxylin. This peculiarity in staining is termed polychromatophilia, and, with the other features of degeneration of the red cells, is shown in Plate II. Distorted erythrocytes (poikilocytes) are common.

Nucleated red cells are an unusual finding in secondary anemia, appearing only in the severer types; they are most commonly encountered in post-hemorrhagic anemia, in the anemia of intestinal parasites, and in that of lead workers, although we have repeatedly found them in the anemia of tuberculosis and syphilis.

Nucleated red cells which are the size of the normal erythrocytes are termed normoblasts. Rarely, one sees an abnormally large nucleated red cell (megaloblast). Nucleated red cells that are smaller than the normal red cells are known as microblasts. It should be further stated that any one or all three varieties of nucleated red cells may appear in a single specimen of blood from a case of secondary anemia.

Leukocytes.—The proper proportion of the normal varieties of leukocytes is often found disturbed in secondary anemia, yet this is by no means a necessary feature.

Summary of Diagnosis.—Progressive weakness, palpitation, dyspnea, pallor of both the skin and the mucous surfaces, with mental hebetude and weakness form the prominent features of secondary anemia.

Lowered viscosity, the absence of rouleaux formation, with pallor of the red cells, constitute the principal characteristics of the blood of secondary anemia. Stained blood shows such endoglobular changes as simple decolorization, punctate basic degeneration, alterations in shape (poikilocytes) and in size (microcytes and macrocytes), and the presence of nucleated red cells and ring-shaped bodies.

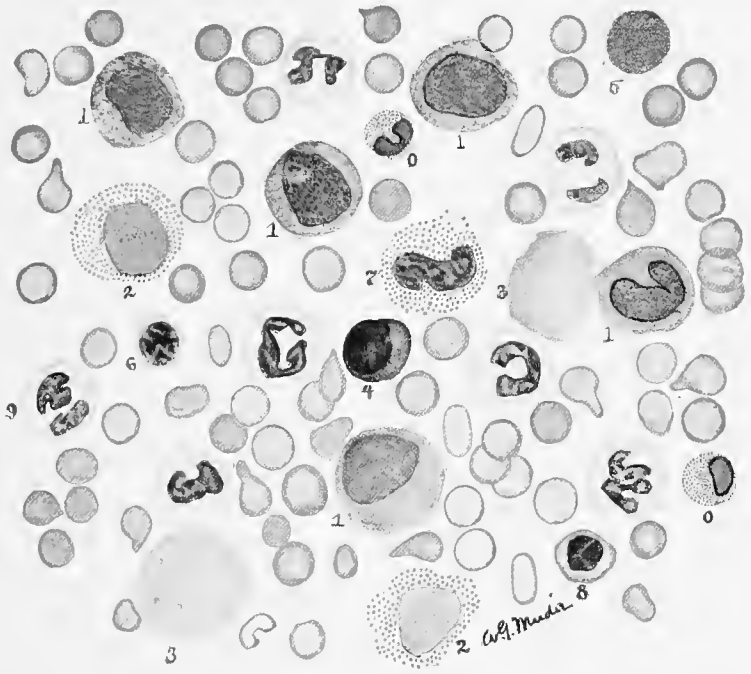
Symptomatic or Secondary Anemia.	Idiopathic or Essential Anemia.
1. Symptomatic blood condition secondary to disease elsewhere.	1. A primary disease of the blood and blood-making organs.
2. Occurs at any age.	2. Occurs, as a rule, during adolescence and in early middle life.
3. Previous or associated history of traumatic or spontaneous hemorrhage, chronic suppuration, prolonged lactation, chronic Bright's disease, carcinoma, chronic lead-poisoning, chronic malaria, gastritis, dysentery, or acute infectious maladies.	3. Previous history not clear. Often follows a profuse hemorrhage or severe mental strain.
4. History of overwork and of insufficient food, sunlight, or fresh air.	4. Negative.
5. May depend upon intestinal parasites when ova are found in feces, or upon malaria when the plasmodia are in the blood. In the presence of Bilharzia, the ova are detected in the bloody urine.	5. Absent.
6. Blood changes are variable, but steadily progressive in malignant disease.	6. Distinctive blood characteristics and profound changes both as to blood-cells and as to hemoglobin.
7. Moderate reduction in both red cells and hemoglobin, the relative proportion being maintained.	7. Marked reduction in both the percentage of hemoglobin and in the number of red corpuscles; there may be a great increase in the number of leukocytes (myelocytes), as in leukemia.
8. General symptoms and signs usually subordinate to those of the primary malady.	8. General symptoms and signs also more characteristic of the form of anemia in question.
9. Gravity of anemia depends on that of the primary disease.	9. Gravity depends on type of blood changes and progressiveness of the condition.
10. Often responds to treatment, depending on cause; in hemorrhage it is of short duration.	10. One variety (chlorotic) is quite curable, but relapses are likely to occur; the other forms are progressive: pernicious anemia is subject to remissions and relapses.

Clinical Course.—This depends—(*a*) Upon the underlying conditions; (*b*) upon the degree of anemia present; (*c*) upon individual surroundings and environment (climate, occupation, city or country life, and age); and (*d*) on whether or not medicinal and hygienic treatment can be properly instituted. Anemia resulting from intestinal parasites, chronic mineral poisoning, underfeeding, intestinal fermentation, and similar conditions disappears rapidly upon removal of the exciting cause. The anemia of such organic maladies as nephritis, valvular heart disease, hepatic cirrhosis, and malignancy is progressive.

LEUKOCYTES.

During health the number of leukocytes in the adult varies between 8000 and 10,000 in a cubic millimeter. Variation in the number of leukocytes depends upon exercise, digestion, bathing, etc.

PLATE IV



Blood of Splenomedullary Leukemia (Boston): 1. Myelocytes; 2, eosinophilic myelocyte; 3, leukocytic shadows; 4, polychromatophilic megaloblast; 5, large mononuclear leukocyte; 6, small lymphocyte; 7, eosinophile; 8, megaloblast; 9, polymorphonuclear leukocyte; 10, small eosinophiles. (Stained with eosin and hematoxylin. Obj. B. and L. one-twelfth oil-immersion.)

Classification.—The normal leukocytes are divided into the following subclasses, and are believed to be derived from two sources, as is shown by the accompanying table:

(A) The myelogenic group (from the bone-marrow):	1. Polymorphonuclear neutrophiles. 2. Eosinophiles. 3. Large mononuclear cells (Ehrlich). 4. Basophiles.
(B) The lymphogenous group (from adenoid tissue):	Lymphocytes, large and small.

Lymphocytes are also produced by the bone-marrow.

A normal differential leukocyte count will give the following percentages: Polymorphonuclear neutrophiles, 62 to 70 per cent.; lymphocytes, 20 to 30 per cent.; large mononuclears, 1 to 10 per cent.; eosinophiles, 1 to 4 per cent.; basophiles, 0.25 to 1 per cent.

Infants display a relatively high percentage of lymphocytes (50 per cent.), whereas the polymorphonuclear cells are proportionately reduced in number.

In disease we have to deal with abnormalities in the total number of leukocytes or in the relative proportions of each variety of white cell present, or with degenerated leukocytes, myelocytes, and eosinophilic myelocytes, the last two always being pathologic.

The accompanying plate (Plate IV) illustrates the different varieties of blood-cells.

Leukocytosis.—Definition.—A condition characterized by an increase in the number of leukocytes present in the peripheral blood.

Varieties and Causes.—*Physiologic leukocytosis* develops after exercise, after parturition, during digestion, after bathing, and during the last months of gestation. There is a slight leukocytosis in infants.

Pathologic leukocytosis is seen after hemorrhage, and in erysipelas, lobar pneumonia, bubonic plague, glanders, acute articular rheumatism, tonsillitis, relapsing fever, malignant endocarditis, and endometritis; it likewise occurs in suppurative conditions, such as acute pyemia, septicemia, cholangitis, and cholecystitis. Leukocytosis also accompanies most of the inflammatory conditions, irrespective of whether or not they involve the deep or superficial tissues; it also results from local irritation, such as is found in appendicitis, osteomyelitis, pyelonephritis, perinephritic abscess, localized peritonitis, epididymitis, conjunctivitis, proctitis, and gangrenous stomatitis; it is occasionally observed in gout, lithemia, toxemia, as, *e. g.*, in lead-workers, and malignancy.

Toxic leukocytosis develops during the course of such conditions as acute yellow atrophy of the liver, hepatic cirrhosis, jaundice, uremia, cinchonism, ptomain-poisoning, illuminating-gas poisoning, chloroform anesthesia, ether anesthesia, acute alcoholic delirium, acute mania, and after the injection of normal salt solution and other chemicals into the blood-vessels.

Drugs.—The number of leukocytes will be found to fluctuate after the administration of certain drugs, and this fluctuation bears a direct relation to the dosage employed, and to whether the drugs were administered by mouth, rectum, or subcutaneously: Ether, chloroform, peppermint, oil of anise, camphor, thyroid extract, gentian, sodium bicarbonate, bismuth, caffein, and quinin. A similar phenomenon follows the administration of liberal doses of castor oil, podophyllin, scammony, etc. It has also been observed that corrosive poisons, opium, chloral, and belladonna are capable of exciting leukocytosis in certain individuals. Intravenous administration of many drugs is followed by an increase in the number of leukocytes.

Leukopenia.—Definition.—*Hypoleukocytosis* is a term used to designate a condition in which the number of leukocytes in the peripheral circulation is decidedly below that of the normal for the individual under observation; and, in the majority of instances, the count is below 6000 cells in a cubic millimeter.

Causes.—Malnutrition and depleting hot baths may be followed by a decrease in the number of leukocytes. Early during the course of tuberculous meningitis, in influenza, uncomplicated tuberculosis, typhoid fever, and malaria, the number of white blood-cells falls below the normal. In many of these previously named conditions a differential count shows that the lymphocytes make up a large percentage of the leukocytes present, and this is especially true late in the course of typhoid fever and in maladies in which starvation has figured prominently. Leukopenia is a conspicuous symptom of splenic anemia, kala-azar, syphilis, and Hodgkin's disease.

Eosinophilia.—Definition.—Eosinophilia is a condition the characteristic feature of which is an absolute and a relative increase in the number of eosinophilic cells in the peripheral blood.

Varieties and Causes.—*Physiologic eosinophilia* is observed during menstruation and in the new-born.

Pathologic eosinophilia occupies a prominent place among the blood changes common to many maladies, among which may be mentioned asthma, fibroid bronchitis, and all conditions accompanied by dyspnea. In cutaneous maladies, such as urticaria, psoriasis, impetigo, prurigo, herpes zoster, ichthyosis, pemphigus, exfoliating dermatitis, and chronic eczema, a variable grade of eosinophilia is the rule.

Malignant Tumors.—The percentage of eosinophilic cells in the blood is often decidedly increased in malignancy, and particularly in sarcoma. The spleen appears to exercise some influence over the occurrence of eosinophilia, since this condition develops after splenectomy has been performed. Hemorrhage into the serous sacs, purpura, chylous ascites, tubercular leprosy, Hodgkin's disease, and leukemia, as a rule, are often accompanied by eosinophilia.

Drugs, when administered in toxic doses, are capable of inducing an increase in the number of eosinophilic cells. This condition is observed after poisoning from phosphorus and after liberal doses of camphor and ether.

Parasitic Eosinophilia.—Soon after infection with animal parasites the percentage of eosinophilic cells in the blood is increased. This phenomenon has been observed in trichinosis, filariasis, and schistosomiasis. Eosinophilia is often a symptom of infection with oxyuris, uncinaria, ascaris, and tape-worm.

Diagnostic Significance.—An increase in the number of eosinophiles in a given specimen of blood is highly suggestive of infection with animal parasites. Eosinophilia is often associated with new-growths along the gastro-intestinal and genito-urinary tracts. Eosinophilia has twice in our experience been seen to follow an acute mastitis. It is rarely found in such chronic conditions as syphilis. Many writers regard an increase in the number of eosinophiles as a favorable symptom in chlorosis, pernicious anemia, and hemorrhage.

Hypoeosinophilia.—It is customary to find the percentage of eosinophiles either moderately or decidedly lowered, and in some instances they may be absent, during typhoid fever, influenza, diphtheria, pneumonia, and septic disease.

Lymphocytosis.—Definition.—An absolute and relative increase in the number of lymphocytes in the peripheral blood.

Classification.—*Simple lymphocytosis* is merely an increase in the percentage of lymphocytes in the peripheral blood. An *absolute lymphocytosis* is an increase in the total number of leukocytes, of which an abnormally large percentage are of the lymphatic variety (lymphatic leukemia).

Varieties and Discussion.—In order to establish firmly the existence of a true lymphocytosis it is customary to study correlatively the number of lymphocytes and other white blood-cells during health and during disease, since the percentage is comparatively high in certain normal individuals. During infancy the percentage of lymphocytes will be found to vary between 40 and 60, and these cells are readily excited to an appreciable increase by feeble manifestations of disease. In the adult lymphocytes constitute from 20 to 30 per cent. of the total number of leukocytes. Lymphocytes are divided into two classes, "large" and "small," by some writers, but we consider all cells answering to the morphologic and tinctorial characteristics of this variety to be lymphocytes. (See Plate VII.)

Clinical Significance.—The blood of children always displays a relatively large number of lymphocytes as compared with that of adults. Lymphocytosis is present in splenic tumor, in whooping-cough before the characteristic whoop appears, and in measles during convalescence. Lymphocytosis and glandular enlargement are characteristic features of obscure cases of syphilis, of uncomplicated tuberculosis, and of malnutrition from a variety of causes, among which are rickets, scurvy, diarrhea, and gastritis. The diagnosis of lymphatic leukemia should be cautiously made in children, for a decided leukocytosis may occur as the result of the maladies previously named.

PROGRESSIVE PERNICIOUS ANEMIA

(IDIOPATHIC ANEMIA).

Pathologic Definition.—A disease of the blood characterized by a faulty production (hemogenesis) and a progressive destruction (hemolysis) of the red corpuscles. In addition there are advancing anemia, apparent preservation of superficial fat, a lemon-yellow color of the skin, retinal hemorrhages, and a tendency to the development of sclerosis of the spinal cord. The marrow of the long bones shows red patches throughout. The fat is of a yellow color and the muscles are a peculiar bright red. All the viscera are poor in blood, pale, and at times shriveled. The liver may give a reaction for iron.

Varieties and Causal Factors.—Pernicious anemia may, for purposes of description, be divided into three classes: (A) Persons suffering from pernicious anemia in whom it is impossible to detect any cause for the condition, either during life or at postmortem. It has been claimed that the idiopathic form of anemia is dependent on increased hemolysis, yet it is difficult to show satisfactorily that this is in reality true. In other cases hemogenesis is apparently normal, whereas in still others there appears to be an inherited tendency to lowered hemogenic power. The pathologic process upon which this form of anemia depends has not been clearly proved, hence the exciting causes are purely theoretic.

(B) Cases dead of pernicious anemia in whom it was impossible to detect an appreciable cause for the condition during life, but in which the actual etiologic factor was disclosed at autopsy. In certain cases the causal factor may be found to be animal parasites inhabiting the intestines, blood, arteries,

veins, or cellular tissues, *e. g.*, uncinaria, tape-worm, filaria, plasmodium of malaria, schistosomum, etc. Modern clinical methods have made possible the recognition of many of these causes during life. At autopsy an atrophic condition of the stomach, chronic gastritis, and malignant growths may be found, but these too may be recognized by a study of the secretory and digestive power of the stomach. It is questionable, however, whether the functionating power of the stomach and the pathologic conditions of the gastro-intestinal mucosa are not the result of the anemia, rather than the conditions that originally excited such blood dyscrasia.

(C) Anemias that appear to be either directly or indirectly traceable to mental shock, parturition with the loss of a large quantity of blood, hemorrhage, chlorosis, or such depleting conditions as diarrhea and dysentery. In this class of anemias are to be included cases that have developed amid unhygienic surroundings; the latter, however, cannot be numerous, since, in our experience, at least 50 per cent. of the cases developed among the well-to-do class and in private practice. We have seen one case of pernicious anemia in a negro and another in a half-bred Indian.

Age.—Until recently pernicious anemia was believed to be a disease developing after the age of forty. Griffith, however, has cited a number of instances in which pernicious anemia developed in individuals under the age of twelve years. In our practice we have seen a patient of twenty-nine and another of thirty-one years. In the light of our present knowledge of the bacteriology of the blood, and owing to the ease with which such examinations may be conducted, it would appear that this particular field of clinical research bids fair to make clear our uncertain views on idiopathic anemia.

Principal Complaint.—The patient is seldom able to determine any fixed date on which the trouble began, but states that he has, for a number of months or years, noticed a progressive weakness, shortness of breath, wakefulness, and possibly flabbiness of the muscles, all of which became gradually worse. An abrupt onset is rare, although a few instances have been recorded. In certain cases languor and anorexia are the most prominent symptoms, whereas in others constipation alternating with diarrhea constitutes a distressing feature. Headache, vertigo, mental depression, failing vision, attacks of faintness, and tinnitus aurium are among the annoying symptoms.

Thermic Features.—A moderate degree of fever is sometimes present. A temperature of 102° F. is the average in the milder grades of anemia, while in the more severe types, and for some days preceding death, the temperature may be normal or subnormal.

Nervous Phenomena.—Paresthesia, twitchings, spastic paralysis of the limbs, and abnormal reflexes of the lower extremities, with a possible absence of the knee-jerks, may be found. Inability to control the sphincters is occasionally seen in those cases in which there are other evidences of involvement of the spinal cord. Multiple neuritis affecting the nerves of both the forearm and the leg has been observed in advanced cases of pernicious anemia, but in all patients in whom wrist-drop and foot-drop were present some form of arsenic had been administered for an indefinite time, and may have been responsible for the development of these particular symptoms.

Physical Signs.—Inspection.—Early during the course of pernicious anemia and during an intermission the skin may present nothing characteristic, but when the anemia is at its height, the skin assumes a lemon-yellow tinge, is dry and lusterless, and the hair and nails appear to

be poorly nourished. The mucous membranes (lips, gums, tongue, and conjunctivæ) are pale, and late during the course of this malady slight puffiness of the eyelids and edema of the ankles occur. If a single extremity be permitted to rest in one position for a prolonged time, edema or cyanosis is likely to develop.

The general nutrition appears to have been preserved, and the patient is seldom emaciated. When the subject is placed in a position to bring out markedly the pulsation of the various large vessels, they will be found to display abnormal throbbing, which is particularly noticeable over the carotid, subclavian, and brachial arteries.

The apex-beat is rapid, feeble, and diffuse, and there is distinct pulsation over the base of the heart; pulsation is at times detectable in the epigastrium. Capillary pulse is often demonstrable.

An ophthalmoscopic examination shows the retina to be extremely pale, and in the severer forms of anemia there are retinal hemorrhages, which are responsible for the failing vision.

Palpation.—The bony structure is well covered with soft, flabby tissue, and the muscles are extremely soft and often tender. The knee-jerks are usually exaggerated, yet the reverse condition may exist. Tenderness along the course of the nerve-trunks of the lower limbs and of the forearms may occasionally be present, but this tenderness may not be a feature of pernicious anemia, since it always occurs late during the course of the disease and after the administration of large doses of arsenic. In two cases observed there were distinct evidences of an arsenical neuritis.

Percussion reveals nothing of special diagnostic value unless there has been an effusion into the pleural or pericardial sacs or the evidences of consolidation are manifested.

Auscultation discloses the presence of a soft systolic murmur (hemic), best heard over the base of the heart, and not well transmitted. This murmur diminishes during the stage of remission, but returns with each successive relapse. A venous hum may be audible in selected cases. When pulmonary congestion and bronchopneumonia complicate the condition, we may get fine, moist, and crackling râles over the bases of the lungs.

Laboratory Diagnosis.—The fresh blood exudes somewhat slowly at the site of puncture, is pale, almost watery in consistence, and when placed between glass slides, its viscosity is seen to be diminished. The oxygen-carrying power of the entire blood is greatly reduced, since the percentage of hemoglobin is between 15 and 50 during the active stage; although it may reach from 50 to 75 per cent. during the stage of remission. The specific gravity is always low, and bears a close relationship to the percentage of hemoglobin. Each erythrocyte is overcharged with hemoglobin, the greatest reduction affecting the number of red cells. It is the rule, therefore, to find the color-index high in pernicious anemia, although it almost invariably falls gradually when there is any amelioration in the general symptoms, but rises again with each approaching relapse.

During the active stage the number of red cells may fall to 1,000,000 or even 500,000 in a cubic millimeter, but during the stage of remission they often rise to from 2,500,000 to 3,400,000.

A microscopic study of the fresh blood shows many of the red cells to be of unusual size, while a few are extremely small. All the red cells are deeply stained with hemoglobin unless the blood is taken during the latter stage of the malady, when there may be only a peripheral band of coloring-matter in the cell cytoplasm. The erythrocytes are often distorted, and

may assume a variety of shapes, many of them becoming oval or elliptic, whereas others will display jagged outlines.

Stained blood, when placed under a microscope, is seen to contain nucleated red cells (megaloblasts, microblasts, and normoblasts); the non-nucleated erythrocytes are found to be stained unevenly, some appear as overstained with the eosin, while others are feebly stained. (See Plate V.)

During the stage of remission but few blood changes may be detectable by the microscope; in fact, we have seen instances in which not even a suspicion of pernicious anemia could be found from a microscopic examination of the blood. In making a prognosis it is well to estimate the number of nucleated red cells in a cubic millimeter (see Method of Estimating, p. 334), since a large number of nucleated red cells is of serious import.

The *leukocytes* are always diminished in pernicious anemia, and a differential leukocyte count shows an increase in the percentage of lymphocytes and a diminution in the polymorphonuclears. An increase in the eosinophilic cells is occasionally noticed during the active stage. Leukocytosis sometimes develops as a result of complications, or just prior to death, when myelocytes are numerous.

Megaloblasts are seldom found, except in pernicious anemia. (Plate V.) Normoblasts and microblasts, on the other hand, are often found in secondary anemia.

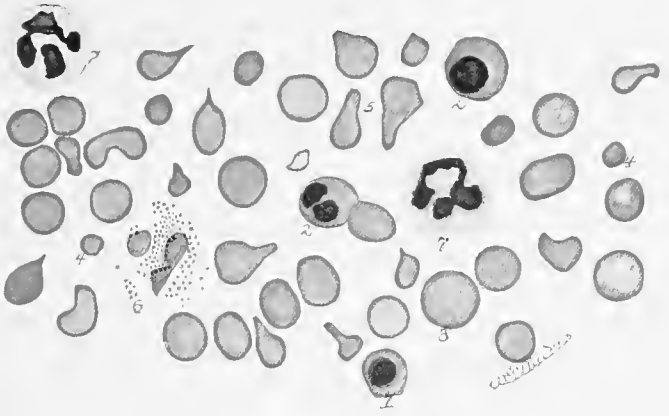
The *urine* is about normal in quantity, of high color, and often of low specific gravity. It may contain a moderate amount of albumin, but this finding is by no means characteristic of the disease under consideration. In pernicious anemia the urine is, as a rule, charged with pathologic urobilin, which may be detected by both chemic and spectroscopic examination. Urines containing pathologic urobilin develop a green fluorescence on the addition of a few drops of an alcoholic solution of zinc chlorid. Uric acid is at times increased, and it is the rule to find the urea content high. Owing to intestinal decomposition, indicanuria is usually present.

Course.—Occupation figures largely in the progress of pernicious anemia, since those who are able to take rest and recreation are found to do better than those who are actively employed or who are repeatedly subject to extreme grief and anxiety. We have had a patient under observation for over eight years, during which time he has had but three relapses, each relapse having followed severe mental strain.

Summary of Diagnosis.—The clinical characteristics of the affection, particularly their steady progression with remissions, are quite as important as is a microscopic study of the blood. The clinical significance of the laboratory findings are set forth in the following table, modified from Boston's "Clinical Diagnosis":

Fatal Issue Early.	Protracted Course.
1. Progressive anemia; interval between remission short or absent.	1. Remissions of prolonged duration.
2. Color-index high.	2. Color-index slightly increased.
3. Increase in diameter of red cells, with a tendency toward development of oval and elliptic forms.	3. Red cells of normal size.
4. Marked degenerative changes in the erythrocytes.	4. Little degeneration present.
5. Megaloblasts numerous, exceeding normoblasts.	5. Megaloblasts few. Normoblasts in excess.
6. Relative decrease in polymorphonuclear leukocytes.	6. Percentage of polymorphonuclear cells about normal.
7. Lymphocytosis present.	7. Absent.

PLATE V



Blood of Pernicious Anemia (Boston): 1. Normoblast; 2, megaloblasts, one showing two nuclei; 3, macrocyte; 4, microcyte; 5, poikilocytes; 6, ruptured eosinophiles; 7, polymorphonuclear leukocytes. (Specimen stained with eosin and hematoxylin. Obj. Spencer one-twelfth oil-immersion.)

Blood of Chlorosis (Boston): 1. Normal-sized erythrocyte stained deeply; 2, poikilocyte; 3, crenated erythrocyte; 4, normal cells stained feebly. Note basophilic leukocyte in left half of plate. All red cells stain less deeply than do the corresponding cells in pernicious anemia. (Specimen is stained with eosin-hematoxylin and methylene-blue. Obj. Spencer one-twelfth oil-immersion.)

Differential Diagnosis.—It is with great difficulty that we are enabled to make more than a provisional diagnosis of pernicious anemia through an examination of the blood alone, since such conditions as gastric carcinoma, gastric atrophy, intestinal parasites, mineral poisoning, and long-standing syphilis produce blood changes that strikingly resemble those of pernicious anemia. The presence of anemia due to **intestinal parasites** is confirmed by a careful examination of the feces. The history and the presence of well-marked punctate basophilia serve to eliminate **mineral poisoning** (except lead). The accompanying table shows the distinguishing features between the various types of anemia:

Progressive Pernicious Anemia.	Obscure Gastric Carcinoma or Gastric Atrophy.
1. The blood shows characteristic changes, and the red corpuscle count falls to or below 1,000,000 per c.mm.	1. Blood shows characteristics of secondary anemia, and the count does not fall to 1,000,000, as a rule.
2. Leukopenia and relative lymphocytosis common.	2. There may be leukocytosis or a relative increase in the polymorphonuclear cells.
3. Gastric pain not prominent, and usually absent.	3. Gastric pain suggestive of carcinoma. (See p. 499.)
4. Lemon tint of skin common.	4. Skin of a pale, muddy color, or only slightly jaundiced (saffron-yellow).
5. Adipose tissue fairly well preserved.	5. Progressive emaciation.
6. No glandular enlargements palpable.	6. Supraclavicular or inguinal glands may be palpable.
7. No physical signs referable to stomach.	7. There may be an area of increased resistance and tenderness over the stomach.
8. Examination of gastric contents after test-meal negative.	8. Examination of gastric contents shows deficiency or absence of free hydrochloric acid and the presence of lactic acid and the fatty acids.
9. Duration varies with the length of remissions. Some improvement may follow judicious treatment.	9. Fatal in nine months to one and one-half years. Condition becomes steadily worse until death.

LEUKEMIA.

Pathologic Definition.—A disease of the blood-making organs, either acute or chronic, characterized by a persistent increase in the total number of leukocytes, with the presence of large numbers of myelocytes or a great increase of the lymphocytes, coupled with a relative decrease in the number of polymorphonuclear elements. In the myeloid type the bone-marrow is extensively involved and there is splenic enlargement. Late during the course of the disease there is edema of the face and extremities, and effusion into the peritoneal cavity often occurs. The muscles and viscera are pale, and degeneration is not unusual. The lymphatic type shows hyperplasia of the lymph-glands, with an increase in the lymphocytes. The visceral and other changes closely resemble those of myeloid leukemia. In selected cases the changes that occur in both the myeloid and the lymphatic varieties are present simultaneously. Cases of acute lymphatic leukemia may terminate abruptly as the result of a large hemorrhage. A case under our care coming to autopsy showed about twenty hemorrhages into the cerebral tissue, varying in size from that of a grain of wheat to that of a hickory-nut.

Clinical Forms.—(A) **Myeloid leukemia** is a condition in which there is splenic and hepatic enlargement, with pathologic changes in the

bone-marrow and a large number of myelocytes in the peripheral blood. It may be acute or chronic.

(B) The **lymphatic type** may be either acute or chronic, and is associated with enlargement of the lymphatic glands, and sometimes with moderate enlargement of the spleen and liver. In lymphatic leukemia the increase in the number of leukocytes concerns, for the most part, the mononuclear cells (lymphocytes), but with few myelocytes. While acute leukemia is seen most commonly in children, it may occur during adolescence, in which case splenic enlargement develops rapidly, with appreciable enlargement of the lymphatic glands. Fussell and Taylor have collected 56 such cases from the literature.

(C) In this class we will consider those cases that appear at the onset to be purely of the lymphatic variety, but later develop myeloid manifestations.

Myeloid Leukemia.

Predisposing and Exciting Factors.—The *exciting cause* of

Fig. 153.—Glands of the Neck and Occiput and the Regions that should be Examined to Ascertain Cause for Glandular Enlargement.

myeloid leukemia is not thoroughly understood, but from recent investigations it would appear that this type of the disease is due to infection.

The myeloid form of leukemia is usually seen during early adult and middle life (from twenty-five to forty), yet it may occur at the age of fifty and even as late as seventy.

The lymphatic type is commonest in the young, and it is the rule to find pure lymphatic leukemia in children or before the age of twenty. Mixed types of leukemia may develop at any age, but this variety, too, is commonest in the young. Pseudoleukemia and other blood conditions more or less closely allied to true leukemia will be discussed elsewhere.

The disease not infrequently follows injury over the spleen (five such cases coming under our observation), intestinal ulceration, stomatitis, pseudoleukemia, malaria, and syphilis of the bones. Heredity is believed to play an important rôle in leukemia.

Principal Complaint.—**Chronic leukemia** is characterized by an insidious onset, and during the first few months of the disease it is usually

regarded as a simple anemia. Languor, anorexia, faintness, dyspnea, ringing in the ears, and dizziness are the chief complaints early in leukemia. Rarely, these symptoms are but slight, and the patient makes no complaint except of progressive weakness and of a peculiar appetite. Painful priapism, frequent night emissions, nausea, vomiting, hemoptysis, and epistaxis are among the initial symptoms. (See tables, pp. 373–376.)

As the disease progresses the symptoms become more pronounced, enlargement of the left side of the abdomen, swelling of the face and feet, and sometimes general edema are added. There is also feverishness at night (*vide* Thermic Features). Dimness of vision is an annoying symptom, and the patient complains of hemorrhoids, large quantities of blood being passed with the dejecta; cutaneous ecchymoses and pruritus are also present. Gangrene of the mucous membrane of the rectum, with its characteristic odor, may occur at times.

Dysentery developing late in the course of chronic leukemia is often a

Fig. 154.—Glands of the Face and Anterior Portion of the Neck and Regions that should be Examined for Glandular Enlargement.

distressing feature, and increases the already existing weakness. Facial neuralgia is present in a limited number of cases, and local paralyses result from either hemorrhage or leukemic growths in the central nervous system.

Thermic Features.—After the disease has reached its height slight fever is present, usually fluctuating between 99° and 101° F., although it may reach 103° F. in severe cases.

Physical Signs.—**Inspection** discloses a peculiar bluish, muddy, or waxy pallor of the skin, edema of the face, with a certain amount of distortion of the features, unilateral distention of the abdomen, hemorrhoids, edema of the feet, and puffing of the hands and fingers. The patient's gait is feeble, tottering, and uncertain, while his general attitude is that of exhaustion; cyanosis is often present.

Palpation reveals a peculiar sensation of the skin, roughness of the hair, edema, and the presence of a tumor-mass in the left hypochondrium. Hepatic enlargement to from one to three inches below the costal margin is also found. The spleen may extend to the umbilicus, or, as we have seen

in several cases, it may occupy the greater part of the brim of the pelvis. The cervical and inguinal glands may be enlarged, and are not, as a rule, adherent to the skin. Rarely, nodular bony growths are to be felt developing from the ribs or from other flat bones.

By the aid of **percussion** it is possible to confirm a large part of the information, obtained by inspection and palpation, as to the increased area of splenic and liver dullness. The area of cardiac dullness may also be increased because of the presence of dilatation or pericardial effusion. Rarely, there are to be found areas of dullness in the chest that probably depend upon the presence of glandular enlargement. Bilateral movable dullness the result of pleural effusion may be present.

Auscultation reveals a soft systolic murmur audible over the base of the heart, and not transmitted (hemic). This murmur is present early during the course of leukemia, and becomes accentuated with the progress of the disease, but, like the so-called "hemic murmur" of other blood diseases, its diagnostic value is limited.

Auscultatory percussion (see Fig. 240) serves as a practical and accurate method of outlining the spleen, liver, and heart, and also aids in determining the size of leukemic or glandular enlargements where such pathologic conditions are present within the chest or abdomen.

Acute leukemia is an uncommon form of the disease characterized chiefly by an exaggeration of all the early manifestations of chronic leukemia; it runs a rapid course, terminating fatally within a few months. Practically all the symptoms and signs present in chronic myeloid leukemia occur during a comparatively short period. No plausible explanation for the clinical course pursued by acute leukemia can be given.

Laboratory Diagnosis.—*Fresh Blood.*—Upon puncture of the finger the blood exudes somewhat slowly from the wound, is pale, often resembling thin pus, and displays nearly the normal coagulation power and an increased adhesiveness, which is shown by the difficulty with which it is spread upon a glass surface.

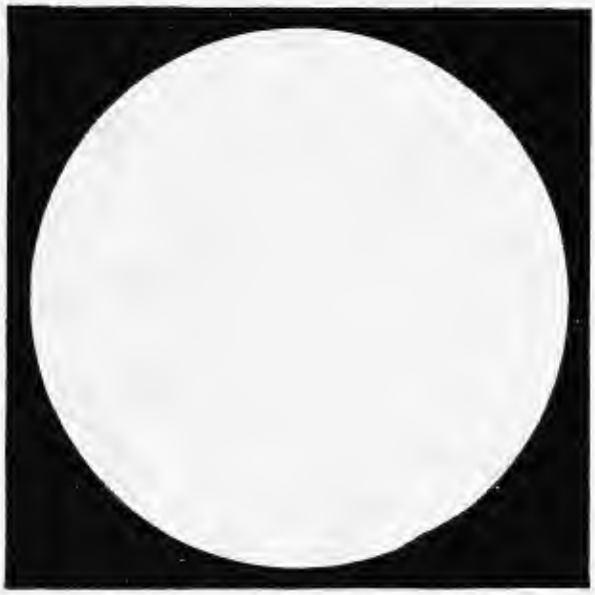
The *hemoglobin* is reduced to from 75 to 40 per cent., but this reduction is a relative one, and is in direct correlation with the number of erythrocytes present; hence the color-index approximates the normal. The number of red cells in a cubic millimeter will be found to vary between 2,000,000 and 3,500,000, except in advanced cases, in which their number may fall below 2,000,000.

An increase in the number of leukocytes in a cubic millimeter (60,000 to 1,000,000) constitutes the characteristic feature of myeloid leukemia. The ratio between the red and the white cells varies from 1 : 250 to 1 : 2, which is in striking contrast to the normal ratio of 1 : 500. In extreme cases the number of leukocytes in a cubic millimeter may equal that of the erythrocytes, and Sorsen cites a case in which the ratio of leukocytes to erythrocytes was 3 : 2.

A microscopic study of the living blood discloses the presence of red cells arranged in small clusters (Plate VI) throughout the field; their protoplasm is pale, and they do not present the normal luster. There is great variation in the size of the red cells, and poikilocytes are common, as are also macrocytes and microcytes.

The leukocytes form a prominent microscopic feature of living leukemic blood, and are seen to be arranged in large clusters and dense aggregations (Plate VI). The majority of the large leukocytes are of the mononuclear variety, although small mononuclear cells and large polymorphonuclear cells

PLATE VI



Fresh Preparation from the Blood of a Case of Leukemia ($\times$ 550): Large mononuclear leukocytes of immature form (Grawitz).

with feebly granular protoplasm are also present. In advanced leukemia, Charcot-Leyden crystals are occasionally found (Fig. 31, p. 80).

The *stained blood* shows the same features mentioned under the consideration of the living blood, but the tinctorial properties of the leukocytes and of the red cells should also be studied in making a careful diagnosis of myeloid leukemia. Even during the early stage of leukemia the red blood-cells stain feebly with eosin, and many of them show only a peripheral band of stained cytoplasm, while their central pale spot is abnormally large. Practically, all the manifestations of degeneration of the erythrocytes are demonstrable in leukemic blood. (See Secondary Anemia, p. 356.)

Nucleated red cells (macroblasts, normoblasts, microblasts, p. 376) are commonly encountered, the rule being to find a few cells representing each type. Macroblasts whose nuclei are undergoing karyokinetic changes are more often seen in leukemic than in any other variety of pathologic blood.

Leukocytes.—At first glance leukemic blood is seen to contain an abnormally large number of mononuclear leukocytes, and in myeloid blood 20 per cent. or more of these cells belong to the variety known as myelocytes; their appearance is shown in the accompanying plate. The lymphocytes and polymorphonuclear cells stain normally, but they are less numerous than normal.

A differential leukocyte count serves to separate the various types of leukemia, and further enables one to follow the progress of the disease, for the general symptoms bear a close relation to the relative percentage of myelocytes. The blood from an ordinary case of myeloid leukemia contains about 30 to 60 per cent. of myelocytes, but their number may not exceed 15 per cent.; eosinophiles, 2 to 4 per cent.; polymorphonuclear cells, 10 to 40 per cent., with few transitional forms and basophiles. In some cases basophilia is a marked feature.

Illustrative Case of Acute Leukemia.—C. C. M., male, aged nine years; came under our care at the Medico-Chirurgical Hospital of Philadelphia.

Family History.—Father living at the age of thirty-six and mother at forty-two. Three older children of family are comparatively healthy. A younger sister has organic heart disease, which developed after repeated attacks of acute articular rheumatism.

Previous History.—Has had measles, mumps, and whooping-cough. Had a severe sore throat one year ago, although no physician was consulted at the time; one week later, however, a younger sister developed scarlet fever, and there were several other cases of this disease in the immediate neighborhood. For nearly a year past the child has displayed a disinclination for play, and he frequently complained of feeling tired; when out playing he would leave the other children, go to his home, and lie down upon a couch for from one-half to two hours. The mother also noticed that for eight months past the child's appetite was poor, and, as she expressed it, he merely "picked" at his food.

Social History.—Attended school from the time he was six years old until a few months ago, when he became so easily exhausted that it was deemed advisable that he remain at home. Prior to this no physician had been consulted, for the child looked healthy, and his apparent indisposition was attributed to a dislike for school.

Present Illness.—When first seen at the hospital, the mother stated that the child had been confined to the house for the past four weeks; that he appeared to be feverish at night, vomited often, and was subject to severe attacks of nose-bleed, following which he would be markedly prostrated. Constipation was present. During his stay in the hospital he passed a moderate amount of blood by the rectum, and attacks of epistaxis were repeated about twice a week. Vomiting was also an occasional annoyance. The child showed no inclination for exertion, not even to move about the bed, and when assisted to sit up in bed or in a chair, violent attacks of cardiac palpitation ensued. Dyspnea was evident even when the child uttered short sentences, and became extreme upon slight exertion.

For the past few weeks there has been some pain in the upper portion of the abdo-

men, at times distinctly localized to the region of the spleen or to the hepatic area. Cough, accompanied by quite free expectoration, was present after sleep, and violent cough accompanied attacks of dyspnea.

The patient was unusually irritable, frequently cried, and often gave expressions of distress when moved about the bed. There had been some impairment of the vision, and an ophthalmoscopic examination gave evidence of deposits of questionable nature in the retina. He complained of a sense of oppression in the frontal region, and headache was present at times. He slept poorly, and never felt rested after sleeping. Attacks of vertigo, cardiac palpitation, and syncope became more and more frequent with the progress of the disease, and the condition terminated in coma.

After admission to the hospital the temperature ran an irregular course, fluctuating between 99° and 101° F.; practically one month later the fever was less marked, although two days before the fatal termination the temperature rose to 103° F., and at this time signs of bronchopneumonia appeared.

Physical Examination.—*General.*—The skin presented a peculiar cachectic (muddy) appearance. The face was rigid, the expression fixed, the abdomen prominent, and swelling of the hands and lower extremities occurred. The respirations were unusually short and rapid—thirty a minute. There were few small ecchymoses into the skin of the hands, lower extremities, and the mucous membrane of the mouth. There was moderate enlargement of the axillary, cervical, and inguinal glands. The general attitude was that of weakness and exhaustion.

Local Examination.—*Palpation.*—The inguinal, cervical, and axillary glands were enlarged and nodular, and the skin was freely movable over them. The apex impulse of the heart was extremely feeble, and pressure caused pitting over the lower extremities as high as the knees and in the loins; the skin of the hands and forearms also pitted upon pressure. The physical signs of effusion into the peritoneal cavity were also prominent. (See Ascites, p. 567.) The pulse was weak, readily compressible, and irregular as to both force and time, the beats numbering 120 to 140 a minute.

Percussion.—Early during the illness percussion of the chest gave negative results, but later areas were isolated in which the percussion-note was impaired over the bases of both lungs. The area of cardiac dullness was increased, and the outline of such area inverted, the base of the triangle being directed downward—a clinical fact that points strongly to the existence of pericardial effusion. Movable dullness, which was replaced by a tympanitic note, was conspicuous over the abdomen. (See Physical Signs of Ascites, p. 567.) The area of hepatic dullness was moderately increased, extending to two inches below the costal margin. Owing to the presence of fluid in the peritoneal cavity it was not possible to outline the area of splenic dullness accurately, but on performing auscultatory percussion the organ was found to be enlarged.

Auscultation.—The heart-sounds were weak, and appeared to have lost their muscular quality, the first sound approximating more or less closely the second sound. Throughout the entire illness numerous moist and bubbling râles were head over both lungs, particularly late during the course of the disease.

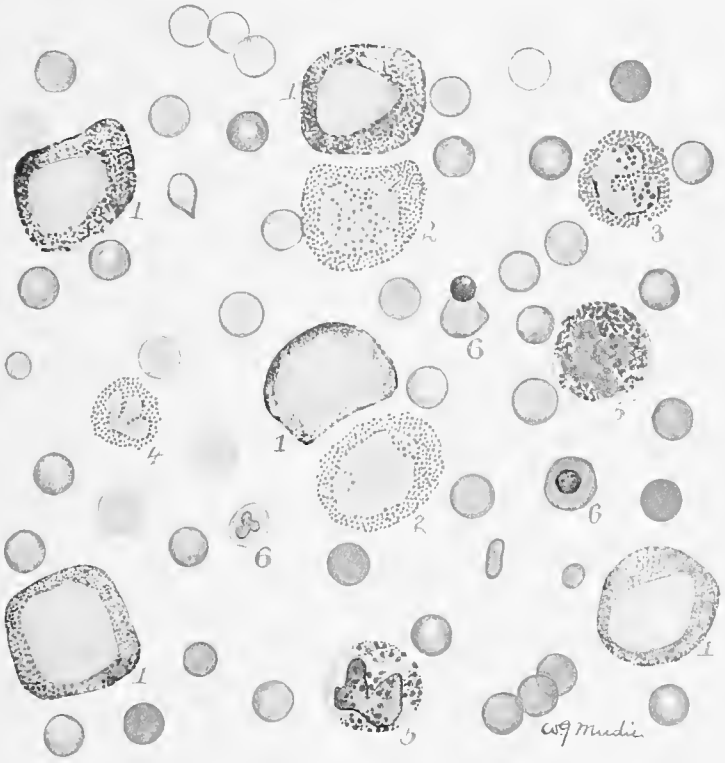
Laboratory Findings.—A blood examination made upon the second day of the patient's stay in the hospital showed: Red cells, 2,130,000; white cells, 87,300; hemoglobin, 52 per cent. A differential leukocyte count gave lymphocytes, 65.3 per cent.; myelocytes, 4 per cent. Blood examinations were made weekly for a period of ten weeks; the last showed: Red cells, 1,780,000; white cells, 360,000; hemoglobin, 35 per cent. At this time a differential leukocyte count showed 64 per cent. of lymphocytes and 18 per cent. of myelocytes. There was also well-marked degeneration of the red cells. The quantity of urine voided during the day varied between 20 and 30 ounces; it contained a small amount of albumin, many leukocytes, and at times red blood-cells.

Diagnosis by Induction From Clinical Data.—While such clinical features as progressive weakness, dyspnea, edema, and a peculiar complexion, accompanied by hemorrhage from the mucous surfaces, strongly suggested the nature of the disease, a diagnosis was not made positive until a study of the blood was completed, and upon the evidence obtained from a hematologic study a diagnosis of unmistakable leukemia was made.

Course of the Disease.—The patient's general condition progressed from bad to worse, terminating in death within a period of eleven weeks, which, together with the time he was confined to bed before coming to the hospital, gives a total duration of fifteen weeks. It is probable, however, that the illness continued over a somewhat longer period. Autopsy was not permitted.

Summary of Diagnosis.—A history of repeated mild attacks of dizziness, the symptoms of anemia, the urine being free from albumin,

PLATE VII



Blood of Myelogenic Leukemia (Boston): 1. Myelocytes; 2, eosinophilic myelocytes; 3, eosinophile; 5, mast-cell; 6, nucleated erythrocytes, one with clover-leaf nucleus and one with nucleus on cell's margin. (Stained with Jenner's stain. Obj. B. and L. one-twelfth oil-immersion.)

is highly suggestive of leukemia. Progressive weakness, edema, hemorrhages from the mucous surfaces, and later splenic enlargement, with the presence of an abnormally large number of leukocytes (myelocytes) in the peripheral blood, form the distinguishing features of myeloid leukemia.

Differential Diagnosis.—Leukemia is to be distinguished from **leukocytosis** by the fact that there is seldom more than a moderate increase in the number of leukocytes in the latter condition, and this increase concerns, for the most part, the polymorphonuclear neutrophiles, with no myelocytes. The leukocytosis of children may at times chiefly affect the lymphocytes, but here again myelocytes are unusual findings. **Malignant disease of the lymph-glands, malaria,** and **passive congestion of the spleen,** due either to cirrhosis of the liver or to valvular heart disease with tricuspid regurgitation, may give symptoms resembling those of leukemia, but the blood-findings in these maladies are the same as those of secondary anemia (pp. 373, 374, and 375).

Nephritis.—At the onset of the disease leukemia is probably oftenest mistaken for nephritis, owing to a decided edema of the eyelids and face; here again an examination of the blood and an analysis of the urine will serve to separate the two maladies.

Splenic anemia (anemia with splenomegaly) is quite indistinguishable from myeloid leukemia, except as the result of an examination of the blood, which will reveal the characteristic features—splenic tumor with leukopenia.

Chronic mineral poisoning, *e. g.*, the anemia following lead intoxication, may resemble leukemia, but the history of exposure, the detection of lead in the urine, neuritis with wrist-drop, and a leukocyte count not exceeding 30,000 serve to separate plumbism from leukemia.

Amyloid Disease.—In amyloid disease the skin presents a waxy pallor, and edema of the face and extremities, enlargement of the spleen and liver, as well as many of the subjective symptoms of leukemia are present. A history of either syphilis or prolonged suppuration is suggestive of the true nature of the disease. An examination of the blood with a differential leukocyte count, however, is necessary in order to separate this condition from leukemia. In amyloid disease the leukocyte count is often high,—25,000 to 50,000,—but, as a rule, the polymorphonuclear cells show the greatest increase, while there is a less decided increase in the lymphocytes. We have seen from 2 to 10 per cent. of myelocytes in the blood of amyloid disease.

Fatty Liver.—Chronic tuberculosis with fatty infiltration, or, oftener, fatty degeneration of the liver with associated enlargement of the spleen, is commonly encountered in the tuberculosis wards of the Philadelphia Hospital, and is distinguished from leukemia by its chronic course, the existence of pulmonary cavity, characteristic cough, expectoration, and a leukocytosis of about 15,000 to 40,000, with the presence of but few myelocytes.

Chloroma (Green Cancer).—A sarcomatous growth involving the bones of the orbit, and characterized by the formation of secondary growths in the skin and viscera, which are greenish in color. The disease may resemble either lymphatic or myeloid leukemia in certain respects, depending entirely upon the location of the metastases. Orbital pains, conjunctival hemorrhages, exophthalmos, faulty audition, and deformity of the orbit distinguish chloroma from leukemia. Hematologically, the blood of chloroma resembles that of true leukemia.

The features that distinguished leukemia from pernicious anemia, secondary anemia, and chlorosis are set forth on pp. 373–375.

Lymphatic Leukemia.

It is difficult to show conclusively that many cases of leukemia are not at first of the lymphatic variety, since the disease begins somewhat insidiously, and a blood examination is not obtained until it is well advanced.

Causation.—Lymphatic leukemia may occur at any age, but the majority of cases that have come under our observation developed during childhood and early adolescence. One case seen by us, however, occurred in a woman seventy-six years old.

Principal Complaint.—The temperature and physical signs resemble closely those described under Myeloid Leukemia. The distinctive characteristics are general glandular enlargement (moderate), and the late development of splenic and hepatic overgrowth. Lymphatic leukemia is said to develop more rapidly than the myeloid type, except in the case of the so-called "acute leukemia" of adults.

Laboratory Diagnosis.—The *hemoglobin* and the *erythrocytes* are proportionately reduced, the latter falling to between 3,500,000 and 2,000,000 in a cubic millimeter. The leukocytes fluctuate between 50,000 to 450,000 in a cubic millimeter, and even a higher degree of leukocytosis has been recorded. The ratio of red to white cells may be 1 : 200 or even 1 : 2.

A microscopic study of the fresh blood reveals the presence of a great number of small and large lymphocytes, which often occur in clusters and appear to have crowded the red cells into groups of from four to twenty each.

Stained Blood.—The red cells stain feebly and show a variable degree of degeneration. (See Secondary Anemia, p. 357.) Nucleated red cells are often found in this disease.

A differential leukocyte count reveals the chief characteristic of lymphoid blood, namely, that from 50 to 95 per cent. of the total number of white cells is lymphocytes. The increase in the relative proportion of lymphocytes is accompanied by a corresponding decrease in the polymorphonuclear elements. Either the large or the small form of lymphocytes may predominate. It has been quite generally maintained that when a high percentage of large lymphocytes is present, the case runs a rapid course, but this statement has not been confirmed.

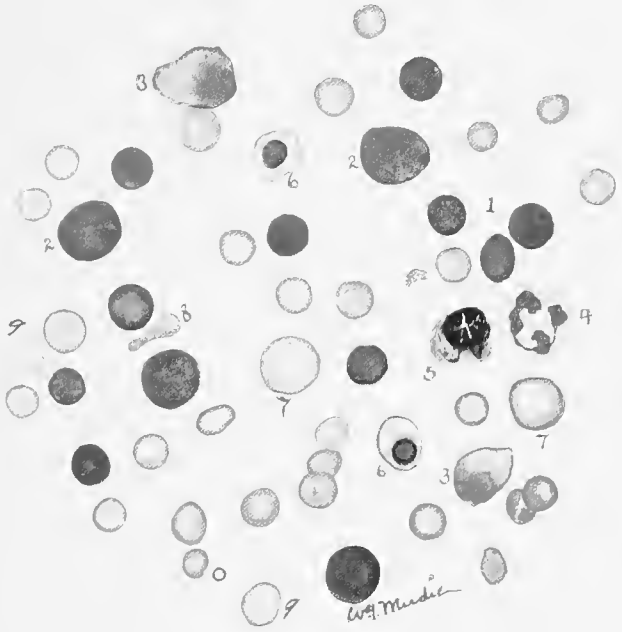
The urine is, as a rule, apparently normal until the disease is well advanced, when it may contain albumin and many leukocytes.

Summary of Diagnosis.—Lymphatic leukemia is recognized chiefly by enlargement of the superficial glands and lymphocytosis.

Differential Diagnosis.—Lymphatic leukemia may be confounded with *pseudoleukemia* (Hodgkin's disease), but the latter is not attended with leukocytosis and runs a more chronic course. Moreover, there is early involvement of the glands, which appear in grape-like clusters.

The anemia of *malnutrition*, commonly seen in children, resembles closely the early stage of lymphatic leukemia, and in one instance observed at the Philadelphia Hospital it was impossible to determine, from the first few blood examinations, whether or not we were dealing with lymphatic leukemia: the leukocyte count was above 60,000 in a cubic millimeter, and of these, 50 per cent. were lymphocytes. This child was given blood tonics and improved greatly, his blood being practically normal four months later. A similar variety of anemia may follow certain of the infectious diseases, as, *e. g.*, scarlet fever, measles, and exfoliating dermatitis, but the history, coupled with the results of treatment, will show the true nature of these conditions.

PLATE VIII



Blood of Lymphatic Leukemia (Boston): 1. Small lymphocytes; 2, large lymphocytes; 3, degenerated lymphocytes; 4, polymorphonuclear leukocytes; 5, megaloblast, showing fragmentation of nucleus and polychromatophilia of protoplasm; 6, nucleated red cells (megaloblasts); 7, macrocytes; 8, poikilocyte; 9, erythrocytes of normal size; 0, microcyte. (Stained with eosin and hematoxylin. Obj. Spencer one-twelfth oil-immersion.)

CHLOROSIS.

Pathologic Definition.—A blood disease characterized by imperfect development of the genitalia and anomalies in the blood-vessels together with pronounced alteration in the circulating blood and by anemia of the body tissues.

Predisposing and Exciting Factors.—Age and Sex.—Young females, fourteen to nineteen years, are most often attacked. Males may rarely display many features of this malady. Cases have been seen by us between twenty-five and thirty-five years, but in all such cases there was a history of one or more attacks earlier in life.

Previous attacks figure prominently as a predisposing factor, two or more attacks being rather common.

Occupation and Sunlight.—Chlorosis is most common in girls who work indoors and in buildings where there is a limited amount of sunlight. Employment in imperfectly ventilated buildings is doubtless a contributing factor. Overwork of whatever nature gives rise to chlorotic anemia.

Personal cleanliness is claimed to figure in selected cases, the condition arising in those who are careless in this respect.

Insufficient sleep is doubtless a factor in the production of chlorotic anemia, and masturbation may be a cause in some cases.

Food.—Many cases are poorly nourished either as the result of insufficient nourishing food or from gastric derangements which impair the appetite and at times interfere with perfect assimilation.

Climate.—Chlorosis is frequent among young females who have recently changed their residence from a tropical to a colder climate. Those from the British Isles (especially Ireland) and from southern Europe frequently suffer from chlorosis when coming to the United States.

Toxins absorbed during obstinate constipation may be a cause, as may also anxiety and grief.

Principal Complaint.—Early there is languor, indisposition to mental or physical exertion, irritability, weakness, depression, and fatigue following moderate exertion. Headache, cardiac palpitation, and constipation appear early and increase in severity as the condition advances. Dyspnea is common and amenorrhea the rule.

The appetite is poor and a perverted appetite is present. The patient may be seen eating particles of earth, etc. There is at times a craving for sour and spiced food. Eructations of gas and rarely vomiting are seen.

Physical Examination.—The subcutaneous fat is retained. The peculiar greenish-yellow tint of the complexion is striking. There may be edema of the ankles. The scleræ are often pearly or bluish-white. The nails show pallor, as do also the cheeks, tongue, and lips. Visible pulsations of the carotid vessels are frequent, and a pulsation at the base of the heart and in the peripheral veins is observed.

Palpation.—The skin and the extremities are frequently cold, owing to sluggish heart action. The pulse is usually full and easily compressible.

Auscultation.—Systolic murmurs, soft and "whiffing" in character, are heard at the base of the heart. There is a venous hum or bruit de diable —a soft continuous murmur heard over the large cervical veins.

Laboratory Diagnosis.—Hyperacidity of the gastric fluid is common. The urine is of low specific gravity, pale, and contains indican.

Examination of the blood shows: The hemoglobin ranges from 70 per cent. to 35 per cent. in severe cases. The number of red corpuscles is not greatly reduced, and is from 3,700,000 to 4,100,000 per cubic millimeter, but

in severe cases the count may be 1,900,000. The leukocytes are only slightly increased in number (8000 to 8500 per c.mm.). Microscopically, the red cells are seen to be paler than normal, and somewhat altered in size and shape. Some are distinctly larger than usual (macrocytes), but the majority are slightly undersized (microcytes). Irregularity in shape (poikilocytosis) is seen in the red cells and an occasional normoblast may be observed. There is usually a relative lymphocytosis.

Summary of Diagnosis.—There is a decided reduction in the percentage of hemoglobin, without a corresponding change in the number of red cells. Vertigo, palpitation, and pallor when seen in young females are important. Constipation and a perverted appetite, as well as environment, are worthy of consideration in formulating a diagnosis.

CHARACTERISTIC DIFFERENCES IN LEUKEMIC BLOOD.

Myeloid Leukemia.	Chronic Lymphatic Leukemia.	Acute Lymphatic Leukemia.
1. Red cells moderately reduced—3,000,000.	1. Moderate reduction—2,000,000 to 3,000,000.	1. More marked reduction early during course of malady.
2. Nucleated red cells, including megaloblasts, present.	2. Not common.	2. Normoblasts quite common.
3. Leukocytes, 150,000 to 500,000 or more in a cubic millimeter.	3. 100,000 to 300,000.	3. 30,000 to 200,000 the rule.
4. Myelocytes, 30 to 60 per cent.	4. Myelocytes few.	4. Myelocytes few.
5. Eosinophiles common, also eosinophilic myelocytes.	5. Eosinophiles few.	5. Eosinphiles rarely found.
6. Neutrophiles commonly seen.	6. Less common.	6. Scanty.
7. Lymphocytes—large and small—12 to 30 per cent.	7. Small lymphocytes, 60 to 90 per cent.	7. Lymphocytosis—large forms predominant, many irregular cells.

THE DISTINGUISHING SYMPTOMS AND PHYSICAL SIGNS IN SECONDARY ANEMIA AND THE ESSENTIAL ANEMIAS.

Pernicious Anemia.	Chlorosis.	Leukemia.	Secondary Anemia.
1. Progressive weakness, with anorexia, and gastric disturbances, with constipation alternating with diarrhea.	1. Great weakness coming on somewhat suddenly. A perverted appetite, with obstinate constipation.	1. Weakness develops slowly, but is progressive. Anorexia the rule.	1. The development of the anemia depends entirely upon the primary disease with which it is associated.
2. There may be aching and mild pains in the extremities.	2. Absent.	2. Pain, when present, depends upon pressure from an enlarged liver or spleen.	2. Always dependent upon the primary disease.
3. Palpitation an annoying feature late in the disease.	3. Present upon slight exertion throughout the disease.	3. Great cardiac discomfort late in disease, usually with cyanosis.	3. Not a prominent feature, but may be present upon exertion.

THE DISTINGUISHING SYMPTOMS AND PHYSICAL SIGNS IN SECONDARY ANEMIA AND THE ESSENTIAL ANEMIAS.—(*Continued.*)

PERNICIOUS ANEMIA.	CHLOROSIS.	LEUKEMIA.	SECONDARY ANEMIA.
4. Headache quite common; patient cannot concentrate mental power. Even reading produces exhaustion.	4. Common, and intense occipital pains upon exertion. The jar of walking may cause headache, and the patient complain that her heels are too high. Patient irritable, wakeful, and nervous.	4. Headache not a characteristic feature. Mental condition dull.	4. When due to such chronic conditions as nephritis, headache is prominent. Insomnia and irritability are prominent features.
5. Inspection: Skin, lemon-yellow tint. Superficial fat well preserved.	5. In severe types there may be slight puffing of the ankles.	5. Puffiness of the eyelids and some distortion of the features, due to edema of the face. Swelling of the ankles and lower extremities early; also backs of hands and fingers. Abdomen prominent.	5. Edema is unusual except in anemia due to the Necator americanus, when the distortion of the face is equally marked with that seen in leukemia.
6. Effusion into the serous sacs uncommon, and when present, affects the pleura.	6. Absent.	6. Pleural and peritoneal effusions common.	6. Absent the rule.
7. Failing vision and retinal hemorrhages the rule.	7. Absent.	7. Retinal hemorrhage has been reported.	7. Retinal hemorrhage rare, except in the anemias of diabetes and nephritis, where they are common.

LABORATORY FINDINGS BY A STUDY OF FIXED AND STAINED BLOOD IN HEALTH, SECONDARY ANEMIA, AND THE ESSENTIAL ANEMIAS.

FINDINGS IN NORMAL BLOOD.	PERNICIOUS ANEMIA.	CHLOROSIS.	LEUKEMIA.	SECONDARY ANEMIA.
1. Form and size of the red cells as shown by Plate II.	1. Poikilocytosis common, with increased diameter of the erythrocytes characteristic.	1. Extreme degree of distortion and abnormally small cells the rule.	1. Moderate poikilocytosis and great variation in size of erythrocytes.	1. Poikilocytosis in extreme types; the size of cells varies greatly—both microcytes and macrocytes are found.

LABORATORY FINDINGS BY A STUDY OF FIXED AND STAINED BLOOD IN HEALTH, SECONDARY ANEMIA, AND THE ESSENTIAL ANEMIAS.—(*Continued.*)

FINDINGS IN NORMAL BLOOD.	PERNICIOUS ANEMIA.	CHLOROSIS.	LEUKEMIA.	SECONDARY ANEMIA.
2. The red cells stain evenly except for their central portion, where there is a small circular area which is feebly stained.	2. The cytoplasm may stain unevenly, or the entire cell may overstain. Occasionally understained cells are seen. Polychromatophilia displayed by megaloblasts.	2. All cells feebly stained, many as mere shadows. Overstained cells rare.	2. Most cells understained. Polychromatophilia and basic degeneration common.	2. Many of the cells are understained; few stain as normal, and others as mere shadows. Punctate areas of basic degeneration common.
3. Nucleated red cells absent.	3. Common—both megaloblasts and normoblasts.	3. Normoblasts occasionally seen.	3. Normoblasts common; megaloblasts unusual, except late in disease.	3. Normoblasts not an unusual finding. Megaloblasts extremely rare.
4. Differential leukocyte count shows percentage of the different cells to bear the normal relation.	4. Lymphocytes increased.	4. Lymphocytes may show a slight increase, and in a few instances slight eosinophilia has been noted.	4. In the myeloid type myelocytes compose 30 to 60 per cent. of the leukocytes. In the lymphatic type 50 to 95 per cent. of the white cells are lymphocytes, with few myelocytes.	4. The polymorphonuclear cells are usually above the normal. Eosinophilia is found in mineral poisoning, in intestinal parasites, and is occasionally seen in the anemia of infectious maladies.

PSEUDOLEUKEMIA

(HODGKIN'S DISEASE; GENERAL LYMPHADENOMA).

Pathologic Definition.—A variety of anemia characterized by a progressive hyperplasia of the superficial lymph-glands, occasional lymphoid growths, and enlargement of the spleen and liver, with an absence of the degenerative blood changes characteristic of lymphatic leukemia. The blood picture is that of a secondary anemia with leukopenia in afebrile cases and leukocytosis during the febrile period.

Varieties.—(A) Anemia in which involvement of the lymphatic glands is a leading feature.

(B) Anemia displaying the blood changes of pseudoleukemia (secondary anemia), with splenic enlargement and but slight involvement of the axillary, inguinal, and cervical glands.

Exciting and Predisposing Factors.—Bunting and Yates * have

* Archives of Internal Medicine, August, 1913, p. 236.

described at length their findings in cultural studies made from the involved glands in Hodgkin's disease. Billings and Rosenow* have conducted a series of investigations which tend to confirm the reports previously cited. A gram-staining, non-acid-fast, polymorphous, diphtheroid bacillus corresponding to the bacillus of Frankel and Much is a rather constant finding in smear preparations from the involved glandular tissues. These bacilli have been cultivated from the infected glands by Bunting and Yates and also by Negri and Mieremet. The bacillus of Bunting and Yates does not appear in pure cultures in the majority of instances, but is found associated with a staphylococcus. It has been further determined by Rosenow that the staphylococcus develops in pure culture aërobically, and that the bacillus develops when cultivated anaërobically. The conditions that materially predispose to the development of pseudoleukemia are as yet unknown. Males are affected in 75 per cent. of cases, and the disease seems especially prone to attack young and middle-aged persons. This is shown by Gowers' analysis of 100 cases, of which 30 were under twenty years, 34 between twenty and forty years, and only 36 developed after the age of forty.

Heredity is a doubtful predisposing factor. Flexner and other writers have suggested that certain protoplasmic bodies present in the large glandular nodules may be concerned in the production of pseudoleukemia. Still other writers, as, *e. g.*, Sailer and Musser, believe tuberculosis of the glands to be the initial lesion.

Pseudoleukemia has been known to follow ulcerative stomatitis, chronic irritations of the skin, local glandular swelling, malarial intoxication, glandular tuberculosis, and syphilitic infection, but the actual etiologic relationship that these maladies bear to pseudoleukemia is not known.

Principal Complaint.—The patient usually consults his physician for unilateral enlargements involving the glands of one side of the neck, particularly the submaxillary and cervical glands. In the course of a few months these groups of glands extend until they eventually involve both sides, and later other glands, such as the axillary and inguinal, show enlargement.

The patient does not manifest any marked constitutional depression until the disease has progressed for some time, when he complains of languor, failing strength, headache, giddiness, palpitation on slight exertion, dyspnea, loss of appetite, constipation, and at times of edema of the ankles and feet. The gastro-intestinal symptoms (diarrhea, constipation, hemorrhoids, etc.) are ofttimes annoying, and are suggestive of lymphoid growths in the stomach and in the intestines. Deafness, due to the interference of glandular growths in the pharynx, pressure paraplegia, and neuralgic pains are among the distressing symptoms.

Mucous hemorrhages, *e. g.*, epistaxis, may be an annoying feature late during the course of pseudoleukemia.

Physical Signs.—**Inspection** reveals pallor or a peculiar bronzing of the skin and mucous surfaces; at times jaundice, due to obstruction of the common duct, is present. Pruritus and erythema are not unusual. There is enlargement of the superficial glands, which are arranged in grape-like clusters and in huge bunches. There are edema of the feet, swelling and cyanosis of the arms and hands, swelling and blueness of the head and face—all of which congestions result from pressure by enlarged glands. There may be marked dyspnea, and the patient assumes the semirecumbent posture,

* Journal Amer. Med. Assoc., December 13, 1913, p. 2122.

owing to effusion occurring into the pleural sacs. The cardiac impulse is rapid and diffuse, and pulsation over the carotid and brachial arteries is at times present.

Palpation shows the enlarged glands to be freely movable beneath the skin, although rarely they may become adherent. The heart impulse is feeble, rapid, and wavy, and the pulse is soft, thready, and weak. Inequality of the radial pulse due to the pressure of glands on the axillary and cervical arteries is not an uncommon finding. Unilateral sweating of the face, the result of pressure upon the cervical sympathetic, is occasionally observed. When peritoneal effusion exists, fluctuation may be detected. The liver is rarely enlarged sufficiently to make it palpable. In an average case of pseudoleukemia the spleen can be felt below the costal margin, and in the splenic form of this malady the spleen is decidedly enlarged, while the lymphatic glands are but moderately or not at all increased in size.

Percussion reveals nothing of special interest unless there is effusion into the serous sacs. Glandular growths in the chest or in the abdomen are often detected by percussion, while auscultatory percussion enables one to outline the tumor masses accurately.

Auscultation.—The heart-sounds are rapid and irregular, and hemic murmurs are common. The breath-sounds may be enfeebled, exaggerated, or variously modified by pressure of enlarged glands which encroach upon the respiratory tract.

Laboratory Diagnosis.—Uncomplicated cases of pseudoleukemia are indistinguishable from those of true leukemia except as the result of a hematologic study. During the first months or even for a year the hemoglobin is but slightly reduced (75 to 80 per cent.), but from this time on it falls with the progress of the disease and may reach 50 to 30 per cent. At first the red cells are practically normal, but a gradual reduction follows closely the reduction in hemoglobin. The red cells reach 3,500,000 to 2,000,000 in a cubic millimeter in the average case.

There may be a variation in the number of leukocytes present in the peripheral blood, but in uncomplicated cases the leukocytes seldom rise above 18,000.

A differential leukocyte count shows such increase to affect chiefly the polymorphonuclear elements, myelocytes being unusual; eosinophilia, however, is not uncommon. In cases in which the leukocyte count is normal an increase in the percentage of lymphocytes is the rule. In our study of the blood conditions in a fairly large series of cases of glandular enlargement we found it impossible to make a diagnosis of pseudoleukemia upon the blood findings alone. Further, it was often found to be practically impossible to distinguish, hematologically, between syphilis, malignant disease, tuberculosis of the glands, and pseudoleukemia. In a review of many cases in the literature on the subject, in which a high grade of leukocytosis obtained for a prolonged period during the latter part of the disease, it was questionable whether such cases were not in reality instances of true leukemia from the onset.

Summary of Diagnosis.—A history of glandular enlargement affecting first the submaxillary glands and those of the neck, with gradual advancement and involvement of the glands of the opposite side, as well as of the axillary and inguinal regions, favors pseudoleukemia. A negative history of syphilis, normal areas of splenic and hepatic dullness, and the blood findings of secondary anemia are highly suggestive of the glandular type of pseudoleukemia.

Differential Diagnosis.—Pseudoleukemia is indistinguishable from **true leukemia** unless a study of the blood be made, which must include a differential count of the leukocytes. A high percentage of myelocytes, with a corresponding decrease in the polymorphonuclear cells, is positive of true leukemia. **Obscure syphilis** sometimes resembles pseudoleukemia, and is to be differentiated from this disease by a history of infection or of heredity.

Tuberculosis of the various systems of glands closely resembles pseudoleukemia. The involvement of additional glands is usually less rapid in tuberculosis than in pseudoleukemia; tuberculous adenitis is a disease of the young. Again, when the disease is tuberculous, the unilateral infection seldom spreads from the neck if the initial enlargement is there. Tuberculosis is more likely to affect the submaxillary than the cervical glands. The presence of abscesses and sinuses favors glandular tuberculosis.

Suppurative and ulcerative conditions involving the hand or scalp not infrequently cause unilateral enlargement of the superficial glands, but a history of prolonged suppuration and the presence of a unilateral infection serve to exclude pseudoleukemia.

Inanition.—In children in whom malnutrition appears to be the most prominent feature glandular enlargement is not infrequent, and may be confused with pseudoleukemia. The blood in inanition always displays an abnormally high percentage of lymphocytes, and the polymorphonuclear cells are seldom increased. A history of dietetic errors or of some preceding infection, *e. g.*, gastritis, dysentery, etc., and the improvement of the child's general condition upon the institution of judicious treatment, are the cardinal features that exclude the existence of pseudoleukemia.

Malignant disease of the superficial glands is at first indistinguishable from pseudoleukemia, but the rapid course and the tendency to involvement of the liver are opposed to a diagnosis of pseudoleukemia.

Benign cystic epithelioma, although a rare disease, may be confused with the glandular enlargement of pseudoleukemia. In benign epithelioma the initial lesion may be situated in any portion of the body, from which point small nodular masses develop in the skin, these nodular growths following the course of the lymphatics. Benign epithelioma develops more slowly than does pseudoleukemia, and the nodules are located within the skin, while in pseudoleukemia the skin is not adherent to the nodules. For a further discussion of the salient points of difference between pseudoleukemia and other blood maladies see tables on pp. 373–376.

Pseudoleukemia with splenic enlargement must be distinguished from **splenic anemia** (Banti's disease), and here it may be stated that, in our opinion, some of the cases of pseudoleukemia with splenic enlargement reported are, in reality, suffering from this form of anemia; we are also inclined to believe that, with our improved methods of hematologic study, cases of Hodgkin's disease with splenic enlargement as the leading symptom will be comparatively rare. Idiopathic enlargement of the spleen and splenic tumor from whatever cause may be confused with the splenic type of pseudoleukemia.

Clinical Course.—While pseudoleukemia usually continues over a period of several months or years, typical cases terminate fatally in from two and one-half to three years. During the course of the disease, however, marked remissions occur, but are always followed by more severe exacerbations, until late in the disease, when the periods of remission become shorter and those of the active stage longer. Indeed, Hodgkin's disease may terminate in lymphatic leukemia.

Among the unfavorable manifestations are: Great weakness, rapid glandular enlargements, emaciation, interference with the respiratory or circulatory systems as the result of pressure from glandular tumors, profound anemia, and general edema. A perceptible diminution in the size of the glandular masses not infrequently takes place late in the disease and is of serious prognostic omen. When infectious maladies complicate pseudoleukemia, the patient usually succumbs within a few days, and even local derangements may result disastrously.

ANÆMIA INFANTUM.

Definition.—A blood disease affecting children, and characterized by splenic enlargement and symptoms closely resembling those of leukemia, but displaying no marked tendency toward a fatal issue. This malady was first studied by von Jaksch.

Predisposition.—Children during the second year of life are most susceptible to the disease, but it is not infrequently seen at three and four years of age.

Principal Complaint.—The child does not thrive, and such gastro-intestinal symptoms as vomiting, anorexia, and diarrhea are present. The mother affirms that increasing weakness and pallor have been prominent features since the onset of the illness.

Thermic Features.—In a case observed at the Philadelphia Hospital mild irregular fever (99°–101° F.) was observed.

Physical Signs.—Upon **inspection** there is to be detected extreme pallor, an anxious expression, puffiness of the eyelids, edema of the ankles and feet, emaciation, increased respirations, and a diffuse cardiac impulse. The entire abdomen may be abnormally prominent, but more commonly there is a unilateral bulging, corresponding to the position of the spleen.

Palpation.—The pulse is often weak and thready, and late in the disease it may become rapid and dicrotic. The heart impulse is weak, although somewhat diffuse, and the muscles are soft and flabby. In the splenic region the round edge of the organ may be felt, and may extend to the median line of the abdomen; at last the spleen occupies the greater portion of the left superior abdominal quadrant. At times it is possible to detect a decided notch in the advancing margin of the spleen, and the organ may be felt to rise and fall with respiration.

Percussion reveals nothing of special interest, save that it is confirmatory of palpation in ascertaining the size of the spleen and of the liver.

Auscultation.—The heart-sounds are feeble, and often accompanied by a soft systolic murmur (hemic). There is hurried respiration, which, owing to interference from enlargement of the spleen, becomes shallow.

Laboratory Diagnosis.—The blood exuding from a puncture resembles that of secondary anemia. The hemoglobin will be found to vary between 75 and 45 per cent., whereas the red cells number between 3,000,000 and 1,500,000 in a cubic millimeter. Leukocytosis forms one of the characteristic features, and a count of from 30,000 to 100,000 leukocytes in a cubic millimeter is the rule. Decided fluctuation in the number of leukocytes is also an equally characteristic feature of infantile anemia. The ratio of white to red cells in severe types of the disease may reach 1 : 200.

A microscopic study of the living blood shows rouleaux formation to be imperfect, and the cells are often equally disseminated throughout the field. There is also great variation in the size and form of the erythrocytes.

Stained blood shows the red cells to be poor in hemoglobin and greatly

distorted; their size varies, microcytes and macrocytes being common. Abnormally large cells whose centers are practically without stain are plentiful.

Nucleated red cells (megaloblasts, microblasts, and normoblasts) are commonly found in infantile anemia. A differential count of the leukocytes shows a lymphocytosis. Myelocytes are occasional findings.

Summary of Diagnosis.—In a child having a history of progressive weakness, pallor, emaciation, the physical signs of splenic tumor, anemia, and leukocytosis the diagnosis is clear. The blood changes of profound secondary anemia further favor the diagnosis of infantile anemia.

Differential Diagnosis.—Abdominal tumor is not an unusual finding in children; these are often the result of **sarcomatous growths of the kidney,** from which infantile anemia must be differentiated. The history of a gradual onset, the marked secondary anemia, together with the physical signs,—*e. g.*, freely movable tumor,—favor infantile anemia. Sarcoma displays a less well-marked anemia and seldom shows a high grade of leukocytosis.

Lymphatic leukemia differs from infantile anemia in that the former is found at any period of life, and displays an extraordinary number of lymphocytes and enlargement of the lymph-glands.

Myeloid leukemia may at times resemble infantile anemia, but here the age and extraordinarily high percentage of myelocytes serve to exclude the latter disease.

Course.—Despite the grave type of anemia present, which is at first progressive, over 50 per cent. of the cases are amenable to treatment.

SPLENIC ANEMIA.

General Remarks.—Splenic anemia was first described by H. C. Wood, and was generally considered a distinct type of blood disease. Some writers, however, regard splenic anemia as dependent upon some pre-existing condition, as a consequence of which splenic enlargement and secondary anemia occur. Although splenic anemia has here been classed as a distinct variety of blood malady, we admit that this classification is open to criticism. (See Splenomegaly.)

Classification.—Technically speaking, the so-called splenic anemia is but a single type of the disease, but this is divided into three quite distinct stages: (*a*) The initial stage, which displays a somewhat high grade of anemia, with muscular weakness. (*b*) A stage characterized by atrophy of the liver and splenomegaly, with a variable degree of pain in the region of the spleen. It is during this stage of the malady that the patient often complains of other symptoms. (*c*) A stage in which progressive asthenia exists, terminating in death.

Principal Complaint.—The patient complains of prostration, loss of weight, gastro-intestinal disturbances, such as gastritis, frequent paroxysms of vomiting, hematemesis, shortness of breath, and palpitation. There may have been hemorrhages into the skin, and bloody urine may have been passed. A variable degree of pain, due to pressure from splenic enlargement, and edema of the ankles and feet, due to the same cause, are commonly experienced.

Physical Signs.—Inspection.—The skin and mucous membranes are pale, except in rare instances, in which the skin has become pigmented. The flesh appears to have been well preserved, while the gait is feeble and shows evidence of weakness. Slight edema of the eyelids and of the ankles

and feet develops late. The abdomen shows bulging, restricted to the splenic region.

Palpation reveals nothing of interest beyond outlining the hypertrophied spleen and showing, as it does, an absence of the liver at the costal margin. Rarely ascites exists, when it may be possible to elicit fluctuation. The pulse is weak.

Percussion, including auscultatory percussion, is but confirmatory of palpation.

Auscultation.—The heart-sounds are feeble, at times rapid, and hemic murmurs are to be heard after decided anemia exists.

Laboratory Diagnosis.—The hemoglobin shows decided reduction, often falling to below 50 per cent., while the red corpuscles display a less marked decrease and are, as a rule, above 3,000,000 in a cubic millimeter. Leukopenia is the characteristic feature.

Microscopically, the most conspicuous feature of the fresh blood is the extreme pallor of the red cells. There is also a high grade of poikilocytosis.

Stained blood reveals the characteristics of a severe secondary anemia (p. 356). Nucleated red cells (normoblasts) are common.

Urine.—Pressure from splenic tumor often causes renal congestion, in which instance the urine is of high specific gravity, high color, and, microscopically, it may be found to contain both red and white cells, as well as hyaline and granular casts. Albuminuria develops in cases in which renal congestion is marked.

Summary of Diagnosis.—In a case of splenomegaly with profound progressive anemia and showing the blood changes of chlorosis *with leukopenia,* splenic anemia is to be considered. A negative history of *syphilis, prolonged suppuration, malaria,* and *tuberculosis* is necessary, since any one of these maladies may cause blood changes resembling those known to occur in splenic anemia. The absence of glandular enlargement further supports the diagnosis of splenic anemia.

Differential Diagnosis.—**Myeloid leukemia** differs from splenic anemia in showing a leukocyte count above 60,000, of which a high percentage of the cells (30 to 60) are myelocytes. Rarely, indeed, cases of leukemia are seen in which the leukocytes fall to near the normal number, and remain there for an indefinite period, and it is only with great difficulty that this form of leukemia can be differentiated from splenic anemia during the stage of remission.

Pseudoleukemia with splenic tumor resembles splenic anemia, but glandular involvement, a feature characteristic of Hodgkin's disease and absent in the latter condition, serves to separate these diseases.

Enlargement of the spleen probably of **malarial origin** is to be distinguished from splenic anemia by the history alone, and unless this is very clear, it is doubtful whether these conditions can be differentiated from each other.

Banti's disease (cirrhosis of the liver with splenomegaly) is the concluding stage of splenic anemia.

Pernicious Anemia.—The small size of the spleen and the abnormally high ratio of hemoglobin to red cells, coupled with the increased diameter of the individual red cells, are features strongly suggestive of pernicious anemia.

HEMOPHILIA.

Definition.—A condition characterized clinically by an inherent tendency to repeated profuse hemorrhages from the mucous surfaces, with the

exception of the uterus, and to uncontrollable hemorrhages from the skin, mucous membranes, and deeper structures, following slight injury. The true pathology of hemophilia remains uncertain.

Causation.—Hemophilia may manifest itself during any period of life, but is oftenest seen in young adult females. The ratio of females to males is 10 : 1.

Principal Complaint.—These patients seldom complain of ill health, and the first evidence of the existing trouble is the onset of a profuse hemorrhage, which appears in the form of epistaxis, hemoptysis, or hematemesis, and in the passing of blood-stained feces and bloody urine. The patient often gives a history of swelling of the larger joints and a tendency to the development of large ecchymotic spots after having received but a slight injury. Even the prick of a pin or a similar trivial injury is followed by a hemorrhage into the skin, which at times becomes sufficiently severe to cause a true hematoma. Hemorrhages frequently occur into the buccal mucous membrane, especially beneath the tongue and upon the cheeks.

Physical Signs.—Inspection.—The patient is often a well-nourished individual who gives no evidence of disease; but when hemophilia is associated with splenic enlargement or disease of the liver or other viscera, there may be pallor and the general features of anemia may be manifest. Areas of ecchymoses may be detected, and swelling of the large joints, which at times show fluctuation, has been reported.

The mouth is most likely to display hemorrhages, yet in a large percentage of cases the physical examination, and even the patient's general complaint, gives no clue to the malady. Further systematic physical examination confirms inspection.

Laboratory Diagnosis.—In two cases studied by us we were unable to detect any distinctive feature between the blood of hemophilia (with anemia) and that of scurvy, purpura, and grave secondary anemia. Upon slight puncture of the finger the blood exudes freely, and when placed upon a glass surface, it appears to possess nearly the normal amount of adhesiveness. In one case observed by La Place a microscopic study of the living blood showed it to be normal, whereas the second observation disclosed a high grade of secondary anemia, the hemoglobin and red cells being proportionately reduced, and the other characteristics of secondary anemia (see p. 356) being in evidence. In the cases observed, 4 per cent. and 2 per cent. respectively of myelocytes were present.

Summary of Diagnosis.—A history of repeated ecchymoses following slight injury, and of profuse bleeding as a result of a trivial abrasion of the skin and mucous surfaces, or from slight injury to the nose, is highly suggestive of hemophilia. The family history is also of importance, since hemophilia is transmitted by the female, although it may be found in males. Other features characteristic of hemophilia are that serious hemorrhage from the uterus is unusual, and that hemorrhages are not likely to occur either during menstruation or pregnancy. Afebrile swelling of the joints is not unusual in hemophilia.

Differential Diagnosis.—Hemophilia is to be distinguished from **purpura hæmorrhagica, peliosis rheumatica, scurvy,** and conditions resulting from **inanition.** The preëminent feature that separates hemophilia from these conditions is the history of profuse hemorrhage following slight injury to the body tissues. When hemophilia exists in a person who is otherwise perfectly healthy, the diagnosis is comparatively clear, but after repeated hemorrhages it is commonly difficult and practically impossible to

distinguish between hemophilia and the maladies just named. Profuse **hemorrhage of the new-born** differs from hemophilia in that the former is preceded by jaundice. The family history is second in importance to hemorrhage on slight injury in diagnosing hemophilia.

Course.—The patient seldom attains the age of fifty, but usually succumbs to the secondary effects of the hemorrhages. It is possible, however, for subjects of hemophilia apparently to recover and to pass a period of many years with no recurrence of the bleedings—and, in fact, they may enjoy perfect health.

PURPURA.

General Considerations.—A purely symptomatic condition in which, owing to some alteration either in the blood proper or in the vessels or tissues, there are punctate areas of discoloration due to minute extravasations of blood into the skin or mucous membranes. In severe cases a more copious extravasation of blood, followed by ecchymoses, takes place.

Clinical Varieties.—(1) **Primary purpura,** of which two types are known to exist:

(*a*) *Simple purpura* (purpura simplex).

(*b*) *Arthritic purpura,* of which there are two subdivisions—*hemorrhagic purpura* (purpura hæmorrhagica) and *peliosis rheumatica.*

(2) **Secondary purpura,** in which hemorrhages into the skin and mucous membranes develop during the course of some malady in which purpura does not usually occur:

(*a*) *Purpura of acute infectious diseases, e. g.,* measles, variola, whooping-cough, cerebrospinal meningitis, mountain spotted fever, septicemia, ulcerative endocarditis, typhoid fever, and scurvy.

(*b*) It may accompany such *chronic conditions* as Hodgkin's disease, tuberculosis, jaundice and jaundice of the new-born, hemophilia, nephritis (chronic interstitial), leukemia, chlorosis, secondary anemia, and pernicious anemia.

(*c*) Purpura is occasionally seen to occur during the *anemia of malignant*

FIG. 155.—PURPURA.
Child showing purpura hæmorrhagica, treated at the Philadelphia General Hospital, 1909.

disease (cancer, sarcoma) and in that following *locomotor ataxia, grave hysteria,* and *myelitis.*

(*d*) It may follow *convulsions, violent exercise, paroxysmal coughing* and *vomiting.*

(*e*) *Drug purpura* follows the administration of lethal doses of mercury, belladonna, potassium iodid, ergot, quinin, copaiba, etc. Again, introduction into the system of certain *venoms*, as by the bite of the rattlesnake, copperhead, and cobra, as well as the bites from insects, tarantula, scorpion, spider, etc., are frequently followed by numerous extravasations of blood into both the skin and the mucous surfaces.

Primary Forms.—*Simple Purpura.*—In this form of purpura the causal factor is evidenced through such predisposing influences as puberty. Acute infectious diseases may contribute toward its development, but hemorrhages into the skin not infrequently occur in persons who apparently enjoy good health.

Arthritic purpura (peliosis rheumatica) is commonly associated with lesions of the heart and other serous membranes, but conclusive evidence that the purpura is rheumatic in nature is often lacking. Males at about the age of puberty appear to be especially susceptible to this form of purpura. The patient often complains of arthritic pains, anorexia, angina, and headache, with a variable degree of swelling and tenderness in the joints. Polyarthritis appears to characterize this form of purpura.

Henoch's Purpura.—This variety of purpura appears in the young, but, like the arthritic type, it too accompanies swelling and tenderness of the joints. In addition to purpura, erythema multiforme is also present. Such gastro-intestinal symptoms as anorexia, vomiting, intestinal colic, and diarrhea also occur.

Hemorrhagic purpura is oftenest encountered in poorly nourished girls at about the age of puberty. The first evidences of this condition are pallor, progressive weakness, and palpitation, which continue to progress until the purpuric eruption appears. At first, minute extravasations of blood into the skin and mucous surfaces occur, but later there is severe bleeding from the nose, lungs, stomach, and bowel. In this type of cases impoverishment of the blood is extreme and a fatal termination is the usual outcome.

Secondary Purpura of the New-born.—Hemorrhage into the skin and mucous surfaces may manifest itself but a short time after birth, or there may be, at this time, a profuse hemorrhage from the umbilical cord. This form of purpura is characterized by extreme jaundice, and there are usually symptomatic indications of syphilis. Hemorrhage into the urinary tract also occurs, and the urine contains blood, bile, and methemoglobin.

Many of these cases are doubtless due to infection conveyed through the umbilical cord, and while all cannot be regarded as syphilitic in origin, a certain percentage probably belong to that class.

Morbus Maculosus Neonatorum.—This term is applied to a non-syphilitic purpura of the new-born which is characterized by jaundice, high temperature, gastro-intestinal symptoms, albuminuria, hematuria, etc. In rare instances bleeding from the bowel and mouth is seen. Minute hemorrhages into the conjunctiva are not unusual.

Principal Complaint.—This varies greatly with the degree of purpura manifested in each particular case. Discomforts are experienced by even the milder cases of purpura, among which should be mentioned malaise, restlessness, mental dullness, and such gastro-intestinal derangements as anorexia, coated tongue, headache, constipation, and diarrhea. A history of having taken certain drugs, of urticaria, or of overexertion may be obtained. In severe types of purpura prodromal symptoms appear two or more days before the eruption is manifest, although occasionally the latter may develop abruptly with extensive cutaneous ecchymoses which increase rapidly. In cases in which hemorrhages from the mucous surfaces of the

mouth, nose, and intestines are profuse, prostration becomes extreme, and the patient complains of muscular tenderness and pains in the limbs, abdomen, and chest. Palpitation, vertigo, dyspnea, and headache result from extreme anemia.

Thermic Features.—In peliosis rheumatica there may be fever, which ranges between 99.5° and 102° F. In simple purpura there is but slight fever, and the temperature is often normal. In secondary purpura the temperature is practically that of the preëxisting malady, so that this symptom is of little diagnostic importance in this type of the affection. In purpura hæmorrhagica there is, as a rule, moderate fluctuation between 100° and 103° F., while in the severe forms a temperature of 104° and 105° F. is occasionally seen. In the purpura of the new-born a temperature of 100° to 102° F. is to be expected, and when jaundice of pronounced character exists, it not unusually reaches 104° to 106° F.

Physical Signs.—Inspection.—In practically all forms of purpura hemorrhages into the skin appear in the form of petechiæ (pin-point hemorrhages), vibices (streak-like hemorrhages), or ecchymoses (hemorrhages varying from the size of a pea to that of a horse-chestnut). At first minute hemorrhagic spots, bright red in color, are to be seen surrounding the hair; but later they change gradually and become yellowish brown, nearly yellow, and eventually disappear. The spots are not materially altered when pressure over them is made, nor by stretching the skin, a feature that serves to differentiate petechia from other skin eruptions.

Purpura displays a tendency to occur in successive crops, and after several of these have appeared, great variation may occur in the color of the hemorrhagic spots, all of which were unaltered by pressure. The lower extremities are more commonly the seat of the eruption than is the surface of the trunk. In rare cases these hemorrhages terminate in gangrene. There may be some slight oozing of serum from the surface of large hemorrhagic extravasations.

Palpation.—In rheumatic purpura the joints are frequently enlarged, and fluctuation over the large articular surface is not unusual.

Laboratory Diagnosis.—The blood findings of purpura closely resemble those seen in the various types of secondary anemia, and are, therefore, of no special importance in establishing a diagnosis.

Urine.—In neurotic patients the quantity of urine voided in the twenty-four hours is above the normal, and it may contain blood and red and white cells. Hemoglobinuria is found at times, and in secondary purpura, in which there is an associated nephritis, renal casts are encountered.

Diagnosis and Differential Diagnosis.—The diagnosis rests entirely upon the appearance of minute hemorrhages into the skin and mucous membranes. Purpura must be distinguished from the hemorrhages occurring in profound anemia and in scurvy, in which cases the history usually serves to differentiate them from the first-named disease.

Clinical Course.—Purpura of whatever type is always of grave significance, and when it develops during the course of another malady, it renders the prognosis of that condition more unfavorable. The degree of hemorrhage into the skin, coupled with the general condition of the patient, naturally controls, in the main, the duration and gravity of this affection.

SCORBUTUS (SCURVY).

Remarks.—A constitutional disorder resulting from dietetic errors, and characterized pathologically by decided impoverishment of the blood, with degeneration of the red cells, a peculiar sponginess of the alveolar mucous

membranes, with a liability to hemorrhages into and from the mucous surfaces and into the skin, coupled with a brawny induration involving the flexor muscles of the lower limbs. Extensive hemorrhage may be found in the region of the large joints (knee), and such hemorrhages at times occur beneath the periosteum. Both the viscera and the tissues contain an abnormally small quantity of blood, and hemorrhages into the viscera and into the serous surfaces are by no means uncommon.

Clinical Varieties.—Two distinct varieties of scorbutus are to be recognized: (*a*) The scurvy of adults, and (*b*) that occurring in infants.

Predisposing and Exciting Factors.—A prominent factor in the production of scurvy is the long-continued use of a dietary practically devoid of vegetables; it has also been claimed that the absence of salts unbalances the metabolic equilibrium. These cases of scorbutic anemia often show an absence of hydrochloric acid in the stomach-contents both before and after the symptoms of scurvy have appeared. Again, the total acidity of the gastric juice has been shown to be below that of the normal in certain instances, but this finding is by no means constant. For some time prior to the development of the true symptoms indigestion is a prominent feature.

Such other factors as unhygienic surroundings (filth, insufficient sunlight, exposure to cold and wet, too active muscular exercise and overwork, with insufficient fresh air), heredity, and mental anxiety, occasioned, for example, by the loss of friends, financial reverses, etc., engender a soil upon which scorbutus may develop.

Age.—Scurvy is far more common at the extremes of life, being found oftenest in children who are nourished upon artificial foods. It is also common in the aged, and among individuals who develop an eccentric appetite for certain foods; it also occurs among those who, because of some gastric malady, are unable to eat fruits. The disease may, however, occur at any time in life, but it is uncommon during the second, third, and fourth decades; it rarely develops among the insane and among persons who are continually confined, as, *e. g.*, in prisons and in homes.

Principal Complaint.—Scurvy is a condition that develops insidiously, one of the first marked manifestations being an enlargement of one of the knees, which becomes tense, acquires a purplish tinge, and in every particular resembles an ordinary bruise. For some time it has been observed that the patient became progressively weaker, and that the mental faculties have been likewise dulled; despondency may also have been observed. Early in the course of scurvy there are arthritic pains, which may be severe, and the pains in the muscles and along the course of the long bones are often excruciating. The anemia, which is at first mild, becomes marked, and the patient suffers from such additional symptoms as palpitation, shortness of breath, swelling of the gums, and bleeding from the mucous surfaces, especially of the gums, where there is a continuous oozing of blood. Hemorrhage from other mucous channels—epistaxis, hemoptysis, hematemesis, melena—is occasionally seen. Hematuria is of uncommon occurrence. In several instances loosening of the teeth has occurred.

Among other annoying features are a foul breath and disordered taste, which is most pronounced upon awakening from a night's sleep. The patient has little or no desire for food, and complains of constipation, though in rare instances diarrhea may develop. During the entire course of the disease the patient complains of pain, which may at times be severe, and again dull, or there may be merely a sense of discomfort; lancinating pain is experienced when the clothing presses upon swollen joints.

Thermic Features.—When fever is present in scurvy, it is usually due to the development of some complication, *e. g.*, bronchopneumonia. A subnormal temperature is by no means unusual.

Physical Signs.—Inspection.—The patient does not, as a rule, become emaciated, but the skin is pale and there is an apathetic expression; the face, as well as the fingers and feet, appear to be edematous. The gait is unsteady, and indicative of weakness; the skin is lusterless, yellowish or muddy in color, with, there may be, yellowish or greenish areas, which probably result from profuse hemorrhages. Minute hemorrhages into the skin are the rule, and these appear first upon the legs, around the hair; when the hemorrhages are profuse, they spread over a somewhat large surface, studding the skin with ecchymotic spots. If the hemorrhages are unusually severe, they are termed ecchymoses. In severe types of scurvy vibices (streak-like hemorrhages) are not unusual, and in one case observed by us bullæ were numerous. Swelling in the region of the joints is often the result of subperiosteal hemorrhages. Later, the skin displays a nodular appearance, due to the infiltration of blood into the tissues, which blood has been partly absorbed; it is possible at this stage to see hemorrhagic areas, varying from a bright red to a brownish yellow or yellow in color.

Upon **palpation** the muscles are found to be soft and flabby, the skin, especially of the face, is often edematous, and the ankles and swollen joints are usually hard. The enlargements seldom fluctuate, since the hemorrhage is, as a rule, subperiosteal. Great tenderness is elicited over the swollen areas. The mucous membrane of the mouth, and particularly that of the gums, has a spongy feel, and bleeds when pressure is made upon it. The pulse is soft and small, the heart impulse is feeble, and palpitation and accelerated pulse follow the slightest exertion. In cases of long standing bone necrosis may occur, and the epiphyses separate from the shaft.

Auscultation.—In uncomplicated cases the heart action may be rapid and irregular, and a soft systolic murmur is heard over the base of the organ.

Special Features.—Profound mental depression is a prominent feature, and insomnia often causes serious annoyance. Meningeal hemorrhage is occasionally observed. Failing vision, retinal hemorrhages, night blindness and day blindness, as well as delirium, are among the unusual findings in scurvy.

Laboratory Diagnosis.—Urinary symptoms vary in direct proportion to the severity of the condition; thus the quantity of the urine is diminished, the color is high, and there is a high specific gravity (about 1.020); phosphates in excess of the normal are also commonly present. The solid constituents are said to be correspondingly diminished, although it has been stated by certain observers that the normal chlorids are increased. Albuminuria is not uncommon, but nephritis, when present, should be regarded as a complication.

The blood changes in scurvy are the same as those of secondary anemia. When the skin is punctured the blood oozes freely, is pale, and of almost watery consistence.

The percentage of *hemoglobin* is decidedly reduced, and may vary between 75 and 40 per cent., or even lower in extreme cases. The red cells show a corresponding reduction in number. Leukocytosis is not an essential feature in scurvy, although in several cases studied by us it developed

when the disease was at its height, but its occurrence may have been due to some intercurrent condition. In other cases observed we found the leukocytes to be practically normal.

A microscopic study of the living blood shows that rouleaux formation is imperfect. The cells are often disseminated equally throughout the field, and there may or may not be great variation in the size of the erythrocytes.

Stained blood displays practically all the features common to degeneration of the red cells, *e. g.*, discoloration, unequal distribution of the hemoglobin, punctate basophilia, poikilocytosis, etc. In mild cases we have seen lymphocytosis, but the results of the examinations have been so varied in different cases that it cannot be said that there is any constant finding in the leukocytes of scurvy.

Infantile Scorbutus.—Pathologic Definition.—A disease of children characterized pathologically by changes practically identical with those previously described under Scorbutus.

As in the scorbutus of adults, the exciting cause of this malady is not known. Some writers believe infantile scorbutus to be identical with rickets, and consider it under the head of hemorrhagic rickets, whereas others hold that it has no connection with this malady. The majority of observers, however, believe that rickets predisposes to the development of scurvy, but there is little to prove that these two diseases are dependent on the same etiologic factor.

History and Principal Complaint.—Practically all cases of infantile scorbutus are found to occur in children who are nourished artificially. It is claimed that the affection is more common among children nourished upon certain prepared foods. It is equally likely to follow the use of sterilized milk, malted milk, and the like.

Age.—The disease usually develops during the first ten months of life, but may be found to occur as late as the eighteenth month; it is occasionally seen in older children. The majority of cases, however, develop before the fourteenth month. This form of scurvy is equally common among both the well-to-do and the poor.

The skin presents the features characteristic of the scurvy of adults. Emaciation is often pronounced, and the general symptoms of inanition are in evidence. Children with scurvy generally shriek when moved, and cry bitterly while being dressed or bathed; the chief point of extreme tenderness appears to be in the lower extremities. Peculiar, imperfectly cylindric enlargements of the femora are often observed, and these are, as a rule, extremely tender. One leg is generally involved at a time, and within a short period the other leg shows a slight deformity. At first the child rests with his legs flexed, but if there are repeated hemorrhages or epiphyseal separation, the legs may be straightened or often somewhat curved, owing to these anatomic changes. Rarely, involvement of additional bones may follow in somewhat rapid succession, but these lesions are, as a rule, less pronounced than those of the legs.

If the teeth have appeared, they are seen to be surrounded by the characteristic spongy gums. Cutaneous hemorrhages may develop in the limbs, and particularly about the face and eyes; in severe cases the conjunctivæ may show ecchymoses.

Summary of Diagnosis.—A history of dietetic errors, swollen and spongy gums, progressive weakness, mental hebetude, and tenderness over the bones and epiphyses, with hemorrhages into the skin, make the diag-

nosis of scorbutus clear. The fact that improvement follows the institution of dietetic treatment further supports the diagnosis of scurvy.

Differential Diagnosis.—The history will serve to separate scurvy from *purpura.* Scurvy may be confused with *rickets* and since the two conditions are often present simultaneously, the distinction becomes quite difficult. The involvement of the lower extremities, with limited motion, extreme tenderness, partial paralysis, and the occurrence of large, more or less cylindric, subperiosteal swellings strongly favor the existence of scurvy. The peculiar gloss of the skin over these cylindric enlargements, the fact that the skin does not pit on pressure and is not hot to the feel, and that a permanent deformity of the bone remains after the swellings become absorbed, further support the diagnosis. The sponginess of the buccal mucous membrane, so characteristic of scurvy, is absent in true rickets. Hemorrhages into the skin are also seen in *leukemia.*

Acute lymphatic leukemia often resembles scurvy, and we have seen several cases of scurvy in which the lymphocyte count and the enlargement of the lymphatic glands resembled those found in acute leukemia. The general course of the disease, the rapid improvement under treatment, and the fact that the leukocyte count seldom exceeds 60,000 in scurvy, will serve as points for the differentiation of these conditions.

Course.—In mild and even in moderately severe cases rapid recovery follows the institution of proper hygienic (dietetic) treatment. The presence of complications—*e. g.,* bleeding, effusion into the pleura or pericardium, pulmonary infarction, and nephritis—adds materially to the gravity of this malady. Although the majority of these patients go on to convalescence within a period of a few weeks, cases are rarely to be seen that linger over a long period and show but little result from treatment. This type of the malady is found in the aged and may terminate fatally.

PARASITOLOGY OF THE BLOOD.

During the course of acute infectious diseases the microörganisms that cause the disease often circulate in the blood-stream. These organisms may be—(1) vegetable parasites (various bacteria and fungi) or (2) animal parasites (filaria bancrofti and other species of the genus filaria, the various forms of malarial parasites, trypanosoma gambiense, spirocheta recurrens, and spirocheta duttoni). (See Bacteriuria, p. 703.)

Method of Detection of Bacteria in the Blood.—In order to prove the existence of a bacteriemia it is necessary to remove about ten cubic centimeters of blood from one of the superficial veins, preferably the median basilic, and to distribute the blood so obtained in three small Erlenmeyer flasks, each of which contains about 30 c.c. of sterile bouillon or glucose bouillon, and in three shake plates of glucose-agar or plain agar.

The bend of the elbow is scrubbed with soap and water, then washed with water, alcohol, and 1 : 1000 solution of mercury bichlorid, and a wet bichlorid dressing applied and allowed to remain for one hour. Then the nurse or an assistant applies a tourniquet above the elbow, so as to make the veins prominent, and cuts the bandage holding the dressing. Then the operator, with sterile hands, removes the dressing and plunges the needle of a previously sterilized antitoxin syringe into the distended vein, withdrawing the necessary amount of blood. This is immediately distributed between the flasks and the melted agar tubes; the latter are plated and all are incubated for twenty-four hours. At the end of the incubation period the flasks and plates are studied according to usual bacteriologic methods.

The following organisms have been recovered from the peripheral blood: Streptococcus pyogenes, staphylococcus pyogenes, bacillus pyocyaneus, diplococcus pneumoniæ, gonococcus, tubercle bacillus, bacillus coli communis, bacillus enteritidis, bacillus typhosus, bacillus paratyphosus, bacillus pneumoniæ (Friedländer), bacillus pestis, bacillus anthracis, bacillus mallei, micrococcus melitensis, etc.

The organism discovered in 1873 in the blood of patients suffering from relapsing fever, by Obermeier, and called spirillum obermeieri, is now known as spirocheta recurrens. It is generally accepted as the cause of relapsing fever of the European type. Similar organisms have since been found in connection with African tick fever (spirocheta duttoni), the Indian form of relapsing fever (spirocheta carteri), yaws (treponema pertenue), and syphilis (treponema pallidum); the last two organisms are, however, not found in the blood. Spirillar organisms are also found in the mouth and the vagina (spirocheta refringens) and sometimes in the pus of abscesses. Their position in the natural orders is subject to dispute, one group of observers believing them to belong to the bacteria and another group believing them to be protozoa.

Clinical Significance.—The isolation of a bacterium from the circulating blood is positive evidence that that organism is responsible for the acute infection from which the patient is suffering. In cases of acute ulcerative endocarditis it has been observed that more patients recover when the staphylococcus pyogenes is the cause of the disease then when the streptococcus pyogenes is the offending organism.

DISEASES OF THE DIGESTIVE SYSTEM.

DISEASES OF THE LIPS, TONGUE, AND MOUTH.

HERPES LABIALIS.

Pathologic Definition.—An eruption of the lips that first appears as a crop of vesicles that later unite to form thick crusts.

General Remarks.—In appearance, herpes labialis resembles closely herpes affecting other portions of the body, and its tendency to become more extensive than herpes occurring elsewhere is usually dependent upon the fact that the patient is continually irritating these parts by picking at them. When the eruption affects the corners of the mouth, irritation is produced by eating and talking.

Clinical Significance.—Herpes labialis is a symptom of diagnostic value in three acute infectious maladies—*e. g.*, cerebrospinal meningitis, lobar pneumonia, and malaria. Herpes may, however, be present in practically all febrile conditions, and is also associated with acute rhinitis, acute gastro-intestinal catarrh, certain febrile maladies, and neuritis, chronic gastric catarrh, and helminthiasis.

LABIAL ECZEMA.

Definition.—An eczematous condition affecting the lip, at or near the junction of the mucous membrane with the skin, in the median line, and at the angles of the mouth. It is characterized clinically by a branny or scaly desquamation, with a tendency toward the appearance of deep fissures, which are extremely painful upon the taking of warm and hot foods.

General Observations.—In severe cases these eczematous areas bleed readily and are extremely painful. Eczema of the lips appears to be aggravated by exposure to cold, and is not infrequently a part of a general gastro-intestinal irritation. Like eczema affecting other portions of the body, it is capable of being excited by certain bacteria and by fungi.

EPITHELIAL DESQUAMATION OF THE TONGUE (GEOGRAPHIC TONGUE).

Pathologic Definition.—A disease characterized by an extensive desquamation of the lingual epithelium from the dorsum and margins of the tongue, the formation of small circular or crescentic, reddened blotches, with slightly elevated grayish margins (lingual psoriasis), and by an absence of salivation, pain, or irritation.

Principal Complaint.—Epithelial desquamation is seldom seen by the physician in its early stages, and the patient rarely complains of any inconvenience. The peculiar condition of the tongue is, as a rule, detected

accidentally. The initial patches seen upon the tongue are grayish, and give the tongue a thickened appearance. There is an increase in the size of the initial patches, which may result in the detachment of large areas of epithelium, which leave behind a bright-red surface. A few days after the detachment of the scales of the epithelium the red zone is seen to be surrounded by an elevated grayish border, which may vary in width from $\frac{1}{20}$ to $\frac{1}{12}$ inch. These reddened, crescentic areas are most likely to be situated near the base of the tongue and along its lateral margins; they may cross the tongue transversely, and eventually cover the entire surface of the tongue. With the fading and disappearance of the older patches new ones are seen to form upon different portions of the tongue, and this change of epithelial covering may continue for weeks, months, or even years.

Simple desquamation of the lingual epithelium, the cause of which is unknown, is to be distinguished from desquamation due to infection (see Gonorrheal Stomatitis) and the so-called geographic tongue.

ACUTE GLOSSITIS (GLOSSITIS ACUTA).

Pathologic Definition.—An acute parenchymatous inflammation of the tongue resulting from injury to the organ, as, *e. g.*, from a burn, a sting, a bite, a wound, etc.; less often it is due to infection complicating mercurial and other forms of stomatitis.

Principal Complaint.—The tongue is extremely painful, so that talking, chewing, and swallowing excite great distress. In the majority of cases there is a continuous dribbling of saliva, although, rarely, the tongue is dry.

The patient complains of great discomfort, owing to pronounced swelling of the tongue, inability to open the mouth, and dyspnea. He is unable to take any solid food, and in some instances hot and extremely cold foods excite pain. After the disease has existed for some days discomfort is felt at the angles of the jaw, in the lingual regions, and along the sides of the neck.

Thermic Features.—The temperature of the mouth may range between 101° and 104° F. In one case studied at the Philadelphia Hospital the temperature of the buccal cavity was above 103° F. for seven days, while the rectal temperature never exceeded 101° F.

Physical Signs.—Inspection.—The tongue is red and swollen, and its surface may be glazed; it displays deep grooves and fissures, or the papillæ may be unusually prominent and covered with a thick, tenacious exudate. The tongue presses against the teeth, which have worn deep notches into its margin. The sublingual and cervical glands are swollen, and may be sufficiently enlarged to cause decided distortion of the features.

Palpation confirms inspection with regard to the lymphatic enlargements (sublingual, cervical).

Clinical Course and Duration.—In the majority of instances acute glossitis goes on to recovery in from seven to twelve days. Lingual abscess and diffuse pharyngitis rarely complicate glossitis, and when present, materially increase the gravity of this affection.

CHRONIC GLOSSITIS.

Remarks.—A chronic superficial inflammation of the dorsal surface of the tongue, provoked by the excessive use of alcoholic spirits, tobacco, and highly spiced foods. The patient seldom complains of any decided inconvenience, and the disease causes no pain. The surface of the tongue is

rough and glazed at certain points, and dull and lusterless at others. Crossing the dorsum of the tongue are deep grooves and fissures that seldom, if ever, bleed, and are not sensitive to mild irritation.

TONGUE-SWALLOWING.

Definition.—A condition seen during infancy, in which the tongue is turned backward into the pharynx and interferes with respiration.

Predisposing and Exciting Factors.—Suffocation from obstruction to the respiratory passages by the tongue may occur in children previously healthy, although it is more commonly seen during the course of whooping-cough, and following epileptiform seizures. The conditions that allow tongue-swallowing to develop are: (*a*) An abnormally long frenum and (*b*) a relaxed condition of the tongue. It should be remembered that, particularly in children of lowered vitality, the base of the tongue may fall back into the throat, so that it partially or entirely covers the epiglottis. A similar condition may develop in adults during complete anesthesia.

Hennig reports a case in which sudden death followed an epileptic attack, and at autopsy the tip of the tongue was found tightly wedged in the esophagus. Holt states that he has repeatedly seen serious cases of dyspnea in infants, the result of tongue-swallowing.

ULCERS OF THE FRENUM.

Definition.—These ulcers are situated between the attachment of the under surface of the tongue and the buccal cavity beneath.

Predisposing and Exciting Factors.—Whooping-cough is the most potent factor in the production of ulcers of the frenum. Friction against the central incisors appears to be necessary to its production, although we have seen ulcer of the frenum before the lower incisors have appeared. Malnutrition also favors the development of the ulcer, and in adults this condition may develop in such chronic afebrile states as diabetes, nephritis, and valvular heart disease. It is often associated with acute and chronic maladies in which cough is a prominent symptom, *e. g.*, in croupous pneumonia, pulmonary tuberculosis, and those conditions in which the digestion is materially impaired, as, for example, in chronic gastritis, obstructive jaundice, and dysentery.

The ulcer may be confined to the frenum, or may extend to the tongue or to the tissues of the mouth, attaining a size of from $\frac{1}{8}$ to $\frac{1}{2}$ inch in diameter. Ulcers of the frenum are yellowish gray in color, and surrounded by an elevated margin.

ACUTE CATARRHAL STOMATITIS (STOMATITIS ERYTHEMATOSA).

Pathologic Definition.—A disease of the mouth characterized pathologically by a catarrhal inflammation of the mucous membrane of the buccal cavity and an excessive secretion of the salivary and muciparous glands.

Predisposing and Exciting Factors.—**Age** serves as a potent predisposing factor, the disease being most common in children at the time of the eruption of the first teeth, although catarrhal stomatitis may be found at any age. **Traumatism,** even though slight, is often followed by a somewhat extensive catarrhal inflammation of the buccal mucosa. The ingestion of irritating foods, *e. g.*, hot and highly spiced articles, also con-

tributes toward the production of stomatitis. Catarrhal stomatitis is frequently an annoying complication during the course of measles, influenza, tuberculosis, scarlet fever, diphtheria, and acute gastritis; it is also encountered late during such chronic afebrile conditions as nephritis, diabetes, hepatic cirrhosis, valvular heart disease, and chronic dysentery.

Principal Complaint.—The patient is restless and continually annoyed by the increased flow of saliva, which may be clear or blood stained, but is always tenacious. In severe cases the saliva may pour from the mouth and cause violent irritation of the lips and face. Rarely, especially in the case of adults, the excess of saliva may be swallowed.

The patient shows no inclination to take food, although he may experience a feeling of hunger. Children, as a rule, refuse the breast and the bottle. When stomatitis occurs during the course of acute infectious maladies, the most prominent symptoms of the initial infection are intensified.

Physical Signs.—Inspection.—The greater portion of the buccal mucous membrane is infected, and the capillaries are markedly dilated; here and there over the surface small hemorrhages are to be seen. Swelling of the mucous membrane is also present, and is most in evidence along the insertion of the teeth, although in severe cases the lips may be edematous. The mucous follicles are swollen, and in virulent types small cysts form in the mucosa.

The tongue is heavily coated, its edges are intensely congested, and the papillæ are slightly elevated. When stomatitis complicates scarlet fever or other acute infections, it is not uncommon for large flakes of epithelium to slough from the surface of the tongue. Deep fissures in both the tongue and the lips are occasionally seen.

Palpation.—The mucous membrane of the mouth and lips is hot to the touch, and after the disease has progressed for from two to four days, the glands beneath the jaws become swollen and tender. (See Drug Eruption, p. 401.)

Laboratory Diagnosis.—The flow of saliva is greatly increased, and the secretion is acid in reaction. If the condition progresses to ulceration, the saliva becomes blackened in color, alkaline in reaction, and emits a fetid odor.

Microscopically, many desquamated epithelial cells, erythrocytes, leukocytes, and tissue débris are to be seen, and a profusion of bacteria and fungi are also present. It is quite difficult to show specifically that any one or more of the bacteria recovered from the saliva or the ulcers have any etiologic significance in catarrhal stomatitis.

Clinical Course and Duration.—Acute catarrhal stomatitis runs a short course, reaching its acme by the end of the second day, and terminating in recovery in from four to seven days. In those in whom the general nutrition is greatly reduced, as in Bright's disease, etc., this condition may tend toward chronicity.

HERPETIC STOMATITIS (Follicular, Vesicular, Aphthous Stomatitis).

Pathologic Definition.—A self-limited disease, characterized by congestion of the buccal mucous membrane, with the appearance of small, isolated, yellowish or yellowish-white patches, which subsequently break down, to form superficial ulcers. In the severer types these ulcers may coalesce, with the formation of extensive ulceration, whose edges are ragged.

Predisposing and Exciting Factors.—The *exciting cause* of herpetic stomatitis is unknown, yet the consensus of opinion is that it is

bacterial in origin, although Holt and Forchheimer are strongly inclined toward the belief that it is nervous in origin. Children under one year are more susceptible than either older children or adults. Catarrhal conditions of the gastro-intestinal tract, dentition, malnutrition, anemia, and the exanthemata seem to predispose to aphthous stomatitis.

Principal Complaint.—Soreness of the mouth is the most constant complaint in herpetic stomatitis. Pain is particularly acute, and often prevents the child from taking food, and even adults may find it impossible to take solid food for a period of several days. Talking may be difficult and even painful. The ingestion of hot and cold foods is followed by intense lancinating pains, and there is considerable soreness along the angles of the jaw.

Physical Signs.—Inspection.—The most characteristic finding in aphthous stomatitis is congestion of the mucous membrane of the mouth, which exhibits a variable number of small superficial ulcers that appear in successive crops. These ulcers may be found on the interior of the cheek or of the lips, and, in fact, they may be distributed over any portion of the mucosa of the mouth and pharynx. The number of ulcers present at a given time may vary within wide limitations, and ulcers at various stages of development are always to be seen during the acute stage of this affection. The *general features* are slight fever, anorexia, and irritability.

Characteristics of the Ulcers.—These ulcers vary in size from that of a pin's head to a millet seed, seldom attaining the size of a pea. They are first yellowish in color, but later present a dirty grayish hue, and we have repeatedly seen ulcers that in many respects resemble diphtheric membrane. Small ulcers are usually surrounded by a red areola, and close inspection is necessary to discover that there is a distinct excavation, since at first glance the ulcerated surface appears to be slightly elevated. Many ulcers may coalesce to form one large irregular patch, but this is by no means a common finding.

The tongue is, as a rule, swollen, while its edges may be greatly congested. Small ulcers frequently appear underneath the tongue and on the frenum.

Clinical Course and Duration.—Herpetic stomatitis terminates in recovery in from five to fourteen days. There is a decided tendency toward relapses in both children and adults who are not well nourished.

FETID STOMATITIS (ULCERATIVE STOMATITIS; RIGGS' DISEASE).

Definition and Remarks.—A disease of the mouth characterized by ulceration of the mucous membrane at its junction with the teeth. The disease does not appear before the development of the teeth or after their removal; it shows a tendency to extend.

Predisposing and Exciting Factors.—Age is a predisposing factor, adults being the more susceptible. Ulcerative stomatitis may make its appearance during convalescence from infectious maladies, *e. g.*, scarlet fever, typhoid fever, influenza. The use of improper foods and of food that has undergone putrefaction is said to contribute toward the production of fetid stomatitis. The disease is infrequent among the better classes. Neglect of the toilet of the mouth and of the teeth tends decidedly toward the production of stomatitis. Ulcerative stomatitis is a symptom of scurvy, and follows the prolonged use of such metallic substances as lead, mercury, and phosphorus.

Principal Complaint.—The patient complains that his mouth is sore, and that the gums bleed readily upon the slightest irritation. He

is unable to take highly spiced or hot foods, and talking may cause a variable amount of discomfort. The *general features* attending the condition indicate lowered vitality, and moderate fever is generally observed.

Physical Signs.—Inspection.—Ulcerative stomatitis may attack any portion of the buccal mucous membrane, but is oftenest located at the margin of the gum (most commonly opposite the lower incisors), from which point it extends in every direction. In mild cases the gums are swollen, spongy, and of a deep reddish or purple hue, and bleed when irritated either by food or by the tooth-brush. This congestion of the gums may encircle the incisor, canine, molar, or all the teeth. In many instances it affects only the gums of one side of the mouth, but in the vast majority of cases bilateral involvement occurs. The ulcerative process may rarely extend to and involve the lips, roof of the mouth, and soft palate. In fetid stomatitis the conditions are most favorable for a general septic infection of the mouth, and this occasionally follows, in which case the entire mucous membrane becomes the seat of an acute, often purulent, inflammation.

The red and congested membrane surrounding the teeth later assumes a grayish or dirty hue, and there may be a peculiar yellowish deposit, which, when removed, leaves behind a bleeding surface. After ulcerative stomatitis has existed for some time the gums retract and the teeth become loosened. Instances are recorded in which necrosis of the jaw occurred. In a case studied at the Philadelphia Hospital edema of the gums was so marked that only the tips of the incisor teeth were visible after the disease had progressed for one week. The mouth is usually bathed in saliva (often blood-stained), which commonly dribbles from between the lips. Hemorrhagic saliva and bleeding from the buccal cavity is a rather common feature during some stage in the course of the following general conditions, with or without local disease:

Scurvy,
Hemophilia,
Purpura,
Mercurialism,
Splenomedullary leukemia,
Pernicious anemia,
Lymphatic leukemia,
Hodgkin's disease,
Splenic anemia,
Phosphorus-poisoning,
Tuberculous gingivitis,
Yellow atrophy of the liver,
Arsenic-poisoning,
Plumbism (chronic).

Among the local conditions causing bloody sputum are:

Traumatism,
Aphthous stomatitis,
Ulcerative stomatitis,
Gangrenous stomatitis,
Vincent's angina,
Noma,
Epithelioma,
Myeloid sarcoma,
Alveolar abscess,
Papilloma,
Pyorrhea alveolaris,
Actinomycosis.

The tongue is swollen to such a degree that its edges bear the imprints of the teeth. The dorsum of the tongue is heavily coated with a grayish or yellowish-gray fur. The skin and the mucous membrane of the conjunctivæ are pale, and other evidences of secondary anemia are present. (See Drug Eruption, p. 401.)

Palpation.—Marked enlargement of the submaxillary lymph-nodes is felt beneath the jaw, and the nodes are tender and painful upon pressure. The gums are soft and spongy.

Laboratory Diagnosis.—Scrapings from the surface of these ulcers will be found to contain, microscopically, great numbers of epithelial cells,

mucus-corpuscles, leukocytes, red blood-cells, and many varieties of bacteria, among which the streptothrix, streptococcus, diplococcus, staphylococcus, and spirillum of Vincent deserve special mention.

Clinical Course and Duration.—Ulcerative stomatitis frequently displays but little tendency toward recovery, and such cases may last for weeks or months; where, however, treatment is judiciously applied, recovery follows in from seven to fifteen days.

ULCERATIVE STOMATITIS WITH ANGINA.

Remarks.—Many cases of this particular type of stomatitis have recently been reported, and in each a bacteriologic study of the saliva disclosed the presence of a fusiform bacillus and a spirillum, which are usually regarded as the bacillus and spirillum of Vincent.

Ulcerative stomatitis with angina does not differ markedly in its clinical aspects from the type of stomatitis previously described, except that angina forms a prominent symptom. An analysis of the recorded cases furnished sufficient evidence to show that Vincent's bacteria occupy a prominent place in the production of stomatitis with angina. Several writers, however, have described this condition without mentioning Vincent's organisms, and since their microscopic reports appear to be complete, it is scarcely reasonable to believe that they would have overlooked these large bacteria if they had been present. In our opinion, a conservative view is the only safe one to accept in regard to these organisms as the causal factor in the production of ulcerative stomatitis, since reliable records exist in which no mention is made of this organism.

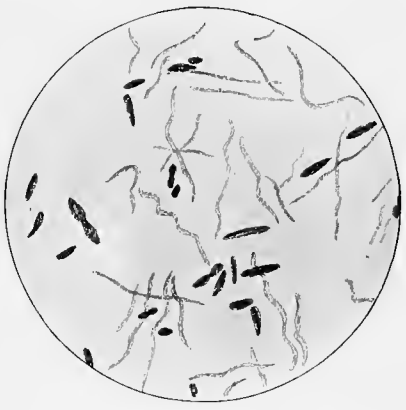


Fig. 156.—Bacillus and Spirillum of Vincent, from Case of Ulcerative Stomatitis (Boston).

The Bacillus of Vincent.—A straight, fusiform bacillus, expanded at its center, with pointed extremities, measuring 6 to 12 microns in length.

Method of Detection.—Smear the saliva thinly over the slide and permit it to dry in the air; after this the specimen should be fixed by passing it three times through the flame. Stain with anilin oil, gentian-violet water, methylene-blue, or, if preferred, by Gram's method. Most of the bacteria decolorize by Gram's process, although a few supposedly degenerated forms retain a violet hue.

Spirillum of Vincent.—This spirillum is slightly longer than the bacillus of Vincent, and is found to occur in the stained specimens either singly, or more often in clusters, and occasionally in dense aggregations. In the living specimen Vincent's spirilla are seen to be motile. The tinctorial properties of this spirillum are similar to those of Vincent's bacillus.

GONORRHEAL STOMATITIS.

Remarks.—An unusually rare condition in which stomatitis follows infection of the mouth by the gonococcus. The disease was first described by Dohrn and Rosinksi, who found it in the new-born. The lesions are in no way characteristic of gonorrheal infection, and a microscopic study of the secretion is necessary in order to make a diagnosis.

Laboratory and Differential Diagnosis.—Small portions of the exudate should be smeared thinly upon the slide and stained for gonococci. (See p. 898.) It must be borne in mind that diplococci are normally present in the buccal secretion, and these are at times microscopically indistinguishable from the gonococcus.

In order to determine that an infection of the mouth is due to the gonococcus, cultures must be made. The distinctive difference between the gonococcus and the diplococcus of the normal buccal secretion is that the former develops only when planted upon a special medium and is Gram-negative, whereas other diplococci will develop in blood-serum, bouillon, and agar, and are Gram-positive.

DIPHTHERIC STOMATITIS.

In grave cases of diphtheria the false membrane may extend from the throat into the oral cavity, and may involve the lips. (See Diphtheria.)

GANGRENOUS STOMATITIS (NOMA).

Pathologic Definition.—An acute inflammatory disease of the mouth, first involving the mucous surface of the cheek, and characterized by rapidity of development, extensive destruction of the tissues of the mouth with gangrene, and often perforation of the cheek and an early fatal termination.

Remarks.—Noma is a somewhat rare disease, and although considered with diseases of the mouth, it is by no means limited to the buccal cavity, but may develop in the genitalia of the female. Holt has detected it in the external auditory meatus.

Exciting and Predisposing Factors.—Joseph Sailer recovered diphtheria bacilli from the scrapings of exudate of extensive gangrenous processes of the buccal cavity. This observer's findings have since been confirmed by us in two cases, both in children, in whom death resulted from cardiac paralysis. Holt found pure cultures of streptococci, whereas in a case studied by Cornil and Babes, as well as in those reported by Ranke, a streptococcus was the prominent organism.

Noma most often complicates measles or other diseases of childhood. It is likely to develop in ill-nourished children, such malnutrition being the result of gastro-intestinal catarrh, whooping-cough, and bronchitis. It may be seen to follow other forms of stomatitis. The disease is doubtless mildly contagious, and may affect several children of a home or an institution.

Age figures prominently as a predisposing factor, although we have seen one case occurring in a man aged fifty-two years.

Principal Complaint.—The child is unable to eat, and presents the general appearance of being very ill. Early during the course of noma the patient may be in a fairly good physical condition, but as the disease progresses he grows rapidly worse, and in the course of two or three days there are profound prostration, mental dullness, and a weak, rapid pulse, which soon becomes dicrotic and easily compressible. Diarrhea may develop on the third day, and greatly increases the prostration. The breath is very foul, the odor being that of gangrenous tissue. Not infrequently this odor of the breath is the first thing that attracts attention to the condition.

Pain is uncommon, although in rare instances it may be quite severe. Hemorrhage from the arteries of the cheek is one of the annoying complications, and is not infrequently the immediate cause of death.

Thermic Features.—The temperature reaches 101° to 103° F. by the second day, and as the disease progresses fever becomes rapidly higher, until, by the end of the third day, the temperature may reach 104° to 105° F. A subnormal temperature is a precursor of a fatal issue, and develops in from six to eighteen hours before death.

Physical Signs.—**Inspection.**—The initial lesion appears as a dusky spot upon the outer surface of the cheek or lip. Inside the lip or cheek there is a corresponding area, which is dark, greenish-black in color, and surrounding this necrotic mass the mucous membrane is swollen. The gums may become involved, the teeth loosen, and in some reported instances they have fallen out. Necrosis may involve the alveolar process of the lower jaw or perforate the cheek or lip. As a rule, perforation affects but one cheek, although bilateral perforation has been described.

Palpation.—At the margin of the gangrenous area, and for some distance surrounding the teeth, the tissues are tense and somewhat edematous. This infiltration may extend to the tissues of the entire face.

Laboratory Diagnosis.—Scrapings and cultures from the gangrenous débris will be found to contain a profusion of bacteria, among which the streptococcus is most common. The staphylococcus, diphtheria bacillus, and pseudodiphtheria bacillus may also be recovered. The leptothrix, large diplococci, various forms of bacilli, and the spirochæta dentium have been discovered in the scrapings of gangrenous stomatitis. In 1905, in a series of extensive studies, Harrmann reported his findings, attributing the rapid pathologic changes of noma to infection with the spirochæta. There is no known method by which the spirochæta of Harrmann can be distinguished from the spirochæta dentium that is present in normal buccal secretion and in the secretion of the mouth in other conditions, *e. g.*, catarrhal stomatitis and gingivitis.

Summary of Diagnosis.—This depends, for the most part, upon the presence of a rapidly progressing ulceration with a tendency toward development of gangrene of the buccal mucous membrane or of other mucous surfaces, together with marked prostration, and high fever.

Course and Duration.—Seventy-five per cent. of all cases of noma terminate fatally between the fourth and tenth days. The prognosis is rendered more grave when noma develops in ill-nourished children. In favorable cases recovery is unusually slow, and there is marked deformity of the face, due to the extensive cicatrization.

PTYALISM (SALIVATION).

In ptyalism the quantity of saliva secreted daily will be found to exceed the normal—between two and three pints.

Predisposing and Exciting Factors.—An increased flow of saliva occurs during the early months of pregnancy. Salivation is also one of the symptoms of inflammation of the buccal mucous membrane, with the exception of thrush. The saliva is increased at the time of the eruption of the first teeth, in alveolar abscess, and in trigeminal neuralgia. It is not unusual to find the flow of saliva increased during hysteria, and during the menstrual period of hysteric individuals. Salivation has also been seen to occur in hydrophobia. Ptyalism is likewise a symptom of such acute infectious diseases as typhoid fever, variola, and typhus fever, and rarely is it to be seen during the course of epidemic parotitis (mumps).

Drugs increase the flow of saliva; among these should be mentioned

acids, alkalis, cantharides, antimony, copper, gold, mercury, iodin, the iodids, muscarin, pilocarpin, and tobacco.

True ptyalism is to be distinguished from the dribbling of saliva that occurs in idiocy, diphtheritic paralysis, and chronic bulbar and facial paralyses.

MINERAL (MERCURIAL) STOMATITIS (SALIVATION; PTYALISM).

Pathologic Definition.—An acute inflammation of the buccal mucous membrane surrounding the teeth, characterized by an increased flow of saliva following the prolonged use of mercury or of the iodids.

Principal Complaint.—The first symptom is tenderness of the teeth, the patient complaining that they are too long and that, while masticating his food, they are extremely sensitive or even painful. A few days later he complains of a peculiar metallic taste and of great annoyance from the excessive flow of saliva, which may be so markedly increased as to cause a constant dribbling from the mouth. The patient may be unable to take solid foods. In a small percentage of cases diarrhea develops after ptyalism has become pronounced.

Physical Signs.—Inspection.—The gums are red, considerably swollen, and upon palpation are found to be spongy and extremely tender. The tongue is red, swollen, heavily coated, and may be the seat of numerous small ulcers. The gums recede from the teeth, the teeth loosen, and in extreme cases they fall from their sockets; rarely, necrosis of the jaw follows.

Course and Duration.—The prognosis as to recovery is favorable, a cure usually following after withdrawal of the exciting drugs. In mercurial stomatitis the duration is from ten days to four or five weeks, and recovery is materially hastened by judicious treatment.

DRUG ERUPTION.

Lesions of the mucous membrane of the mouth are occasionally seen after the administration of quinin, phenacetin, antipyrin, and aspirin. This class of ulcers appear as erosions that display well-defined margins with overhanging borders. They are painful to the touch, and are seen, as a rule, in persons who display idiosyncrasy for these drugs. (See also Chronic Plumbism, p. 979; Ptyalism, p. 400; and Catarrhal Stomatitis, p. 394.) Bismuth when administered in large doses is at times followed by a bluish ulceration of the gums, which most often makes its appearance about the wisdom teeth. Following such ulceration there is liable to be an extensive pigmentation of the surrounding mucous membrane. Rarely these ulcers display a false membrane.

ACUTE PHARYNGITIS (PHARYNGITIS ACUTA SIMPLEX).

Pathologic Definition.—An acute inflammation of the mucous membrane of the pharynx, which may be either primary or secondary to inflammatory processes elsewhere, or to acute infectious conditions—*e. g.*, influenza, scarlet fever, measles, diphtheria, tonsillitis, and smallpox.

Predisposing and Exciting Factors.—Acute pharyngitis may be due to many causes, among which are exposure to cold and wet, the inhalation of irritating gases, and lowered vitality; it may develop during

the course of an acute or chronic infection. Certain writers assert that pharyngitis is frequently rheumatic in character.

Age.—It is most frequently seen during early adult life—between the seventeenth and thirty-fifth years.

Principal Complaint.—Among the initial symptoms may be chill or a series of slight chills, following which there are moderate fever, anorexia, headache, slight acceleration of the pulse, dryness of the throat, and, at times, of the entire buccal cavity; these are followed by an increased secretion, and later there may be expectoration of mucopus. The catarrhal condition spreads rapidly over the entire throat, and swelling and edema of the fauces, palate, tonsils, and base of the tongue occur.

Usually the inflammation extends to the Eustachian tube, and there is a temporary embarrassment of hearing. In severe cases the mucous secretion may contain a small quantity of blood. Pain at the angle of the jaw is an early and almost a constant symptom, and is decidedly increased by movement of the jaw. The patient describes a peculiar scratching sensation in his throat, as though something were there that should be expelled. Efforts to clear the throat and coughing give no relief. Constipation and headache occur in pharyngitis, and the latter is seldom relieved until the bowels have been freely moved.

In children under ten years of age the initial symptoms and, in fact, the entire course of the disease, may be severe, and associated with marked constitutional symptoms, *e. g.*, a temperature ranging between 102° and 105° F., extreme pain on swallowing, acceleration of the pulse, and increased respiration. If the inflammation extends to the larynx, the cardinal symptoms of laryngitis will be added. (See Laryngitis, p. 34.)

Physical Signs.—Inspection.—There may be some swelling at the angles of the jaw and temporary enlargement of the lymph-nodes in that region. The mucous membrane of the pharynx is at first bright red and dry in appearance, but later it is seen to be covered with a tenacious mucus or with a mucopurulent exudate, which partially conceals the intensely congested condition of the pharyngeal surface.

Edema of the mucous membrane, and often of the pillars and of the soft palate, is apparent. The pharyngeal follicles become acutely inflamed, standing out as glistening nodes.

Summary of Diagnosis.—The diagnosis is based upon the fact that there are pain and tenderness of the throat, with but moderate glandular enlargement; and while markedly inflamed, no false membrane is present. The constitutional symptoms are not so well marked as in tonsillitis, where there is an equal amount of involvement of the throat.

Cultures from the mucous membrane of the pharynx may show the presence of streptococci, diplococci, and bacilli, but no diphtheria bacilli can be discovered.

Microscopically, the mucoid exudate of the pharynx reveals the presence of various forms of bacteria, erythrocytes, leukocytes, and pus-cells.

Differential Diagnosis.—The condition is to be distinguished from the acute pharyngitis of measles and scarlet fever. (See differential table, p. 857.)

Clinical Course and Duration.—The majority of cases terminate in recovery in from three to seven days. One attack predisposes to subsequent seizures, particularly in poorly nourished individuals.

CHRONIC PHARYNGITIS.

Pathologic Definition.—A chronic inflammation of the mucosa of the pharynx, characterized anatomically either by hypertrophy or by atrophy of the follicles, although both processes may exist in the one case.

Varieties.—Among these are chronic hypertrophic nasopharyngitis, follicular pharyngitis, and simple nasopharyngeal catarrh. Chronic pharyngitis is a condition for the specialist, hence it will not be discussed at length here.

Predisposing and Exciting Factors.—Among these are overwork, either mental or physical, and repeated attacks of acute pharyngitis. It is common in inveterate smokers, public speakers, and in singers.

Principal Complaint.—The chief symptoms are a continual desire to clear the throat, and a sensation as of dropping or irritation in the pharynx.

Upon examination the mucous membrane of the posterior wall of the pharynx is found to be hyperemic, dusky, and studded with isolated round bodies that correspond to the enlarged lymphatic follicles. In *pharyngitis sicca*, or dry pharyngitis, this mucous surface is dry and glistening.

ANGINA LUDOVICI.

Definition.—A phlegmonous inflammation of the floor of the mouth and pharynx,with unilateral swelling at the angle of the jaw, difficult respiration, pronounced nervous symptoms, and sore throat.

Predisposing and Exciting Causes.—Actinomycosis, diphtheria, streptococcic infection of the mouth or neighboring glands, scarlet fever, and traumatism are among the causes of the complaint.

Principal Complaint.—The initial symptom is sore throat, which early becomes intense, and continues throughout the course of the disease. The submaxillary glands and glands at the angle of the jaw, together with the adjacent cellular tissue of the neck, throat, and mouth, are involved by the phlegmonous process. Talking and eating decidedly increase the pain. Edema of the pharynx and of the tissues of the neck is followed by difficult respiration.

The *fever* may be high and continuous, or may assume the hectic type. The *pulse* is accelerated, and may become weak and dicrotic. *Nervous symptoms* may be absent during the early stages of Ludwig's angina, but may appear later; coma is not uncommon.

Pus-formation usually follows within a few days, and unless prompt surgical measures are instituted, extensive sloughing of the tissues of the neck is likely to occur.

Clinical Course and Duration.—Difficult respiration and pronounced nervous symptoms, when present, make the prognosis grave. Milder cases recover in from ten days to three weeks, but there is a tendency for relapses to occur. Owing to difficulty in respiration many of the patients become victims of bronchopneumonia, which is doubtless the real cause of death in a large percentage of the fatal cases.

RETROPHARYNGEAL ABSCESS.

Remarks.—An accumulation of pus in the cellular tissue between the pharynx and the vertebral column. The condition is characterized clinically by difficult or impeded breathing, dysphagia, cough, and a variable degree of pain and soreness.

Predisposing and Exciting Factors.—Age is a predisposing fac-

tor, the condition being most common among children. It not infrequently follows acute infections of the throat, *e. g.*, scarlet fever, diphtheria, and tonsillitis. Disease of the vertebræ may predispose to the development of pharyngeal abscess, although this type is somewhat rare, except in children.

Summary and Differential Diagnosis.—**Inspection** reveals a congestion or bulging of the pharyngeal wall, whereas **palpation** elicits fluctuation and, rarely, pulsation. Retropharyngeal abscess may be confounded with aneurism of that region, as in two cases in which a diagnosis of aneurism of the pharynx was made. In a third case that came to our notice an aneurism projecting into the pharynx was ruptured by the passing of an esophageal bougie.

Differential Diagnosis.—The essential clinical differences between retropharyngeal abscess and aneurism are:

Retropharyngeal Abscess.	Aneurism.
1. History of some acute infection of the throat or adjacent structures.	1. Absent.
2. Common in children.	2. Seen after middle life.
3. No evidence of general atheroma.	3. Atheroma the rule.
4. Upon inspection, the pharynx is greatly inflamed, but the tumor does not pulsate.	4. Tumor pulsates.

Prognosis and Duration.—The prognosis is favorable, the majority of cases rupturing spontaneously and the contents of the abscess being either expectorated or swallowed. In abscess the result of necrosis of the vertebra the prognosis is that of Pott's disease.

THE ESOPHAGUS.

METHODS OF EXAMINATION.

Esophagoscopy. — Esophagoscopy is a method of examining the esophagus by means of tubes introduced through the mouth. Retrograde esophagoscopy is a term used to describe the examination of the lower end of the esophagus by the aid of tubes introduced through the abdominal wall. (For a description of the esophagoscope see Gastroscopy, p. 446.)

Anatomic Consideration of the Esophagus with Reference to Esophagoscopy.—No attempt will be made here to give a detailed anatomic description of the esophagus, but only those points concerned in an esophageal study will be alluded to. The measurements of the esophagus have been found by different observers to vary widely, and this is due in part to the fact that the measurements have been made from different landmarks, and that the esophagus is seldom found to be exactly in the same position at different examinations, even when made upon the same patient. "The only really fixed point is at its junction with the posterior pharyngeal wall" (Jackson). This peculiarity of the organ is dependent upon its degree of motility and its capability to contract and expand, together with the effect produced by the action of deglutition and the reverse action of regurgitation. The esophagus will also be found to vary greatly both in caliber and in length at different ages. For practical purposes, the measurements of the esophagus

are always made with the upper central incisor teeth as a starting-point. The accompanying table, compiled by Jackson, is based upon the observations of Stark:

LENGTH OF THE ESOPHAGUS AT DIFFERENT AGES.

Age.	Teeth to Cricoid.	To Bifurcation.	To Cardia.	Length of Whole Esophagus.
Birth	7 cm. (2¾ in.)	12 cm. (4¾ in.)	18 cm. (6¾ in.)	10 cm. (4 in.)
1 Year	10 cm. (4 in.)	14 cm. (5½ in.)	22 cm. (8¾ in.)	12 cm. (4¾ in.)
2 Years	10 cm. (4 in.)	15 cm. (6 in.)	23 cm. (9 in.)	13 cm. (5⅛ in.)
5 Years	10 cm. (4 in.)	17 cm. (6¾ in.)	26 cm. (10¼ in.)	16 cm. (6⅜ in.)
10 Years	10 cm. (4 in.)	18 cm. (7 in.)	28 cm. (11 in.)	18 cm. (7 in.)
15 Years	14 cm. (5½ in.)	23 cm. (9 in.)	33 cm. (13 in.)	19 cm. (7½ in.)
Adult	15 cm. (6 in.)	26 cm. (10¼ in.)	40 cm. (15¾ in.)	25 cm. (10 in.)

The caliber of the esophageal lumen is also subject to relatively greater variations than is the length of the organ; the diameter of this canal is found to vary greatly at different distances from the upper teeth. There are four distinct points at which the diameter of the esophagus should be considered, and that are of importance in attempting the introduction of the esophagoscope. These are best learned from the appended table, originally compiled from Stark's measurements:

DIAMETERS OF THE ESOPHAGUS AT THE FOUR CONSTRICTIONS.

Constriction.	Diameter.	Vertebra.
Cricoid:	Transverse, 23 mm. (1 in.) Anteroposterior, 17 mm. (¾ in.)	Sixth cervical
Aortic:	Transverse, 24 mm. (1 in.) Anteroposterior, 19 mm. (¾ in.)	Fourth thoracic
Left bronchus:	Transverse, 23 mm. (1 in.) Anteroposterior, 17 mm. (¾ in.)	Fifth thoracic
Diaphragm:	Transverse, 23 mm. (1 in.+) Anteroposterior, 23 mm. (1 in.)	Tenth thoracic

As has been stated in describing the introduction of the gastroscope (p. 448), the upper esophageal constriction, located at the introitus, concerns us chiefly in esophagoscopy. (See accompanying table.) Second in importance is the normal constriction present at the hiatus œsophageus (diaphragmatic constriction; see Fig. 157). It is apparent that the portion of the esophagus passing through the diaphragm may be greatly altered as the result of contraction or relaxation of the muscular fibers immediately surrounding the esophagus. Consequently, complete relaxation, rigidity, and spasm of the diaphragm are important factors in considering this method of clinical observation. (See also Gastroscopy, p. 448.) Generally speaking, in children, a tube having a diameter of 7 mm., and in adults one of 10 mm. diameter, will be found to pass directly through the esophagus.

Position.—Beginning at a level of the bifurcation of the trachea, the esophagus curves around the aorta and descends somewhat to the left, passing through the esophageal opening of the diaphragm close to the vertebræ.

(See Fig. 157.) The subphrenic portion of the esophagus also deviates to the left, and has a somewhat wide range of mobility, and it is the mobility of this portion of the tube that makes it possible for us to introduce the straight gastroscope.

Normal Appearances.—The esophageal picture changes materially in form in various portions of the tract. The introitus œsophagei is closed by the constriction produced mainly by a contraction of the pharynx, producing a backward pressure of the cricoid cartilage, which at all times, except during the act of swallowing, lies in direct contact with the posterior pharyngeal wall.

The cervical portion of the esophagus has the appearance of a transverse slit, due to the collapse of the walls from before backward, and opens ahead of the tube, showing a more or less flat anterior and posterior wall, meeting at the sides. This often opens and closes with the respiratory movements. Upon entering the thoracic esophagus the esophagoscope

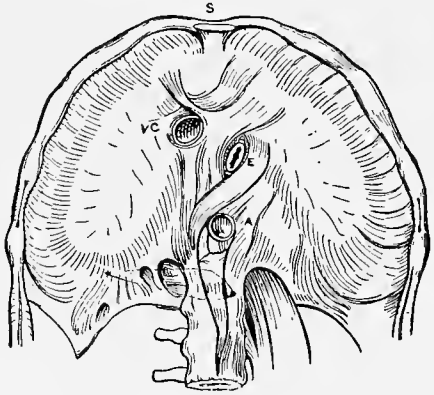


FIG. 157.—UNDER SURFACE OF THE DIAPHRAGM (Jackson).

E, Hiatus œsophageus; note the direction of its axis; *A*, aortic opening; *VC*, opening for vena cava; note direction of tendons and muscular fibers.

reveals a more or less oval or quadrangular opening, into the depths of which the observer looks. This opening is considerably smaller than the entire esophageal lumen, and increases in diameter during inspiration. If the instrument is moved slightly from side to side, the apparent size of the esophagus is also changed.

At the diaphragm the lumen again assumes the form of a slit, the axis being placed obliquely from the right posteriorly to the left anteriorly. (See Fig. 157). The subphrenic portion of the esophagus is less affected by respiratory movements, but may be closed by movements of the diaphragm and of the abdominal viscera.

When folds appear in the esophageal wall, they are probably due to faulty technic in manipulating the instrument.

The color of the normal esophageal mucosa varies greatly in different individuals, in the same person at different times, and still more does the apparent color vary with the form of illumination employed. For these reasons a detailed description of what may be seen will not be given here.

Technic of Esophagoscopy.—The examination of the upper end of the esophagus is not attended with any difficulty, and, technically, it is the same as direct laryngoscopy. The patient is given no food for at least eight hours, and is then directed to brush his teeth with soap and chalk, and to rinse his

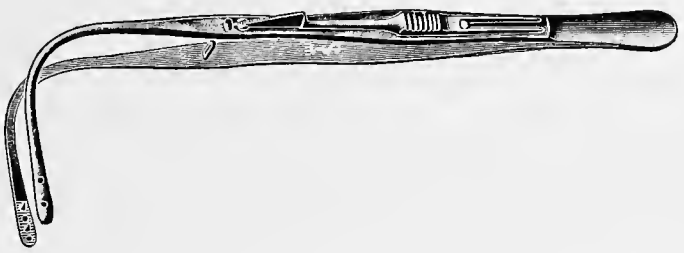


Fig. 158.—Sajous' Cotton-holding Forceps for Preliminary Cocainization of the Larynx and Pharynx (Jackson).

mouth every two hours with 30 per cent. alcohol. He should wash his face thoroughly with soap and water, paying particular attention to beard or mustache if these are present; he should rinse them first with water and then with 1 : 1000 mercury bichlorid solution.

Anesthesia.—Local anesthesia is sufficient for the performance of esophagoscopy, especially when only the upper portion of the canal is to be examined. This is effected by a 4 per cent. solution of cocain, applied with a swab of cotton held in a Sajous forceps (Fig. 158). After a few moments the tubular speculum is carried in until the epiglottis appears, when an application of the cocain is made to all visible adjacent structures. The instrument is passed posteriorly to the epiglottis, and brings into view the interior of the larynx and the introitus œsophagei, which are to be touched with cocain. In children the application of a cocain solution is to be cautiously made, and even adults may display an idiosyncrasy to the action of the drug.

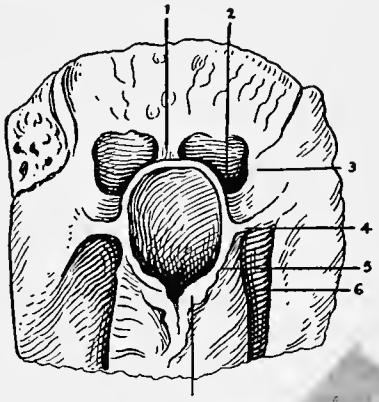


Fig. 159.—Base of the Tongue and Upper Border of Normal Larynx, Viewed from Behind (Jackson).

1, Median glosso-epiglottic fold; 2, right glosso-epiglottic fossa; 3, lateral glosso-epiglottic fold; 4, pharyngo-epiglottic fold; 5, aryepiglottic fold; 6, right pyriform sinus, by way of which esophagoscope should be passed.

As the instrument passes back of the epiglottis the arytenoids are brought into view, and are seen to lie in contact with the posterior pharyngeal wall. A slit is observed, and the end of the instrument is inserted far enough into this slit to reach below the arytenoids and engage posteriorly to the cricoid cartilage, when the pyriform fossæ will be visible (Fig. 159), with the pushing forward of the cricoid cartilage by the instrument the upper portion of the esophagus comes into view.

Passing of the Esophagoscope.—The passing of the esophagoscope differs in no way from the passing of the gastroscope. (See p. 448.)

Counterindications.—These are practically the same as those to be considered in the introduction of the gastroscope (p. 453), but in this connection especial attention must be called to the existence of acute esophagitis.

Diseases of the Esophagus.—Stricture of the esophagus may be recognized by other methods than esophagoscopy, yet an actual inspection at the point of stricture is often of value, and is a direct guide as to the character of the disease in question. Again, the size of the lumen of the esophagus at the point of stricture is accurately determined by the esophagoscope, a feature of great clinical importance in those cases in which the esophageal bougie cannot be satisfactorily introduced. Ulcers and new-growths of the esophagus may also be detected by this clinical method.

Dilatation of the esophagus is recognized with a greater degree of certainty by the aid of the esophagoscope than by other methods, since in this condition the esophageal wall disappears from view during the act of respiration. Consequently, a rhythmic disappearance and reappearance of the esophageal wall is pathognomonic. Again, in diverticula there is commonly found either a benign or a malignant growth at some portion of the esophageal wall.

Foreign bodies lodged in the esophagus may be accurately located and even removed by the aid of the esophagoscope.

X-RAY EVIDENCE IN DISEASES OF THE ESOPHAGUS.

By George E. Pfahler, M.D.

The tissues forming the esophagus do not differ much in density from the surrounding tissues; therefore, of itself it cannot be demonstrated by the ray. By means of a bismuth mixture, however (bismuth subcarbonate, one part, to two of milk, broths, or gruels), one can obtain an outline of the lumen of the esophagus, and thus determine its course, its movements, and the presence of any constrictions. One obtains the most information concerning the esophagus by fluoroscopic examination.

One must keep in mind that there are three physiologic constrictions: the first is at the cricoid cartilage; the second is on a level with the fourth thoracic vertebra at the arch of the aorta; and the third is below the diaphragm where the esophagus enters the stomach. These physiologic constrictions can be demonstrated by allowing the patient to swallow capsules or pills of bismuth, which will be found to lodge for a longer or shorter period of time at these points.

Displacements of the esophagus will occur in those affections which are accompanied by displacements in the other mediastinal organs—pleural adhesions, pleural effusions, pneumothorax, tumors of the lung or vertebræ, aneurisms, etc. This additional evidence may help to make the diagnosis in doubtful cases.

Stenosis of the esophagus may be due to compressions from without (aneurisms, mediastinal tumors, adhesions) or to constriction from within (cicatricial, carcinomatous, spasm). The former have been discussed under Mediastinal Diseases.

In the study of a *stenosis* one can follow a metallic sound down to the constriction, or, much better, one can follow bismuth mixtures which are swallowed naturally by the patient. I first follow a liquid mixture (milk and bismuth), then a thicker mixture (kefir and bismuth), then the kind of

food that the patient says he cannot swallow, and give the bismuth liquid on top until the location of the stricture has been determined. After this has passed or been regurgitated, one can give various sizes of pills or capsules, until the size of the constricted lumen has been determined.

In *carcinoma* of the esophagus one can usually determine the location, outline, and extent of the constriction. The lumen is irregular, and except in the terminal stage the constriction is not complete. There will always occur trickling of the liquid bismuth mixture through the orifice. The ultimate passage of the food will depend upon the degree of stenosis. Additional information may often be obtained by studying the influence of the ingested mixture upon the neighboring structures.

Spasm of the esophagus, on the other hand, is more apt to be a complete constriction, which after a time relaxes entirely, allowing the food to pass without obstruction. Occasionally one can see the constriction move upward carrying the bismuth mixture ahead of it.

Dilatation of the esophagus and its degree can be determined by filling the lumen above the constriction with bismuth mixture. The amount of dilatation will depend upon the degree of constriction, and the duration.

Diverticulum of the esophagus can usually be recognized, but this will depend upon the ability to fill its cavity with the bismuth mixture. One may get it to fill at one time and not at another. It may vary in size, shape, and location. Its walls will be smooth. The most important evidence, however, is obtained by watching it empty itself. A stenosis empties from the bottom; a diverticulum from the top.

The *movements* of the esophagus are interesting. The peristalsis can be seen to carry solid food down to the cardiac orifice. Liquids can usually be seen entering the cardiac orifice by spurts synchronous with the cardiac pulsations. In a stenosis of the cardiac orifice with dilatation above, I was able to see several bismuth capsules churned about one another in the attempt to force them through the constriction.

DISEASES OF THE ESOPHAGUS.

ESOPHAGITIS.

Pathologic Definition.—An inflammatory condition, either acute or chronic, involving the mucous or submucous or both coats of the esophagus.

General Remarks.—The esophagus, owing to the protection afforded by its particular location, function, and histologic structure, is less prone to be attacked by the diseases known to affect other mucous surfaces.

When, however, disease of the esophagus occurs and is localized to the mucous or submucous coats, the symptoms will in general be those of similar affections of other mucous surfaces, with this difference, that sooner or later dysphagia is likely to occur. A study of diseases of the esophagus consequently resolves itself into a differentiation of all other conditions in which there is difficulty in swallowing or interference in any way with the passage of food to the stomach.

For present purposes the symptomatology of affections of the esophagus in general will be considered here, and we will discuss later, under separate headings, the special symptoms belonging to each particular disease.

Principal Complaint.—Pain is by far the most distressing and com-

mon symptom of disease of the esophagus. In acute inflammation the pain is severe, and is distributed over the region of the neck, beneath the upper portion of the sternum, between the shoulders, and for some four to six inches along the vertebral column; a similar pain is experienced in periesophageal inflammation. If the esophageal inflammation is acute, the patient complains of a variable degree of stiffness of the neck.

Sharp, lancinating pain, burning in character, results from an acute inflammation of the mucous surface of the esophagus, and in many instances is caused by the passage of hot or highly acid foods to the stomach. Dysphagia, while most annoying to the patient, is not always accompanied by pain. Pressure upon the esophagus may in rare instances excite continuous pain or pain upon swallowing, but it is usually localized and not of an acute character, unless solid food is passing.

Progressive weakness and emaciation are among the most constant symptoms of disease of the esophagus, and are more pronounced when either obstruction or malignant disease is present.

Cough accompanies practically all pathologic conditions of the esophagus or of the adjacent structures, from pressure exerted upon the recurrent laryngeal nerve, the bronchi, or the trachea. The conditions that commonly give rise to such irritation and cough are thoracic aneurism, carcinoma of the esophagus, and enlarged mediastinal glands.

There is more or less constant annoyance from the accumulation of secretion in the mouth, and the patient expectorates a frothy, viscid mucus. Mucus from the esophagus is not expelled by coughing, but is readily loosened by merely clearing the throat. Generally speaking, the patient complains of profuse expectoration when there is either acute or chronic inflammation of the esophageal mucous membrane.

ESOPHAGEAL HEMORRHAGE.

Definition.—The escape of either arterial or venous blood from the esophageal mucous membrane, which may later be ejected through the mouth or find its way into the stomach.

Exciting and Contributing Factors.—The commonest cause of hemorrhage is a varicosity of the veins of the mucous membrane of the esophagus. In this connection esophageal hemorrhage is not readily explained, since it accompanies cirrhosis of the liver, chronic nephritis, and certain diseases in which splenic tumor is present. Minute hemorrhages from the esophagus are highly suggestive of carcinoma, and especially is this the case when a variable degree of obstruction is present. Foreign bodies, either lodged in or while passing through the esophagus, may be the cause of bleeding, and ulceration of this canal may also give rise to hemorrhage.

Characteristics.—Blood from the esophagus is bright red in color, and always alkaline in reaction; it is not ejected by vomiting, but is brought up by "clearing of the throat."

Physical Examination of the Esophagus.—**Inspection** of the esophagus is possible only with the aid of the endoscope, and may, in some instances, show evidence of ulceration or of varicose veins.

Palpation of the esophagus immediately above the clavicles may be performed from the sides of the neck. From this point the esophagus, when distended or enlarged, will be felt immediately behind the trachea. In periesophageal abscess or dilatation of the esophagus a peculiar soft, tumor-like mass can be felt in this region, and in marked dilatation a pear-shaped tumor may be seen in the neck.

The passing of an esophageal sound furnishes the most valuable evidence as to the condition of the esophagus.

Cautions.—(1) The bougie should be protected at its point by an olive-shaped expansion.

(2) The instrument should be introduced gently, and should pass through the tube without the operator making any decided pressure.

(3) If the question arises as to whether the bougie has entered the esophagus, the patient should be directed to speak, and if he is able to do so, it is evident that the sound has not entered the larynx.

(4) The normal constriction of the esophagus corresponds to the level of the fourth thoracic vertebra, and is *ten inches* from the incisor teeth; therefore all bougies should have an indelible mark at this point in order to guide the operator. It is also well to have the bougie graduated in inches from the olive-shaped expansion to the tip. A mild contraction is noticed when the bougie passes the cricoid cartilage.

(5) When the bougie has passed a constriction in the esophagus, it should be removed slowly and with extreme gentleness until it has again passed the point of constriction.

(6) The entrance of the esophagus into the stomach corresponds to the level of the eleventh thoracic vertebra.

General Remarks.—The esophagus may be obstructed at almost any point, either as the result of disease within the canal, from pressure by tumors in the mediastinum or by foreign bodies lodged in the tube. Organic disease usually affects the upper half of the esophagus. In dysphagia due to paralysis of the esophagus the bougie passes into the stomach without interruption. In dysphagia due to spasm the tube will be found to pass *slowly*, and without any decided pressure, into the stomach. In stricture of the esophagus following ulceration the bougie will not pass easily.

The location of an obstruction possesses a certain value in the diagnosis; *e. g.*, an obstruction of the esophagus located five or six inches from the incisor teeth is commonly of cicatricial origin, whereas an obstruction nine inches from the teeth is highly suggestive of carcinoma. If the passing of the bougie excites pain at any particular point along the esophagus, this is doubtless the seat of disease, and the nature of the pathologic condition is suggested by the point at which the irritation exists.

Auscultation.—Hanburger* was the first to call attention to auscultation as a means of diagnosis in constriction of this canal, and this observer has reported in detail sounds heard over both the normal and the diseased esophagus.

Method.—Place the stethoscope over the pharynx, at the side of the neck, over the hyoid bone, or at the left of the spinous process of the vertebra, as low as the first thoracic vertebra. A loud, gurgling sound is heard when the patient swallows liquid. This sound, however, is of short duration. On placing the stethoscope over the left edge of the sternum, or just to the left of the vertebral column, liquids may be heard to pass from the mouth into the stomach. If there is a constriction or decided lessening of the caliber of the esophagus at any part of the canal, a peculiar gurgling sound, resembling that heard when water is poured from a bottle, is audible at a level with such constriction. If there is complete obstruction of the esophagus, the characteristic sound of swallowing is not heard below the point of such obstruction. In dilatation of the esophagus the sound

* Jahrbücher der k. k. Gesellschaft der Aerzte in Wien, Bd. xviii.

produced by swallowing liquids resembles that of rain beating against a glass window-pane. The sound normally audible when the stethoscope is placed along the spine over the course of the esophagus during the act of swallowing liquids is best appreciated by listening to this sound.

ACUTE ESOPHAGITIS.

Predisposing and Exciting Factors.—These have been alluded to under General Remarks. The condition may, however, result from extension of a catarrhal process from the pharynx; it is not infrequently associated with certain acute infections, *e. g.*, typhoid fever, and is particularly common in diphtheria, scarlet fever, and pneumonia. Practically all diseases arising within the esophagus are capable of causing an acute or a chronic inflammation of the lining mucous membrane. In a few instances the formation of small pustules has been noted, disseminated over the mucosa of the esophagus.

Principal Complaint.—The patient complains of more or less constant pain, more particularly upon swallowing either solid or liquid foods. Following deglutition there is at times a dull pain or a sense of weight immediately beneath the sternum. Regurgitation of food is by no means uncommon, and dysphagia is also likely to occur. As a rule, a large quantity of mucus is mixed with the regurgitated food, but pus and blood are rather uncommon findings.

Summary of Diagnosis.—Pain localized immediately beneath the sternum, and intensified by the passage of food to the stomach, is highly suggestive of esophagitis. The ejection of blood and mucus also points strongly toward inflammation of the esophagus.

Course and Duration.—As a rule, cases of acute esophagitis subside in from a few days to three weeks, but when the condition complicates a pharyngeal or gastro-intestinal malady, the prognosis may be less favorable. In necrotic and in purulent types of the disease the constitutional symptoms are pronounced, and death may result.

CHRONIC ESOPHAGITIS.

Pathologic Definition.—A chronic inflammatory process involving the mucous coat of the esophagus, and following repeated attacks of acute esophagitis.

This condition, as stated, may result from repeated attacks of acute esophagitis, or it may depend, in part at least, upon dilatation of the veins of the esophagus, the result of valvular heart disease, myocarditis, chronic interstitial nephritis, or hepatic cirrhosis. The symptomatology of the chronic form differs from that described under Acute Inflammation of the Esophagus only in degree.

Esophagoscopy.—The esophagoscopic findings show paling and mottling of the mucous surface of the organ. There is also present a somewhat thick, tenacious mucus, that covers the entire mucous surface.

ULCER OF THE ESOPHAGUS.

Pathologic Definition.—An acute or chronic inflammatory process, with ulceration, involving first the mucous coat of the esophagus, and possibly extending to the submucous and muscular coats.

Predisposing and Exciting Factors.—Ulcer of the esophagus may follow simple catarrh of the esophageal mucosa, diphtheritic catarrh of the esophagus, and pressure from mediastinal tumors; in bed-ridden patients pressure exerted opposite the level of the cricoid cartilage may give rise to ulcer at this point. Esophageal ulcer may develop during the course of the acute infections, *e. g.*, pneumonia, acute gastritis, acute ulcerative stomatitis, and typhoid fever. There may be ulceration of the esophagus, simulating closely that detected in the stomach, but such ulceration is, as a rule, situated near the cardiac extremity of the tube.

Esophagoscopy.—The ulcer may be inspected by means of the esophagoscope, and in this way the exact character of the lesion is determined. The esophagoscope should not be employed when ulcer follows the taking of irritating and corrosive substances. Local treatment may also be applied through the esophagoscope. (See p. 404.)

Summary of Diagnosis.—Ulcer of the esophagus is difficult to diagnose antemortem, since practically all the symptoms of acute catarrh of the esophagus are also present in this condition. In addition, ulcer is characterized by intense pain on swallowing, the pain appearing to be localized immediately beneath the upper border of the sternum and along the spine on a level with the lower border of the scapula. It is seldom that esophageal ulcer perforates the tube, but if perforation should occur there is emphysema of the surrounding tissues, and particularly of the region of the neck. Emphysema is an almost positive sign of perforation of the esophagus.

Complications and Sequelæ.—Perforating ulcer usually leads to abscess of the mediastinum and of the tissues of the neck. In rupture of the esophagus complicating carcinoma, ulcer, or traumatism, the symptoms are pain, violent vomiting, a weak, rapid pulse—in fact, the general symptoms of shock. Doubtless the vast majority of ulcers of the esophagus heal spontaneously, and, as a result of this process, a dense layer of cicatricial tissue is formed that, in time, produces a local diminution of the caliber of the esophagus.

ESOPHAGEAL DIVERTICULUM (PHARYNGOCELE).

Definition.—A circumscribed sacculation involving one or more coats of the esophageal wall.

Varieties.—(*a*) Pressure diverticula, which may be either congenital or acquired; and (*b*) the so-called traction diverticulum.

Predisposing and Exciting Factors.—Excluding those cases of diverticulum of the esophagus that are congenital, the predisposing and exciting factors in pressure diverticula are "localized lesions in the muscular coat of the esophagus," which, in the majority of cases, are produced by rapid eating or by the swallowing of large particles of food—the lodging of foreign bodies in the esophagus, and the like.

Sex figures prominently, since males are far more susceptible than females.

Age also deserves consideration. The condition is seldom seen in children unless it is of congenital origin.

Location.—The sac occurs most often on the posterior wall, at or near the junction of the esophagus and the pharynx, where the muscular coat of the canal is weakest. When the muscular coat becomes expanded or thin, the mucous membrane tends to protrude forward between the muscular fibers, forming an apparent hernial sac. A characteristic of esophageal diverticulum is the fact that its size increases gradually.

Traction diverticula occur only in children, and are situated at a level with the bifurcation of the trachea; they protrude from the anterior wall of the esophagus.

Principal Complaint.—The patient is at first conscious of the fact that a portion of his food lodges too high, and he describes this peculiar sensation as being relieved when he stretches or makes general pressure upon the neck. He may also observe a small mass or bulging in the neck, which is often relieved by gentle manipulation. Tumor of the neck is a not infrequent symptom, and whenever there is a history of fluctuating tumor in this locality, the possibility of pharyngo-esophageal diverticulum should be foremost in the mind of the diagnostician. In certain cases the patient is able, by pressing upon the neck, to cause a portion of the previously taken food to regurgitate into the mouth—a positive sign of dilatation of the esophagus. When the sac becomes filled with food, pressure exerted externally may excite vomiting, which is accompanied by severe strangling. In those cases in which vomiting is a frequent symptom irritation and soreness of the pharynx and the esophagus are likely to be associated. Soreness may be so marked as to prevent the patient from taking sufficient nourishment, and consequently the symptoms of malnutrition develop. The distended sac may exert sufficient pressure on the nerves of the neck to cause dysphagia and alteration in the voice. Pressure upon either the superior or the recurrent laryngeal nerves causes paroxysmal coughing, dyspnea, and hoarseness.

Esophagoscopy.—An esophagoscopic examination enables one to obtain a clear conception of the degree of dilatation existing in a given case. The esophagoscopic findings in diverticulum are quite characteristic, since there is a rhythmic disappearance from view of the esophageal wall with the act of respiration. (See Esophagoscopy, p. 404.) Again, either benign or malignant tumor is frequently found to involve some portion of the esophageal wall. Stricture may exist at the lower portion of the sacculation, and in such cases it is possible to estimate the actual caliber of the esophagus at the point of constriction.

Summary of Diagnosis.—A tumor-like mass that appears suddenly in the neck and disappears upon pressure and gentle manipulation or after paroxysmal coughing is highly suggestive of diverticulum. If the esophageal sound may be passed directly into the sac, the instrument is palpable through the neck, and the sensation offered to the operating hand is entirely different from that experienced when the sound passes into the stomach.

Duration.—Most cases are of prolonged duration, and recovery is practically impossible, unless operative interference is employed. In those patients in whom the nutrition remains good the prognosis as to life is favorable.

STRICTURE OF THE ESOPHAGUS.

Pathologic Definition.—A condition by which the caliber of the esophagus is diminished at any given point throughout its length; this narrowing may be congenital, the result of disease within the esophagus (ulceration) or of extra-esophageal pressure.

Predisposing and Exciting Factors.—It is to be borne in mind that, normally, there is a moderate constriction of the esophagus ten inches from the teeth, and that any decided alteration in the caliber of this tube elsewhere is pathologic. Aside from congenital deformity of the esophagus, stricture oftenest results from epithelioma, and is second in importance only to congenital deformities as a cause of stricture of the esophagus. Less

often stricture of the esophagus may depend upon an excess of cicatricial tissue produced in the process of healing of an esophageal ulcer. Corrosive substances taken into the esophagus may give rise to extensive ulceration and sloughing of the mucosa.

A few instances are recorded in which stricture of the esophagus followed typhoid fever, and it is fair to suppose, at least, that typhoid ulceration took place in the esophageal mucous membrane. Certain authors hold that syphilitic ulceration is occasionally responsible for esophageal stricture. True gastric ulcer situated at the junction of the esophagus with the stomach has been reported.

Principal Complaint.—This will be found to vary greatly with the degree of stricture present. Probably the first complaint is of inability to swallow large morsels of food, or such food may cause the patient some discomfort in its passage into the stomach. In practically every case the sufferer notices that this difficulty in the swallowing of solid food increases gradually until finally he is able to take only liquids. The pain, discomfort, or pressure, as the patient is apt to describe it, felt when swallowing solid food is localized to one particular point, immediately beneath the sternum or between the scapulæ. It will be observed that as the lumen of the esophagus becomes narrower, increased effort will be required to make the food pass through the tube to the stomach.

Spasm of the esophagus may be responsible for temporary esophageal stricture, and has been considered under the head of Neuroses of the Esophagus. The degree of pain accompanying stricture of the esophagus will be found to vary greatly with the cause of the stricture.

After the lumen of the esophagus has become markedly lessened the patient usually expectorates a large quantity of mucus and regurgitates particles of food that have remained in the esophagus for hours. Food and secretion ejected from the esophagus are alkaline in reaction, and other chemic evidences are also present to show that they do not come from the stomach. Rupture of the esophagus is best considered in connection with dilatation of this tube. (See Esophageal Dilatation.)

The patient complains chiefly of progressive weakness and loss of flesh, which in advanced cases is extreme. In persons suffering from stricture of moderate degree malnutrition does not occur.

Esophagoscopy.—By means of this method it is possible to determine not only the location of the stricture, but also the actual caliber of the esophagus at the point of constriction. The condition of the esophageal wall immediately surrounding the stricture is also of importance in considering the treatment, since sclerotic changes might yield to the bougie, whereas in the presence of malignancy such procedure would be contraindicated. The degree of dilatation of the esophagus above the point of constriction is also of interest. (See Esophageal Diverticula.)

Diagnosis and Laboratory Diagnosis.—Regurgitation of food is the most positive sign of stricture of the esophagus, and in cases in which the constriction is located in the upper portion of the esophagus it occurs almost immediately after eating. If the constriction is situated near the stomach, food is regurgitated in from one to four hours after it is taken. The more marked the degree of dilatation above the stricture, the later does the regurgitation of food occur. Solids and liquids regurgitated from the esophagus are alkaline in reaction, and show that they have not been acted upon by the gastric juice, but the changes effected by the mixture of saliva are in evidence. There is an absence of both free and combined hydrochloric acid.

Auscultation (see p. 411) is of value in diagnosing the stricture. The most conclusive evidence, however, of the existence of stricture is obtained by the introduction of the esophageal bougie.

Differential Diagnosis.—Stricture of the esophagus, although apparently easily diagnosed, must be differentiated from spasm of the esophagus and obstruction the result of external pressure—*e. g.*, from large bronchial glands, thoracic aneurisms, mediastinal abscess, enlarged thyroid, and large pericardial effusion, in all of which conditions the bougie passes quite easily into the stomach. In external pressure from glands, or an enlarged thyroid gland, chronic pleurisy with extensive adhesions, caries of the vertebræ with spinal distortion, the bougie may be passed into the stomach, but its passage excites severe pain. If constriction of the gullet is due to neuroses, the bougie's progress is suddenly arrested, but soon passes the apparent stricture without extra force. In neuroses the bougie in its transit meets with obstructions at different portions of the tube. The *x*-ray may be of inestimable value in distinguishing between stricture and obstruction due to pressure from thoracic tumors and from bone disease.

Caution.—An esophageal bougie should never be introduced when disease of the heart or of the blood-vessels (aneurism) is present, since such procedure is likely to be accompanied by disastrous results.

Syphilis and traumatism to the esophagus favor the formation of stricture, and old age and a history of carcinoma likewise point to a similar condition in the wall of the tube. Gastric ulcer, typhoid fever, and dysentery may influence slightly a diagnosis of stricture.

DILATATION OF THE ESOPHAGUS.

Definition.—A condition in which any portion of the entire esophageal canal becomes expanded.

General Consideration.—A variable amount of dilatation practically always follows stricture of the esophagus, and these expansions vary in direct proportion to the degree of stenosis present. (See Stricture.)

General dilatation of the esophagus without stricture is certainly a rare condition, and is distinguished from the localized form by the fact that the esophageal bougie passes directly into the stomach.

The symptoms of dilatation of the esophagus are practically those of stenosis of the tube, except that pain is less common.

SPASM OF THE ESOPHAGUS (ESOPHAGISMUS).

Predisposing and Exciting Factors.—Probably the best examples of spasm of the esophagus are seen in hysteria, epilepsy, and hydrophobia. In practically all cases the patient is of a neurotic temperament. Less often spasm may be reflex in nature, and secondary to disease of the stomach—*e. g.*, gastric atrophy and dilatation. Spasm occurs occasionally in aged males, and only in those of nervous temperament.

Principal Complaint.—Difficulty in swallowing is the only inconvenience from which the patient suffers. As a rule, he is able to take solid foods except during the spasm, when they cannot pass through the esophagus. Pain is much less common and more mild than in true stricture of the esophagus.

The patient first complains that he is choking, and this uncomfortable feeling continues until the food has passed the point at which spasm occurs. In many persons of hysteric temperament a variable degree of choking is

experienced irrespective of the ingestion of food, and such spasm may be induced by excitement.

Diagnosis and Differential Diagnosis.—The facts that hysteric temperament exists and that the attacks are intermittent are positive evidences of the nature of the condition. The freedom from pain and the characteristic vomiting also favor the existence of spasm.

Spasm of the esophagus is differentiated from stricture by the fact that the bougie is usually grasped tightly within the esophagus at a given point, but if permitted to remain for a few seconds, it will pass the constriction without any special effort upon the part of the operator.

Esophagoscopy.—An esophagoscopic examination shows absence of disease of the organ. In one case studied at the Philadelphia Hospital it was possible, by gliding the bougie through to the esophagus, to excite spasm at different portions of the canal—a positive sign of neurosis.

CARCINOMA OF THE ESOPHAGUS.

Pathologic Definition.—A malignant growth usually developing primarily from the esophageal mucosa, and characterized later by the formation of stricture.

Predisposing and Exciting Factors.—Age is a most prominent factor in carcinoma of the esophagus, the condition being extremely uncommon before the fortieth year. Males are more often afflicted than females. The prolonged use of alcohol is believed to exert some influence. An esophageal ulcer forms a favorable site for the development of epithelioma.

Principal Complaint.—The patient complains of dysphagia, which increases from time to time until it is practically impossible for him to take solid food. Simultaneously with the increasing dysphagia the patient complains of progressive emaciation and of weakness, with or without pain. He regurgitates portions of the food eaten, and such ejecta are often blood-stained, containing a large amount of mucus, and in rare instances shreds of diseased mucous membrane. The food is regurgitated either immediately after attempting to swallow or from ten to fifteen minutes later, the length of the interval varying with the location of the carcinoma. In atypical cases the dysphagia may be slight, or the symptoms may subside as the result of disintegration of the carcinomatous growth. Not uncommonly the patient displays other manifestations of carcinoma, *e. g.*, involvement of the cervical glands.

Esophagoscopy.—It is possible by this method to recognize malignancy of the esophageal wall at an unusually early date, and before there are extensive changes in that portion of the esophagus above the lesion. In carcinoma of the esophagus of long standing the employment of the esophagoscope is attended with a certain degree of danger, since in these cases extensive ulceration is likely to be present. (See Gastroscopy; also *X*-Ray Diagnosis, p. 408.)

Laboratory Diagnosis.—The ejected material is acid in reaction until stenosis develops, when it becomes alkaline. Lactic acid may be present, but hydrochloric acid is absent. Mucus is always present. Red and white blood-cells, epithelial cells, yeast-cells, fat-globules, starch-granules, particles of food, and shreds of necrotic tissue are among the microscopic findings.

Summary of Diagnosis.—Mediastinal tumors may give rise to symptoms that closely simulate those caused by carcinoma of the esophagus, but the degree of emaciation and the anemia are less marked than in

carcinoma. The introduction of a stomach-tube may facilitate the making of a diagnosis, since shreds of mucus and particles of carcinomatous tissue may be dislodged by the tube and recovered in the fluid thus obtained. There is always great danger of perforation when either the stomach-tube or the bougie is introduced into the esophagus.

Course.—The patient's general condition grows progressively worse, and complications, *e. g.*, bronchopneumonia, pulmonary gangrene, esophageal perforation, and hemorrhage, are likely to occur. The disease is fatal, the majority of cases terminating in from three to twelve months after the first manifestation of symptoms.

THE STOMACH AND INTESTINES.

EXAMINATION OF THE ABDOMEN.

TOPOGRAPHY.

Ballance divides the abdomen arbitrarily into regions. He bounds the entire abdomen with a circle, the center of which is the umbilicus. (See Fig. 160.) We have found that this division of the abdomen requires slight

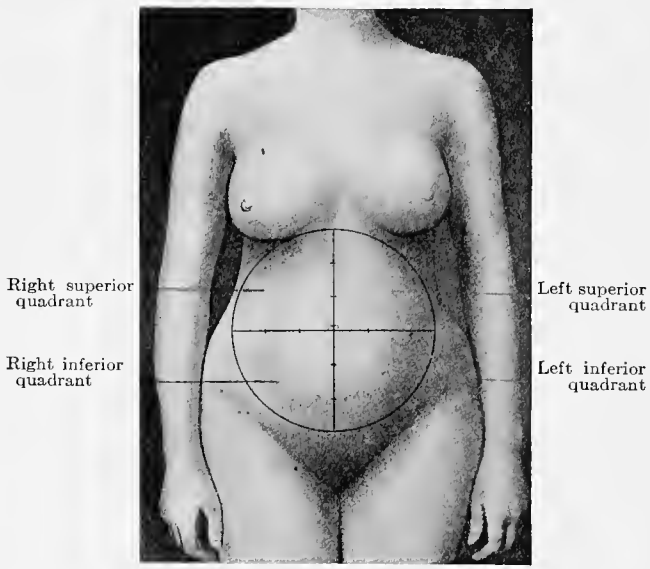


FIG. 160.—ARBITRARY REGIONAL DIVISION OF THE ABDOMEN.

modification, necessitated by reason of the difference in stature of the individuals examined. Thus in conducting our examinations we make the vertical diameter of the circle extend from the tip of the ensiform cartilage to the pubic articulation (Fig. 160), and the horizontal diameter extend to

the margins of the abdominal wall, at a point approximately midway between the ensiform cartilage and the top of the pubic arch.

This arbitrary outline, in the average individual, will be found nearly to transcribe the circle, whereas in a tall person the vertical diameter will exceed that of the transverse, and, again, in the obese, the transverse diameter will be found to far exceed that of the vertical. In those displaying an abnormal amount of abdominal fat the umbilicus may be found some distance below its normal location, and in such cases our transverse arbitrary division must be taken at a point near the center of the vertical line extending from the ensiform cartilage to the pubic articulation.

This circular outline of the abdomen is divided by a transverse and a vertical line into four regions (Fig. 160): Those above the level of the umbilicus we speak of as the superior right and superior left quadrant respec-

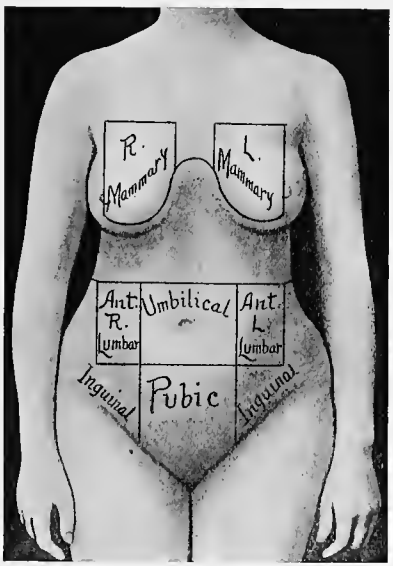


Fig. 161.—Arbitrary Division of Abdomen and Mammary Region.

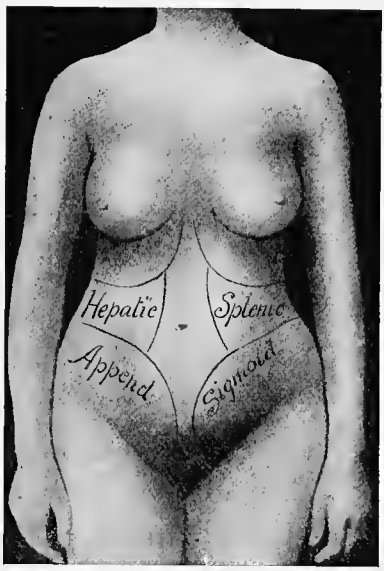


Fig. 162.—Special Arbitrary Division of the Abdomen.

tively; and of those below the umbilical level as the inferior right and inferior left quadrant respectively. It is often necessary to refer to a viscus or tumor as occupying one of these regions, and to qualify this statement by referring to other anatomic landmarks of the abdomen.

The advantage of this arbitrary division is further exemplified by supposing that a tumor exists in the left inferior quadrant, and that the center of this tumor is three inches from the junction of the transverse with the vertical line at the umbilicus, and one inch below the line drawn between the umbilicus and the left anterior superior spine (Fig. 164). The size may now be readily determined by taking measurements from the center of the tumor.

Viscera that are bisected by either the transverse or the vertical lines are referred to in a description of them as being situated in either the median or the vertical line, as, *e. g.*, the bladder. Viscera and tumors are also spoken of as occupying a position a definite number of inches above or below the transverse line or to the right or left of the median line, and a certain num-

ber of inches from the pubis, the ensiform cartilage, or the umbilicus (Figs. 160, 161, 164).

Thus, in the case of a growth situated in the right inferior quadrant, this portion of the circle is further divided by a line extending from its center to the right anterior superior spine of the ilium. Again, when locating a tumor or an area of tenderness or of pain in the superior right or left quadrants, these may be subdivided by a line extending from their center to any bony structure present in this region, *e. g.*, ascertain the costochondral articulation, and the exact location of the growth or area of tenderness or resistance may be marked upon this line subdividing the quadrant. It may be that the point in question is a definite distance to one or the other side of

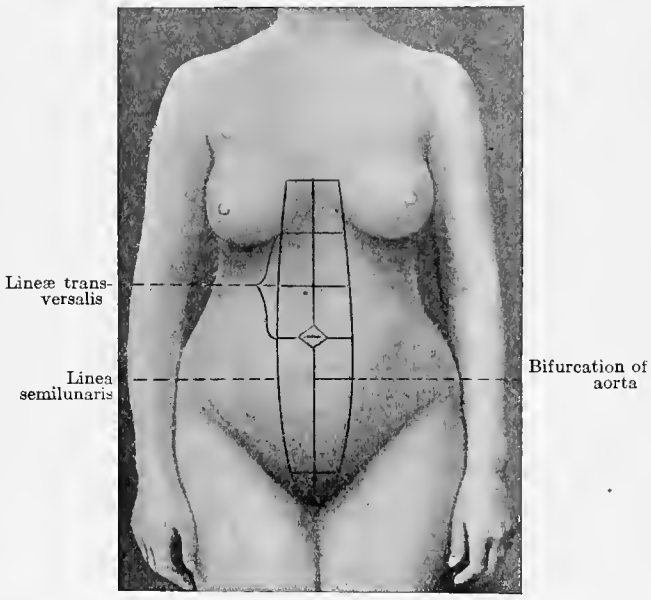


FIG. 163.—LANDMARKS OF THE ABDOMEN.

this subdividing line. Of further diagnostic service is it to employ a corresponding arbitrary division of the back. Observing the divisions of the abdomen, the normal position of the umbilicus and the fixed bony structures located at or near the periphery of the circle are employed to designate the exact location of a given abdominal tumor or area of tenderness (Fig. 163).

Within the superior right quadrant are two layers of organs, a superficial and a deep layer. In the superficial or anterior group are the right lobe of the liver, the gall-bladder, the hepatic flexure and portions of the ascending and the transverse colon, the head of the pancreas, and the pyloric end of the stomach.

The deep or retroperitoneal layer contains the greater portion of the right kidney and the suprarenal body.

The inferior right quadrant contains a portion of the ascending colon, cecum, vermiform appendix, right ovary, right Fallopian tube, and a portion of the uterus and of the bladder. At the margin of the arbitrary circular

boundary is the right inguinal ring. Upon deep inspiration the right kidney is forced down for about half its length.

Within the superior left quadrant will be found, in the superficial layer, the left lobe of the liver, the spleen, the greater portion of the stomach, the splenic flexure of the colon, a portion of the transverse colon, the descending colon, and the tail and about two-thirds of the body of the pancreas. The deeper layer contains the upper portion of the left kidney, the pelvis of the kidney, and the suprarenal body.

In the inferior left quadrant are the left ovary, the left Fallopian tube, a portion of the uterus and of the bladder, the descending colon, and the sigmoid flexure.

According to Holden, the left kidney is situated within the left inferior quadrant only upon deep inspiration. Along the bony margin we find the left inguinal ring.

The position of the small intestines is so variable that they may be found occupying portions of each arbitrary division of the abdomen.

FIG. 164.—LEFT ABDOMINAL HEMISPHERE.

DATA OBTAINED BY QUESTIONING THE PATIENT.

This particular class of symptoms will be considered fully under the general complaint for each abdominal malady. Inquiry should be made as to whether or not the patient suffers from local or general sensations of fullness, heat, burning, and pain. The local sensation of weight or of an abnormal degree of fullness in the abdomen is always suggestive of tumor, enlargement of organs, or displacement of a viscus.

The sensation of heat, or of a more or less constant burning, with or without pain, is usually associated with the presence of inflammatory affections of the abdomen, *e. g.*, carcinoma, pyosalpinx.

Character of Pain.—Pain may be either localized or general, dull or lancinating, continuous or intermittent.

Abdominal pain may begin suddenly, being extremely severe from the start, this being best exemplified by the pain of renal and of hepatic colic; or it may begin with slight sensations of discomfort, nausea, or faintness, and progress steadily until a severe type of pain is experienced—*e. g.*, uterine colic, intestinal colic (lead colic).

The sudden development of acute pain points toward inflammation or perforation of some hollow abdominal viscus, although it is often a symptom

of flatulent distention of the abdomen, intestinal obstruction, enteralgia, gastralgia, and all types of colic. Sudden pain developing during the course of gastric ulcer, typhoid fever, and allied gastro-intestinal conditions points strongly toward intestinal perforation. Chronic pain is indicative of peritoneal adhesions or of a somewhat acute or chronic inflammatory process. Abdominal pain, chronic in character, is seen to occur in intestinal neuroses, general neurasthenia, hysteria, insanity, locomotor ataxia, and lead-poisoning.

Localized pain is a symptom of gastric ulcer, pyosalpinx, and abdominal affections in which a variable degree of localized peritonitis is present.

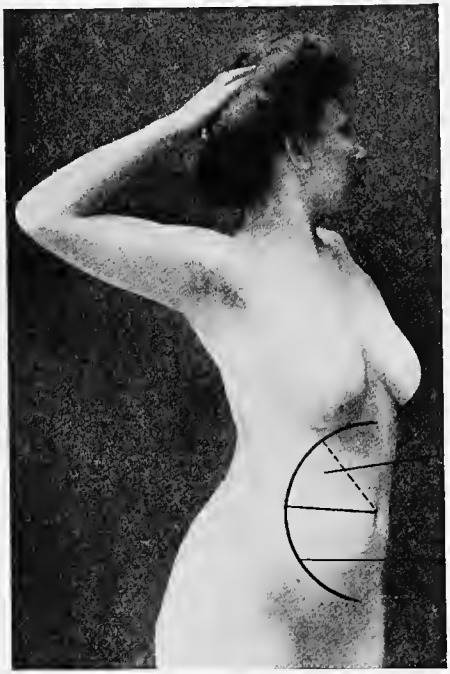


FIG. 165.—RIGHT ABDOMINAL HEMISPHERE.

The *abdominal pain* may be general in diffuse peritonitis, intestinal colic, and rheumatism of the abdominal muscles and fascia, the last-named condition ofttimes causing intense suffering, the muscles being tender and hypersensitive to movement, as *e. g.*, in laughing, coughing, and the like.

A dull, boring pain is associated with the presence of a large stone in the renal pelvis, carcinoma of the retroperitoneal glands, and carcinoma of the abdominal organs. Dull pain is seldom reflected from the seat of its greatest intensity.

Lancinating pain is continuous in general peritonitis and in inflammatory conditions in which the peritoneum shows decided involvement; it is also a symptom of carcinoma involving any of the abdominal organs.

The neuralgia and pain ofttimes associated with herpes zoster will be found to follow the distribution of certain spinal nerves. Neuralgic pains are to be distinguished from other forms of abdominal pain by the well-marked areas of tenderness, and by the fact that symptoms of neuritis may be associated.

Again, pain may result from affections of the skin and abdominal wall, *e. g.*, traumatism, abscesses, and ulceration. Pain situated in the abdominal wall may be dependent upon disease of its bony structures, as the ribs, vertebræ, or pelvis. Pain referable to disease of the vertebræ is usually limited to the median line, is intermittent in character, and oftenest described at a definite point between the ensiform cartilage and the umbilicus. We have seen several cases, both in private and in hospital practice, in which this particular type of abdominal pain was excited by pressure of an abdominal aneurism.

Special attention should be called to abdominal pain when present in children. We have considered such pains in the order of their frequency of occurrence:

(*a*) Dysperistalsis of the intestine, including enterospasm, usually excited by irritation. Obstruction or nervous incoördination is by far the commonest cause of abdominal pain in children under one year of age.

(*b*) Disturbance with the motor function of the stomach, together with pyloric spasm, cardiac spasm, and hour-glass contraction are not infrequent causes of abdominal pain during early life.

(*c*) Spasm of the sphincter of the bladder.

(*d*) Acute catarrhal dysentery.

(*e*) Reflex or referred abdominal pain is rather common in children, and may result from disease of the pleura, pericardium, and lung.

(*f*) Spinal caries.

(*g*) Renal colic.

(*h*) Appendicitis and peritonitis.

INSPECTION OF THE ABDOMEN.

One of the most important points to be remembered in making an inspection of the abdomen is that diseases involving extra-abdominal organs, *e. g.*, valvular heart disease, may exhibit among their symptoms an abnormal distention of the abdomen. In such conditions the distention is due to ascites. The reverse condition, *i. e.*, abdominal contraction, is a symptom of meningitis and of systemic poisoning, as seen in lead-workers.

Any distention of the abdomen is always suggestive either of unequal muscular development or of disease; in the latter case the form of distention points somewhat directly to the viscus affected. The condition of the skin, whether it be smooth or rough, dry or moist, and the degree of dilatation of the superficial veins should all be taken into consideration in formulating a diagnosis of abdominal affections. The superficial veins of the abdomen are dilated together with the general dilatation of the veins of the lower extremities, or such distention may depend upon some obstruction to the deeper venous blood in the pelvis or thorax—*e. g.*, organic heart disease, cirrhosis of the liver, adhesive pyelophlebitis, pressure upon the vena cava exerted by tumors of the abdomen or of the thorax. As a rule, the veins are found to be prominent in ascites and in most condi-

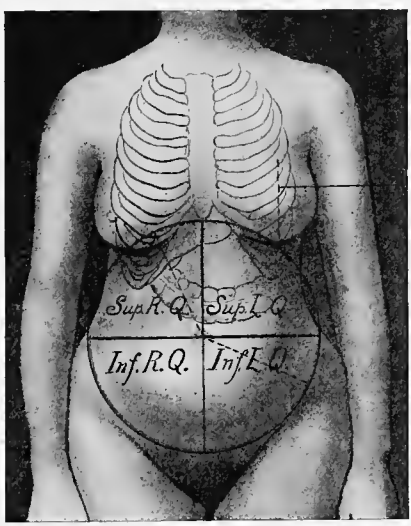


Fig. 166.—Arbitrary Division of the Abdomen, Showing Relation of Transverse Colon.

tions causing abdominal distention, regardless of whether or not there has been a decided atrophy of the abdominal wall.

A knowledge of the thickness of the abdominal wall is also of decided clinical importance. A thin abdominal wall depends, for the most part, upon an absence of adipose and muscular tissue, and in any given case this may have gone so far as to be due in part to atrophy of the muscular structure. The cause of muscular atrophy should be ascertained, and will be found, in the vast majority of instances, either to depend upon intra-abdominal pressure or to be associated with nutritional affections in which there is pronounced emaciation—*e. g.*, diabetes, tabes dorsalis, paretic dementia. In the first class of cases should be placed frequent pregnancies, large ovarian tumors, and repeated attacks of ascites, all of which conditions tend to produce a variable degree of atrophy of the abdominal muscles, tuberculosis,

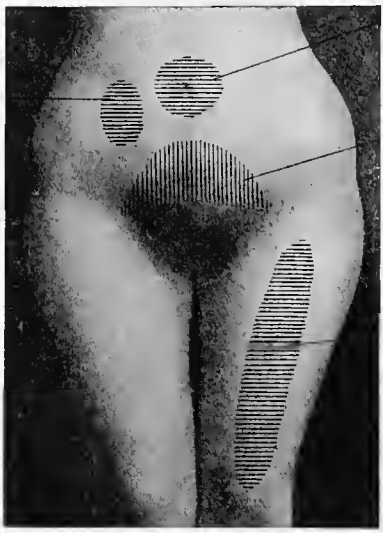


FIG. 167.—AREAS OF PAIN.

and gastro-intestinal catarrh of long standing. When the abdominal wall is thin, the superficial veins are perceptible and may be abnormal.

Further, abdominal distention may result in a separation of the rectus muscles, with hernia-like protrusions between. In several cases seen by us such hernias have followed the exciting conditions previously named; in one case, that of an Italian treated in the wards of the Philadelphia Hospital, there was pronounced abdominal hernia, the result of enlargement of the liver and of the spleen following malarial infection.

Great importance attaches itself to the presence or absence of these hernial projections, and particularly is this true in considering the question of aspiration of the peritoneum. Hernia may be most confusing, as when it appears in portions of the abdomen not commonly affected.

A thick abdominal wall may depend, in part, upon extraordinary muscular development, upon an unusually heavy deposit of fat, or, lastly, upon edema. In health the abdominal thickness is due to muscular development, the position of the umbilicus remaining unchanged. Excessive abdominal

fat causes decided folds in the abdomen below the umbilicus, and the umbilical depression will be found below its normal position.

The skin has a somewhat dull appearance, and there are likely to be numerous striæ, but the superficial veins are not prominent. Occasionally, there is an extraordinary dilatation of the veins in the region of the umbilicus, and this mass of dilated veins is referred to as the "caput medusæ."

Movements of the Abdomen.—Under normal conditions the abdominal movements may depend upon respiration, vascular pulsation, gastric movement, intestinal movements (peristalsis), fetal movements, and the changes of position of floating viscera or tumors coincidentally with the change of the patient's position.

Abdominal movement or the abdominal type of breathing is increased by organic disease of the lungs, a large quantity of fluid in one or other pleural cavity, mediastinal tumor, thoracic aneurism, and in conditions that materially inhibit a lateral expansion of the lungs, as, *e. g.*, pleural adhesions and emphysema.

Under normal conditions the upper half of the abdomen rises and falls synchronously with inspiration and expiration. The respiratory expansion is diminished by the presence of a large quantity of fluid in the peritoneal sac, tuberculosis of the peritoneum, general or local peritonitis, peritoneal adhesions the result either of disease or of operative interference with the abdominal viscera, and by abdominal tumor. Conditions that interfere with the descent of the diaphragm also lessen the abdominal expansion—*e. g.*, ascites, tumors occupying the upper portion of the abdomen, and enlargement of the liver and of the spleen.

In paralysis of the diaphragm a reverse phenomenon occurs with the respiratory movements—*e. g.*, the size of the abdomen diminishes with inspiration, whereas a feeble degree of expansion takes place during expiration. A peculiar respiratory movement of the abdomen is also observed in the presence of obstruction to the larynx and upper respiratory tract, resembling somewhat closely that seen in paralysis of the diaphragm.

If there is enlargement of either the liver or the spleen, the enlarged organ will be seen to rise and fall synchronously with respiration, and the movement of such viscus is usually referred to as "the shadow." Movements of the abdomen are of great value in making a diagnosis of tumors either of the liver or the spleen, since tumors that are not attached to either of these organs are but little, if at all, influenced by respiration. Exceptions to this general rule, however, occur—*e. g.*, when a tumor of the right kidney is seen to fall and rise synchronously with respiration, a phenomenon readily explained by the fact that in such cases the kidney is permanently adherent to the liver.

Vascular Movements.—Abdominal movement due to vascular pulsation is usually observed in the median line, the exception being in the case of pulsation of the liver, a feature sometimes seen to accompany tricupsid regurgitation. In subjects in whom the abdominal wall is thin, pulsation of the aorta is perceptible; and either localized or diffuse pulsation is to be seen when there is aneurism of the abdominal aorta or of one of its branches. Decided pulsation near the umbilicus is suggestive of aneurism of the celiac axis.

Tumors of moderate size overlying the aorta may give rise to movement of the abdominal wall, such movements being synchronous with the impulse of the artery. Epigastric pulsation in the median line or slightly to the left may be the result of a dilated right ventricle. (See Cardiac Dilatation, p. 300.)

Gastric Movements.—The movements of both the stomach and the intestines may be seen through the abdominal wall, and are especially marked when there is a high grade of peristalsis. The peristaltic movements are increased in dilatation of the stomach and gastroptosis, in which conditions they appear in somewhat rhythmic succession, and usually extend from left to right. (See p. 440.) Movements of the large intestine will be seen to follow the course of the colon, and may appear on either side or in the median line, at the top of the abdomen. In coloptosis the movement of the colon may be in the median line, on a level with or below the umbilicus.

All movements of the intestines, and even those of the stomach, may be increased by any condition in which obstructive lesions of the lumen of the bowel exist. In complete intestinal obstruction a reverse peristaltic wave is commonly visible, becoming more and more evident until there is regurgitation of the contents of the bowel into the stomach, with vomiting of fecal material. Movement of the small intestines may be greatly increased after the ingestion of certain foods—*e. g.*, unripe fruit—and such drugs as jalap, elaterium, sodium phosphate, and magnesium.

Contour of Abdomen.—The general contour of the normal abdomen is familiar to every physician, but in order to obtain the valuable knowledge to be gained by inspection, it is necessary for him to get a front, lateral, and three-quarter view of this portion of the body.

When there is general enlargement of the abdomen, the increase is nearly symmetric. Uniform enlargement results from ascites, provided the abdominal wall is thick and muscular; but if the abdominal wall is relaxed, the contour may be more or less pear-shaped when the patient is standing, and again flattened along the lateral boundaries when the recumbent posture is assumed. The abdomen is pendulous when the enlargement is dependent upon fat deposited in the abdominal wall.

Localized enlargement of the abdomen causes the surface to be irregular at some given point, and will be further discussed in conjunction with diseases of the abdominal viscera. The abdomen may be uniformly distended in hysteria, and a prominence of the lower portion of the abdomen may be dependent upon retention of the urine.

A retracted or scaphoid abdomen is seen during the course of chronic maladies, such as lead-poisoning (Fig. 221), carcinoma, tuberculosis, and diabetes. In these maladies the abdominal wall is found to be very thin, and consequently an undue prominence of the viscera, particularly of the liver, may produce local enlargement.

The size of the abdomen will be found to vary greatly in different persons, this variation depending not only upon the thickness of the abdominal muscles or the deposit of fat in the abdominal wall, but also upon the amount of fat deposited in the omentum, and upon the caliber of both the small and the large intestines.

The caliber of the small intestine is greatly increased in persons who eat heavily and whose habits are sedentary. Prominence of the abdomen due to the deposit of fat or to enlargement of the intestine is to be distinguished from the enlargement accompanying true obesity, since in the latter condition there is a general deposit of fat throughout the subcutaneous tissues.

Abdominal prominence due to new-growths, ascites, pregnancy, and cysts is easily distinguished from the foregoing types of enlargement, since in the former conditions there are likely to be evidences of a variable degree of emaciation.

Causes for general and local enlargement of the abdomen are divided for

clinical study into the following: General enlargement, local enlargement, enlargements due to oversize of solid viscera; to distention of hollow viscera; to fluid; to cystic and solid tumors; to physiologic causes, and to abnormalities (tumors and overgrowths) in the abdominal wall.

GENERAL ENLARGEMENT.

Distention of the bowel and stomach by gas,
Distention of hollow viscera following surgical operation,
Ascites (serous),
Ascites hemorrhagic or chylous,
Obesity,
Adiposis dolorosa,
Intestinal obstruction.

LOCAL SWELLING (UPPER HALF).

Cancer of liver,
Sarcoma of liver,
Cyanotic liver,
Fatty liver,
Amyloid liver,
Leukemic liver,
Malarial liver,
Enlargement of spleen,
Aneurysm of abdominal aorta,
Hypertrophic cirrhosis.

LOCAL SWELLING (LOWER HALF).

Perinephritic abscess,
Sarcoma of kidney,
Hydronephrosis,
Pregnancy,
Uterine fibroid,
Displacement of stomach and intestine.
Ovarian cyst,
Ectopic gestation,
Malignancy,
Distention of bladder,
Enormous dilatation of stomach,
Dilatation of bladder.

ABDOMINAL WALL.

Phantom tumor,
Nodules in adiposis dolorosa.

EXAMINATION OF THE STOMACH.

General Remarks.—The stomach is an expanded portion of the alimentary canal, the food being retained in this expansion partly for the purpose of effecting solution, and partly in order to become thoroughly saturated by the secretions of the mouth and of the stomach. The stomach-wall is made up of three coats—an interior or mucous coat, a middle or muscular layer, and an external or peritoneal coat. When one or more of these coats become diseased, definite symptoms arise; in many types of gastric disorder all the coats of the stomach may be involved.

It must be remembered that the mucous and muscular coats of the stomach-wall are richly supplied with blood-vessels, and that erosion or congestion of the wall is likely to be followed by an extravasation of blood into the cavity of the stomach. The stomach-wall is also abundantly supplied with nerves, and its relation to the pneumogastric nerve, as well as to numerous sympathetic plexuses, may give rise to certain symptoms. Disease of remotely situated organs may display, as the most prominent symptoms, those manifestations also referable to disease of the stomach.

CHARACTER OF EVIDENCE TO BE OBTAINED BY INQUIRY.

Family History.—Heredity is seldom an important factor in diseases of the stomach, except in two conditions, *e. g.*, gastric neuroses and gastric carcinoma.

Social History.—In but few diseases is special inquiry into the social condition of the patient of such vital clinical importance as in gastric affections. Many stomach disorders have their origin in neurasthenia, or in the various forms of dissipation and overwork, *e. g.*, overeating, improper mastication, insufficient exercise, excessive mental strain, overwork, and the abuse of alcohol. They may also arise as the result of the improper functioning of some other organ, as, for example, the liver, pancreas, intestines, kidneys, heart.

(*a*) The time consumed for the ingestion of a meal and the character of the food eaten are matters of special importance. Overeating, and particularly the ingestion of rich and highly seasoned foods, is one of the commonest causes of gastric disorders. Too frequent eating and the habit of taking food between meals are highly detrimental, since the stomach is not permitted to get sufficient rest. Thorough mastication is also important. Unless the food is well mixed with the saliva, starch digestion is materially impaired. Again, the taking of alcoholic stimulants and the excessive use of narcotics and tobacco tend to inhibit digestion.

(*b*) **Exercise and Occupation.**—Persons of sedentary habits are especially prone to develop gastro-intestinal disorders; for this reason the amount of exercise taken daily must be carefully ascertained in order to determine the nature of the disease present.

Occupations that do not allow sufficient outdoor exercise to be taken, or that necessitate frequent or continuous exposure to such toxic substances as lead, arsenic, mercury, and gases, constitute a common cause of gastric disorders. A history of the ingestion of corrosive substances also points strongly toward gastritis. Occupation is of further importance to the clinician, since cooks, clerks, seamstresses, tailors, shoemakers, and carpenters are particularly subject to gastric catarrh and to gastric ulcer.

(*c*) **Mental Strain and Overwork.**—Overwork, either mental or physical, interferes with the proper functioning of the stomach; thus the practice of doing heavy work immediately or within an hour after taking a full meal lessens the activity of the gastric glands and consequently diminishes the gastric secretion. Those who do not rest after the ingestion of a full meal sooner or later develop gastric inactivity.

(*d*) **Local affections,** either intra-abdominal or extra-abdominal, may occur either as a complication of or as a sequel to, disease of the stomach. Impairment of hepatic function of whatever nature gives rise to the development of a variable degree of indigestion. Pathologic changes in the liver that interfere with the return circulation from the stomach are frequent, and are best exemplified by atrophic hepatic cirrhosis, with ascites.

Pancreatic disease may manifest gastric insufficiency as one of its symptoms, and *nephritis* and diseases of the *intestine* may also give rise to the symptoms of indigestion. Organic heart disease, when sufficiently pronounced, is a cause of venous stasis of the gastric mucous membrane, which interferes with gastric secretion.

(*e*) **Age.**—Early adult life—fourteen to thirty years— predisposes to the development of gastric disorders, but such predisposition is probably due to lack of care both as to the character of the food ingested and the regularity with which such foods are taken. In women gastric maladies are common at the menopause.

(*f*) **Sex** exercises but slight influence on diseases of the stomach.

(*g*) **Previous Diseases.**—Gastric disease may follow certain of the infectious conditions, when it is a direct result either of the preceding infection

or of imperfect innervation. Any prolonged illness, whether acute or chronic, that materially impoverishes the system predisposes to the development of gastric disease.

(*h*) Finally, any abdominal growth that causes pressure upon the sympathetic nerve-supply of the abdomen may provoke the symptoms of gastric disease. Relaxation of the muscles of the abdominal wall, by permitting displacement of the stomach or of other abdominal viscera, is often followed by symptoms of gastritis; therefore a history of the previous existence of ascites, abdominal tumor, and repeated pregnancies is of great importance to the clinician.

LEADING FEATURES AND THEIR SIGNIFICANCE.

Disordered Appetite.—(1) **Anorexia.**—*Definition.*—The appetite is impaired, or there may be no desire whatever for food.

Anorexia is an early symptom in nearly all infections, and also occurs during the course of many chronic afebrile and febrile maladies. In disease of the stomach, however, the presence or absence of anorexia is of great importance in formulating a diagnosis. Loss of appetite may be present in organic disease of the stomach, and more particularly in carcinoma, and is not infrequent in gastric neuroses with gastric hyperesthesia. When the appetite is greatly impaired for prolonged periods, food is often repugnant, and the patient may go for days or even weeks without sufficient food to provide for the general bodily nutrition, and, as a consequence, the symptoms of malnutrition develop.

Prominent among the causes of anorexia are: Excessive mental strain, mental shock, anxiety, imperfectly prepared foods, and starvation. The use of such drugs as digitalis, strophanthus, the salicylates, opium, the iodids, etc., may be followed by anorexia.

(2) **Bulimia, also known as Hyperorexia.**—*Definition.*—A symptom characterized by an excessive appetite for practically all kinds of food.

Bulimia is nearly always pathologic in origin, although some persons who are apparently healthy consume extraordinary amounts of food both at and between meals. During convalescence from typhoid fever and other prolonged febrile conditions the appetite is, as a rule, excessive. In diabetes mellitus one of the earliest symptoms is overeating, and this usually persists until late in the course of the disease. Bulimia is an occasional symptom in neuroses of the stomach, neurasthenia, and hysteria, and in these conditions epigastric distress and even pain are alleviated by the taking of large amounts of food. A child whose intestinal tract is infested by lumbricoid worms often develops this symptom. An abnormal appetite may develop after hemiplegia, monoplegia, epileptic seizures, or during the course of meningitis, migraine, ataxia, Huntingdon's chorea and other nervous conditions, and in certain types of insanity.

In a case of carcinoma of the retroperitoneal glands, recently seen, this peculiar type of appetite persisted for several months.

(3) **Polyphagia** is a condition in which the feeling of satisfaction after a meal is so delayed that there is a constant desire for more food.

(4) **Parorexia or Perverted Appetite.**—(*a*) *Malacia* is a condition in which there is a desire for highly spiced and acid foods, *e. g.*, pickles, salads, mustard, pepper, and the like. This symptom is present in neurasthenia, chronic gastritis, intestinal putrefaction, chlorosis, and the secondary anemias of young women.

(*b*) *Pica* is an abnormal craving for substances other than natural food. such as dirt, crayon, the wood from pencils, blades of grass, straw, and the like. The condition is suggestive of neurasthenia and hysteria, and is seen in children infected with intestinal parasites, and in girls at or near the time of puberty. It is also encountered in chronic gastritis and in anemic individuals.

(*c*) *Allotriophagia* is a desire for disgusting substances, such as urine and feces; it has been seen in insane persons.

Thirst.—An excessive thirst may result from a variety of conditions, many of which are not connected with the stomach.

Physiologic thirst develops as a result of free perspiration, and is intensified by the taking of drugs that induce purging, *e. g.*, aloin, elaterium, and magnesia.

Pathologic thirst is one of the cardinal symptoms of diabetes mellitus, and follows either acute or chronic conditions in which there are excessive evaporation and combustion, such as typhoid fever and scarlet fever. Thirst also occurs in any condition that causes rapid removal of a large quantity of the liquid elements of the blood, *e. g.*, a hot bath, hemorrhage, diarrhea, dysentery, and vomiting.

Thirst is a prominent feature of acute gastritis, and the desire for liquids is increased during the course of chronic gastritis, this symptom becoming markedly intensified whenever an exacerbation of the latter disease occurs.

The ingestion of certain substances increases the desire for liquids, *e. g.*, alcohol and alcoholic substances and highly seasoned foods, particularly salt meats and fish.

Taste.—In diseases of the stomach, particularly in acute and chronic gastric catarrh, the taste is abnormal (offensive). An offensive taste is a prominent symptom of the form of acute gastritis known as *biliousness.* The taste is perverted, dull, or absent during the fastigium of typhoid fever, at the height of acute gastritis, and during acute exacerbations of chronic intestinal catarrh. In practically all febrile conditions in which the tongue is coated the sense of taste is markedly impaired, and the patient desires only highly spiced and acid foods.

Chronic diseases of the tonsils, pharynx, esophagus, and posterior nares are usually accompanied by an unpleasant taste upon rising in the morning, although in some instances it may be present throughout the day. In dilatation of the esophagus, gastrectasis, pulmonary abscess, pulmonary gangrene, and abscess of the liver that has ruptured into the lung the taste is somewhat sweet, and more pronounced after coughing, vomiting, and clearing the throat.

A highly acid taste is suggestive of hyperacidity of the stomach, and is frequently seen in gastric ulcer, and in conditions dependent upon an excess of free hydrochloric acid. In dilatation of the stomach with atrophic gastritis and in carcinoma an acid taste is extremely common, and is usually dependent upon increased acidity, due to an excess of lactic and of butyric acid. An alkaline taste is occasionally described by hysteric individuals.

Lastly, taste may be modified or absent as a result of disease of the nerves of special sense, particularly those of taste and of smell. Whenever the sense of smell is absent, the taste is greatly modified, and, as a rule, much less acute than in health.

Pyrosis and Regurgitation.— **Definition.** — The eructation of either liquids or gases, which cause a burning sensation in the esophagus, throat, and mouth. These symptoms are generally associated with some

pathologic condition of the stomach. There may be eructation of gas, liquids, and particles of undigested food, the condition then being known as *pyrosis with regurgitation.* There are exceptional instances in which the fluid that rises to the mouth is alkaline in reaction and comparatively tasteless. Pyrosis must be distinguished from vomiting; in the former the fluid is ejected without any effort upon the part of the patient, and without either gastric discomfort or pain. The fluid brought up, when alkaline, may consist of saliva that has accumulated in some sacculation or expanded portion of the esophagus. Saliva when ejected possesses the power of digesting starches.

Alkaline fluids are regurgitated during or immediately after the ingestion of food, whereas acid liquids are more likely to be ejected one or more hours after food has been taken.

Pyrosis is a symptom of overeating, acute gastritis, alcoholic gastritis, chronic gastritis with fermentation, gastric ulcer, and other conditions in which there is hyperchlorhydria or hyperacidity of the gastric contents from whatever cause.

The regurgitation of foods from the stomach may be accompanied by a burning sensation and the eructation of a variable amount of gas or liquid. At certain times gastric fluid alone is regurgitated, whereas at others large quantities of food are brought up. When the patient chews the regurgitated food and swallows it again, the condition is termed rumination; this is a symptom of neurasthenia, hysteria, and insanity.

Hiccough.—Definition.—A peculiar clicking sound that follows spasm of the diaphragm and the rushing of air through the glottis.

Hiccough persisting for prolonged periods is of unfavorable prognostic significance. It occurs as the result of overeating, the ingestion of too highly seasoned foods, alcoholic beverages, and unripe or decomposing fruits and vegetables. When it occurs after the ingestion of a full meal, it seldom lasts more than a few minutes.

Hiccough is frequently an expression of exhaustion or severe toxemia with marked nervous atony, *e. g.,* uremia and intestinal putrefaction. It is an unfavorable symptom in either local or general acute peritonitis.

FIG. 168.—TRACING OF RESPIRATORY MOVEMENTS IN A CASE OF HICCOUGH.

Hiccough may be an annoying symptom during the course of any variety of nephritis, erysipelas, brain tumor, and myocarditis. Occasionally it accompanies Cheyne-Stokes respiration. (See p. 51.)

Nausea usually precedes vomiting, although in many persons vomiting is apparently impossible, whereas nausea may be a most annoying symptom. Nausea is a feature of many extragastric conditions, but it may follow the taking of certain foods or irritating substances. The odor of a certain food or liquid often excites nausea, and many persons are nauseated at the mere sight of certain forms of food. Hyperacidity of the stomach-contents is a frequent cause of nausea, as are also disease of the esophagus, chronic pharyngitis, and chronic postnasal catarrh.

Nausea may follow the accumulation of toxins within the stomach, and

not infrequently occurs after traumatism to the head, at the sight of blood, or on witnessing an accident. It is a common symptom of chronic interstitial nephritis, general arterial sclerosis, organic heart disease with tricuspid regurgitation in which there is an associated venous stasis of the gastric mucosa. Nausea is occasionally seen in cirrhosis of the liver, obstructive jaundice, and chronic enterocolitis. Abdominal or thoracic tumors pressing upon the sympathetic nervous system, *e. g.*, ovarian cysts, abdominal aneurisms, and uterine fibroids, may excite nausea and vomiting.

Whenever the cause of nausea is in doubt, an examination of the external auditory canal should be made, since pressure upon the membrana tympani may give rise to this symptom. Eye-strain, due to defects either in the cornea or in the deeper structures of the eye, may also induce temporary attacks of nausea.

Vomiting is a phenomenon due to a coincident, spontaneous contraction of the abdominal muscles and diaphragm, together with a relaxation of the muscles at the cardiac end of the stomach. Spasm of the muscles at the cardiac portion of the stomach serves to explain why many persons are unable to vomit even when there is violent retching, due to contraction of both the diaphragm and the abdominal muscles. The center for vomiting is said by physiologists to be located in the medulla oblongata, contiguous to that for respiration. Nerves from practically all parts of the body, and particularly from the liver, intestine, kidneys, lungs, esophagus, uterus, and bladder, and from the special sense centers, convey impressions to the center for vomiting, and this fact serves to explain why vomiting is so often reflex in character.

The vomiting of acute infections, *e. g.*, scarlet fever or small-pox, may be dependent upon a specific irritant circulating through the vomiting center, or upon an associated congestion of some other portion of the body that has direct communication with that center. The latter explanation serves, in a measure, at least, to explain the vomiting of uremia, auto-intoxication, and allied conditions. The question has arisen as to whether or not the vomiting of hysteria is central in origin.

In attempting to determine the actual origin and significance of vomiting careful inquiry must be made into its nature, duration, time of development, the manner in which it began, what is believed to have precipitated the first attack, the quantity of vomitus ejected each time, the character of the material vomited, the condition of the bowels, and the patient's general health.

Types of Vomiting.—(*a*) The vomiting of acute infections and inflammation of the stomach.

(*b*) The vomiting of chronic conditions of the stomach: (1) Chronic gastritis. (2) Vomiting of gastric ulcer. (3) Vomiting of gastric carcinoma. (4) Vomiting of atrophic gastritis with dilatation.

(*c*) Vomiting of acute infectious diseases.

(*d*) Cerebral vomiting.

(*e*) Reflex vomiting: (1) Cyclic vomiting. (2) Vomiting of peritonitis. (3) Vomiting of pulmonary tuberculosis. (4) Uremic vomiting.

(*a*) The **vomiting of acute gastritis** is quite characteristic, since it develops after the free ingestion of indigestible foods and alcoholic liquors; the use of such drugs as opium, the bromids, the salicylates, the iodids, and toxic doses of arsenic and mercury.

Nausea is a precursor of the vomiting of acute gastritis, and epigastric distress or pain is usually present.

The patient is greatly weakened by the act of vomiting, and often, in addition to exhaustion, his skin becomes cold and covered with beads of

perspiration. The first vomitus contains particles of undigested food, and after the stomach has been relieved of this, the patient vomits only mucus, to which is added a large quantity of saliva. If the vomiting continues, the patient ejects a greenish-yellow material which owes its color to the admixture of bile. After repeated attacks of vomiting the vomitus is not infrequently streaked with blood.

The conditions known to cause vomiting of blood are: 1. Acute gastritis. 2. Gastric ulcer. 3. Gastric carcinoma. 4. Ulcer of the esophagus. 5. Carcinoma of the esophagus. 6. Varicosity of the esophageal veins. 7. Prolonged vomiting of doubtful cause. 8. Cirrhosis of the liver. 9. Organic heart disease with tricuspid regurgitation. 10. Introduction of corrosive substances into the stomach. 11. Tuberculosis of the stomach. 12. Profound anemias, either primary or secondary. 13. Vicarious menstruation. 14. Blood swallowed and coming from the mouth, throat, or upper air-passages. 15. Extensive cutaneous burns.

(*b*) **Vomiting in Chronic Conditions of the Stomach.**—(1) The *vomitus of chronic gastritis* differs from that seen in acute inflammation of the stomach in appearance, chemic reaction, and effect upon the patient. "I have repeatedly found the material vomited in chronic gastric catarrh to be acid in reaction, unless, as occasionally happens, the vomiting takes place several hours after eating, when it is sometimes faintly alkaline or neutral" (Anders).

Vomiting does not cause extreme prostration in chronic gastric maladies, and even though marked emaciation is present, it is unsafe to conclude that gastric carcinoma exists without making a chemic analysis of the stomach-contents.

If the vomitus of chronic gastritis should display an abnormally high total acidity, special quantitative tests should be made to ascertain the amount of free hydrochloric and lactic acid present. Acid salts, lactic acid, and butyric acid may be in excess, and the amount of hydrochloric acid may be greatly below that of the normal. (See Tests for Gastric Contents, p. 464.) The characteristic vomitus of the different types of chronic gastritis will be further considered together with a description of this affection.

(2) *Vomitus of Gastric Ulcer.*—Vomiting is one of the most constant symptoms of gastric ulcer, and occurs either immediately after or within from one-half to one and one-half hours after the taking of food. It is always preceded by acute pain, which subsides as the vomiting ceases.

In ulcer the vomitus contains particles of the food previously ingested, and in approximately 30 per cent. of all cases blood is present. If careful analyses of the vomitus for the presence of blood are repeatedly made, the evidences of minute hemorrhages will probably be found.

The so-called characteristic features of the vomitus of ulcer are an excess of free hydrochloric acid and the presence of blood. In contradistinction to the vomitus of chronic gastritis, in which the total acidity may also be above the normal, the vomitus of ulcer rarely, if ever, contains lactic and butyric acids in appreciable amounts.

(3) *Vomiting of Carcinoma.*—Gastric carcinoma is almost always accompanied by vomiting, although it may not occur until the carcinomatous process has attacked the gastric wall; as a rule, however, vomiting takes place early, and continues until a fatal termination occurs. The location of the carcinoma and the degree of ulceration or of sclerosis of the gastric wall materially influence the time of its occurrence and the character of the vomitus. The macroscopic appearance of the vomitus of carcinoma is not characteristic, as was formerly held.

The frequency of vomiting will be found to vary greatly, depending upon the size of the carcinoma and upon its location on the stomach-wall. Vomiting occurs a variable time after the taking of food, and is preceded by a deep-seated boring pain, which is not relieved by emptying the stomach.

If the carcinoma is situated at the cardiac portion of the stomach, vomiting takes place almost immediately after the ingestion of food, but if the lesion is near the pylorus, it is deferred for some hours. In carcinoma of the pylorus with a variable degree of obstruction dilatation of the stomach follows, and in such instances copious vomiting may occur only once or twice during the week. If there is oozing of blood into the stomach, the vomitus is brownish or bluish in color—the so-called "coffee-ground" vomitus of gastric carcinoma.

Chemically, the vomitus of gastric carcinoma shows a deficiency in free hydrochloric acid, and, indeed, in many instances free hydrochloric acid is absent. A still more constant finding is that of lactic acid, which is usually present in large amounts, as is also butyric acid.

Caution.—A diagnosis of carcinoma of the stomach cannot be made from an analysis of the vomitus alone, since similar findings are rarely met in the gastric fluids of other chronic conditions in which malnutrition and neurasthenia figure prominently.

(4) *Vomiting of Gastric Dilatation.*—In this condition the character of the material ejected from the stomach is in many ways characteristic of the pathologic changes that have taken place. In striking contrast to the vomiting of gastric ulcer and of gastric carcinoma, we here find that the patient does not vomit at any stipulated time after the taking of food, nor does the character of food taken in any way influence the time of vomiting. Depending upon the degree of dilatation, an abnormally large quantity (from two to eight quarts) of partially digested food is ejected. The vomitus often contains particles of food that have been taken days before, and that have remained in the stomach without undergoing decided alteration. If the degree of gastric dilatation is great, free hydrochloric acid may be absent from the vomitus, whereas lactic and fatty acids are, as a rule, present.

(*c*) **Vomiting of Acute Infections.**—Virulent forms of infection, such as small-pox, scarlet fever, yellow fever, or pneumonia, are often ushered in by vomiting, and the severity of the vomiting is directly dependent upon the severity of the type of infection.

The vomitus of the acute infections at first contains undigested food, but later it is nearly mucoid in consistence. If the attack of vomiting continues over a period of several hours, blood may be ejected. When the vomitus contains blood, before retching has occurred, hemorrhagic forms of infection are to be suspected.

Chemic analysis of the vomitus of the acute infections shows that the normal quantity of free hydrochloric acid is present, and at times traces of lactic acid are also demonstrable. There is nothing characteristic of the vomitus of acute infections, and it is often with great difficulty that one is able to distinguish this form of vomiting from that caused by dietetic errors.

(*d*) **Cerebral Vomiting.**—At times the physician encounters severe vomiting that is dependent upon pathologic cerebral conditions, *e. g.*, brain tumor, acute meningitis, chronic meningitis, and early in apoplexy. The vomiting of apoplexy is associated with definite symptoms and signs of cerebral hemorrhage, whereas other types of cerebral vomiting are not accompanied by characteristic symptoms.

Vomiting of Locomotor Ataxia.—During the course of this disease violent

attacks of vomiting and pain occur periodically, which are known as "gastric crises." The vomitus of ataxic crises, in addition to containing particles of undigested food, is highly acid in reaction.

If the vomiting should continue for a prolonged period, there are profound exhaustion and anuria. In asthenic cases of ataxia circulatory collapse frequently follows these gastric crises.

(*e*) **Reflex Vomiting.**—*Definition.*—A condition in which vomiting occurs without appreciable pathologic change in the stomach.

As previously stated, vomiting may be induced reflexly, and be dependent upon disease in organs remotely situated from the stomach, or it may be caused by the sight of certain substances, as blood and horrible scenes, or by certain odors. Pain of whatever nature is one of the commonest causes of this symptom.

In determining whether or not the vomiting is of reflex nature, it is necessary for the physician to ascertain the existence or non-existence of disease or irritation of other organs than the stomach. When it is possible to determine the seat of an irritation, an impression of which is in turn conveyed to the vomiting center, the removal of such irritation is followed by relief. Probably the best example of reflex vomiting is that occurring in early pregnancy, before the uterus has risen above the brim of the pelvis, while it exerts pressure upon the pelvic sympathetic nerves. It must be borne in mind that any abdominal growth causing similar pressure may excite vomiting.

(1) *Cyclic Vomiting.*—A pathologic type of vomiting first described by Lyden, characterized by its sudden onset and the severity of the retching. Snow has suggested that the cause of these attacks is probably a gastric neurosis, whereas other writers hold that it is quite difficult to distinguish cyclic vomiting from the vomiting of uremia and that of toxic origin. The vomitus first contains the contents of the stomach, but later becomes mucoid in character. Chemically, the first vomitus is practically normal, but later it may contain no hydrochloric acid. In adults cyclic vomiting is not accompanied by a rise in the temperature, but in children fever is generally present.

Among the symptoms associated with cyclic vomiting are extreme prostration, retraction of the abdominal muscles, and a tendency toward circulatory collapse. After the vomiting has persisted for an hour or more the expression becomes anxious, the cheeks are sunken, and the extremities are beaded with cold perspiration.

(2) *Vomiting of Peritonitis.*—Reflex vomiting may be caused by both acute and chronic peritonitis. The vomitus of peritonitis is likely to contain bile after the stomach has become empty. In persons suffering from renal, hepatic, and uterine colic it is often difficult to determine whether or not peritonitis is present, but the fact that this form of vomiting usually subsides with the disappearance of the pain would indicate that the peritoneum is not involved.

(3) *Vomiting of Pulmonary Tuberculosis.*—In pulmonary tuberculosis with cavity formation the patient usually experiences a paroxysmal attack of coughing soon after rising in the morning, and during this attack the throat becomes so irritated that the contents of the stomach are ejected. It must be remembered that gastric ulcer may be present in those suffering from pulmonary tuberculosis, in which case the characteristic features of the vomiting of the latter condition may also be present.

(4) *Vomiting of Uremia.*—This type of vomiting seldom, if ever, occurs

unless albumin or casts, or probably both, are present in the urine. It should not be forgotten that a urine of low specific gravity may contain neither albumin nor casts, and yet the patient suffers from attacks of uremic vomiting. Again, it is of further interest to note that these patients do, at some time or other, show both albumin and casts in their urine, but the albuminuria is, as a rule, intermittent, and the amount of albumin passed with the urine is slight.

Uremic vomiting occurs most often during the morning hours, but may take place at any time during the day or night. Vomiting commonly follows several hours' exposure to cold, and also occurs after partaking of a meal rich in albumins.

In practically all persons suffering from uremic vomiting the arterial tension is high, the pulse is slow but wiry, the heart-sounds are forcible, and there is evidence of a variable degree of cardiac hypertrophy.

Pain.—Types.—Epigastric pain may be moderate, severe, or intense. The terms *cardialgia*, *gastrodynia*, and *gastralgia* are used to designate these pains. They are all somewhat synonymous, and all are used in a more or less restricted sense by various writers.

Cardialgia properly means neuralgia of the stomach, but is sometimes defined as "severe paroxysmal pain in the epigastrium in the absence of gastric lesions" (Anders).

Gastrodynia is a term applied to severe, cramp-like pains in the stomach region. The phenomenon may or may not be associated with organic disease of the stomach.

Gastralgia means pain in the stomach.

Flatulence is a term applied to an accumulation of gas in the stomach and intestines.

Attacks of epigastric pain often begin as a mere sensation of discomfort in the epigastrium, which gradually increases until distinct painful sensations of varying severity are experienced. Attacks of pain are seen in atonic dyspepsia, catarrhal gastritis, the gastric neuroses, gastric ulcer, gastric carcinoma, localized peritonitis, etc.

During the paroxysm, the patient displays a variable degree of shock, the severity of which is dependent upon the degree of pain and the length of time it has existed. In severe cases the skin becomes cold, clammy, and beaded with perspiration. The expression is anxious, and the pulse is weak and rapid.

Pain of Ulcer.—During the active stage of ulcer soreness upon pressure over the epigastrium is a constant finding. Upon taking food the discomfort is immediately aggravated, and localized pain occurs. The pain may radiate from the point of localized tenderness to the back, and is most intense just to the left of the spinal column and along the inner border of the scapula. In atypical cases the pain of ulcer may not develop for from one-half to one hour after food is taken.

In uncomplicated cases of gastric ulcer the pain is appreciably lessened, and oftentimes disappears after vomiting. During the course of the disease the patient may develop gastralgia, when the pain will be reflected for considerable distance over the abdomen to the back, and in severe cases down the arm.

Pain of Gastric Carcinoma.—Pain is one of the most constant symptoms of carcinoma, although it is not invariably present. In two autopsies performed by us the patients had never complained of pain during their illness. The pain of carcinoma is less definitely localized than is that of ulcer,

and may be reflected over the abdomen to the back. It is not increased immediately after the ingestion of food, but becomes most intense in from two to four hours later. Gastralgic attacks are not unknown in case of carcinoma, and vomiting does not tend to relieve the patient.

Pain of Acute Gastritis.—In this condition the pain varies in direct relation to the degree of gastric involvement present. After the ingestion of acids or of other highly irritating substances the pain is acute and is best described as an epigastric burning. If this symptom follows the too free use of rich foods, alcoholic liquors, and tobacco, the patient complains of a dull pain accompanied by nausea. In acute gastritis pain is excited by making deep pressure over the epigastrium.

Pain of Gastric Fermentation.—Hyperacidity and excessive fermentation may induce acute epigastric pain with overdistention of the stomach, but tenderness over the epigastrium is seldom present.

Epigastric Pain not Connected with the Stomach.—Cramp-like pain, either mild or intense, situated slightly to the right of the median line and radiating to the right shoulder, is highly suggestive of **hepatic colic.** In severe cases the pain may be reflected over the entire epigastrium, and rarely runs down along the right side of the abdomen, simulating the pain of appendicitis. Hepatic colic is paroxysmal in nature, the attack lasting from ten minutes to several hours, The pain of gall-stones subsides suddenly, and is usually followed by localized tenderness over the epigastrium.

Carcinoma of the common bile-duct or of the liver (with localized peritonitis) may be accompanied by a more or less constant pain, localized to the epigastrium, and radiating to the back and over the abdomen.

In *pancreatic disease* epigastric pain is one of the chief symptoms. In acute pancreatic hemorrhage the pain is sudden and intense; collapse, followed by death, soon follows.

Pancreatic colic is marked by a somewhat characteristic, cramp-like pain, localized in the epigastrium. This pain is to be distinguished from that of hepatic colic by the fact that in the former disease the patient is likely to have diarrhea and often complains of salivation.

Carcinoma of the head of the pancreas is often the cause of severe and almost continuous pain in the epigastrium. This pain may be boring or lancinating in character, depending upon the degree of peritoneal involvement present.

The development of a dull pain in the epigastrium in from two to five hours after the taking of a full meal points quite strongly toward pancreatic disease and intestinal indigestion. The pain of pancreatic disease is not continuous, as is that of gastric carcinoma.

Thoracic aneurism and duodenal carcinoma may each cause a distinct boring pain near the ensiform cartilage; and tuberculosis of the vertebra, with necrosis, is not infrequently a cause of epigastric pain.

Cardiac Palpitation.—A decided increase in the heart's action, with pulsation over the greater part of the left chest, is at times a symptom of flatulence. Palpitation may follow overeating or the ingestion of improper foods, and is also a symptom of chlorosis, secondary anemia, and hysteria. Overstimulation from the too liberal use of alcohol or narcotics (tobacco) is often the cause of palpitation.

Palpitation is a symptom of cardiac disease, and in approximating the actual significance of this symptom, it is necessary for the clinician to exclude practically all the conditions previously mentioned.

Dyspnea.—Shortness of breath often follows the too free use of rich

foods or overdistention of the stomach by gas; it is a late symptom in gastric ulcer, gastric carcinoma, and chronic gastritis, in which conditions it is dependent on the associated anemia.

Among the extragastric maladies that may cause dyspnea should be mentioned cardiac disease, nephritis, pulmonary disease, and all types of primary and secondary anemia.

Constipation.—Constipation may result from disease of the stomach, and not infrequently occurs in gastric ulcer and early during the course of gastric carcinoma. In chronic gastritis, especially when there is dilatation of the stomach, constipation is the rule. New-growths situated in the stomach-wall or pressing upon the pylorus and the duodenum are often responsible for obstinate constipation. Constipation may be a precursor of acute gastritis.

Diminished motility of the stomach favors the development of constipation. The variety of food and the amount and character of exercise taken are prominent factors in making definite deductions as to the cause of constipation.

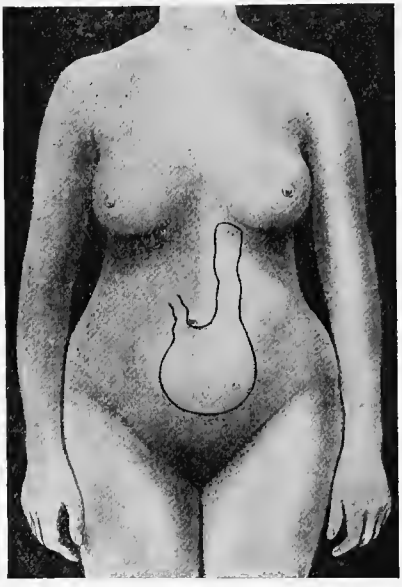


Fig. 169.—Sacculated Form of Gastric Dilatation (from clinic, Howard Hospital).

Diarrhea may develop late during the course of gastric carcinoma, gastric ulcer, and chronic gastritis, in which diseases it is probably due to gastric and intestinal fermentation. If the motor power of the stomach is excessive, food is propelled from the stomach into the intestine before gastric digestion has been completed, and diarrhea results.

Gastric neuroses and alterations in the gastric juice, *e. g.*, lack of sufficient hydrochloric acid, are followed by diarrhea. Diarrhea may also be a temporary symptom of both acute and chronic gastric catarrh. (See Diarrhea, p. 514.)

Drowsiness.—The patient may become extremely drowsy after the ingestion of a liberal meal if chronic gastritis or constipation is present. In all gastric derangements accompanied by constipation mental dullness and a tendency to sleep during the day, with insomnia at night, are among the symptoms. A marked feature of disordered sleep is that the patient sleeps equally well or better when sitting than when he assumes the recumbent posture.

PHYSICAL EXAMINATION OF THE STOMACH.

General Remarks.—Inspection.—The patient should be placed in the recumbent posture. The physician should sit so that the superior surface of the abdomen is on a level with his eyes, and then inspect the surface closely from both the right and the left side. He should then stand at the head of the bed and inspect the abdomen from the thorax to the pubes, and, lastly, he should stand at the foot of the bed and view the surface of the

body from the pubes to the thorax (Fig. 7, p. 46). These three positions should be taken in all cases when inspecting the abdomen, and if the eye is on a level with the patient's body, any abnormality that may be present will readily be detected.

Inspection with the patient in the erect posture is ofttimes of great value. (See Fig. 170.)

In persons who have a small amount of abdominal fat, and in whom the stomach is well distended, the outlines of this viscus are readily traced with the naked eye. The first thing to be observed is the lower curvature, noting particularly at what level it is seen, *e. g.*, if the greater curvature is below the umbilicus, gastric dilatation or gastroptosis is present (Fig. 172). If the lower gastric line is above the umbilicus, the stomach is not greatly dilated, and may be of normal size. An abnormal bulging in any portion of the epi-

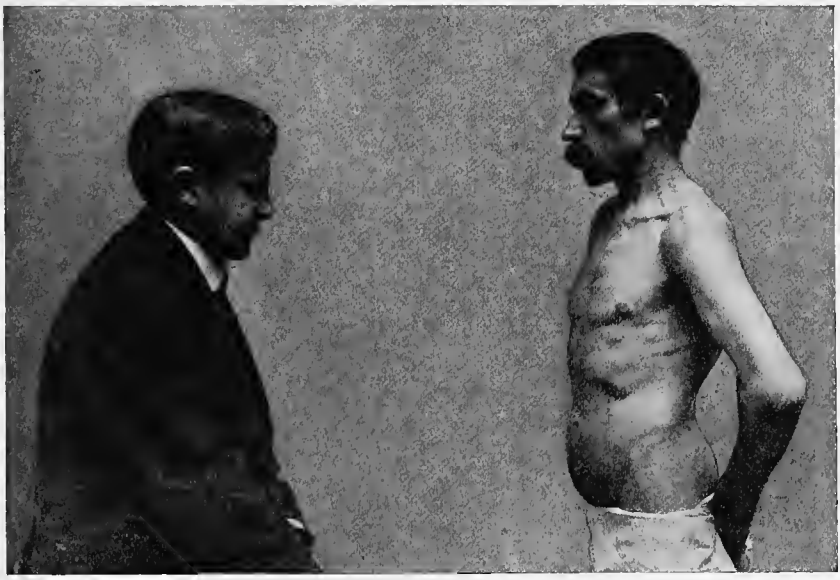


Fig. 170.—Inspection of the Chest and Abdomen. Patient standing.

gastrium, and particularly when near the ensiform cartilage, is highly suggestive of carcinoma of the pylorus. Gastric dilatation and bulging at the pylorus are two common signs of gastric carcinoma. When a dilated stomach is markedly distended, there is a distinct furrow or groove extending along the course of an imaginary line drawn from the umbilicus to the left nipple; this depression in the abdominal wall corresponds to the lesser curvature of the stomach. A furrow on the abdominal wall below the umbilicus and to the left of the pubes corresponds to the position of the greater curvature.

It will be readily understood that the physical signs of gastric dilatation just described are subject to great variation, which depends, first, upon the degree of dilatation; second, upon the rigidity or flaccidness of the abdominal wall; and, third, upon the degree of gastric distention,

Peristalsis.—Peristaltic waves are seen over the epigastrium, and cor-

respond to the movements of the stomach; these waves may be produced by applying either cold or electricity to the abdomen. Normally, the peristaltic waves (Fig. 172) should be seen in the upper portion of the epigastrium, but if there is marked gastric dilatation, they are seen to come from beneath the ribs on the left, and to extend toward the umbilicus (Fig. 172). If an abdominal tumor is present, the peristaltic waves may be accentuated over the tumor, but if the tumor is anterior to the stomach, the waves are absent at that particular point. The peristaltic waves and, in fact, the position of the stomach, are readily outlined with the naked eye when the stomach is distended by air or gas. (See Gastric Dilatation, p. 505. Also landmarks of abdomen, pp. 418–420.)

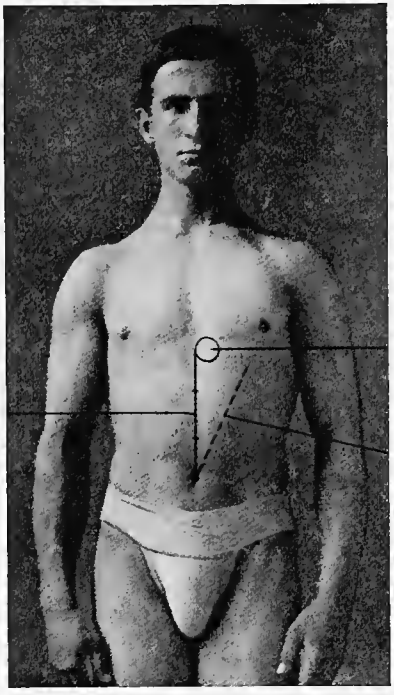


FIG. 171.—SHOWING DIVISION OF ENSIFORM AND UMBILICAL LINE.

Palpation.—The patient should preferably be in the recumbent posture, with his thighs slightly flexed upon the abdomen, in order to relax the abdominal wall. Place the hand upon the abdomen, and let all pressure be made with the fingers pressing equally along their entire length (Fig. 173). It is well to make slight rotary movement of the broad hand, increasing the pressure with each rotation, but between the acts of increased pressure the hand should not be lifted from the abdominal wall; in this way deep pressure is made without exciting contraction of the abdominal muscles.

To ascertain whether or not an abdominal mass is movable with a change of position of the patient, it is necessary to place the patient upon his side and in the knee-chest position, when, if floating abdominal tumors are present,

they will come to the abdominal parietes. Deep palpation will also determine whether or not an abdominal mass moves with respiration.

Epigastric pulsation is of great importance, not only in diseases of the stomach, but in cardiovascular derangements, *e. g.*, thoracic aneurism, abdominal aneurism, tricuspid regurgitation, and cardiac dilatation. Pulsation of the liver is usually the result of tricuspid regurgitation, but may be seen in hepatic abscess. Epigastric impulse may be transmitted as the result of a solid mass overlying the abdominal aorta. In neurasthenic persons, and in those in whom the abdominal wall is extremely thin and relaxed, a wavy pulsation of the epigastrium is quite common.

Increased resistance of the abdominal wall results physiologically from overdevelopment of the muscular coat of the abdomen, and may also be depen-

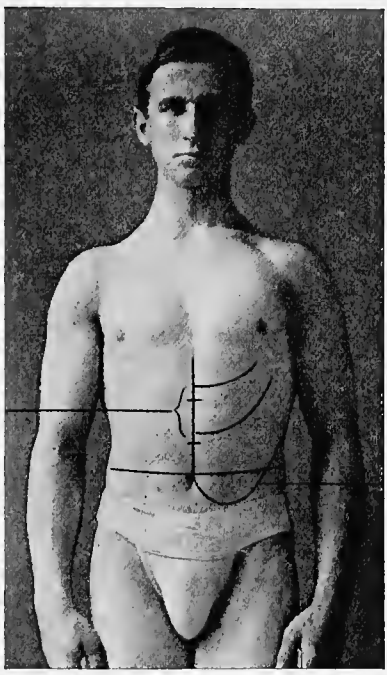


FIG. 172.—UPPER AND LOWER NORMAL WAVE OF STOMACH AND GREATER CURVATURE IN EXTREME DILATATION.

dent upon an excess of abdominal fat. Distention of the stomach is marked by a decided increase in the resistance of the upper portion of the abdominal wall, and if the stomach is diminished in size and the abdominal resistance is increased, carcinoma or tuberculosis with a localized involvement of the peritoneum is to be suspected. Increased abdominal resistance is encountered late during the course of atrophic gastritis, in ascites, and in the presence of uterine and ovarian growths. A localized increase in the resistance of the wall of the abdomen, when referred to disease of the stomach, usually occupies the upper right portion of the epigastrium, and is suggestive of pyloric carcinoma.

Tumors of the stomach, liver, spleen, pancreas, and peritoneum may cause

an increased resistance over the epigastrium. Tumor of the stomach is the only one of the previously named group that is capable of descending *en masse* as it develops; *e. g.*, carcinoma involving the pylorus may be found as low as the umbilicus, and rarely it is seen below this point. The degree of descent of a carcinoma of the stomach-wall depends upon the associated peritonitis,

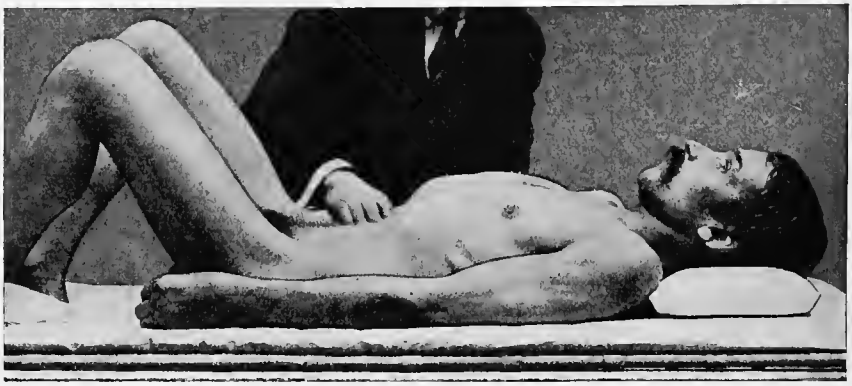


FIG. 173.—POSITION OF PATIENT AND OF OPERATOR FOR ABDOMINAL PALPATION.

and upon whether or not adhesions to other epigastric structures exist. If a carcinoma of the stomach is adherent to the diaphragm, the liver, or the spleen, it will display a variable amount of movement with respiration.

Combined palpation of the stomach with an internal exploratory exami-

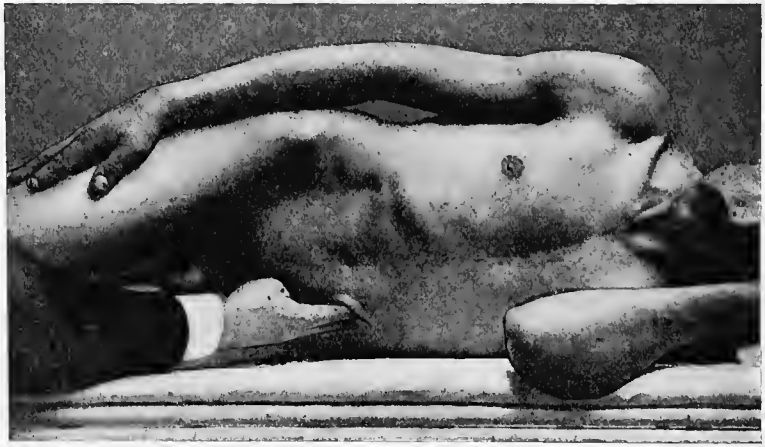


FIG. 174.—ABDOMINAL PALPATION TO DETECT CHANGE OF POSITION OF ABDOMINAL GROWTHS WITH THE CHANGE OF POSITION OF THE PATIENT.

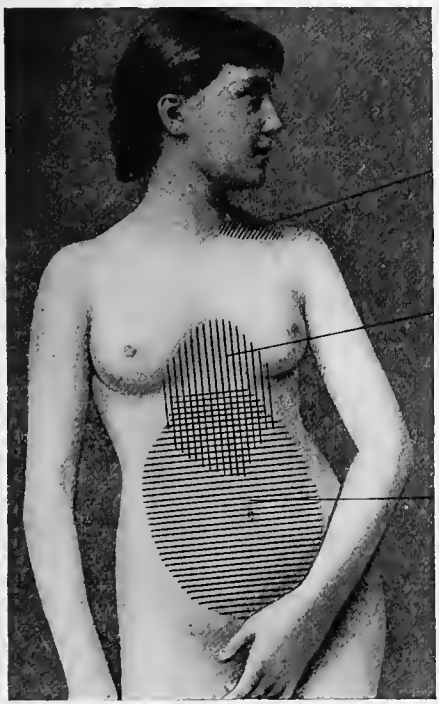
nation may be resorted to in order to determine the actual size of the organ, but this measure is rarely necessary.

Palpation and Pain.—Tenderness over the epigastrium is found in acute gastritis, gastric ulcer, gastric and pancreatic carcinoma, malignant disease of the common bile-duct, acute hepatitis, pancreatic calculus, hepatic

colic, and in any condition that may have associated localized peritonitis. It is impossible for one to attach too great diagnostic importance to epigastric tenderness or to pain that is excited by deep palpation.

In gastric ulcer the tenderness is usually localized, whereas in practically all other conditions in which epigastric tenderness is one of the symptoms it is less markedly localized in direct proportion to the extent of acute or chronic peritonitis present.

Percussion.—Normal Position of the Stomach in the Adult.—The position of the stomach will be found to vary greatly within certain limits. These variations, as they occur during infancy, childhood, and in early adolescence, are shown in the accompanying illustrations (Figs. 176, 177, 178). When food is taken the stomach changes its position, and the greater curvature is rotated forward and slightly upward.



Involvement of the diaphragm, transverse colon, and splenic and hepatic flexure.

Vertical lines outline area of gastric pain. Carcinoma, ulcer, gastralgia, neuroses, acute gastric catarrh, hyperesthesia, duodenal ulcer. Pancreatic disease causes pain near the center of this area, as do also pneumonia in children and disease of the vertebræ.

Abdominal pain. Where the vertical lines cross the transverse lines the pain is excited by conditions named above. Over area of transverse line lead colic, mucous colic, flatulency, Dietl's crises (floating kidney), appendicitis (initial stage), intestinal obstruction, intestinal perforation, mercury poisoning, crises of locomotor ataxia, pneumonia (in children), strangulated hernia, hyperesthesia, rheumatism of the abdominal wall, and rarely Löebstein's carcinoma, abdominal aneurism. Raynaud's disease, and acute pancreatitis.

FIG. 175.—DISTRIBUTION OF PAIN.

For convenience of study Obrastzow's division of that portion of the abdomen between the ensiform cartilage and the umbilicus is to be recommended. An imaginary line extending from the ensiform to the umbilicus is divided into three equal parts. In normal men and women the stomach will be found between the umbilicus and the ensiform, and the lower border of the stomach will usually correspond to the inferior third of this line.

The upper border of the stomach is taken at the left parasternal line; at this point it is normally found at the lower border of the fifth or upper border of the sixth rib, although instances are recorded in which the superior boundary of the stomach corresponded to the fourth rib and to the sixth inter-

costal space respectively. The upper border of the stomach is a trifle lower at the left nipple-line than at the parasternal line, but is usually found at the fifth interspace or the sixth rib. In the anterior axillary line the superior boundary of the stomach corresponds to the seventh interspace.

Traube's semilunar space is that portion of the left chest overlying the stomach. It is bounded above by the lower border of the heart, the left lung, and the liver; below by the margin of the ribs, and to the left by the spleen.

Tympany.—In outlining the stomach by percussion it is most satisfactory to have the patient in the recumbent posture, with his thighs at right angles to the body, in order to relax the abdominal wall. In infancy the stomach is almost cylindric in outline, and occupies an oblique position, but at the age of one year it will be found in a transverse position (Fig. 176). It is of great importance to ascertain the change in shape of the stomach when the patient suffers from gastric dilatation.

Percussion-note.—As the stomach always contains some air, a tympanitic note is obtained by percussing over this hollow viscus, and while this note varies greatly, depending upon the degree of distention of the stomach by gas, etc., it is always elevated in pitch, but lower than that of the colon, metallic in character, and displays a certain distinctive quality known as "stomach tympany."

Auscultatory percussion (Fig. 179) may be employed in determining the exact outline of the stomach, although this diagnostic measure is of far greater value for outlining the solid abdominal viscera and new-growths.

Conditions That May Increase the Area of Stomach Tympany.—
A. Those depending upon alterations in the size and shape of the stomach.
B. Pathologic conditions not directly connected with the stomach.

A.

1. Overeating.
2. Gastroptosis.
3. Dilatation of the stomach.
4. Starvation.
5. Cardiac stenosis.

B.

1. Fibroid induration of the left lung.
2. Adhesive pleurisy (left side).
3. Contraction of the liver.

The area of stomach tympany is diminished in—

1. Left-sided pleural effusion.
2. Fibroid induration of the stomach-wall.
3. Hour-glass contraction.
4. Left pyopneumothorax.
5. Enlargement of the liver.
6. Enlargement of the spleen.
7. New-growths of the mediastinum.
8. Abdominal tumors, *e. g.*, ovarian cyst, uterine fibroid, carcinoma of the left kidney, echinococcus cyst, pregnancy, and ascites.

Auscultation.—Auscultation is of value in determining whether or not stenosis of the esophagus or disease of the stomach exists. (See Esophageal Stricture, p. 416.) The gurgling sound, audible when the patient swallows liquids, and heard only by placing the stethoscope over the esophagus, is always followed, in from five to ten seconds later, by a second sound, which is caused by the escape of the fluid from the esophagus into the stomach. This second sound is usually referred to as the "deglutition gurgle," or murmur. If there is obstruction to the cardiac portion of the esophagus, the "deglutition gurgle" takes place later, and is materially modified.

By placing the ear over the stomach, either anteriorly or at the back, it is possible to get a decided splashing sound when the patient is shaken

rapidly from side to side. The presence of the succussion splash is positive evidence that the stomach is partially filled with liquid, but it must be remembered that the splash alone is to be taken only as corroborative evidence of other physical signs and symptoms.

The succussion splash may be elicited by external manipulation of the abdominal wall; this is accomplished by placing the left hand over the pylorus and the right hand just at the costal margin in the anterior axillary line, when, by alternating pressure at these points, the succussion splash is produced. Percussion along the margin of the ribs and over the left superior abdominal quadrant is often sufficient to cause a splashing sound.

The succussion splash over the abdomen is at times very loud, and may be heard for some distance from the patient, although the best means of eliciting this sound is for the operator to place his ear against either the abdomen or the back.

Significance.—Under normal conditions, when 100 cubic centimeters of water are taken before retiring, the succussion splash cannot be elicited in the morning. If the succussion splash is present under such conditions, an atonic condition of the stomach-wall exists. The succussion splash should be audible during digestion, and the sounds are of pathologic significance when they are present three hours after the ingestion of an ordinary meal. If the splash is present five to seven hours after liquid food has been taken, it is positive evidence that there is gastric dilatation or defective gastric motility.

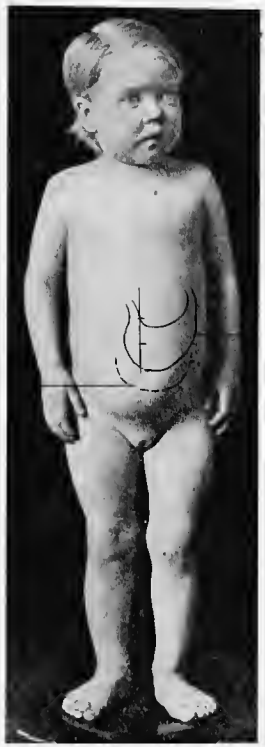


Fig. 176.—Lower Normal Position of Stomach in Child.

Under normal conditions the splash is heard above the umbilicus. In dilatation of the stomach and in gastroptosis it is audible below the umbilicus. In children who are poorly nourished a splashing sound is also audible below the transverse umbilical line. The exact location at which the succussion splash is heard is of value in determining the lower boundary of the stomach, and this is best attained after inflating the organ.

Caution.—Great care should be exercised in differentiating the succussion splash caused by fluid in the stomach from that the result of air and fluid in the left pleural sac. (See Pyopneumothorax, p. 166.)

GASTROSCOPY.

Definition.—Gastroscopy is a clinical method of inspecting the interior of the stomach by means of tubes that serve as specula. Ordinarily, the natural passage (esophagus) is chosen for conducting this method of examination, although occasionally abdominal wounds and fistulæ are utilized.

Instruments for Gastroscopy.—The Gastroscope.—In order to examine the stomach, an 80 cm. tube is frequently required, although in many cases a 70 cm. by 10 mm. one is sufficient. It is impossible to illuminate well the field of view by any form of light projected through the proximal

FIG. 177.—SHOWING HIGH NORMAL POSITION OF STOMACH IN CHILD.

FIG. 178.—LATERAL AREA OF STOMACH TYMPANY IN THE CHILD.

end, since there is too great a loss for practical work. With Jackson's gastroscope (Fig. 180) the length of the tube is immaterial—the view is as good at the end of an 80 cm. tube as is that of a 45 cm. esophagoscope of the same diameter.

The tubes are illuminated by a small "cold" lamp carried down to the extremity by a light-carrier. The chief advantages of this form of illumination are:

The light being in the tube, the object is always illuminated, regardless of the movements of the patient or of the operator. The lamp may occa-

sionally become smeared with blood, which necessitates withdrawing the light-carrier and cleaning the lamp.

The illumination is as powerful and the view as clear through an 80 cm. gastroscope as through a shorter tube.

A great advantage is gained by oblique light, due to the location of the lamp at one side of the orifice of the tube. "The darker shades of red do not throw back rays strongly, so that in case of a long tube, with the light at one end and a dark-red object at the other, the light traveling twice the tubal length, the greatest skill and the utmost perfection of every detail of apparatus are absolutely essential for results" (Jackson).

A small object or an instrument introduced into the tube does not cut off any light, as the light is beyond the instrument.

Each tube is fitted with a handle, which is a time-saver as compared to attaching a handle each time a different tube is inserted.

FIG. 179.—METHOD OF OUTLINING THE STOMACH BY AUSCULTATORY PERCUSSION.

"The tubes for general use are fitted with an auxiliary drainage canal which maintains a dry, clean condition at the distal end of the tube. Occasionally a tube is needed without this auxiliary drainage, but only rarely, as in the case of passing a narrow stricture." The secretion is removed by pressure maintained in a bottle, by aspirating syringe, the bottle being connected at intervals with the outer end of the drainage canal. If the drainage canal becomes obstructed, an extra drainage-tube may be inserted and removed as needed. The extra drainage-tubes are especially useful for blowing in medicaments or bismuth for Roentgen-ray localization. A number of accessory instruments have been devised, and for a description of these the reader is referred to Jackson's monograph.

In the wall of the gastroscope, as in the esophagoscope, there are two small auxiliary tubes or canals. Both of these canals open into the main tube, close to the distal end. One canal ends near the handle, in a tip for the

attachment of rubber tubing connected with the aspirating apparatus previously referred to, and this keeps the field clear of all fluids and prevents smearing of the lamp. In some cases quantities of fluid must be pumped out of the stomach.

The other canal is for the light-carrier, already alluded to, which is a small, removable, double conductor, carrying the lamp to the distal end of the instrument, where it sheds its light at close range at the point where needed, leaving every object between it and the observer's eye in darkness.

The exterior of the tube is not graduated, and the depth is to be measured with a sterilized steel rule by noting the distance between the proximal (outer) end of the tube and the upper teeth,

An obturator or mandrin with a projecting conic end is fitted to facilitate the passing of the inferior pharyngeal constrictor, especially for those unfamiliar with esophageal work.

Technic of Gastroscopy.—In order to introduce the gastroscope with any degree of ease and safety to the patient, complete anesthesia is essential. Certain writers recommend that, in heroic patients, local anesthesia, by means of cocain, may be used, but in no case does cocain prevent retching and spasm of the diaphragm, which temporarily interrupts the examination. Ether anesthesia has been employed extensively by Jackson and others, and it is suggested that chloroform should be an ideal anesthetic in this particular work, although it does not seem to have been extensively

Fig. 180.—Chevalier Jackson's Bronchoscope, Esophagoscope, and Gastroscope.

employed. Irrespective of which anesthetic is selected, morphin administered hypodermically will be found of assistance.

Preparation of the Patient.—It is desirable to have the gastro-intestinal tract as nearly empty as possible, and no solid or semisolid food should be allowed for at least twelve hours before the gastroscope is introduced. When the gastroscope is to be introduced in patients having no evidence of pyloric stenosis, liquids, *e. g.*, black coffee, may be allowed by mouth eight hours before the introduction of the instrument.

Position of the Patient.—Place the patient upon an ordinary operating table, and lower his feet approximately fifteen inches. This position will be found to assist the operator greatly, and the only objection to it is that the aspirating bottle must be attached to the instrument in order that any excess of fluid may be drained through the gastroscope. After the instrument has been passed into the stomach, the entire plane of the table is again changed, so that the head is about 30 cm. higher than the feet.

Passing of the Gastroscope.—The operator must be gentle in all his movements, and if the tube does not pass readily, it is not in the right position, not rightly directed, or not sufficiently well lubricated with vaselin. The proximal end of the instrument should be held lightly between the fingers of the right hand, and the handle directed horizontally to the right. (See Fig. 181.) The forefinger of the left hand should be introduced into the right glosso-epiglottic fossa posteriorly to the lateral glosso-epiglottic fold, and,

if possible, into the right pyriform sinus. Guided by the finger the instrument is forced to follow the same route, and naturally glides toward the median line. The cricoid cartilage may be reached in children, and pressure made directly upon it. (See Fig. 182.) In the adult traction is made upon the tissues in the right epiglottic fossa. Certain authors recommend passing the esophagoscope by threading it over an esophageal bougie.

Position of the Patient's Head.—The neck of the patient is bent backward (Fig. 183) in order to straighten the cervical curvature, or rather to bring the axis of the oral cavity to that approximately parallel with the esophagus, and it is also possible, with the head in this position, to pass the superior teeth more readily. When bending the neck of the patient, the angle should be as much as possible at the upper cervical vertebræ, thus straightening the oropharyngeal angle. If the position of the patient's head appears to cause oppression, it may be found necessary to elevate it. The exact position of the patient's head is one of the most difficult features in the introduction of the gastroscope.

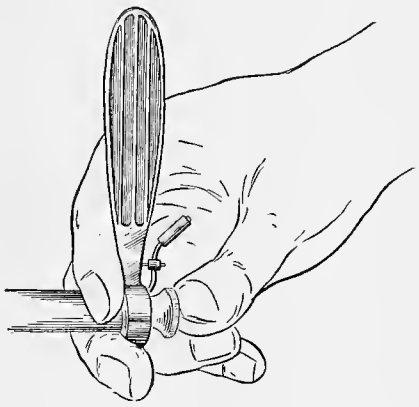


Fig. 181.—Position of the Right Hand for Introduction of the Gastroscope. View from Above by the Operator Looking Downward (Jackson).

Directions for Assistant.—In all this work safety demands that the mouth, pharynx, and esophagus be brought into a straight line, not by a leverage-like action of the instrument, but by holding the head steadily in extreme extension with the mouth widely open. Not only does lateral pressure add to the operator's difficulty, but it also prevents him from

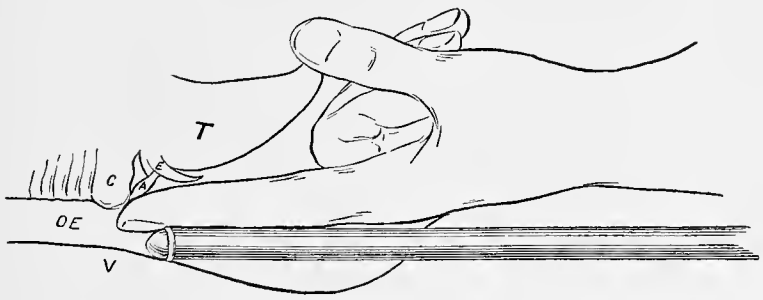


Fig. 182.—Position of the Left Hand in Starting the Gastroscope or Esophagoscope (Jackson).

determining what the point of the instrument is touching. No mouth-gag is always self-retaining, and a slight slip while the tube is in position may have serious consequences. Therefore, a second assistant should be detailed to hold the head and steady the mouth-gag. In order that the assistant be out of the operator's way, it is necessary that he shall hold the head at arm's length. (See Fig. 183.) The patient is drawn forward

until the tops of his shoulders clear the table four to six inches, and the mouth-gag is inserted on the left side. The assistant's left foot is supported on a stool (Fig. 183), 26 inches lower than the top of the table; his right forearm is passed beneath the patient's neck, supporting it; his right hand grasps the mouth-gag, drawing it strongly backward. "His left hand rests on the left knee, grasps the head strongly at or in front of the bregma, bending it backward and exerting a certain degree of upward pressure. The exact proportion of backward and upward pressure cannot be described, but is readily appreciated on trial, especially if the assistant has actually experienced the difference in sensation when the hand hangs free and when it is properly supported in extreme extension" (Boyce).

FIG. 183.—POSITION OF PATIENT AND SECOND ASSISTANT FOR BRONCHOSCOPY (Jackson).

After the introitus is passed, the obturator is removed, the cord is attached to the light-carrier by the bayonet fitting, which, by rotation, is used as a switch to turn the current on and off. The rheostat on the battery is to be regulated to full illumination when the instruments are prepared. Turning the bayonet fitting lights the instrument, and the passing from this point is under the guidance of the eye. The sense of touch is used to note resistance, which, if felt, means that such resistance must be overcome by skill, since the application of force is dangerous. Once started, the passage of the instrument down the esophagus is comparatively easy if the following important points are observed:

(*a*) The instrument must have been well lubricated before starting.

(*b*) The tube must be guided by the eye, so as to follow the esophageal lumen.

(*c*) The pinching of the tube by the teeth must be avoided, in order that the tube shall be free to move as needed to follow the axis of the esophageal canal as it is seen.

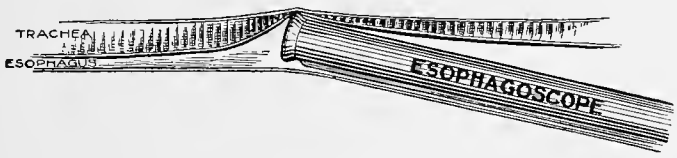


FIG. 184.—DIAGRAM SHOWING OCCLUSION OF THE TRACHEA BY FAULTY DIRECTION OF THE GASTROSCOPE (Jackson).

After passing the introitus, care must be taken to raise the head of the patient slightly to prevent the tube pressing upon the trachea (Fig. 184). This is readily noticed if the passing is done by sight.

In finding the lumen, the normal respiratory movements are of great assistance. The way often seems to be completely blocked ahead by what appears to be the esophageal wall, but with the next inspiration a lumen appears in one or other quadrant of the tube, where a few bubbles are seen.

"The introitus passed, only two points will give any trouble. The first is at the hiatus diaphragmaticus, the second, the bend of the abdominal esophagus to the left. The hiatus is passed by placing the long axis of the elliptic cross-section of the tube from the right posteriorly forward toward the left anteriorly. This is easily done by placing the handle of the gastroscope in the direction of the visual axis of the patient, if he were looking forward (if erect) to the left (see Fig. 157)" (Jackson).

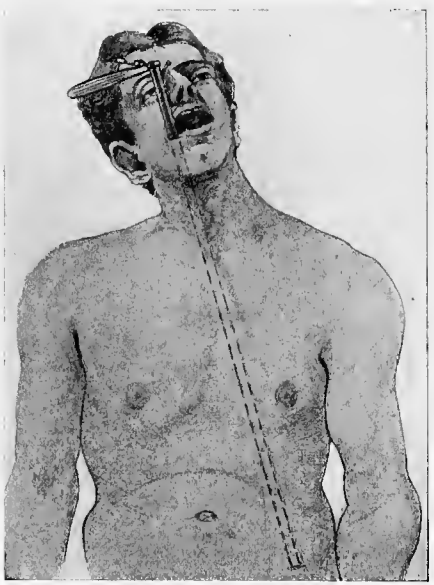


FIG. 185.—HEAD AND NECK MOVED TO THE RIGHT TO REACH THE LEFT LIMIT OF THE EXPLORABLE AREA (Jackson).

Passage of the abdominal esophagus is facilitated by moving the head to the right (Fig. 185), and the lumen is carefully watched and followed. If serious difficulty is experienced in passing the hiatus, it will be found that anesthesia is not complete. After the tube has entered the stomach a systematic plan of exploration is to be followed. An area of from one to six centimeters of gastric mucosa is visible at one time, so that a systematic plan of tube travel brings all portions of the explorable mucous membrane into view.

"There are two plans of exploration, both of which should be carried out. First, the gastroscope should be passed down carefully and gently to the greater curvature, inspecting the anterior and posterior walls. At times these walls do not seem to be fully collapsed ahead of the tube, and one will have to be examined first, then the other. Then the tube is withdrawn, inclined slightly laterally in the same plane, then pushed gently downward again in a new series of folds. This is repeated until the extreme pyloric limit is reached. To reach this limit, the head and neck of the patient are moved to the left, with the tube below the cardia.

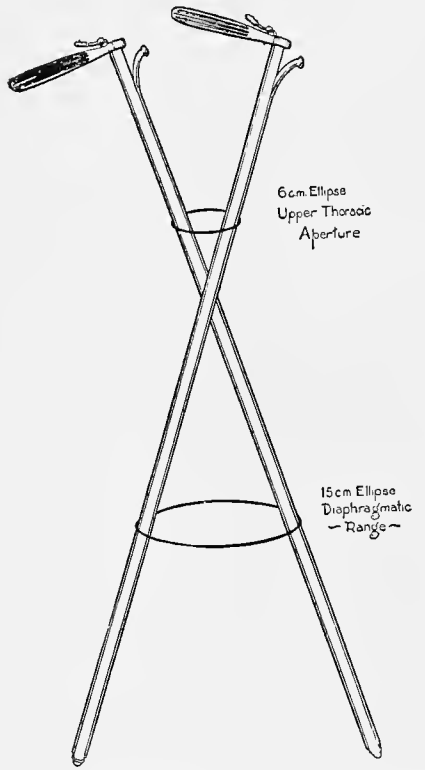


Fig. 186.—Showing Extreme Right and Left Positions of the Gastroscope (Jackson).

"After the whole possible range has been covered in this way, we proceed to the second plan. The tube is passed down until the extremity touches the wall of the greater curvature in the extreme left of the possible field. Then the tube is moved slowly along the greater curvature, but not in close contact therewith, until the extreme right is reached. Withdrawing the tube a centimeter or two, the field is slowly swept again in the same plane, but at a higher level, and so on upward to the cardia. Next the deft fingers of one skilled in abdominal palpation are called upon to manipulate the unexplored portions over in front of the tube. This is sometimes better accomplished by turning the patient upon the side."

Caution.—During the process of turning the patient from side to side the tube must be withdrawn within the esophagus. When the stomach is in its new position, the gastroscope is again pushed downward, and the newly available surfaces are explored. If retching supervenes while the gastroscope is in the esophagus, no harm will result, but when the tube is in the stomach, retching is the signal for immediate withdrawal of the instrument until the distal end is above the diaphragm.

The tendency is for the gastric walls to be dragged along with the tube when the tube is moved, but if the tube is withdrawn slightly before changing its position, the tube then changed, and the instrument reinserted to its original depth within the stomach, the gastric wall will have assumed its normal position and a new field is brought into view.

Explorable Area of Stomach.—This varies with the position of the stomach—the more horizontally the organ is placed, the less of its surface can be viewed by the gastroscope (one-third to one-half). The degree of anesthesia is also an important factor in governing the extent of the explora-

DESCRIPTION OF PLATES IX AND X.

GASTROSCOPIC VIEWS (CHEVALIER JACKSON).

1.—Thoracic esophagus. Expiration. Note lumen not entirely closed. Man aged 40.

2, 3, 4, 5, and 6.—Normal stomach. Folds in various positions as seen separating and collapsing ahead of the tube as it is inserted and withdrawn. In Fig. 4 is shown a horseshoe-shaped position of a fold often seen near the cardia, usually to the right. At times seen elsewhere. Compare Figs. 16 and 18, Plate X.

7 and 8.—Stomach. Normal wall of inferior curvature flattened by pressure of the tube-mouth.

9.—Gastritis. Fold in lower right-hand corner is capped by secretion simulating ulcer, before being wiped away.

10.—Gastritis. All folds sponged but one, which shows thick tenacious secretion.

11.—Gastric ulcer seen on edge. Not sponged. Man aged 32. Patient of Dr. Finkelpearl.

12.—Same patient. Scar after healing of the ulcer. Scar shows yellow by engraver's error; it should be grayish, nearly white.

13.—Carcinoma of cardia. Infiltrated, but not ulcerated, hard mass to right of view. Man 38 years. Referred by Dr. Haworth.

14.—Same patient. Further to right than Fig. 13, on lesser curvature. Fungating portion of mass.

15.—Carcinoma of pylorus. Left border of the tumor. Man of 44 years. Referred by Dr. Haworth.

16.—Normal stomach. Three cm. below the cardia. Note horseshoe-shaped fold to the right. Maid of 19 years. Patient of Dr. Lichty.

17.—Normal stomach, farther down, same patient. (Views are never twice alike, no form is meant as typical of locality.)

18.—Normal stomach. Four cm. from cardia. Woman of 33 years.

19.—Normal stomach. Woman of 19 years. Showing diversified forms of folds.

20.—Normal stomach. Transverse trend of folds as greater curvature is approached. Folds are rarely seen as straight as the central one in this view.

21.—Approaching the pylorus. Gastroptotic stomach. (View probably not abnormal.) Woman of 33 years. Referred by Dr. Dranga.

22.—Gastroptotic stomach. (Same patient.) Pylorus hidden by folds.

23.—Folds at fundus (not typical).

24.—Approaching the pylorus. Folds disturbed by tube-mouth. Same patient as Fig. 21.

25.—Same patient, same location, about one minute later.

26.—Same patient, same location, about one minute later. Annular folds of pylorus surrounding prolapsed duodenal folds. Brownish fluid was regurgitated into stomach.

27.—Cancerous (?) infiltration near pylorus. Fluid exuded from triangular slit. Woman aged 26 years. Refarred by Dr. Montgomery.

28.—Gastric ulcer filled with secretion, and seen on edge. Man aged 59 years. Referred by Dr. Goldsmith.

29.—Same ulcer wiped clean. Looking into bed of ulcer.

30.—Cicatrix (?) of stomach, in a man 59 years of age who had a specific history.

31.—Carcinoma of esophagus. Man of 60 years. Referred by Dr. Sanes.

32.—Carcinoma (?) of pylorus. View not at but near the pylorus on greater curvature. Color should be much darker Woman aged 26 years. Referred by Dr. Montgomery.

33.—Carcinoma of pylorus. View at left border. Other portions of growth were spotted with dark brown. Man 46 years of age. Referred by Dr. Walton. Afterward operated upon by Dr. MacClelland, and diagnosis as to size, shape, position, and nature verified.

34.—Another portion of same growth. Mucosa normal, but foldless and hard. (Right in view.)

35.—Cardiospasm. Abdominal esophagismus. Man aged 59 years. Referred by Dr. Goldsmith.

PLATE IX

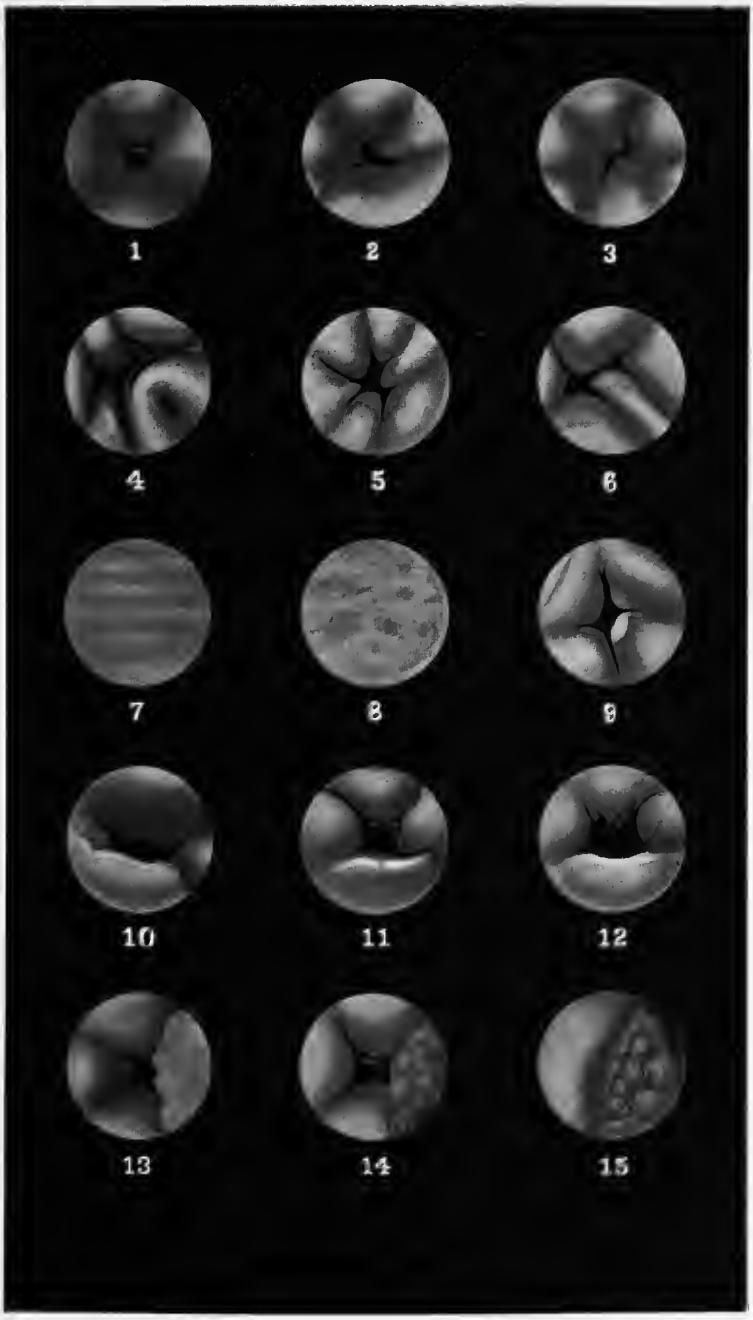
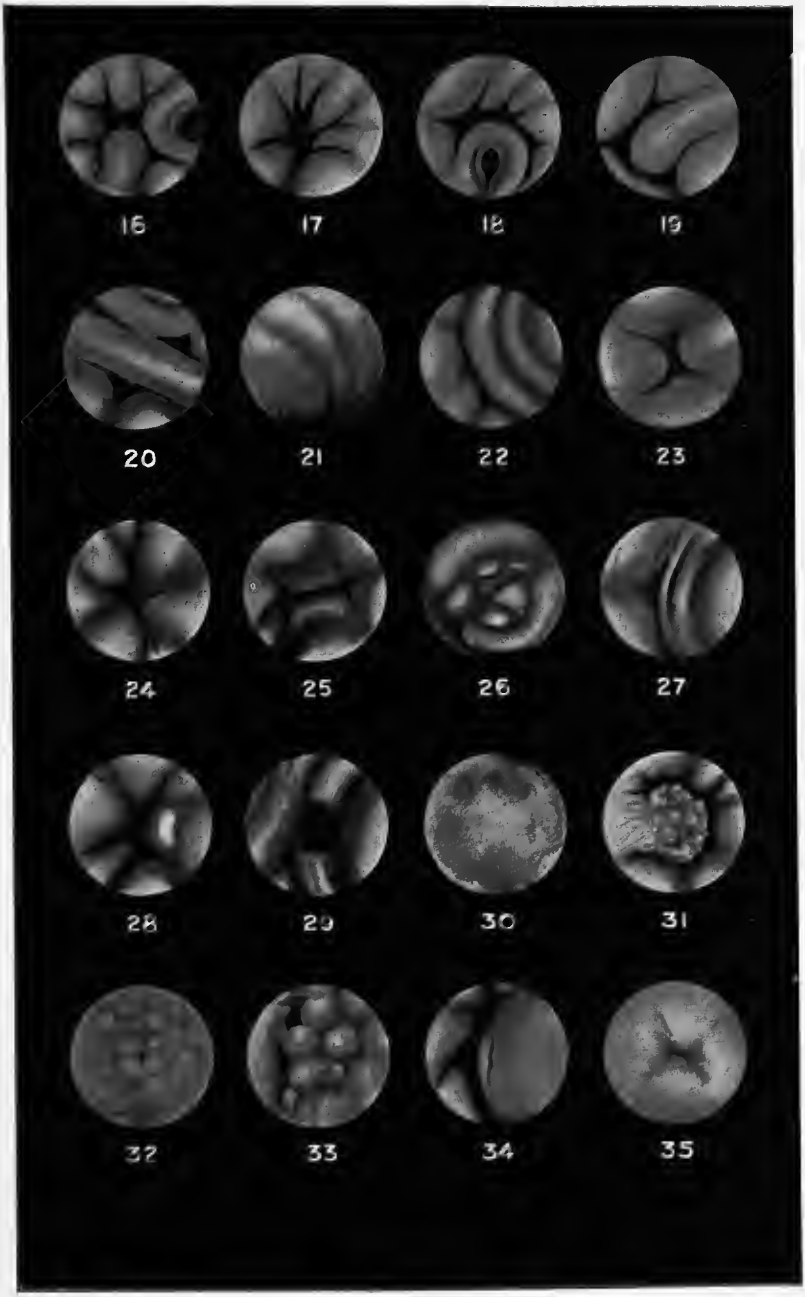


PLATE X



ble area, absolute anesthesia increasing this area materially. A full range of the upper thoracic aperture is available by shifting the head and neck laterally, and Fig. 186 will also explain the results to be obtained by varying the position of the instrument. Moderate anterior and posterior rotation of the patient's head and of the instrument will be found to facilitate the bringing of both the anterior and the posterior walls into full view. Unless the caliber of the esophagus is normal, difficulties are encountered, all of which materially lessen the explorable area of the stomach.

Gastroscopy should not be regarded as an unusually difficult procedure, although considerable skill is necessary in order to attain the best results. The training necessary successfully to use the gastroscope has been compared to that needed for mastery of the ophthalmoscope. The eye soon becomes educated, and if the directions previously given for both patient and assistant are followed closely, but few difficulties will arise.

Contraindications.—Pott's disease and other pathologic changes of the vertebræ may render gastroscopy impossible, and, indeed, are contraindications to the use of the instrument. Gastroscopy is also attended with great danger when there is extensive atheroma of the blood-vessels, and especially when aneurism exists. In our opinion, the procedure is also contraindicated in all pulmonary conditions, for the following reasons:

(1) Prolonged anesthesia is in itself dangerous.

(2) Because extensive adhesions to the esophagus and other mediastinal structures may follow pulmonary disease. Maladies characterized by dyspnea would also contraindicate the use of the gastroscope.

In the profound cachexia accompanying malignant and other chronic maladies the instrument should not be used, because here the percentage of hemoglobin is already low, and at least 20 per cent. or more of this vital substance would be lost as the result of profound anesthesia. Those who have suffered at some earlier date from luetic infection take ether badly; in such cases some risk would attend the introduction of the gastroscope. Valvular heart disease in which any of the symptoms of failure of compensation are present should also be considered.

Gastroscopic Findings.—The accompanying illustrations (Plates IX and X) were prepared by Dr. Chevalier Jackson, who, in addition, suggests that these should not be regarded as the average appearance, but are illustrative of individual cases. The amount of gastroscopic work done is not sufficient to warrant us in drawing any definite deductions as to what represents an average finding in any given type of disease.

X-RAY EVIDENCE OF DISEASES OF THE GASTRO-INTESTINAL TRACT.

By George E. Pfahler, M.D.

It is difficult to cover this subject thoroughly and yet briefly. In general, the gastro-intestinal tract is rendered visible by means of bismuth mixtures, which give an added density of shadow to this canal. The bismuth subcarbonate may be mixed with water, milk, kefir, gruels, or other food (1 oz. to 12 oz. of kefir). Both fluoroscopic and photographic studies are necessary. The fluoroscopic work is unfortunately dangerous to the operator.

One must be familiar with the normal stomach and the variations under normal conditions before much of value can be determined pathologically.

The *filling of the normal stomach* in the standing posture takes place as

follows: the upper pole is outlined usually by a small collection of gas, just beneath the left dome of the diaphragm. The remainder of the stomach is collapsed. Along the right border of the collection of gas the bismuth mixture may be seen entering and giving the appearance of a dark streak. This collects below the gas, making a funnel-shaped shadow. One or two swallows may be retained here for several minutes. Gradually this funnel-like shadow becomes elongated until it reaches the lower pole, curving slightly to the right. At the lower pole the shadow is broadened with the

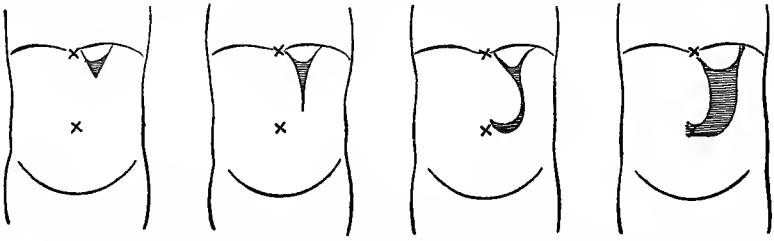
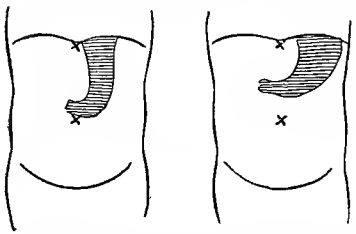


FIG. 187.—SHOWS THE APPEARANCE OF THE STOMACH AS THE FOOD GRADUALLY EXTENDS TO THE LOWER POLE AND THEN FILLS THE ENTIRE STOMACH.
With any further increase the shadow simply increases in area.

accumulation of food, and crosses to, or beyond, the median line, perhaps rising slightly to reach the pylorus (Fig. 187).

The form of the normal stomach will vary with the position, sex, stature, and degree of compression. In the standing posture it is elongated, and resembles the form of the letter "J." In the dorsal recumbent posture it takes more the shape of a cow's horn. It is longer in women than in men. It seems to be longer in women after wearing corsets. It is longer in the tall, thin person, and more vertical than in the short, stout individual.

The lower border of the pyloric sphincter in the standing posture is nearly on a level with the lower pole of the stomach, and is located about an inch above the umbilicus. The normal form of the stomach is found in only about one-third of the healthy subjects examined. This departure from the normal occurs and increases with the age beyond puberty. The lower pole moves downward and is more distant from the pylorus. This is probably due to an increasing relaxation of the tissues and to overfilling the stomach, which drags it downward.



Standing. Lying on back.

FIG. 188.—SHOWS THE CHANGE IN FORM AND POSITION OF THE STOMACH, WITH CHANGE FROM THE ERECT TO THE SUPINE POSTURE.

The **position of the normal stomach** is influenced by so many factors that all must be considered, or mistakes will be made. One must keep in mind the fact that the stomach is attached only at the cardiac orifice, and just beyond the pylorus, and therefore it is a very movable organ. It is supported in part by the abdominal wall and the bed of intestines below. Therefore it will change its position with change in posture, and with modification in the pressure of the abdominal wall, as well as with the movements of the diaphragm during inspiration.

In the *standing posture* the lower pole of the normal stomach will be slightly above the umbilicus, while in the supine, the stomach rises several inches, the shadow of the fundus increases and occupies the left hypochon-

driac region; and the stomach as a whole takes a more transverse position, resembling the classic outline given in most of the old text-books (Fig. 188).

If the subject lies upon the right side, one-half to two-thirds of the stomach will cross the median line.

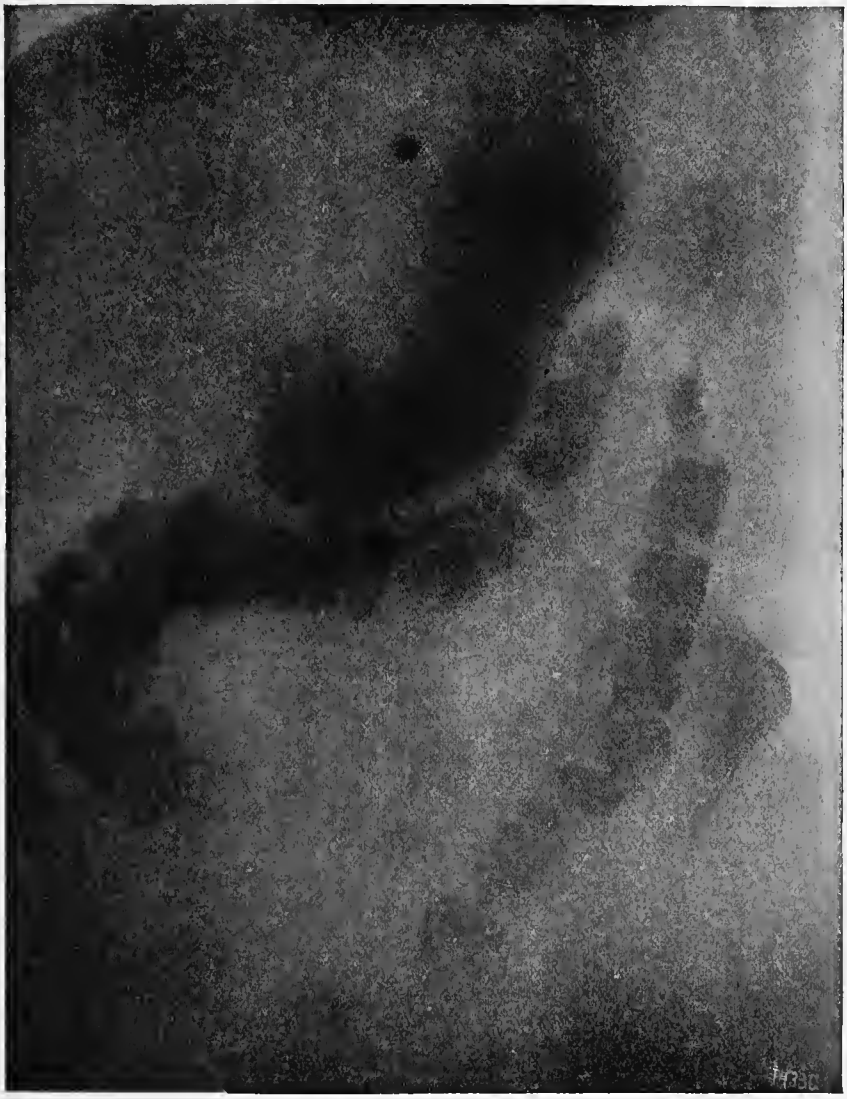


Fig. 189.—Normal Stomach and Bowels in an Adult Fifty Years of Age (Pfahler).

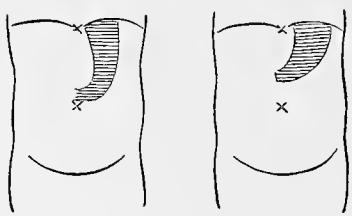
If a person contracts the abdomen, the stomach will be raised several inches. Pressure on the lower part of the abdomen will cause it to rise. The stomach can be moved from side to side by pressure, and even the contents

can be moved about by the pressure of the hand through the abdominal wall. So, too, the pressure of the corset may contract the central part of the stomach, and crowd the lower pole downward.

The *peristaltic contractions* are probably of three different varieties, but only one really concerns us at present clinically. These can be seen to begin at about the junction of the middle and lower third of the stomach by an indentation in the lower border, and are less clearly seen directly opposite, on the lesser curvature. These increase in depth as they move toward the pylorus, varying from one-half to one inch, and at times almost bisecting the stomach-contents. The pylorus itself is indicated at intervals by a clear line which separates the bismuth mixture in the stomach from that which has just passed into the first portion of the duodenum. The peristaltic waves are excited by the presence of food or by massage. The wave requires usually from fifteen to twenty seconds to pass from its origin to the pylorus.

The stomach should empty itself of the normal bismuth meal in from three to four hours. If retained beyond this time, it suggests a decrease in motility.

Preparation of the Patient for a Gastro-intestinal Examination.—Generally the patient should be given a purgative on the night preceding the examination, preferably a bottle of magnesium citrate. It is then best to begin the examination on the following morning with the stomach empty. This is especially important if carcinoma is suspected, for the presence of any food in the stomach may lead to error. Hard fecal masses in the bowels may likewise be misleading. If the examination is begun in the morning, one has the day before him for control observations. In obscure cases, several examinations must be made. In general these examinations are time-consuming and expensive, but unless carefully done, they had better be omitted.



Normally relaxed stomach. Contracted stomach.

FIG. 190.

When simply the positions of the stomach and bowel are to be determined, one can give the bismuth with the breakfast on the preceding morning. This will then be found in the bowel (no purgative to be given in such cases), and at least one examination less will be necessary.

PATHOLOGIC SIGNS.

Disturbance in Motility.—Any condition that will depress the nervous system is likely to be associated with impaired motility and retention of food beyond the normal period. One is likely to find delay in neurasthenia of the depressed variety, in gastroptosis, and in pyloric stenosis. In simple gastroptosis I have seen food retained in the stomach at least eleven hours. In pyloric stenosis I have seen the bismuth mixture retained, in part, for a week. Ordinarily, however, even in pyloric stenosis, the retention does not extend beyond six to forty-eight hours.

The *peristaltic waves* may be absent during an entire examination. This occurs where there is nervous depression or lack of nervous energy. They can usually be excited by massage. Even in marked gastroptosis, the peristaltic contractions may be very deep and strong. When this is true, the patient may be without symptoms. So, too, in pyloric stenosis one often

finds the strongest peristaltic waves, and at times a *reversed peristalsis* may be seen. When tumors involve the stomach-wall, the peristaltic waves will at least be absent at the point of involvement.

Restrictions in Mobility.—In the study of the normal stomach we have

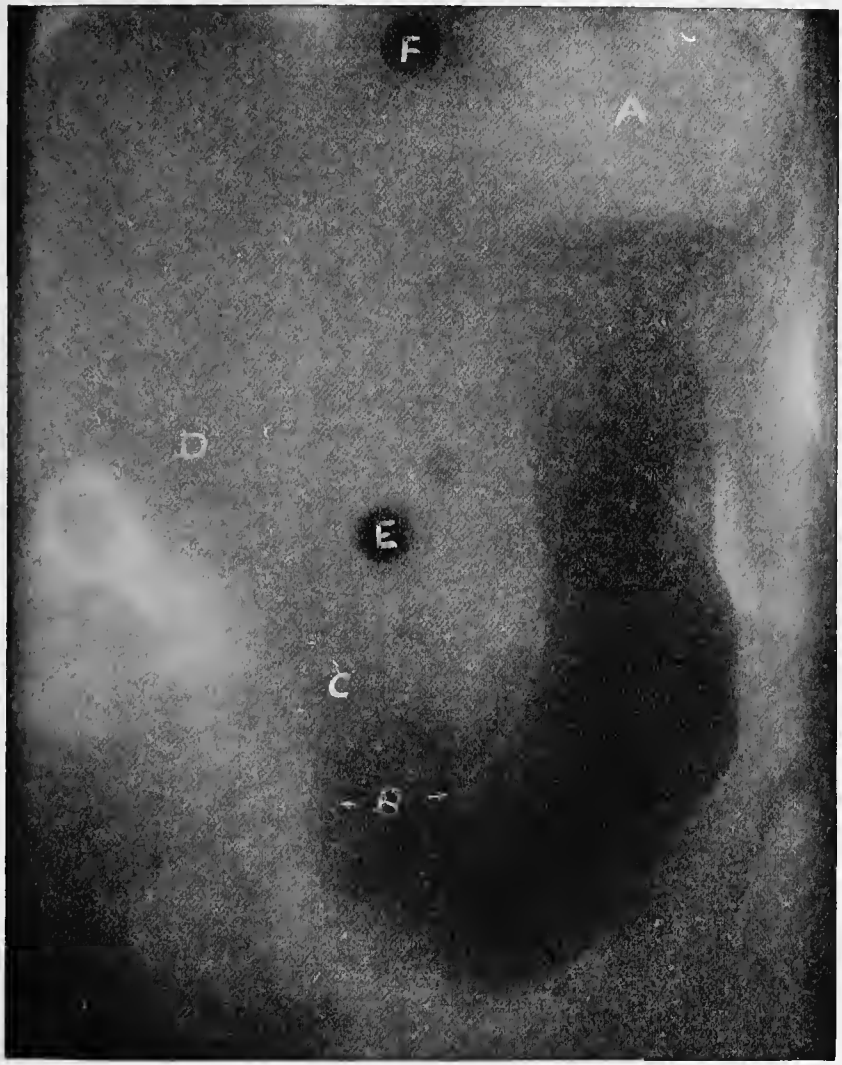


FIG. 191.—GASTROPTOSIS (Pfahler).

A, Gas in upper pole of the stomach; *B*, peristaltic wave—the beginning of the antrum; *C*, pylorus; D, right kidney; *E*, cent on umbilicus; *F*, cent on ensiform cartilage.

learned that it is a very movable organ. Therefore any interference with this mobility, as determined by watching the effects of respiration, change of posture, contraction of the abdomen, massage, or pressure by the hand upon the lower part of the abdomen, will indicate adhesions.

Gastroptosis is a pathologic condition that cannot be divided from the

"normal" stomach by any sharp line. The stomach that we describe as the "normal" is probably the ideal, since health is consistent with a stomach much lower than the "normal." The preservation of the health is probably

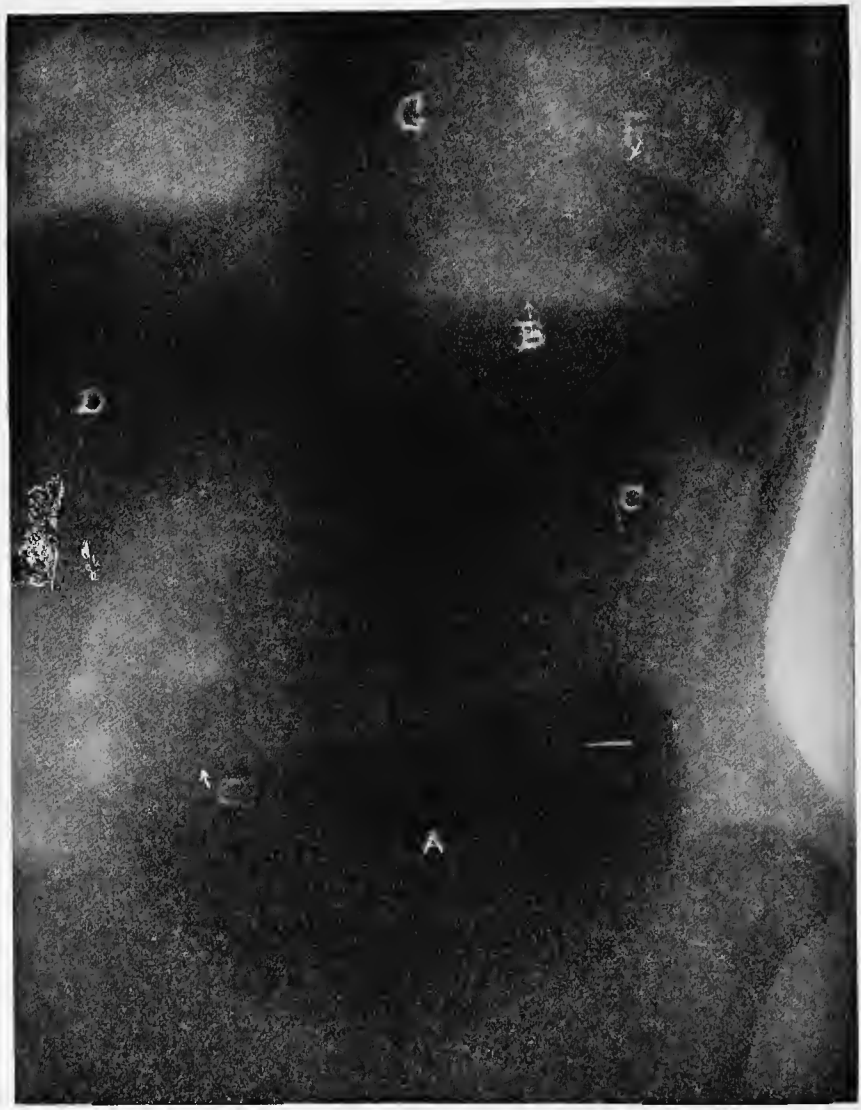


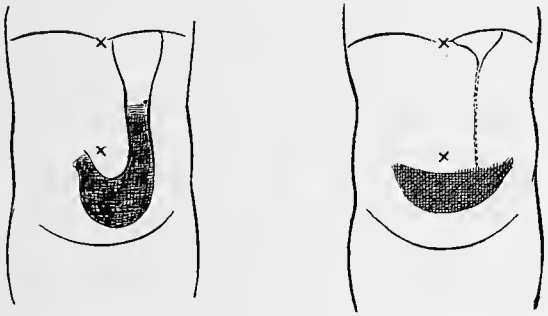
FIG. 192.—GASTRECTASIS DUE TO CARCINOMA OF THE PYLORUS (Pfahler).

A, Bismuth mixed with food, retained after four days; small portions of bismuth were still found at the operation after ten days; *B*, gas in the upper pole of the stomach; *C*, left border of the left kidney; *D*, liver shadow (spots below are due to finger-marks on the negative); *E*, probable shadow of the carcinoma; *F*, left curve of the diaphragm crowded upward by the gas in the stomach.

due to a compensatory activity of the nervous and muscular mechanism of the stomach, and when either of these give way, health fails. In general, if the lower pole of the stomach (in the standing posture) is below the umbilicus,

one can probably class it as gastroptosis. It may even extend downward as far as the pubes. In such conditions the stomach is much elongated, and the pylorus is also found ptosed and movable.

Gastrectasis is, of course, found associated with pyloric stenosis, but it is also found in gastroptosis and in atonic conditions. It is not wise to fill a stomach in order to determine gastrectasis. The stomach is an elastic organ, and therefore can be distended and distorted to a great degree, and



Gastroptosis with gastrectasis. Pyloric stenosis with gastrectasis.

FIG. 193.—SHOWS THE DIFFERENCE IN APPEARANCE BETWEEN A MARKED GASTROPTOSIS AND A MARKED GASTRECTASIS ASSOCIATED WITH PYLORIC STENOSIS.

yet little positive information is obtained and harm may possibly result. By watching the process of filling and the outline of the normal bismuth meal more accurate information can be obtained safely.

In megalogastria due to pyloric stenosis the food drops quickly to the lower pole and there spreads out into a wide semilunar shadow. The upper border of the liquid is level, the lesser curvature cannot be seen, and when the patient is shaken, the liquid can be seen to splash (Fig. 193).

In atonic gastrectasis there is, in addition to the gastroptosis, an elongated stomach with a wide lower pole, but the mixture can be seen to fill the pyloric end. The upper portion (perhaps one-half) of the stomach is distended with gas (usually swallowed air), and this is slow to be eructated. The peristaltic waves are usually absent, but may at times be excited by massage or manipulation (Fig. 194).

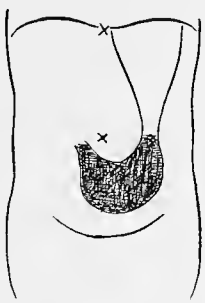


FIG. 194.—ATONIC GASTRECTASIS.

Abnormalities in form of the stomach may result from the above-named pathologic conditions, or may result from pressure upon or adhesions from without, or from cicatrices or neoplasms within the stomach-walls. One must not be hasty in classing changes in the form of the stomach, for deformity may be due to the pressure of collections of gas in the colon, or to misplaced organs (kidney), or to pressure of the spinal column, all of which must be eliminated. This will often require a repetition of the examination on a subsequent day.

Hour-glass contraction may be due to a cicatrix, to adhesions, or to carcinoma, and is recognized by the slow passage of the food from the upper to the lower segment, and finally by the division of the stomach-contents

into two portions. Caution is necessary here. The stomach must be manipulated so as to displace a collection of gas in the bowel. Pressure effects must be relieved, the entire meal should be given, and often the examination must be repeated before a definite decision can be made. I have ob-

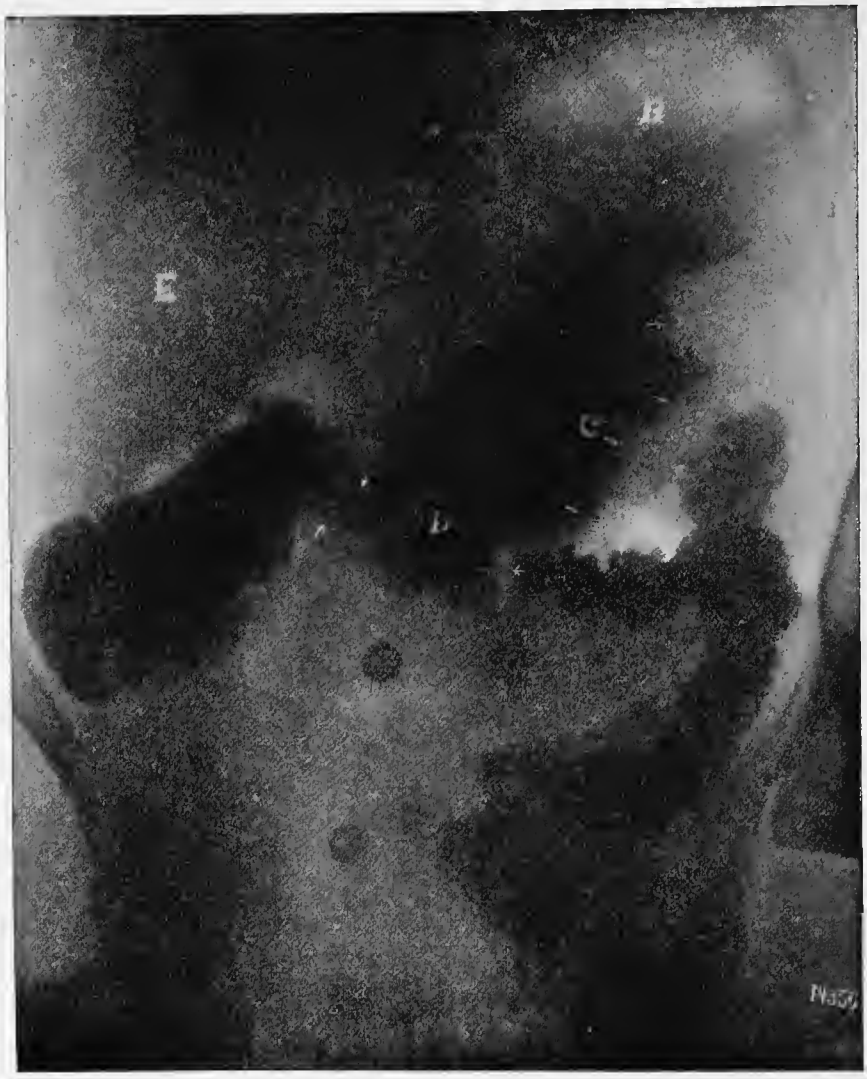


FIG. 195.—CARCINOMA OF THE STOMACH (Pfahler).
A, Ensiform cartilage; *B*, gas in the upper pole of the stomach; *C*, carcinoma; *D*, umbilicus; *E*, liver.

served a tetanic contraction of the stomach (due to ulcer) which gave all the appearances of an hour-glass contraction, and did not disappear during an observation taking an hour.

Gastric carcinoma can be demonstrated more positively and earlier by this method of examination than by any other. It is recognized by its

modification of the course of the first portion of the food through the stomach, by displacement of the stomach-contents, by deforming its outline, and, most important and earliest of all, by an interruption in the peristaltic wave at the point of the disease. In addition, one may find evidence of adhesions which draw the stomach into abnormal positions, or interfere with its normal mobility. The diseased area is more easily demonstrated before much dilatation has taken place.

Diseases of the Bowel.—Evidence of disease of the bowel is found by following the bismuth mixture through the intestinal canal; or in the colon, by injections per rectum. One studies its position, its outline, and the rate of passage of the food. The bismuth mixture makes little, if any, difference in the rate of passage of its contents. Normally, the first part of the food passes through the small intestine in two to three hours, and at the end of four hours a portion is usually found in the cecum. In eight to twelve hours it reaches the first portion of the transverse colon, and at the end of twenty-four hours it has reached the rectum, and at times has passed out. The first bowel movement does not empty the entire colon, but usually only the rectum and descending colon. A portion of the bismuth mixture is often found in the colon after forty-eight hours. I am convinced that the apparent delay in the passage of the bismuth mixture is often due to a readmixture in the colon with the oncoming food, or débris of meals taken subsequently.

Constipation may be studied scientifically in this way; the location of the delay, and often the cause, may be determined. At times this is found to be simply a rectal retention; at others it is due to an apparent "kink" in the bowel, resulting from faulty position; and again it is simply part of a general atony.

Carcinoma will often give obstructive evidence either from below or above or both.

Coloptosis is recognized by a low position of the transverse colon, and accompanying this there is generally a low position of the hepatic and at times of the splenic flexure. Usually there is both a gastroptosis, and an enteroptosis combined, but either may exist separately, or these two conditions may be only a part of a visceroptosis.

GASTRIC CONTENTS.

General Remarks.—Gastric secretion continues normally as long as there is food in the stomach, but during the later stages of gastric digestion the activity of the secretory function of the stomach diminishes. To obtain an accurate knowledge of any pathologic condition of the organ that may be present, an examination of the gastric contents must be made under conditions as nearly like the physiologic as is possible. Reliable results cannot, therefore, be obtained from an examination of the vomitus, but the contents of the stomach must be procured at a definite period after a so-called "test-meal."

The contents of the stomach consist of the fluids secreted by the pyloric and the cardiac glands of the stomach, which provide the active digestive ingredients: the stomach-contents also contain a portion of the buccal secretion, which has gained access to the stomach by swallowing, but at the time we recover the stomach-contents, this secretion from the mouth is considerably altered, owing to admixture with the fluids secreted by the gastric glands.

Test-meals.—Numerous test-meals have been suggested, but those found to be most satisfactory are "the test-breakfast of Ewald-Boas," the "test-dinner of Riegel," and the "Salzer test-meal."

The Ewald-Boas test-breakfast consists of one or two rolls and one cup of tea or water (300 to 400 c.c.). We invariably advise the use of one roll and a glass of water. This meal should be administered early in the morning, when the stomach is empty, before any food or liquid has been taken. The plan that we have adopted is first to wash the stomach thoroughly, making careful note—(1) of the quantity of gas that escapes upon the introduction of the tube; and (2) of the mucus present in this washing.

The test-meal is now given, the patient being directed to eat the bread slowly and to sip the water while eating. About an hour after this meal has been taken the contents of the stomach are to be withdrawn, and analyzed both chemically and microscopically. Hydrochloric acid should be the only acid present.

Riegel's test-dinner consists of from 300 to 400 c.c. (9 to 12 fluid-ounces) of soup, 150 to 200 grams (5 to 6 oz.) of beefsteak, 50 grams (1.7 oz.) of wheat bread or potato, and 200 c.c. (one glass) of water.

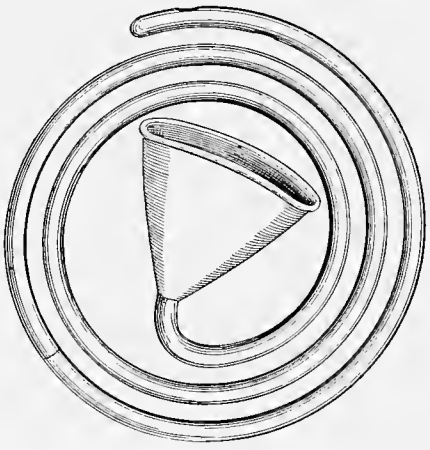


Fig. 196.—Stomach-tube.
Fenestra near tube's end; funnel-like expansion; mark showing distance to be introduced.

Caution.—The steak given at this meal should be chopped fine, lest particles of meat obstruct the lumen of the stomach-tube.

The gastric contents are to be collected by the aid of the stomach-tube in from three and one-half to four hours later.

Salzer Test-meal.—The double test-meal consists of a breakfast of 30 grams (1 ounce) of lean, cold roast-beef, which is chopped sufficiently fine so as not to obstruct the lumen of the stomach-tube; milk, 250 c.c. (8 fluid-ounces); rice, 50 grams (1.7 ounces); and a soft-boiled egg. Four hours later the Ewald-Boas test-breakfast is given, and the gastric contents collected one hour later—five hours after the administration of the first meal.

By means of this double test-meal the gastric juice is obtained when digestion is at its height. The stomach's power of motility is also appreciable, this motility being sufficient, under normal conditions, to remove all particles of meat given with the first meal. If the gastric contents contain particles of meat, there is diminished motility of the stomach.

Recovery of Gastric Fluid.—To obtain the contents of the stomach use a soft, flexible rubber tube with an end opening, or, better, with a closed end and one or more lateral openings. The tube should be indelibly marked at a point 21.5 inches (58 to 64 cm.) from the end introduced, thus aiding the examiner to determine whether or not the tube has entered the stomach.

Introduction of the Stomach-tube.—1. Place the tube in a bowl containing warm water; elevate its funnel at a distance of about two feet above the bowl, and pour at least one pint of warm water through the tube; then allow the entire tube to remain coiled up in the bowl.

2. If the patient is not confined to bed, loosen the clothing around the chest and abdomen, so that the epigastrium is covered by but a single garment. The patient is now directed to be seated comfortably in a chair; when

there is some objection to the patient sitting up, he should be directed to lie on his left side. Place a heavy towel over the patient's chest, and pin it at the back of the neck, in order to prevent the saliva, which always flows freely from the mouth while the tube is in position, from soiling the clothing or causing the patient discomfort.

3. It is most important in introducing the stomach-tube to obtain the patient's confidence, and to assure him that the instrument will not harm him, and that the slight smothering sensation and nauseating effect that its introduction always excites are increased by his own effort, and that if he will try to control the spasm of his throat, the tube will be introduced with greater ease, and the choking sensation will be materially lessened. If this step is skilfully performed, all difficulties are ended and the stomach-tube is introduced with comparative ease in the most hysterical persons; neglected, the tube is always introduced with difficulty.

4. Remove the tube from the water, and without drying grasp it with the thumb, index, and second fingers (Fig. 197). The patient should be directed to open the mouth and to lower the chin slightly, and then about six inches of the tube should be introduced, passing it directly over the tongue to the back of the throat. He is now instructed to swallow, and the tip of the tube will immediately enter the esophagus. Pass the tube gradually with the thumb and finger, taking care to exert force only at the time when the patient swallows—and he should be directed to swallow as often as possible. Continue to pass the tube until the mark on the tube (Fig. 196) reaches the patient's lips; then hold the tube in position with the left hand, and drop the funnel into the receptacle for the fluid. At this point in the operation direct the patient to bend forward and to endeavor to contract and relax his abdominal muscles, or to cough slightly three or four times in succession. If these efforts fail to expel the stomach-contents, the operator should place his left hand over the epigastrium and make gentle pressure, while the patient repeats the expulsive efforts previously described.

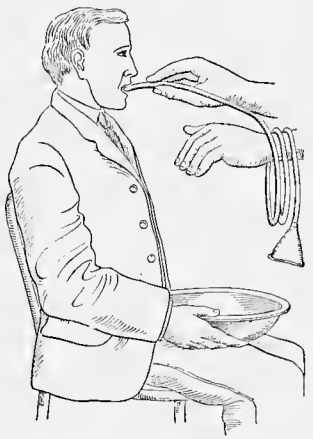


Fig. 197.—Position of the Patient for Introduction of Stomach-tube; also Method of Passing Tube into the Mouth (Boston).

In rare cases it is impossible to recover any fluid by this method, when it becomes necessary to have at hand a glass containing just sufficient warm water to fill the tube from the funnel to the tip. Elevate the funnel and pour this quantity of water into it, and at the moment all the water has entered the tube drop the funnel into a clean bowl, and let both the patient and the operator repeat the expulsive efforts (Fig. 198). The water that was introduced into the funnel will be expelled, and is likely to be followed by a free flow of the gastric contents.

In order to prevent the gastric fluid from becoming diluted with the water the funnel is placed in a second receptacle as soon as the water has escaped; thus the gastric juice obtained will correctly represent the secretion of the stomach. After the gastric fluid has begun to flow, it is not likely to be checked, or at least to stop entirely, until the stomach is emptied of its liquid contents.

Quantity.—The quantity of fluid obtained from the stomach after the ordinary test-meal varies between 20 and 60 c.c.

Microscopic Study.—The sediment of the gastric fluid should be examined microscopically in order to determine whether red blood-cells, leukocytes, shreds of mucous membrane, and particles of undigested food are present. It is also possible to determine, by a microscopic study, to what extent the starch-cells have been broken, and whether or not bacteria and fungi are present (the Boas-Oppler bacillus being common in the fluid from gastric carcinoma). Bile when present colors the leukocytes and epithelium a pale yellow.

Physical Properties of the Gastric Fluid.—The gastric fluid is an almost clear, faintly yellow liquid, having a sour taste, a peculiar odor, and a specific gravity varying between 1.002 and 1.003. The reaction of the gastric juice is acid, owing to the presence of hydrochloric acid, and is found to contain about 0.5 per cent. of solids.

Fig. 198.—Method of Inducing Expulsion of Gastric Contents by Siphonage (Boston).

Chemistry of the Gastric Contents.—The gastric juice consists of water, free hydrochloric acid, ferments, and mineral acids, but an accurate analysis of the normal human gastric fluid is impossible, since this fluid is always contaminated with an admixture of saliva.

Acidity.—It has been satisfactorily shown that the acidity of normal gastric juice depends principally upon the presence of free hydrochloric acid. Experimentally, it has been demonstrated that by determining the amount of chlorin and basic constituents present in the gastric juice, the latter substances, after having been thoroughly saturated, still permit a quantity of hydrochloric acid to remain free.

After the introduction of food into the stomach a varying amount of lactic acid, derived from the carbohydrates and acid salts, is also present; in fact, Ewald considers that the gastric acidity during the early stage of digestion depends to a variable degree upon the presence of lactic acid. Hydrochloric acid is also present during the early stages of digestion, but it probably exists in quite close combination with albuminous substances. When all the albuminous substances present in the stomach have become saturated, hydrochloric acid appears free in the gastric juice, and the quantity of lactic acid gradually decreases, since hydrochloric acid inhibits the development of the microörganisms concerned in the production of lactic acid.

In pathologic conditions the quantity of free hydrochloric acid may display wide variation, and in extreme conditions (gastric carcinoma) it may be absent, or even reach a maximum of 0.5 per cent. (gastric ulcer). The quantity of lactic acid, which, under normal conditions, is but slight or absent during the height of digestion, is often found increased, and fatty acids are likely to be present at this time.

The total acidity of the gastric contents will, therefore, be seen to depend upon the presence of some one or more of the three acids previously mentioned, but a high degree of acidity does not point directly to the presence

of any one acid in excess, and it is, therefore, necessary to determine the presence or absence of the various acids and of acid salts in order to draw definite conclusions as to what particular substance present gives rise to the acidity.

Test for Total Acidity.—Reagents: (1) A decinormal solution of sodium hydroxid (approximately 4 gm. in 1000 c.c. of distilled water); (2) a 1 per cent. alcoholic solution of phenolphthalein. In order to make an accurate decinormal solution of sodium hydroxid it is necessary to balance the solution by titration against a decinormal acid solution (preferably oxalic acid) made by careful weighing and measuring. A solution made by dissolving four grams of sodium hydroxid in a liter of distilled water is only approximately accurate.

We have adopted Jaworski and Ewald's method for the estimation of total acidity, and consider the number of cubic centimeters of decinormal sodium hydroxid solution required to neutralize 100 c.c. of stomach contents to indicate the degree of acidity.

Technic.—1. Place 10 c.c. of the filtered gastric contents in a beaker.

2. Add two drops of the phenolphthalein solution.

3. Add the decinormal sodium hydroxid solution one drop at a time from a buret, stirring the mixture with a glass rod after each addition of the sodium hydroxid solution.

4. At the point where the gastric contents assume a permanent pink color its acidity has been neutralized by the sodium hydroxid solution, and the number of cubic centimeters of the decinormal sodium hydroxid solution employed is read from the buret.

Let us suppose that it requires 4.8 c.c. of the decinormal sodium hydroxid solution to neutralize 10 c.c. of the stomach contents; the decimal point is then removed one place to the right, giving the figure 48, the number of cubic centimeters necessary to neutralize 100 c.c. stomach contents, which figure represents the total acidity of the stomach contents.

Normal Stomach Contents.—Normal gastric fluid will be found to give a figure between 40 and 60 for Americans and Europeans, while 35 is normal for Japanese and natives from eastern Asia. Many clinicians prefer to estimate the total acidity of the stomach contents in terms of hydrochloric acid, which deductions are attained in the following manner, by the well-known laws of molecular equivalents: 1000 c.c. of the decinormal solution contain 4 gm. of NaOH, which is equivalent to 3.65 gm. of HCl. Each cubic centimeter of the solution, then, is equivalent to 0.00365 gm. of free hydrochloric acid. In order to determine the total acidity of a given specimen of gastric contents multiply the number of cubic centimeters of decinormal solution representing the total acidity by 0.00365. Suppose the total acidity of a given specimen was 48. The total acidity in terms of hydrochloric acid would be 48×0.00365, or 0.17. Normal gastric juice contains from 0.14 to 0.24 per cent. of hydrochloric acid.

Hyperacidity.—An abnormally high degree of total acidity is known to accompany the following conditions: gastric ulcer, gastric dilatation, certain gastric neuroses, and when there is a hypersecretion of gastric juice.

Under pathologic conditions the acidity of the gastric contents may not depend upon hydrochloric acid alone. In fact, the total acidity may be extremely high in stomach contents devoid of hydrochloric acid, but abnormally rich in fatty acids, lactic acid, and acetic acid.

Hypoacidity.—Rarely, indeed, the gastric contents is alkaline, amphoteric (changes red litmus-paper to blue and blue litmus to red), or neutral.

The acidity of the gastric contents is lowered in the mucous forms of chronic gastritis and when the gastric glands have been destroyed. We have found the reaction of the gastric contents to be neutral upon several occasions, but in each case the patient had taken a large quantity of fluid prior to the recovery of the gastric contents, or else he was suffering from a chronic catarrhal condition of the throat or esophagus and consequently swallowed an excessive quantity of buccal secretion.

When the stomach contents are collected as the result of vomiting, an alkaline reaction is commonly obtained, and this alkalinity is in part, if not entirely, due to the admixture of secretions from the throat, mouth, and esophagus.

Proportion of Hydrochloric Acid.—In health, pure gastric juice has been estimated to contain from 0.2 to 0.3 per cent. of free hydrochloric acid, but quantities of free hydrochloric acid are to be found only at the height of digestion. The exact period at which the height of digestion is reached is governed by the character and the quantity of the food and liquids previously ingested: the less work there is to be accomplished, the sooner will the stomach contents be found to contain free hydrochloric acid following the ingestion of food. After taking Ewald's test-breakfast, the hydrochloric acid appears in the gastric fluid in from thirty to thirty-five minutes, but the point of maximum intensity is usually reached in from fifty minutes to an hour, and may correspond to 0.17 per cent. After the administration of Reigel's meal, the free acid appears after one hundred and thirty-five minutes, and reaches its highest point, corresponding to 0.27 per cent., in from one hundred and eight to two hundred and ten minutes.

Euchlorhydria is a secretion of the normal amount of free hydrochloric acid—varying from 0.14 to 0.24 per cent. This condition is most frequently observed in connection with nervous dyspepsia, and is always to be found when no gastric derangement exists.

Rarely a normal secretion of free hydrochloric acid is found when atony of the muscular wall of the stomach exists. The presence of a normal amount of free hydrochloric acid tends to exclude the existence of chronic gastritis, and favors the presence of neuroses of the stomach.

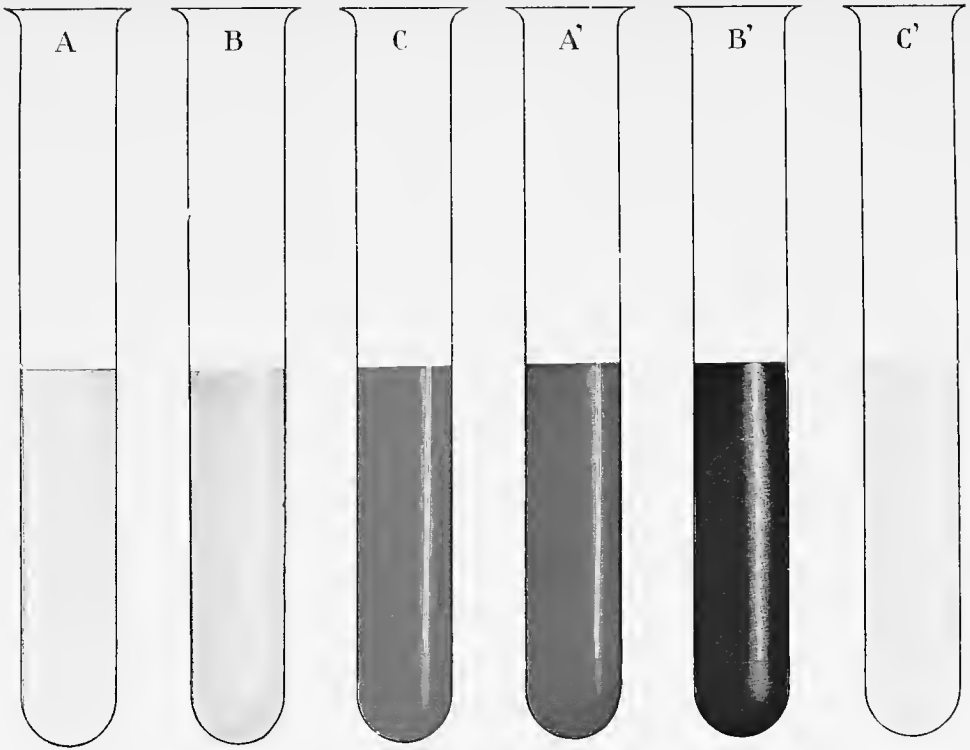
Hyperchlorhydria is a condition in which the secretion of hydrochloric acid averages more than 0.2 per cent. A hypersecretion of hydrochloric acid, as a rule, indicates gastric neurosis, and is commonly encountered in the gastric fluid of neurasthenic persons. A hypersecretion of hydrochloric acid is at times coupled with a continuous hypersecretion of gastric juice.

Hyperchlorhydria is characteristic of gastric ulcer, and is rarely seen in gastric carcinoma.

Hypochlorhydria.—This condition is associated with pathologic states in which the secretory power of the stomach is lessened as the result of disease, *e. g.*, in subacute and chronic gastritis, gastric carcinoma, dilatation, and atony.

Achlorhydria is an absence of free hydrochloric acid. This condition is most commonly seen in gastric carcinoma. Achlorhydria may also be observed in connection with chronic gastritis and in neurasthenic individuals. The degree of acidity of the gastric contents will fluctuate within wide limits during the course of the various chronic diseases, and especially in those accompanied by cachexia, emaciation, and anemia.

Tests for Free Hydrochloric Acid.—The following is a list of reagents, named in the order of their delicacy, by the use of which it is possible to detect free hydrochloric acid in the filtered stomach contents:



A. Gastric fluid to which a 1 per cent. solution of phenolphthalein has been added.
B. Gastric fluid to which a 1 per cent. solution of alizarin has been added.
C. Gastric fluid to which a 0.5 per cent. solution of dimethylamido-azobenzol has been added.
A'. A after titration with a decinormal solution of sodium hydrate.
B'. B after titration with a decinormal solution of sodium hydrate.
C'. C after titration with a decinormal solution of sodium hydrate.

(Boston.)

Dimethylamido-azobenzol	0.02	pro mille.
Phloroglucin-vanillin	0.05	" "
Resorcinol	0.05	" "
Congo-red		
Tropæolin 00	0.3	" "
Emerald-green	0.4	" "
Mohr's reagent	1.0	" "

For general work, the Congo-red and Günzberg tests have been found to be the most satisfactory.

Congo-red Test.—Reagent.—An aqueous solution of Congo-red, 1:1000.

Method.—1. To a half-filled test-tube of water add three drops of the reagent.

2. Grasp the bottom of the tube between the thumb and index-finger, hold it in a clear light, and allow one or two drops of the gastric contents to fall from a pipet. Hold the tube steady to prevent jarring. As soon as the drop of gastric contents comes in contact with the red solution, a blue color is produced, the upper portion of the liquid changing to a pale and then to a dark blue, and, as the gastric contents traverses through the lower, red stratum of the liquid, it leaves behind a faint blue track, collecting at the bottom of the tube in the form of a light-blue sediment.

We have found Congo-red to be satisfactory for the qualitative determination of free hydrochloric acid, since the other acids capable of producing the reaction are seldom, if ever, present in sufficient amounts to cause confusion.

The best test, however, for free hydrochloric acid in the gastric contents is that known as the Günzberg test. It requires the following reagent: Vanillin, 1.0; phloroglucin, 2.0; absolute alcohol, 30.0. One drop of filtered gastric contents is mixed with one drop of the reagent and the mixture is evaporated to dryness by the aid of gentle heat. In the presence of free hydrochloric acid the residue turns a bright pink color. Other acids in the gastric contents produce a dirty yellow color in the residue produced by evaporation.

For the quantitative determination of free hydrochloric acid in the gastric contents ten cubic centimeters of the filtered gastric contents are placed in a beaker and decinormal sodium hydroxid solution is added one-half cubic centimeter at a time. After each addition of the decinormal sodium hydroxid solution the Günzberg test is done, and when it is no longer positive the buret is read. The buret reading is multiplied by ten and the result is the number of cubic centimeters of decinormal sodium hydroxid solution requisite to neutralize the free hydrochloric acid in one hundred cubic centimeters of gastric contents. The result may be expressed by that number, or the number of cubic centimeters of the decinormal sodium hydroxid solution may be multiplied by the factor for hydrochloric acid, the result then giving the actual amount of hydrochloric acid present in the gastric contents.

Example: Suppose in a given specimen of gastric contents 2.5 c.c. of decinormal sodium hydroxid solution were added to 10 c. c. of the filtered gastric contents before the Günzberg test because negative. The free hydrochloric acid would then be $2.5 \times 10 = 25$, or $25 \times 0.00365 = 0.09$ per cent.

If the free hydrochloric acid be subtracted from the total acidity, the result will represent the combined hydrochloric acid and acid salts in the specimen.

In this country a normal gastric contents may be represented by the following figures: Free hydrochloric acid, 20 to 40; total acidity, 40 to 60.

Clinical Significance.—In selecting a diet for an individual in whom the hydrochloric acid is known to be absent on account of structural changes, it is necessary to direct that proteids be given in such form as will permit them to be subject to pancreatic digestion, obviating all possible delay. It is in such conditions that the administration of predigested foods is indicated. On the other hand, in functional achlorhydria a proteid diet may serve to stimulate the secretion of hydrochloric acid.

At times it is found that the quantity of hydrochloric acid secreted is sufficient to satisfy the albuminous affinities of a moderate-sized meal in which the proteids have been limited, and in such cases, when a suitable dietary is followed, digestion may be perfectly carried on.

Ferments and Proferments.—"It is generally conceded that pepsin itself is not secreted as a product of the chief cells of the glands of the fundus, but that the proferment of pepsin (pepsinogen or propepsin) is secreted. Numerous experiments have proved this statement, and shown that in fasting animals the glands of the stomach do not contain pepsin, but a substance which is not destroyed by sodium carbonate, and which is readily converted into pepsin when brought in contact with hydrochloric acid. It is this substance which has been designated under the caption pepsinogen.

"Pepsin is to be recovered from the mucous membrane of the stomach during the stage of digestion, but during the non-digestive stage, zymogen is to be recovered. Zymogen may be found coexistent with the process of digestion. The time at which zymogen is transformed into a ferment is doubtful. A fair amount of evidence exists, however, to show that this change takes place after it has been secreted."

Clinical Significance.—Free hydrochloric acid generally indicates that pepsin is present, and should these two substances be found in the stomach contents, gastric digestion takes place. A simple test to determine this is as follows: (1) Place 25 c.c. of filtered gastric contents in a test-tube, and to it add 0.05 gm. (approximately, one grain) of the white of a hard-boiled egg; (2) heat the mixture to a temperature of 40° C., and observe the change that takes place. Under normal conditions, pepsin and hydrochloric acid being present, the coagulated albumin becomes digested in about three hours. If the albumin is undigested by the mixture, add five drops of dilute hydrochloric acid to the gastric contents, shake well, place at a temperature of 40° C., and observe the result. If digestion of the coagulated albumin takes place only after the addition of hydrochloric acid, zymogen or pepsin was present in the gastric juice, but was inactive until the addition was made, which shows conclusively that free hydrochloric acid was absent from the gastric juice. (3) If the coagulated albumin is unaffected by the gastric fluid after the addition of hydrochloric acid, both pepsin and zymogen are absent from the gastric contents.

Digestion of Carbohydrates.—The gastric secretion is in itself unable to digest carbohydrates, but there is evidence suggestive of the fact that a certain amount of starch is transformed into sugar early during gastric digestion, which digestion is dependent upon the action of saliva taken into the stomach with the ingestion of food. The action of the saliva, however, may be inhibited by 0.12 per cent. of free hydrochloric acid. The transformation of starches into sugar is most active in neutral or mildly alkaline solutions. The ferment of saliva first converts raw starch into soluble starch, then into erythrodextrin, achroödextrin, and eventually into maltose. A high grade of acidity of the gastric contents arrests starch digestion early.

Test for the Degree of Starch Digestion.—Place 10 c.c. of filtered

gastric contents in a test-tube, and to it add a few drops of Lugol's solution (iodin, 0.1 part; potassium iodid, 0.2 part; distilled water 300 parts). This solution causes a blue color to appear if soluble starch amidulin is present; if erythrodextrin, produces a purple color. If no color change develops upon the addition of Lugol's solution, achroödextrin, dextrose, or maltose is present. Soluble starch causes the formation of a blackish-blue precipitate with Lugol's solution.

Achroödextrin possesses a greater affinity for iodin than do any of the other intermediary products. It is well to add Lugol's solution freely in order that some of the intermediary products that require large quantities of iodin for the production of the color may not escape notice.

Further evidence of starch digestion may be obtained by the use of Fehling's solution. Maltose and dextrose reduce the copper when this solution is heated.

Clinical Significance of Reagent for Starch Digestion.—In normal carbohydrate digestion no color is produced with Lugol's solution after a test-meal of bread and water.

The violet color with Lugol's solution shows that starch digestion is imperfect, and it now becomes necessary to determine the cause of such imperfect saccharification. Imperfect starch digestion is caused by an excess of hydrochloric acid, a deficiency of saliva, or imperfect mastication of food.

Slight fat digestion takes place in the stomach, but at present it is not customary to estimate the degree of fat digestion in the gastric fluid.

It has been shown repeatedly that the fatty acids of the gastric fluid are somewhat intermediary, connected with the formation of lactic acid.

Test for Butyric Acid.—Gastric fluid rich in fatty acids emits an odor of rancid butter. Extract 10 c.c. of the gastric fluid with 50 c.c. of ether; evaporate to dryness, and dissolve the residue in a few cubic centimeters of water. Add a trace of calcium chlorid, and if butyric acid is present, it will be precipitated in the form of small, oil-like globules. This precipitation, consisting apparently of oil, will be found to emit a decided odor.

Test for Acetic Acid.—Acetic fermentation may take place in the stomach, although theoretically it is believed to occur only in the presence of alcohol.

Test.—Carefully neutralize the acidity of the gastric contents. If acetic acid is present, sodium acetate will be formed, which will produce a blood-red color on the addition of a 10 per cent. solution of ferrichlorid.

Lactic Acid.—General Remarks.—Appreciable amounts of lactic acid are not present normally in the gastric contents recovered during digestion, but may be ingested with the food.

A decided reaction for lactic acid points to a diminution or an absence of hydrochloric acid. (See Clinical Significance of Hydrochloric Acid, p. 468).

It is generally conceded that the lactic acid found in the stomach is the result of bacterial action. Again, the stomach contents may be found to contain lactic acid during health, but in such instances it has been introduced into the stomach with the food. The bacteria capable of forming lactic acid from carbohydrates are plentiful in the buccal secretion and gastric contents, and induce acid fermentation whenever sugar-bearing substances are ingested.

Granting that a certain amount of lactic acid may be introduced with a normal meal or that sugar-charged foods have been taken, it is possible to find lactic acid in normal gastric contents early during the course of digestion. It is, however, to be borne in mind that lactic acid fermentation

is inhibited by the secretion of hydrochloric acid (0.7 to 1.5 pro mille), either free or combined, and that this inhibition is perceptible, even when the amount of hydrochloric acid present in the stomach contents is very small. It is through this physiologic secretion of hydrochloric acid that lactic acid production is discontinued during the later stage and height of digestion; and it has been authentically stated that lactic acid does not appear in appreciable quantities during the process of normal digestion.

Lactic acid, even when introduced into the stomach as such, may be absent during the later stage of digestion, and when the gastric contents is shown to contain lactic acid early during digestion, its disappearance at the end of the process is doubtless due to an absorption of the lactic acid ingested. Thus, failure to obtain a reaction for lactic acid under normal conditions has been attributed to the presence of free hydrochloric acid, which possibly interferes with the reaction.

Test for Lactic Acid.—In order to ascertain the quantity of lactic acid present, it is necessary, first, to introduce the stomach-tube and to wash the stomach thoroughly, employing from 8 to 12 ounces of water. This water is then withdrawn, and the process repeated several times at a single introduction of the tube. In this way any lactic acid that might be present as a result of retained food substances—a feature of gastric dilatation—is removed. Boas' test-meal (p. 462) should now be administered.

If there is evidence of retention, it may be well to wash the stomach, as just described, in the evening, before the test-meal is given. The stomach contents should be collected the following morning, before any food or drink has been taken.

Caution.—Do not attempt to estimate the lactic acid of the gastric contents unless the stomach has been thoroughly washed prior to the administration of Boas' test-meal.

Uffelmann's Test.—*Reagents.*—(*a*) A 10 per cent. aqueous solution of ferric chlorid. (*b*) Concentrated solution of pure phenol.

1. Place 10 c.c. of water in a test-tube, and to it add three drops of solution (*a*) and three drops of solution (*b*); shake the tube gently, when the mixture will assume a bluish-black color.

2. Add water and shake thoroughly, until a pale amethyst hue results.

3. Grasp the bottom of the tube between the thumb and index-finger, hold in a clear light, and add a small quantity of filtered gastric contents.

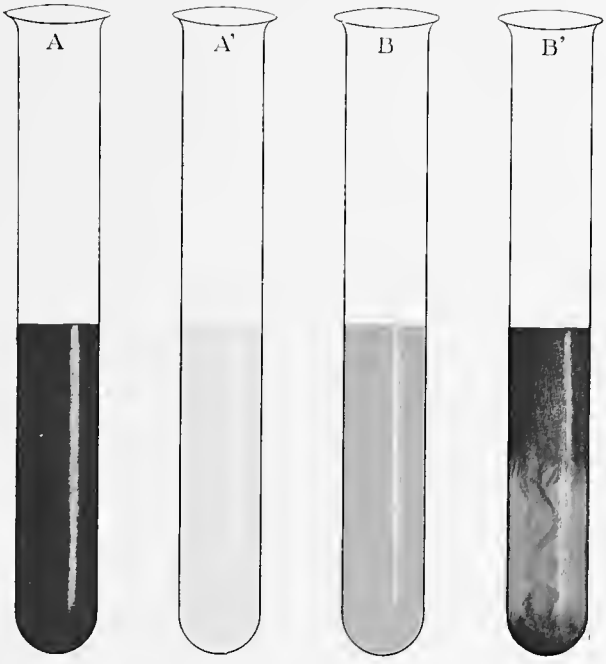
Lactic acid causes the upper stratum of the liquid to change to a canary-yellow color, and to avoid confusion it is recommended that 10 c.c. of gastric fluid be placed in a test-tube and from 30 to 50 c.c. of ether be added. Shake thoroughly, and allow it to stand for a few minutes until the ether separates from the gastric contents; then pour off the ether, and to this ethereal extract add Uffelmann's reagent. In the presence of lactic acid a canary-yellow color appears. (See Plate XII.)

Fallacies.—A reaction occurs with Uffelmann's reagent for lactic acid when an abundance of butyric acid, acid phosphates, glucose, and alcohol, is present in the stomach contents.

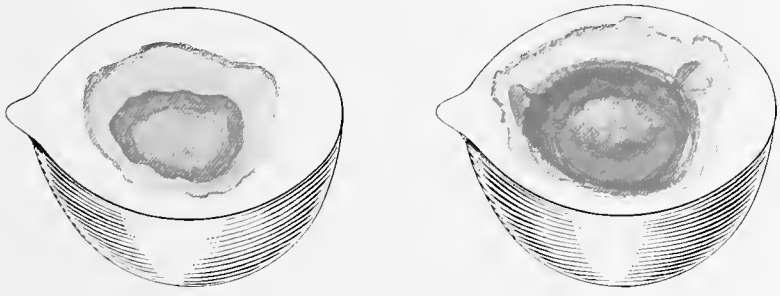
Kelling's Test.—Place 1 c.c. of the filtered gastric contents in a test-tube and dilute with 9 c.c. of water. Hold the mixture in a clear light, and add from one to three drops of a 5 per cent. aqueous solution of ferric chlorid, one drop at a time. Lactic acid causes a greenish-yellow color to appear upon the addition of the reagent.

Acetone is present in the gastric contents when there is organic disease of the stomach or the intestine, and is occasionally present in carcinoma of the pancreas or of the liver.

PLATE XII



A. Uffelmann's reagent.
A'. A after the addition of gastric fluid containing lactic acid.
B. Water to which three drops of Congo-red solution have been added.
B'. Change induced in B when gastric fluid containing free hydrochloric acid is added.
(Boston.)



1. Resorcin test for free hydrochloric acid. 2. Günzburg's test for hydrochloric acid.
(Boston.)

Test—See Urine, p. 656. Either the distillate or the filtered gastric fluid can be employed for this test.

Microscopic Study of the Gastric Contents.

In order to secure any valuable clinical information the gastric contents must be examined microscopically within a few hours after its collection. After a test-meal starch-cells, mucus, epithelial cells, and leukocytes may be found in the gastric contents; but these are present only in small numbers, with the exception of the starch-granules, which are always numerous. Yeast-cells, sarcinæ, and large numbers of bacteria are also found when there is a reduction in the amount of free hydrochloric acid, and even though the total acidity of the gastric contents may be unusually high, bacteria and fungi are present, unless this increase in the acidity is due to hydrochloric acid.

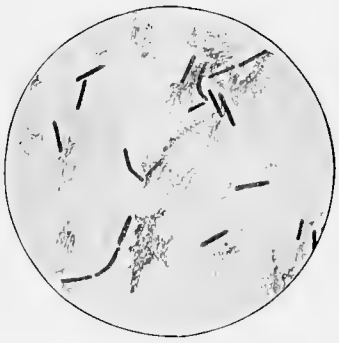


Fig. 199.—Boas-Oppler Bacillus from Near Top of Fluid from Washing in Case of Gastric Cancer (Observation at Pennsylvania Hospital).

Particles of undigested food are recovered from the stomach one hour after a test-meal and also after an ordinary meal. In dilatation of the stomach particles of food may be present that have been ingested some days prior to the recovery of the gastric contents. The epithelial cells and mucus found in the gastric contents may have escaped from the esophagus or come from the throat, and seldom give positive evidence of gastric disease.

Shreds of necrotic tissue and of gastric mucous membrane are rarely detected in the stomach contents, and when present, are always suggestive of gastric ulceration or necrosis. Intestinal parasites—*e. g.*, ascaris lumbricoides, ankylostoma or hook-worm, and segments of the tape-worm—are occasionally found in the vomitus.

The Boas-Oppler bacillus is quite constantly present in the gastric contents during the course of gastric carcinoma. This bacillus (Fig. 199) appears in long, segmented chains, which often assume a somewhat tortuous course; it stains readily with the ordinary anilin dyes, is Gram-positive, and is not motile. This bacillus is found in the gastric contents when free hydrochloric acid is diminished or absent and lactic acid is abundant. There has been some question as to whether or not the Boas-Oppler bacillus is concerned in the production of lactic acid. It is not uncommon to find quite dense aggregations and clumps of these bacilli disseminated throughout the gastric contents.

Clinical Significance.—The bacilli are fairly constant in stomach contents in which lactic acid is plentiful, but they have not been shown to be concerned in the etiology of gastric carcinoma.

The Vomit.—The vomit is usually composed of the contents of the stomach mixed with a variable amount of mucus and saliva. The odor of the vomit is of great diagnostic importance, since in certain conditions it is characteristic; thus a putrid odor suggests pyloric stenosis or ulceration of the stomach; an ammoniacal odor points toward the vomiting of uremia. Certain foods and drugs influence the odor of the vomit (onions, asafetida, garlic), and in phosphorus-poisoning the vomit gives off a decided odor of garlic. Phenol, camphor, and creosote also lend their peculiar odor to the vomit.

VOMITING.

In Acute Infections.

Scarlet fever,
Yellow fever,
Diphtheria (occasional),
Acute nephritis,
Epidemic meningitis,
Whooping-cough (during a paroxysm),
Asiatic cholera,
Small-pox,
Measles,
Typhoid fever (occasional),
Acute dysentery,
Poliomyelitis,
Pulmonary tuberculosis (incipient and advanced stages).

Gastric Causes.

Acute gastritis,
Gastric cancer,
Gastric ulcer,
Pyloric stenosis,
Gastric hour-glass contraction,
Gastric dilatation,
Cyanotic congestion (of cardiac or hepatic origin),
Chronic gastritis,
Following diatetic errors (cholera morbus, cholera infantum).

Renal Causes.

Acute nephritis,
Chronic parenchymatous nephritis,
Chronic interstitial nephritis,
Acute pyelitis,
Floating kidney,
Renal calculus,
Ureteral calculus (during attack of colic),
Nephritic abscess.

Hepatic Causes.

Catarrhal jaundice,
Hepatic calculi (colic),
Acute yellow atrophy,
Acute cholecystitis,
Atrophic cirrhosis,
Hypertrophic cirrhosis.

Intestinal Causes.

Obstruction (stercoraceous vomit),
Acute appendicitis,
Acute catarrhal dysentery,
Acute general peritonitis.
Intestinal colic.

Pancreatic Causes.

Pancreatic calculus,
Carcinoma of head of organ,
Acute pancreatic hemorrhage,
Chronic pancreatitis.

Physiologic.

Pregnancy,
Menstruation.

Shock.

Injuries sufficient to induce shock,
Profuse hemorrhage,
Sudden excitement,
The sight of certain substances.

Drugs and Toxins.

Apomorphin,
Etheranesthesia,
Chloroform anesthesia,
Morphin,
Carbolic acid and corrosives,
Chronic lead-poisoning,
Arsenic-poisoning,
Phosphorus-poisoning,
Illuminating gas-poisoning.

Special Sense.

Certain odors,
Repulsive sights,
Certain tastes,
Hysteric.

Nervous Causes.

Cerebral tumor,
Cerebral abscess,
Concussion,
Cerebral hemorrhage,
Cerebral sinus thrombosis,
Middle-ear disease,
Epidemic meningitis,
Tuberculous meningitis,
Tabes dorsalis (gastric crises),
Increased tension of fluid in spinal canal (uremia and sunstroke),
Epilepsy,
Migraine,
Sea-sickness,
Acute poliomyelitis.

Odor of Stercoraceous Vomiting.—Stercoraceous vomiting is characterized by its fecal odor, which is dependent upon the presence of phenol, indol, and skatol. Fecal vomitus is usually alkaline in reaction.

When vomiting is preceded by nausea, the ejecta will contain an abnormally large proportion of saliva. The time that elapses between the vomiting and the taking of food is of much clinical importance. (See Vomiting, p. 432.) It is also important to distinguish between material ejected from the stomach and that which is regurgitated from the throat or from a dilated or sacculated esophagus. (See Dilatation of the Esophagus, p. 416.)

Bile.—When the vomitus is green in color, bile is generally present, but it is unusual to find bile in the gastric contents that has been recovered through the stomach-tube; we recall but a single instance—that of a neurasthenic female of thirty-five.

Biliary vomiting, when frequent, points to the existence of obstruction either in the lower duodenum or upper portion of the jejunum. The presence of bile in the gastric contents obtained by the stomach-tube is in a measure indicative of pancreatic disease.

(1) Place on a blotting paper a drop of the filtered gastric fluid, and near to it a drop of yellowish nitric acid.

(2) Draw a glass rod through the acid and to the stomach contents.

(3) At the point where the two liquids meet a display of colors develop if bile is present.

Blood.—The vomitus is often blood-stained. (See Vomiting, p. 432.)

DISEASES OF THE STOMACH.

NEUROSIS OF THE STOMACH.

Definition.—A functional condition of the stomach characterized clinically by intermittent and at times periodic attacks of gastric disturbance, the patient displays but few, if any, symptoms between these attacks. Motor or sensory disturbances may be present, or in fact both may develop in the same individual.

Predisposing and Exciting Factors.—Many cases of gastric neurosis are seen to develop in persons who have previously suffered from neurasthenia or hysteria; the condition may also follow prolonged mental strain, overwork, anxiety, business reverses, grief, sudden emotion, excitement, and practically any condition that impoverishes the system.

Emphasis is to be laid upon the fact that not all persons suffering from gastric neurosis are ill nourished, but, on the contrary, many of them appear to enjoy fairly good health. Gastric neuroses are more commonly seen among the moderately well-to-do and the wealthy classes. They are far more common among females than among males. In a large percentage of cases the condition is secondary to some organic lesion that is oftenest located in the genito-urinary tract.

Principal Complaint.—Anorexia is an early and a constant symptom, and after the taking of food the patient suffers from a sense of oppression in the region of the epigastrium. Following the ingestion of certain foods there are eructations of gas, and in some instances particles of food are regurgitated, followed by an intense burning sensation along the esophagus and in the pharynx. Vomiting may occur, but is by no means characteristic of gastric neurosis.

The patient complains at times of a rumbling in the intestine, and of

decided movements felt in the region of the stomach and over the abdomen. Upon examination these movements will often be found to be due to increased peristalsis.

Constipation and attacks of diarrhea, neurotic in character, are among the symptoms of gastric neuroses.

Physical Signs.—**Inspection.**—There may be evidence of abdominal distention or the abdomen may be scaphoid. The movements of the stomach can often be detected through a thin abdominal wall, and if gastric dilatation is present certain irregularities in the abdominal wall are apparent.

Palpation.—The muscles of the abdomen contract quickly in response to any irritation, and the movements of the stomach are exaggerated by abdominal palpation. If pain is present, firm pressure over the epigastrium will generally be followed by relief.

Auscultation.—An unusual amount of gurgling is heard both over the stomach and over the intestine, but such sounds do not appear to be associated with intense abdominal distress.

Laboratory Diagnosis.—In certain selected cases the stomach will be found to contain a large amount of mucus several hours after the ingestion of food or after a night's sleep, whereas in another group of cases in which there is increased motility, it is impossible to recover any liquid from the stomach after a night's rest and the stomach is often found empty forty minutes after the taking of a test-meal.

Chemically, an analysis of the gastric contents in neurosis may vary greatly at different times in the same individual and under practically the same conditions; *e. g.*, hydrochloric acid may be increased, diminished, or even absent—a feature seen not only in gastric neurosis, but in conditions in which there is a marked hysterical tendency.

The quantity of urine voided during the twenty-four hours will also be found to vary greatly and to be influenced—*i. e.*, increased—after nervous excitement. The specific gravity of the urine in gastric neurosis is, as a rule, lower than normal, but bears a more or less close relation to the quantity excreted during the twenty-four hours (the greater the quantity, the lower the specific gravity). Chemically, the percentage of solids present is in direct proportion to the quantity of urine voided. Indican is a common constituent.

The hematologic changes displayed by those suffering from gastric neurosis are of two types—first, that in which there is a moderate grade of secondary anemia (see Secondary Anemia); and, second, that displaying no appreciable evidences of anemia; and, in fact, there may be an increase in the number of red cells in a cubic millimeter, and in the percentage of hemoglobin. The number of leukocytes in a cubic millimeter is of but limited value in this disease, since leukocytosis may result from the ingestion of certain foods, exercise, etc. (See Leukocytosis.)

Summary of Diagnosis.—The diagnosis is based on the following:

(1) The etiologic factors present. In this connection it is important to ascertain what possible causative influence may account for, or contribute toward, the production of the gastric symptoms.

(2) The course and duration of the malady and the absence of positive signs and symptoms suggestive of organic disease of the stomach.

(3) The effect of eating indigestible substances—*e. g.*, whether or not this is followed by dyspnea, palpitation, vertigo, etc.

(4) The character of the pain, and the conditions that excite and relieve pain, are of the utmost importance in distinguishing gastric neurosis from gastric ulcer and gastric carcinoma. (See Epigastric Pain, p. 436.)

(5) The gastric contents obtained after a test-meal shows digestion to be but slightly, if at all, impaired.

Duration.—The duration of neurosis of the stomach depends largely upon the early application of judicious treatment, both medical and hygienic. The majority of cases recover in from a few weeks to several months or a year, although occasionally cases are encountered in which this malady persists for an indefinite period.

Neuroses of Secretion.

Hyperchlorhydria.—Definition.—An abnormal secretory function of the stomach resulting in the production of an excess of hydrochloric acid in the gastric juice.

Predisposing and Exciting Factors.—An excessive quantity of hydrochloric acid is not infrequently seen in persons who have recently undergone great anxiety or who are overworked; it is uncommon in the laborer. A history of dietetic errors—*e. g.*, a too liberal ingestion of rich foods and alcohols—is often obtained.

Sex.—Men are more likely to suffer from this condition than are women.

Varieties.—Hyperchlorhydria may be continuous, but in the majority of instances it is intermittent, lasting for from a few hours to several days. In the intermittent type the amount of hydrochloric acid secreted by the stomach is not above the high normal limit in the free intervals, but during the attacks the quantity of hydrochloric acid may double or treble that of the normal. The intervals between the attacks of hyperchlorhydria vary greatly in different individuals, and generally last from a few days to several weeks or even years.

Principal Complaint.—The first symptom of which the patient complains is an uneasiness in the epigastrium occurring one, two, or three hours after a full meal. He may have noticed that the epigastric discomfort follows the taking of certain foods, but later the discomfort gradually increases until it reaches the stage of acute pain. At a certain interval after each meal, pain occurs. As the disease progresses the pain becomes more and more intense, and in severe cases may develop two hours after the taking of food; it lasts for a period of from two to four hours, during which time acid eructations may occur.

The greater the quantity of hydrochloric acid in the gastric contents, the more severe is the pain, and the less likely is starch digestion to take place in the stomach. The patient often notices that these symptoms are less severe after a meal rich in albumins—a feature readily explained by the fact that an abnormally large proportion of the hydrochloric acid is employed in satisfying these albuminous affinities.

The patient often volunteers the information that his pain is relieved by the taking of alkaline salts.

When hyperchlorhydria has existed for weeks or months, the patient becomes highly nervous, and complains of attacks of vertigo and headache, and of progressive weakness, although he seldom experiences a decided loss of weight.

Physical Signs.—*Inspection.*—The abdomen is of normal contour or may be scaphoid, although there are exceptions to this rule when intestinal tympanites with distention is present. After nervous symptoms have developed, distinct peristaltic waves may be detected.

Palpation.—Deep palpation over the epigastrium usually elicits a slight degree of diffuse tenderness.

Percussion occasionally reveals an increase in the area of stomach tympany.

Auscultation.—A decided splashing sound is frequently audible over the stomach, even in those cases in which dilatation is but slight.

Laboratory Diagnosis.—The characteristic features of hyperchlorhydria are: (1) When the stomach-tube is introduced while the patient is fasting, the organ is found to be empty, or but a small quantity of liquid is recoverable. (2) An increased total acidity of the gastric contents. (3) An abnormally high percentage of hydrochloric acid. (4) Absence of the organic acids and fermentation. (5) Microscopically, few bacteria and fungi are present. (6) Starch digestion is inhibited.

Summary of Diagnosis.—The clinical facts of greatest importance in establishing a diagnosis are the following: (*a*) The age of the patient, the disease being most common in young adults. (*b*) Occupation, students and those doing clerical work being predisposed. (*c*) The time at which pain develops after the taking of food—two to three hours. (*d*) The detection of an abnormally high acidity of the gastric contents, the result of an excess of hydrochloric acid.

Differential Diagnosis.—*Hyperchlorhydria* must be distinguished from *gastric ulcer*, a condition in which the laboratory findings are quite like those of the disease under consideration. The distinctive features between hyperchlorhydria and gastric ulcer are that in the former the epigastric tenderness is diffuse, whereas in the latter tenderness is localized and acute.

The time at which pain develops is also a valuable aid in distinguishing between these two conditions—two or more hours after the taking of food in hyperchlorhydria, whereas in gastric ulcer the pain develops immediately and is not relieved until the stomach is emptied by vomiting. The vomiting of blood is quite common in gastric ulcer (30 to 50 per cent. of cases), but rare in uncomplicated cases of hyperchlorhydria.

Gastro-succorrhœa continua chronica is the name applied to a condition in which there is a continuous secretion of the gastric juice, regardless of whether or not food is present in the stomach. Here the symptoms closely simulate those of hyperchlorhydria, the distinctive feature being that fluid obtained from the fasting stomach does not contain an excess of hydrochloric acid. Contents of the fasting stomach may equal 50 to 100 c.c. in cases where a secretory anomaly exists.

Diagnosis.—Gastrosuccorrhea is distinguished from hyperchlorhydria by the fact that in the former an excessive quantity of hydrochloric acid is found in the fluid obtained from the stomach during the fasting stage only. Another feature of great diagnostic value is that of severe physical shock, which is frequently an antecedent to gastrosuccorrhea.

Duration.—The duration of hyperchlorhydria varies according to the potent etiologic factors present, and may continue for a period of several weeks or months; relapses are not unusual.

Neuroses of Motility.

This type of neurosis is divided into two subclasses—(*a*) Neurosis with increased gastric peristalsis, which is followed by a propulsion of the contents of the stomach into the intestine before it has completely undergone gastric digestion, by the escape of gas from the stomach through the duodenum, and by the paroxysmal escape of gas from the stomach through the esophagus. (*b*) A rare condition that permits partially digested food to escape

from the stomach into the duodenum, and also permits the contents of the duodenum and intestine to be returned into the stomach.

Predisposing and Exciting Factors.—In a large percentage of all cases in which there is eructation of gas from the stomach the patient will be found to be of a hysterical temperament, or at least a neurasthenic. Much of the air is taken into the stomach through the mouth, and is later expelled as the result of increased contraction of the viscus. Authentic cases have been reported in which belching has been due to air expelled from the esophagus, and in which there was an appreciably increased motility or contractility of the stomach.

Principal Complaint.—Eructations of gas, epigastric discomfort, epigastric distention, cardiac palpitation, vertigo, and a feeling of faintness are usually experienced.

Estimation of Gastric Peristalsis.—Empty the stomach by means of stomach-tube and introduce through the tube "Salzer's test-meal, (see p. 462). If gastric motility be normal, it will be found by introducing the tube six hours later that all the lean beef and other food has passed beyond the stomach.

TORMINA VENTRICULI (PERISTALTIC UNREST).

Definition.—A condition characterized by gurgling, which begins during or immediately after the taking of food, and continues for an indefinite period.

Predisposing and Exciting Factors.—Peristaltic unrest is occasionally observed in those cases in which a compensatory hypertrophy of the stomach results from stricture of the pylorus. Cases of this particular type have been seen in which pyloric constriction resulted from carcinoma. Gastric neurosis favors and is at times a causal factor.

Principal Complaint.—Simultaneously with the eructation of gas the patient may complain of a burning sensation in the esophagus and throat, and if continued over a prolonged period, may cause a more or less constant substernal burning. The character of food taken may increase or lessen the degree of pyrosis.

Among the other annoying symptoms of the neuroses of motility may be mentioned the regurgitation of particles of partially digested food into the mouth; these particles are generally remasticated, and again swallowed.

DIMINISHED PERISTALSIS.

Diminished peristalsis may be due to relaxation of the pyloric orifice or of the cardiac orifice of the stomach.

Pyloric relaxation is a decidedly rare form of gastric neurosis, which permits the undigested food to escape into the intestine, and, when the stomach is empty, a reverse current is set up, in consequence of which the contents of the duodenum are returned into the stomach.

Detection.—Pyloric relaxation may be detected by inflating the stomach, when the upper intestine will also be seen to become distended by gas.

ATONY (ATONIC DYSPEPSIA).

This particular type of gastric disease may present itself in the form of a neurosis, although in many instances it is secondary to a chronic type of gastritis. A feature of gastric insufficiency presented by atonic dyspepsia

is that of hypomotility, and it is characteristic of this condition that the food introduced into the stomach remains in the organ too long. (See Significance of Gastric Fluid, p. 461; also p. 477.)

Principal Complaint.—The patient complains of epigastric oppression, distention of the epigastrium, and a tendency toward permanent abdominal enlargement. The appetite is impaired, and at times perverted. Eructations of gas and constipation are the rule.

The gastric fluid obtained six hours after a test-meal containing finely chopped meat will be found to contain chyme and undigested particles of the meal. Diminished motility of the stomach may also be detected by administering a capsule containing potassium iodid (coated with salol or keratin), when the saliva gives a reaction for iodin with starch-water paper as soon as the capsule passes into the duodenum. Alkaline gastric fluid prevents the accuracy of this test. (See p. 477; also Estimation of Peristalsis, p. 477.)

Differential Diagnosis.—**Hypomotility** is to be differentiated from dilatation of the stomach, the distinctive differences between these conditions appearing in the following table:

TABLE SETTING FORTH THE DIFFERENCES BETWEEN GASTRIC HYPOMOTILITY AND GASTRIC DILATATION.

Gastric Hypomotility.	Gastric Dilatation.
1. History of hysteria or neurasthenia.	1. History of gastric carcinoma, gastric ulcer, or pyloric stenosis.
2. A condition common in early adult and middle life.	2. Develops late in middle life and in aged persons.
3. No pain, but a sense of fullness and some epigastric distention.	3. Pain, should pyloric stenosis be due to carcinoma. There is often a history of the pain of ulcer, which may antedate the present trouble by several years.
4. Emaciation is but slight or absent. There is no cachexia.	4. Emaciation the rule. Cachexia common and profound in pyloric carcinoma.
5. The quantity of fluid obtained from the stomach six hours after the meal is approximately the same as that obtained one hour after a meal in the healthy individual (20 to 40 c.c.).	5. An abnormally large quantity of gastric fluid may be recovered at any time during the twenty-four hours.
6. Negative.	6. Gastric contents may contain particles of food that were ingested one or more days prior to the recovery of such contents.
7. Vomiting seldom occurs.	7. Paroxysmal attacks of vomiting every few days or weeks, at which time the patient ejects great quantities of undigested food and of fluid.
8. Chemically, the gastric contents approximates that of normal, although the hydrochloric acid is usually somewhat diminished. Lactic and fatty acids are occasionally present in small amounts.	8. In cases in which dilatation follows carcinoma, free hydrochloric acid is absent, but lactic and fatty acids are present in large quantities. Should the obstruction be due to cicatricial formation from an old pyloric ulcer, free hydrochloric acid is at first in excess, but later diminished or absent.
9. Outline of stomach is seen to be nearly normal.	9. Area of stomach tympany is greatly increased.

Relaxation at the Orifices.

Definition.—A condition that in many respects resembles paralysis of both the pyloric and the cardiac portion of the stomach.

If relaxation involves the cardia, gas and possibly liquids are returned to the mouth, but this regurgitation does not occur unless the stomach is filled. The symptoms from relaxation of the cardiac orifice must be distinguished from those of regurgitation, previously described (see p. 430), in which there are increased motility and possibly spasm of the stomach. If the cardiac orifice is unusually large, increased motility is not essential to such regurgitation. A relaxed condition of the pylorus has also been referred to in this chapter (p. 477.)

Pneumatosis.

Definition.—Pneumatosis is a condition in which the stomach is overdistended by gases.

Remarks.—On account of the gastric overdistention the diaphragm is pushed upward and causes it to exert undue pressure upon the heart, and as a result of such pressure the patient is suddenly seized with either a mild or a severe attack of dyspnea.

The cardinal symptoms accompanying pneumatosis are: dyspnea, in which both inspiration and expiration become difficult; cardiac palpitation, accompanied by the throbbing of the carotid and brachial arteries; a sense of fullness of the head, followed by a tendency toward vertigo, and dimness of vision.

The skin becomes cyanosed, the expression anxious and fearful. If the cardiac distention is not relieved by eructations or by the escape of the gas into the intestine, unconsciousness follows. Attacks of pneumatosis (acute indigestion) may be immediately relieved by the introduction of the stomach-tube into the stomach.

Cardiospasm.

Definition.—A cramp-like pain at the cardiac portion of the stomach. There are two forms of cardiospasm: (1) Acute cramp of short duration; and (2) chronic cramp, which is more or less continuous.

Exciting Causes.—Cardiospasm occurs after the introduction of irritants into the stomach, and may be sufficiently severe to cause a temporary occlusion of the cardiac orifice. In hysterical and neurasthenic persons both acute and chronic cardiospasm may develop. In the chronic type of spasm atresia has been known to follow.

Pylorospasm.

Definition.—A cramp resulting from spasmodic contraction of the ring-like muscles of the pylorus.

Pylorospasm may be either primary or secondary, the latter type being far the more common; it is usually the result of local irritation—hyperacidity, etc.

Nervous Vomiting.

Definition.—A symptom depending upon a reflex neurosis of the stomach, seen at practically all ages, although occasionally more common in adult females of hysterical temperament.

Remarks.—Nervous vomiting does not follow the taking of food nor is it preceded by nausea. The patient seldom complains of a typical vomiting of undigested foods, but of frequently expectorating particles of food.

Neuroses of Sensation.

Gastralgia (Cardialgia; Gastrodynia).—Definition.—Paroxysmal epigastric pain developing without known gastric or nervous lesions.

Predisposing Factors.—Prolonged secondary anemia predisposes markedly to the development of gastralgia. **Sex** is an etiologic factor, females being more susceptible than males. A hysterical temperament and heredity are also believed to be predisposing factors. In many instances gastralgia is believed to be reflex in origin. Overwork, anxiety, and grief often predispose to attacks of the disease. Prolonged hyperacidity of the gastric fluid and other types of gastric neurosis may be followed by paroxysmal attacks of epigastric pain.

Gastralgia is in many instances more or less closely associated with the so-called *nervous dyspepsia*, in which the gastric contents show an excess of hydrochloric acid, but it must be remembered that in such patients hyperacidity, and especially hyperchlorhydria, are remittent. Severe nervous maladies are generally experienced by those suffering from gastralgia. "I believe that a very small percentage of cases are caused by malaria" (Anders).

Principal Complaint.—Paroxysmal attacks of pain, occasionally preceded by anorexia or by a sensation of epigastric oppression, are complained of. The pain is agonizing, and radiates from the epigastrium to the back, and in the more severe types there may be girdle pains surrounding the base of the chest. In mild cases the pain lasts for but a few minutes, whereas in the severe types it may continue for one or more hours. A characteristic feature of the pain is that it is always relieved by the eructations of gas, vomiting, or by the introduction of the stomach-tube. In selected cases the patient may state that she is able to relieve the pain, in a measure, at least, by making firm pressure over the stomach.

Laboratory Diagnosis.—A chemic analysis of the gastric contents will be found to give greatly varying results—*e. g.*, the acidity may be excessive, normal, or subnormal.

Summary of Diagnosis.—The diagnosis of gastralgia is made largely by excluding organic disease of the stomach. The violent spasmodic character of the pain, the fact that the attacks occur at irregular intervals, and the effect of eructations of gas or of the introduction of the stomach-tube (relief of pain), will enable the clinician to form a correct diagnosis.

Differential Diagnosis.—The following table serves to show the distinctive clinical features of gastralgia and of the conditions for which it may be mistaken.

Gastralgia.	Gastric Crises of Ataxia.	Hepatic Colic.	Intestinal Colic.
1. History of previous attacks of epigastric pain at irregular intervals.	1. History of long-standing trouble. Signs and symptoms of locomotor ataxia present.	1. There may be a history of previous attacks which were followed by jaundice.	1. A history of previous attacks of umbilical pain, which always follow the ingestion of indigestible or unripe foods, commonly obtained.

Gastralgia.	Gastric Crises of Ataxia.	Hepatic Colic.	Intestinal Colic.
2. Most common in young females, rarely seen in children or after the fortieth year.	2. Seen in males during early and middle life. Rarely observed in women.	2. Unusual before the age of twenty, common after forty.	2. Common during childhood and in young adults.
3. Pain not excited by food, and develops most often when the stomach is empty.	3. Not affected by food.	3. Most likely to develop within one to three hours after a full meal.	3. Occurs when the stomach is full.
4. Pain is cramp-like, usually of short duration, and when severe, may radiate to the back or partially encircle the base of the chest.	4. Pain resembles closely that of severe gastric pain, encircles chest at about the nipple-line and seldom as low as the umbilicus.	4. Pain develops suddenly, is cramp-like in character, localized to the right hypochondrium, and radiates to right shoulder.	4. Cramp-like pain at the umbilicus and may radiate over entire abdomen.
5. Following the attack the patient is comparatively healthy.	5. Always displays the characteristic signs and symptoms of locomotor ataxia. (See p. 1104.)	5. The conjunctivæ and skin are jaundiced forty-eight hours after the attack.	5. There may be jaundice, due to associated catarrh of the duodenum.
6. Habitual constipation the rule.	6. Constipation the rule, but there is a frequent desire to go to stool. Diarrhea an occasional symptom.	6. Constipation the rule. Stools light or clay-colored.	6. Diarrhea often precedes the pain, and may persist for from six to twenty-four hours.
7. Urine may be that of hysteria, *e. g.*, pale in color, increased in quantity, and of low specific gravity.	7. The urine is likely to contain pus and mucus, the result of an associated chronic cystitis. Power to empty bladder limited.	7. Urine of high color, displays a heavy, yellow froth, and contains bile.	7. Urine scanty, highly colored, and rich in indican.

Hyperesthesia.—Definition.—An increase in gastric sensibility, so marked that mild irritants, when introduced into the stomach, excite discomfort and pain, which may be either dull or burning in character.

Remarks.—A hypersensitiveness of the stomach may accompany both organic and functional affections of the organ. Hyperesthesia may be one expression of a general gastric neurosis or of a general neurasthenic temperament, and is common in girls suffering from chlorosis.

Principal Complaint and Probable Etiology.—There is a sense of epigastric fullness and nausea, followed by vomiting after the taking of irritating foods. In severe cases practically all aliment taken into the stomach excites distress, and on this account the patient refuses to eat. When fasting is continued for several days, however, the epigastric distress increases. In many instances the patient will be cognizant of the fact that the

taking of certain foods, *e. g.*, strawberries, cherries, peaches, and certain shellfish, is followed by an attack of epigastric pain.

The gastric symptoms are often accompanied by such cutaneous manifestations as erythema and urticaria, with intense itching. Typical attacks of hysteria are occasionally observed.

ACUTE GASTRITIS (ACUTE GASTRIC CATARRH).

Pathologic Definition.—A condition characterized by an acute inflammation of the gastric mucous membrane.

Varieties.—(1) Acute catarrhal gastritis, marked by an acute catarrhal inflammatory process involving the greater portion of the gastric mucosa. (2) Acute toxic gastritis, in which the inflammation of the gastric mucosa has been excited by the taking of certain toxic or corrosive substances, *e. g.*, phosphorus, acids, and alcohols. (3) Suppurative (phlegmonous) gastritis, which is characterized by a suppurative process of the gastric mucosa and submucosa, and which may have resulted from either one of the preceding types.

Exciting and Predisposing Factors.—Bacteriology.—Fungi have repeatedly been recovered from the gastric contents, and ulceration of the gastric mucous membrane has been known to follow invasion of the stomach mucosa with the Achorion schönleinii (favus fungus), a case of this type having recently come under our observation. The Oidium albicans (thrush fungus) has also been recovered from the stomach of persons suffering from acute gastritis. Yeast cells and mycelia are not infrequent findings in the gastric contents of this affection, but their pathologic significance is doubtful.

In the vast majority of instances in which acute gastritis attacks those who are in apparent health, the condition is excited by the ingestion of improper foods or by overeating, *e. g.*, an excessive amount of highly spiced and of fatty foods, rich meats, vegetables, nuts, etc. Again, it may follow the eating of decomposing foods, in which case it might be said to be bacteriologic in origin, although in such instances gastritis is not the result of the action of the saprophytic bacteria ingested with the food, but is due to the activity of the ptomains generated by such bacteria. Gastritis may result from the eating of decomposing canned foods, in which bacteria and a variable amount of acetic, lactic, and butyric acids, together with certain ptomains, have been produced. (See Toxic Gastritis, p. 485.)

Acute gastritis frequently follows the too free imbibition of alcoholic stimulants, and may, in selected cases, result from the excessive use of tobacco and other narcotics.

Helminthology.—Attacks of acute gastritis frequently follow the eating of a large amount of pork infected with Trichina spiralis. Children infected with round-worms or tape-worm are subject to attacks of gastritis.

Among other predisposing factors should be considered any condition, either febrile or afebrile, in which impairment of the normal secretory or motor power of the stomach is present. (1) Bad hygienic surroundings; (2) chronic anemia, either primary or secondary; (3) malnutrition and the presence of such chronic maladies as gout, rheumatism, carcinoma, parasitic blood diseases (malaria), tuberculosis, and suppurative conditions; (4) diseases of the liver, *e. g.*, cirrhosis, gall-stone, hepatitis, jaundice (acute or chronic), and disease of the bile-duct, are also prominent predisposing factors; (5) chronic gastric catarrh usually manifests acute exacerbations

among its prominent features; (6) in children, gastritis is exceedingly common when malnutrition is evident, and during the stage of convalescence from the acute infectious diseases including scarlet fever, diphtheria, and typhoid fever; (7) a tendency toward attacks of acute gastritis is seen in adults suffering from typhoid fever, pneumonia, and remittent and intermittent fevers; (8) disease of the pancreas and of the intestines materially predisposes to attacks of acute gastritis.

Principal Complaint.—In the *milder varieties* of acute gastric catarrh (acute dyspepsia) the patient complains of epigastric pain and vomiting. The pain may begin as uneasiness, which is followed by fullness, a sense of pressure, and distress. Among other symptoms are unusual thirst, eructations of gas, and possibly of liquid, which are later followed by an increased flow of saliva. The vomiting is preceded by nausea. The vomitus at first contains the undigested food with a large amount of mucoid material. Should vomiting continue for some time, the ejecta becomes bile-stained, and in severe types may contain blood.

The patient complains of an unpleasant taste, that his tongue feels thickened from its heavy coating, and that there is a peculiar sticky covering over his teeth. The condition of his tongue varies at different times during the day, being now dry and parched, and again, bathed in saliva. Prostration is not a conspicuous symptom.

In the more *severe types* of gastritis the symptoms just described are present, but are more intensified, constitutional symptoms, *e. g.*, prostration, severe headache, mental dullness, cardiac palpitation, numbness and tingling of the extremities, appearing early. Spots appear before the eyes, and while reading the letters become confused. Not infrequently an erythematous eruption appears that may involve any portion of the body or may be general. Intense itching of the skin and urticaria are by no means uncommon.

Thermic Features.—In the milder types of acute gastritis the temperature is but slightly, if at all, elevated, but in severe cases it will be found between 100° and 103° or 104° F. Fever is of but short duration, and usually subsides soon after the stomach is thoroughly emptied.

Physical Signs.—Inspection.—The patient presents an appearance of distress, and if the pain is severe, the cheeks are somewhat sunken, the expression anxious, the attitude that assumed in abdominal cramp, and there may be alternate paling and flushing of the cheeks. An eruption may develop, although it is by no means characteristic of acute gastritis. This eruption may be in the form of rough, erythematous blotches that may coalesce to form a more or less complete erythema. Urticaria may be associated, and the entire surface of the body become involved. Underneath the arms and in the inguinal regions clusters of elevated nodules are often seen.

The tongue is at first red, glazed, and moistened, but a few hours later it becomes dry and parched, and by the second or third day it is heavily coated at the base and center. Tooth-marks at the edges of the tongue are common.

The abdomen is at first somewhat distended; but after vomiting has been severe, it may be scaphoid in outline. Herpes labialis commonly develops during the course of acute gastritis.

Palpation.—Deep pressure over the epigastrium elicits a variable degree of localized tenderness.

Percussion.—This method of diagnosis is of but limited value in acute gastritis. A variable degree of dullness is by no means uncommon early during acute gastritis.

Laboratory Diagnosis.—The laboratory findings are in no way characteristic of this affection. The percentage of free hydrochloric acid may be excessive, diminished, or normal, and the same is true of the total acidity.

The urine is usually rich in indican, highly colored, of high specific gravity, and during the severity of the attack the quantity is lessened. If the patient suffers from intense thirst, which is followed by the taking of large quantities of water, the urinary secretion may be increased, the specific gravity low, and the color light.

Summary of Diagnosis.—The clinical history, and particularly a history of the taking of indigestible foods, alcohols, narcotics, and the like, is important. A sudden rise of temperature, followed by a rapid fall at the end of from the first to the third day, is also of clinical importance. It is necessary for the clinician to recognize acute gastritis when it develops as a complication of some one of the acute infections. The character of the vomiting, the epigastric pain and tenderness, together with the cutaneous manifestations, *e. g.*, pallor, erythema, urticaria, are usually sufficient to enable one to arrive at a diagnosis.

Differential Diagnosis.—Acute gastritis may be confounded with **measles, acute nephritis, epidemic meningitis,** and certain other acute infectious conditions. The following table sets forth the distinctive differential features between acute gastritis, smallpox, and scarlet fever.

TABLE SHOWING THE CLINICAL DIFFERENCES BETWEEN ACUTE GASTRITIS, SMALLPOX, AND SCARLET FEVER.

Acute Gastritis.	Smallpox.	Scarlet Fever.
1. History of dietetic errors the rule, and probably of previous attacks.	1. May be history of exposure to smallpox.	1. May be history of exposure or of an epidemic.
2. No chill.	2. Severe rigor at onset.	2. Chill or a series of chilly sensations.
3. Sense of fullness and distress in the epigastrium, which increases until there is intense pain, which may be reflected over the abdomen.	3. Epigastric pain absent although there may be soreness of the abdominal muscles.	3. No epigastric pain or soreness.
4. Pulse 75 to 100, and of low tension.	4. Pulse 90 to 120, tension fairly high.	4. Pulse strong and wiry at 120 to 140 a minute.
5 The fever is of an irregular type, ranging between 99° and 103° F., and usually subsides when the stomach is thoroughly empty. No secondary fever.	5. Temperature 102° to 104° or 105° F., and of the continuous type early, and falls after the eruption develops. Secondary fever four to six days later.	5. Temperature 102° to 105° F., and of the continuous type, declining by end of first week.
6. Throat symptoms absent.	6. Marked soreness upon swallowing and the entire mucous membrane of the throat is reddened.	6. Intense reddening of the mucous membrane of the throat. The child usually holds his head in one position, and complains of severe angina upon swallowing. There is swelling of the glands of the neck.

Acute Gastritis.	Smallpox.	Scarlet Fever.
7. Diarrhea common.	7. Constipation.	7. Constipation.
8. When in bed the patient usually lies with his thighs somewhat flexed upon the abdomen and the chest inclined forward.	8. Rests upon his back, and hesitates to move about the bed on account of muscular soreness.	8. Patient very restless.
9. Urine free from albumin. Contains indican.	9. Febrile albuminuria present. Hematuria in malignant cases.	9. Trace of albumin early; post-scarlatinal nephritis common after second week, when renal casts are present.
10. With the onset the skin is pale, blanched, cold, and clammy.	10. Face flushed and skin intensely hot.	10. Entire skin flushed, but more marked about the face and neck. Skin is also hot to the touch.
11. Rough, erythematous eruption may appear within the first twelve to forty-eight hours. Eruption is in the form of blotches, and between these are seen slight elevations of urticaria.	11. Eruption is detectable on the second day, when there is a fine erythema on the inner surface of the thighs and arms, and, later, a shot-like eruption over the forehead and about the wrists. Vesicles and pustules appear later (hemorrhagic eruption) in malignant cases.	11. Within the first thirty-six hours the entire body becomes an intense scarlet, so that it is possible to write with the finger upon the chest or back.
12. There may be headache and vertigo	12. Intense headache early, and severe pains in the muscles of the back, loins, and extremities.	12. Headache the rule.
13. Respiratory symptoms negative.	13. Harsh, non-productive cough, with the physical signs of bronchitis.	13. Respiratory symptoms negative unless they occur as a complication.

Duration.—The milder cases of acute gastritis go on to recovery in from one to three days, whereas in the more severe types the duration is somewhat longer.

Complications.—Among the most frequent complications of acute gastritis should be mentioned constipation, which may persist for days or weeks after the attack.

Catarrhal jaundice, probably the result of extension of the inflammatory process to the duodenum, often develops in from two to four days after an attack of acute gastritis. It is important to note in this connection that the symptoms of acute gastritis are almost entirely masked by those characteristic of jaundice. (See p. 605.)

TOXIC GASTRITIS.

Pathologic Definition.—A condition characterized by a violent congestion of the gastric mucous membrane the result of the taking of irritating substances, such as corrosive poisons, phosphorus, antimony and

arsenic, acids, etc., into the stomach. In severe cases there may be localized areas in which there is sloughing of the mucous surface.

Varieties.—(1) Moderately severe toxic gastritis, in which the toxic substances ingested are not corrosive and do not produce either ulceration or sloughing of the gastric mucous membrane, but excite congestion and swelling.

(2) Severe toxic gastritis, caused by the introduction into the stomach of corrosive substances which produce necrosis of the gastric mucous membrane and may be followed by suppuration.

(3) Acute inflammation of the gastric mucous membrane, with edema and swelling, excited by the taking of certain vegetable substances, *e. g.*, peaches, cherries, strawberries, and tomatoes.

Principal Complaint.—This will be found to vary greatly according to the type of gastritis present; even in the milder cases, however, excited by the taking of fruits and vegetables, the onset is somewhat abrupt and violent. The patient vomits incessantly, and suffers from cramp-like pain in the epigastrium. The vomitus at first contains the offending substance, but after there has been severe retching, and particularly after corrosive substances have been taken, the vomitus may contain blood. In severe cases shreds of mucous membrane are expelled with the vomitus. A few hours later diarrhea develops, and, at the same time, there may be intense thirst, burning of the mouth and throat, and dysphagia. After the diarrhea has persisted for a few hours, general symptoms, with prostration, occur.

Thermic Features.—The temperature fluctuates within wide limits, and is governed almost entirely by the character of the exciting substances taken, *e. g.*, in those cases in which stone fruits were the exciting cause, the temperature will be found to vary between 101° and 104° F. Following the taking of corrosive substances the temperature may rise suddenly to 104°, or even higher, but if the quantity taken has been large, the temperature suddenly becomes subnormal, and remains so until death supervenes. In cases with extensive necrosis that are known to recover, a continued type of temperature is the rule.

Circulatory Symptoms.—The heart action is increased in direct proportion to the degree of shock, and in cases in which corrosive substances have been taken, the pulse increases rapidly to from 120 to 160 beats a minute, is weak, thready, dicrotic, intermittent, and compressible. Even in those cases that terminate in recovery the pulse will be found to vary between 90 and 120 beats a minute.

Nervous Phenomena.—Convulsions and the various types of delirium may develop at any time during the course of toxic gastritis.

Physical Signs.—Inspection.—The patient is usually seen in bed; the expression is anxious, the skin is pale and beaded with perspiration, and the respirations are frequent. There is, as a rule, a variable degree of abdominal distention, and the patient rests with the thighs flexed upon the abdomen.

Palpation.—The skin is cold and clammy, and there is tenderness and pain upon deep pressure over the epigastrium.

Laboratory Diagnosis.—The vomitus contains the offending substance. Phosphorus, phenol, and the alkalies are readily detected in the vomited material. The vomitus usually gives a reaction for blood, irrespective of whether or not there has been necrosis of the mucous membrane. *Microscopically*, the vomitus will display red and white blood-cells, and shreds of necrotic tissue may be present.

The quantity of urine excreted is, as a rule, below that of the normal, and following toxic gastritis excited by phosphorus, mercury, and arsenic, these poisonous substances are detectable in the renal secretion. The urine is often bloody, usually of high specific gravity, and after the taking of phenol displays a peculiar smoky appearance and gives off an odor of phenol.

Albuminuria is common, and granular casts, blood-casts, and epithelial casts are rarely present.

The administration of poisons by the mouth may be followed by a rapid disintegration of the blood, in which event the hemoglobin is dissolved out of the red cells, appears free in the blood-plasma, and is excreted by the kidneys. Other evidences of degeneration of the red blood-cells may also be present, depending upon the degree of irritation offered to the gastric and esophageal mucous membrane. Either leukocytosis or leukopenia will be present, the latter occurring when the toxemia is profound.

Summary of Diagnosis.—The history of having taken some substance known to be highly irritating to the gastric mucous membrane, which act was followed by violent vomiting, convulsive seizures, epigastric pain, and a tendency toward circulatory collapse, diarrhea, and intense thirst, are almost positive evidences of toxic gastritis.

The diagnosis is further supported by the detection of eroded surfaces on either the buccal or pharyngeal mucous membranes, and by the discovery, in the vomitus, of the toxic substance taken. If the patient survives the initial shock, there may then be a diminished quantity of urine which is rich in albumin.

Complications and Sequelæ.—The chief complications are acute nephritis and suppurative gastritis. Among the sequelæ should be mentioned stricture of the esophagus and gastric ulcer, although these are uncommon unless corrosive substances have been taken.

PSEUDOMEMBRANOUS GASTRITIS.

Definition.—An inflammatory disease of the stomach characterized pathologically by the production of a false membrane upon the mucosa.

General Remarks.—Pseudomembranous gastritis is always secondary to some acute or chronic malady, *e. g.*, pneumonia, scarlet fever, smallpox, or diabetes; it may also attack debilitated children. The condition is an ex-extremely rare one, and cannot be diagnosticated antemortem.

ACUTE SUPPURATIVE GASTRITIS (PHLEGMONOUS GASTRITIS).

Definition.—An acute inflammation with suppuration of the gastric mucosa and submucosa.

Predisposing and Exciting Factors.—Phlegmonous gastritis is almost as rare as pseudomembranous gastritis, and like it, is a secondary disease. "I have observed pathologic evidences of its presence, however, in two cases that came to autopsy, both patients having died of sepsis. The suppurative process is excited by invasion of pyogenic cocci" (Anders).

The conditions that predispose to suppuration and abscess of the stomach-wall are traumatism, and injury excited by the taking of hot or of irritating foods into the stomach.

Varieties.—(1) Diffuse purulent infiltration of the gastric mucous membrane. (2) Circumscribed inflammation of the stomach-wall (stomach abscess).

Symptomatology.—The symptoms are practically those seen in any suppurative process of a viscus, and they are, therefore, not characteristic of suppurative gastritis.

Thermic Features.—The temperature may be either irregular or of the typhoid type—102° or 104° F. The patient soon enters into the so-called typhoid condition, from which he does not rally.

Epigastric pain, nausea, and vomiting are present in the majority of instances. The detection of pus in the vomitus is in no way diagnostic of suppurative gastritis.

Physical examination reveals but little, if anything, that is of diagnostic value. Instances are recorded in which abscess of the stomach-wall has been palpated through the abdomen, but such cases are extremely rare, and no certain means of diagnosticating this unusual condition is known.

Duration.—The prognosis is extremely unfavorable, the majority of cases terminating fatally in from the fifth to the tenth day.

CHRONIC GASTRITIS (Chronic Catarrh of the Stomach; Chronic Catarrhal Dyspepsia).

Pathologic Definition.—A disease characterized by a chronic catarrhal inflammation of the gastric mucous membrane, which varies greatly in intensity and may, in certain instances, result in atrophy of the gastric glands.

Varieties.—(1) **Simple chronic gastritis,** in which there is but a slight chronic inflammation of the gastric mucosa, and in which, after an Ewald test-meal, the hydrochloric acid is found to be somewhat diminished and lactic acid may be present. Pepsin and rennin are always present after an Ewald test-meal. Acetic acid is occasionally found, and the fasting stomach usually contains some mucus.

(2) **Chronic mucous gastritis,** in which the essential factors are a mild inflammation of the gastric mucous membrane, together with an excessive secretion of mucus.

(3) **Atrophic chronic gastritis** which is characterized anatomically by atrophy of the gastric glands, and clinically by impairment of the gastric function, which is marked by a diminution in, or even an absence of, free hydrochloric acid, pepsin, and rennin.

Exciting and Predisposing Factors.—Chronic gastritis probably oftenest results from—(*a*) repeated attacks of acute gastritis and the prolonged use of highly spiced and rich foods, alcohols, and narcotics. (See Acute Gastritis, p. 482.)

(*b*) Overeating, when continued for prolonged periods, has been shown to excite chronic gastritis, as has the too liberal use of iced water and cold drinks when taken at meal-times.

(*c*) Chronic gastritis may be secondary to a chronic disease elsewhere in the body, *e. g.*, uterine disease, intestinal catarrh (chronic dysentery or chronic constipation), neurasthenia, and conditions in which there are associated neuroses.

(*d*) Mechanic influence, *e. g.*, valvular heart disease, cirrhosis of the lung, pulmonary tuberculosis, cirrhosis of the liver—in short, any condition known to cause chronic venous congestion of the gastric mucous membrane.

(*e*) Not infrequently chronic gastritis is found associated with such chronic afebrile maladies as gout, chronic nephritis, essential anemia, tertiary syphilis, eczema, and diabetes.

(*f*) Repeated attacks of malaria and infection with Amœba histolytica may be followed by chronic gastritis.

(*g*) Chronic gastritis is also a prominent symptom of gastric carcinoma, gastric ulcer, and gastric dilatation, and in many instances it may precede the last-named condition for months or years.

Principal Complaint.—This will be found to vary within wide limitations in different cases and in different phases of the disease. The majority of patients will complain of certain symptoms, among which should be mentioned headache, particularly upon rising in the morning, disturbed sleep, annoying dreams, general mental depression, drowsiness during the day with wakefulness during the night. The patient does not feel rested in the morning, but rises feeling careworn and irritable, and oppressed by a general sense of languor and indisposition to do either mental or physical work.

The appetite is fickle; thus at one time there is anorexia, and following this the appetite is likely to be abnormally great.

The patient experiences a sense of epigastric oppression and fullness after eating which may later be described as a distinct pain. A burning sensation in the epigastrium and substernal burning, with the regurgitation of acid liquid substances into the throat and mouth, are not infrequent complaints. Hiccough may be annoying, and may develop at any hour during the day, although it more commonly occurs one-half to two hours after the ingestion of a full meal. The gas eructated may be offensive. The tongue and mucous membrane of the mouth are parched, and the patient at times experiences an inordinate thirst.

If there is associated gastric dilatation, vomiting is a common feature of chronic gastritis. Morning nausea, with the expectoration of a large quantity of mucus, is common.

In nearly all cases there is constipation, which may be obstinate, and which has usually continued for years, as a rule antedating the gastric symptoms.

A variable amount of cough may be present, and there is clearing of the throat after the taking of food, the result of an associated chronic pharyngitis.

If the patient suffers from chronic gastritis with atrophic carcinoma, the symptoms will include epigastric pain, progressive weakness and emaciation, frequent attacks of vomiting, and progressive anemia. (See Gastric Carcinoma, p. 499.)

Physical Signs.—Inspection.—The patient may be either fairly well nourished or emaciated; the skin and conjunctivæ are slightly pale, except in atrophic gastritis, where there is associated emaciation of the epigastrium, and the entire abdomen may be distended. The tongue appears broad and flabby, its papillæ are enlarged, and the tip and edges of the organ are reddened and show indentations or markings of the teeth. Rarely, the tongue is smaller than normal, and slightly coated at its center; the papillæ are red and prominent, and the edges are thin; still less often the tongue presents a normal appearance.

Palpation.—In selected cases there is epigastric tenderness. The epigastrium is usually found to be full, and excessive gastric motility may be detected.

Percussion.—By this means we are able to outline the boundary of the stomach. In those cases in which there is gastric fermentation or associated gastric dilatation the area of stomach tympany is increased. In cases char-

acterized by sclerosis and atrophy of the stomach-wall there will be a diminished area of stomach tympany.

Auscultation.—Distinct gurgling may be heard over the stomach both when the organ is empty and after a full meal. In mucous gastritis and in chronic gastritis with dilatation a decided succussion splash may be elicited, and may be heard at some distance from the patient's body.

Laboratory Diagnosis.—The vomitus will vary according to the length of time that elapses between the taking of and the ejection of the contents of the stomach. Ordinarily, the vomitus contains undigested food and a moderate amount of mucus. The vomiting of large quantities of mucus irrespective of the taking of food is characteristic of chronic mucous gastritis. In the chronic gastritis of alcoholics the vomitus is usually acid in reaction, although when there is an associated neurasthenic element, the vomitus may be neutral or even alkaline.

The acidity of the vomitus of chronic gastritis may be due to the presence of both free and combined hydrochloric acid, acid salts, and lactic and butyric acids, whereas alkalinity results from the presence of certain alkaline salts.

The laboratory features distinctive of the various types of chronic gastritis have been detailed under Varieties of Chronic Gastritis (p. 488). A fact to be borne in mind is that the various types of chronic gastritis are probably dependent upon a different step in the development of one and the same process. It is exceedingly difficult to estimate, from an examination of the gastric contents, the degree of pathologic change present in the gastric mucous membrane, except in those cases in which there is a marked diminution or an absence in the quantity of free hydrochloric acid.

Summary of Diagnosis.—A diagnosis is attained chiefly from the evidence obtained from the *clinical history, e. g.*, an impaired or a capricious appetite, epigastric fullness and distress, constipation, absence of extreme emaciation, and the prolonged duration of the present malady. A diagnosis is further aided and confirmed by an analysis of the gastric contents. Hyperacidity, when present, is usually dependent upon an excess of lactic and butyric acids. The recovery of an unusual quantity of fluid from the stomach after a night's sleep is positive evidence of the existence of the so-called mucous gastritis.

Differential Diagnosis.—Chronic atrophic gastritis is to be distinguished from **gastric carcinoma,** and, indeed, this is by no means an easy task in many instances, since an analysis of the gastric fluid after a test-meal gives practically the same findings in both conditions. Pain is more constant and more severe in carcinoma than in atrophic gastritis, and emaciation and cachexia are always profound in gastric carcinoma.

Mucous gastritis in which there have been associated attacks of gastralgia may be mistaken for **gastric ulcer.** The vomiting of blood and the detection of blood in a highly acid gastric fluid which shows an excess of free hydrochloric acid strongly favor gastric ulcer. The pain of mucous gastritis is never lancinating unless the stomach is empty, whereas the pain of gastric ulcer is severe immediately after the taking of food, is relieved by vomiting, and is generally absent when the stomach is empty. (See Differential Table, p. 496.)

Duration.—The duration of chronic gastritis varies within wide limitations, the majority of cases not coming under treatment until it has existed for from one to three or more years. Not uncommonly the patient will assert that he has suffered from symptoms of dyspepsia for from fifteen to thirty

years. Singularly enough, the symptoms of gastric indigestion often disappear without treatment between the ages of forty-five and sixty.

Complication.—Gastric carcinoma is probably the most frequent associated disease. Profound anemia and neurasthenia and gastric ulcer may develop.

GASTRIC ULCER.

Pathologic Definition.—A condition characterized pathologically by a clearly outlined ulcer of the wall of the stomach, surrounded at times by a variable amount of inflammation. A single ulcer may be present, but they may be multiple in number. The gross pathologic changes that characterize peptic ulcer are:

(1) It is usually round or oval in contour, and when numerous ulcers unite, the margin is irregular.

(2) At first it is superficial, but later attains considerable depth, and the floor of the ulcer extends to the submucous and even to the peritoneal coat.

(3) Gastric ulcers are usually conical in outline. The base of the cone corresponds to the mucous membrane of the stomach, and the edges of the ulcer slope in toward the apex of the cone. The apex of the cone, which is usually called the base of the ulcer, because it is the deepest part of the lesion, may correspond to the muscular layer of the stomach wall or to the peritoneal coat of the organ.

(4) The ulcers vary greatly in size, the average being that of a dime.

(5) The most frequent site of ulcer is upon the posterior wall, and near the pyloric end of the stomach. Healed ulcers leave permanent cicatrices, which, when situated near the pylorus, may produce pyloric stenosis.

(6) Perforating ulcers often excite dense adhesions to the adjacent viscera, liver, gall-bladder, colon, and duodenum, and a fistulous communication between the stomach and other hollow viscera occasionally exists.

Varieties.—Anatomically, gastric ulcer may be divided into—

(1) **Simple ulcer,** a condition in which there may be one or more ulcers of the gastric mucosa, which do not extend beyond the mucous and submucous coats of the stomach.

(2) **Perforating ulcer,** in which the gastric surface of the ulcer is comparatively small, but the ulcer tends to extend to the deeper coats of the stomach and may even perforate the peritoneum.

Clinically, there may be four types of gastric ulcer: (*a*) Those in which no symptoms are present pointing to disease of the stomach; the ulcer being detected postmortem; (*b*) cases in which the symptoms consist of the sudden development of gastric hemorrhage or of gastric perforation; (*c*) cases in which the symptoms of chronic gastritis and of gastralgia are prominent; and (*d*) typical cases displaying the characteristic symptoms, among which are pain, vomiting, gastric hemorrhage, and localized tenderness.

Exciting and Predisposing Factors.—The exciting cause of ulcer of the stomach is generally conceded to be an embolus or thrombus in one of the gastric arteries supplying the ulcerated area. The conic outline of the ulcer and the fact that its base is directed toward the mucous surface of the stomach serve to explain its thrombotic origin. Gastric ulcer is dependent upon self-digestion of a portion of the stomach-wall the resistance of which has been reduced by the diminished alkalinity of the part originally supplied by an artery that has become occluded. From this point of view, digestion of the stomach-wall is merely a process of removing necrotic tissue.

Among the **predisposing factors** are: (1) Anemia, either primary or secondary, preceded by diminished alkalinity of the circulating blood.

Coexistent with such lowered alkalinity of the blood we frequently find increased acidity of the gastric contents.

(2) *Age.*—The greatest number of cases is seen between the twelfth and thirtieth years, although ulcer may occur before the tenth year, and we have seen it during the fifth decade. Smithies' clinical analysis of 140 surgical cases gives 92 per cent. between the thirtieth and sixtieth years. Welch, in his analysis of 607 autopsies in which gastric ulcer was present, found but a single case under the age of ten. Leber found but 1 case under ten years among 226 cases of gastric ulcer. Rehn found gastric ulcer in children 9, Fenwick, 18, and Cutler 23 times.

In 390 autopsies performed at a hospital in New York, Wollenstein found gastric ulcer 5 times. Holt reports 8, and Dusser 9, cases of gastric ulcer in children, and Adler furnishes the most recent report of a case in a girl of eight.

(3) *Sex.*—Smithies* in his analysis of 140 cases shows 75 per cent. of males.

(4) *Occupation.*—Gastric ulcer is quite common among shoemakers, carpenters, tailors, and seamstresses, in the following of which occupations either an undue amount of pressure is made over the epigastrium or the individual is compelled to stoop forward, and thus forces the posterior wall of the stomach against the spinal column. The affection is common among cooks, in whom it appears to be due to the taking of extremely hot foods and food between meals. Those who overload the stomach with rich and highly seasoned foods are also frequent sufferers from gastric ulcer.

(5) *Toxic and corrosive substances*, when introduced into the stomach, are at times followed by the development of ulcer.

(6) *Traumatism* over the epigastrium is an occasional cause of gastric ulcer.

Principal Complaint.—The patient usually states that she has suffered from dyspepsia and constipation for several weeks or months before consulting a physician. There is a sense of burning in the throat, and often regurgitation of acid liquids and acid gases into the mouth occurs. The appetite is good at first, but later it becomes impaired (75 per cent. of cases), until finally, owing to a diminished desire for food and because of fear, no nourishment is taken. There is also a history of progressive loss of strength together with a moderate degree of emaciation, often 20 pounds without cachexia.

Pain.—The patient suffers from intense epigastric pain, which may at first be localized, but later radiates to the shoulder and back. It is excited by the taking of food, beginning within from one to ten minutes after either solid food or liquids are ingested, and increasing until the stomach is emptied by vomiting or by the food passing into the duodenum. The nature of the pain is characteristic, developing first as a dull ache or burning, or at times a gnawing, sensation. At times it is "sticking" in character, increasing until there is a distinct lancinating pain, which persists until the stomach is empty. Gastralgic pains may also be present in those suffering from gastric ulcer, but they are not detectable until decided prostration and anemia, with neurasthenia, are present. (See Gastralgia.)

The conditions that materially influence the pain of gastric ulcer are—(*a*) Rapid eating, improper mastication, and the ingestion of strongly acid, highly seasoned, and indigestible foods, all of which tend to increase the pain. (*b*) Rest in bed, particularly when the patient lies upon the face, is followed by an appreciable amelioration of the pain; the recumbent posture, however,

* Amer. Jour. Med. Sciences, March, 1913.

slightly aggravates the pain. (*c*) Exhaustion is also followed by increased epigastric pain, as are also anxiety and mental strain.

Location of Pain.—In the majority of instances the patient complains of a distinctly localized feeling of distress about two inches below the ensiform cartilage, although the pain may be referred to a point near the umbilicus. We have seen gastric ulcer several times at autopsies performed upon the bodies of persons who never complained of epigastric pain.

Dorsal pain occurs, and may be intense (Fig. 200). It is usually localized exactly opposite to the anterior painful point; between the tenth and twelfth thoracic vertebræ. The patient seldom, if ever, complains of pain when the stomach is empty unless there is an associated gastric neurosis.

It must be remembered that the distribution and intensity of the pain of ulcer are influenced by the presence or absence of peritoneal adhesions and associated gastritis.

Vomiting occurs in about 30 per cent. of all cases, and takes place shortly

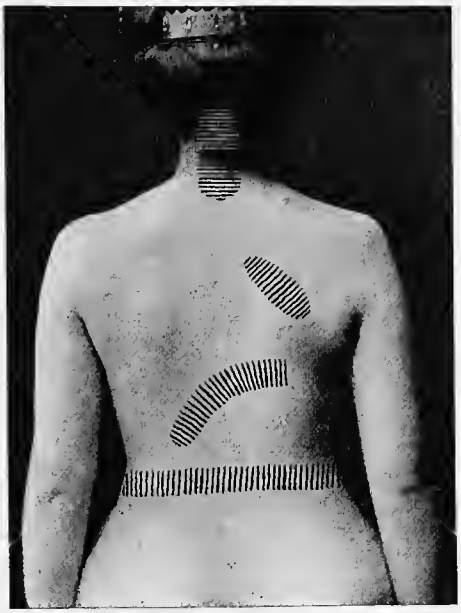


FIG. 200.—PAIN AREAS ON THE BACK.

after eating; it is not attended with retching, and is followed by almost immediate relief. In rare instances blood may be ejected without the occurrence of actual vomiting. Cases have been reported in which patients vomited from one-half to four pints of almost pure blood as the result of hemorrhage from gastric ulcer. Profuse hemorrhage is always followed by intense weakness, vertigo, dizziness, dimness of vision, and the general symptoms of circulatory collapse.

Physical Signs.—Inspection.—The skin and mucous surfaces are pale, there are evidences of emaciation; and the abdomen is scaphoid. The tongue is always glazed, coated at its center, and may show tooth marks at its edges. The buccal mucous membrane is dry at times, and again a hypersecretion of mucus may be present.

Palpation.—In typical cases it is always possible to outline a localized area of tenderness in the epigastrium; this area is about the size of a dime, unless peritoneal adhesions have formed, when it may occupy the entire epigastrium. When gastric ulcer has been present for months or years, a distinct tumor is at times palpable.

Gastroscopy.—With the aid of the gastroscope ulcer involving any portion of the explorable area of the gastric mucous membrane may be detected. By this means both the size and the depth of the ulcer, as well as its location and the degree of infiltration surrounding it, are determined. The secretion covering the base of the ulcer may be secured for microscopic study by passing a bit of cotton over the ulcer.

Laboratory Diagnosis.—The macroscopic appearance of the vomitus is not characteristic unless it contains blood. If a large hemorrhage occurs and emesis takes place soon after the escape of the blood into the stomach, the vomitus will be bright red in color. When the blood extravasates gradually into the stomach and is retained for some hours before it is vomited, the ejected liquid will be black—the so-called "coffee-ground vomit."

Chemically, the vomitus will be found to contain blood in practically all instances, although the quantity may be so small as to be unobservable by the naked eye.

On chemic examination the vomitus displays an excessive acidity. The gastric contents obtained after a test-meal owes its hyperacidity to the free hydrochloric acid present. Lactic acid is generally absent.

The **urine** is usually of low specific gravity and rich in indican.

The **hematologic findings** in gastric ulcer are those of secondary chloroanemia, *e. g.*, moderate reduction in the percentage of hemoglobin and in the number of red blood-cells in a cubic millimeter.

Illustrative Case of Gastric Ulcer.—Female, age twenty-three years; height, 5 feet 6 inches; usual weight, 120 pounds; present weight, 114 pounds.

Family History.—Parents, three sisters, and a brother living. A brother died during childhood, and a sister from scarlet fever at the age of twelve years. No history of malignancy or gastric disease in the family.

Previous History.—The patient had the usual diseases of childhood, including scarlet fever at the age of fourteen years. Has always enjoyed good health until the past year, when she noticed that her appetite was slightly impaired, that she tired easily, and that she was growing nervous.

Social History.—Single, a seamstress, following her usual vocation continuously for the past four years. The room in which she works is poorly lighted and ill ventilated. Her noon meal consists merely of a cup of tea and a moderate amount of pastry.

Present Illness.—She has felt indisposed for the past six months, but during the past two weeks has noticed that she tired more easily and that slight exercise was followed by palpitation. Amenorrhea has been present for the past three months. Eructations of highly acid material and substernal burning are annoying symptoms.

Upon taking food there is severe pain in the epigastrium, which often radiates through the entire upper portion of the abdomen and at times to the back. Pain is aggravated by the taking of solid food, although even a glass of milk may give rise to pain. Occasionally the pain increased in intensity until vomiting occurred, when relief ensued. The patient states that upon two occasions she vomited a small quantity of blood. Pain is affected by the position she assumes; thus, when resting in bed, lying upon her face, the pain is relieved, whereas the supine position often excites pain. She complains of an extremely sensitive spot in the epigastrium, and states that pressure over this area, even the pressure excited by her clothing, often causes pain.

The patient is extremely nervous and is readily excited. The latter symptom is particularly marked when she is to take food, the excitement being probably the result of fear.

Physical Examination.—*General.*—The skin and mucous surface are pale and the complexion is dusky. When sitting, the patient always inclines forward, apparently to relieve the tension of the abdominal muscles. She becomes extremely nervous on the

slightest provocation, and is very irritable. The muscles are flabby, and the skin is somewhat wrinkled. Although the patient is but twenty-three, she presents the appearance of a woman of thirty-two.

Local Examination.—Palpation.—There is a localized area of tenderness in the epigastrium, approximately one inch to the left of the median line; firm pressure over this area excites agonizing pain. The pulse is full, 80 to 90 beats a minute, but not strong.

Auscultation.—The heart-sounds are clear, and on placing the stethoscope over the base of the organ a soft systolic murmur is audible; this murmur is not, however, transmitted in the direction in which organic murmurs are usually to be heard.

Laboratory Findings.—Gastric contents obtained forty minutes after a Ewald test-meal show an excess of free hydrochloric acid (85 per cent.), and the gastric contents also gives a reaction for the presence of blood. Neither lactic nor butyric acid are present. The feces give a positive reaction for blood. Contents of the fasting stomach (40 c.c.) contained many red blood-cells.

The urine is about normal in quantity, highly colored, and rich in indican. The red blood-cells number 3,600,000 a cubic millimeter, the white cells 8100, and the hemoglobin averages 75 per cent.

Diagnosis by Induction from Clinical Data.—The age and occupation of the patient, the occurrence of progressive weakness with some emaciation and amenorrhea were sufficient evidence to prove that the patient was suffering from some serious malady that either interfered with nutrition or permitted an excessive loss through some excretion. Extreme pain in the epigastrium following the taking of food, and the additional evidence that solid food increased the pain, were considered valuable diagnostic features. Much importance attached itself to the history of the vomiting of blood, as well as to the fact that pain was at times relieved when the patient rested in certain positions. A localized area of extreme tenderness in the epigastrium went far to support the diagnosis. The laboratory findings were in themselves characteristic of gastric ulcer, an extremely high degree of acidity, together with an excess of free hydrochloric acid, being present.

Course of Illustrative Case.—The patient continued to follow her usual employment, but for a period of two weeks, despite treatment, grew gradually worse during this period. She was ordered to discontinue work and take abundant outdoor exercise in the sun; a nutritious diet, together with the necessary medicaments to enable her to retain the food was prescribed. She soon showed decided improvement, and was able to return to work in ten weeks.

Summary of Diagnosis.—A diagnosis is made as the result of a careful analysis of the clinical history, *e. g.*, the presence of etiologic factors and of such symptoms as pain, vomiting of blood, gastralgia, secondary anemia, constipation, and progressive weakness. It is also supported by the characteristic laboratory findings, *e. g.*, an excess of free hydrochloric acid, and the presence of either small or large quantities of blood in the vomitus. Feces may give reaction for occult blood. The fact that the local symptoms are intensified by the ingestion of food is characteristic. Again, the prolonged duration and tendency toward remission are confirmatory of chronic ulcer.

Differential Diagnosis.—Gastric ulcer may be distinguished from **gastric carcinoma** by the clinical history and by the knowledge obtained from an analysis of the gastric contents. (See Differential Table, p. 502.) Although hemorrhage is generally conceded to be of great diagnostic value in gastric ulcer, it is often misleading, and should be regarded as but one factor in a group of symptoms that all point strongly toward ulcer. The vomiting of blood may be due to a variety of causes. (See Acute Gastritis, various forms.)

The hemorrhage of ulcer is differentiated from that due to any of the conditions mentioned under Acute Gastritis by the distinctive symptoms of this affection.

Gastralgia may be mistaken for gastric ulcer, and, indeed, when ulcer develops in those of a neurasthenic temperament, the diagnosis may be extremely difficult, and may be attained only as the result of an analysis

of the gastric contents after a test-meal. The accompanying table, modified from Anders, shows the distinctive features between the gastric neuroses and gastric ulcer.

Gastric Ulcer.	Gastralgia.
1. History of occupation in which pressure is made over the stomach, or the eating of hot foods.	1. Patient of a neurasthenic type, and gastric symptoms often follow severe mental strain and anxiety.
2. Rare before puberty or after the age of thirty-five.	2. Seldom seen before the twentieth year, and most frequent near the menopause and after the fortieth year.
3. Progressive prostration and emaciation develop early.	3. Less marked, and may be absent.
4. There is vomiting of blood in from 30 to 50 per cent. of all cases.	4. Absent.
5. Pain is excited by the taking of food, and develops immediately or within a few minutes after the food has entered the stomach. The position of the body may influence pain.	5. Pain is experienced several hours after a meal, or when the stomach is practically empty, unless the patient suffers from gastric ulcer and gastric neurosis, in which case pain is also excited by the taking of food. Position has no effect on the pain.
6. Food excites pain.	6. Food relieves pain.
7. Pressure over the epigastrium excites pain, although rarely it causes slight relief.	7. Firm pressure is invariably followed by an amelioration of the pain.
8. The administration of nerve sedatives tends to lessen the pain.	8. Pain is less severe or absent after such treatment.
9. Gastric contents obtained after a test-meal show an excess of hydrochloric acid.	9. No constant findings in gastralgia. Fluid may display hyperacidity or be neutral or alkaline in reaction.
10. Regurgitation of acid fluid to the mouth and a burning sensation beneath the sternum are common.	10. Substernal burning less common; regurgitation of liquids and foods often present, and rumination is occasionally seen.
11. In ulcer of long standing a tumor may be distinctly palpable in the epigastrium.	11. Tumor absent.
12. In patients displaying a tumor or constriction of the pylorus gastric dilatation is present.	12. Dilatation absent.

The following table sets forth the distinguishing features between hemorrhage from the stomach and hemorrhage from the throat and lungs, but these are not sufficient evidence on which to make a differentiation between hemorrhage from the lungs and hemorrhage from the esophagus.

Hemorrhage from the Stomach.	Hemorrage from the Throat and Lung.
1. History of organic disease of the stomach or of cardiac embarrassment with tricuspid regurgitation.	1. History of pulmonary disease with cough and expectoration.
2. Previous attacks of eructations of gas, liquids, and food that excited substernal burning.	2. Paroxysmal attacks of coughing, which develop upon rising in the morning.
3. A study of the regurgitated fluid shows it to contain either hydrochloric or lactic acid, and particles of undigested food may be present.	3. Material alkaline in reaction, and may contain tubercle bacilli and shreds of elastic tissue.
4. Epigastric uneasiness precedes hemorrhage from the stomach, and nausea, faintness, and an acid taste are also experienced.	4. Substernal oppression and discomfort, an annoying sensation in the throat, and a saline taste precedes hemorrhage from the lung.

Hemorrhage from the Stomach.	Hemorrhage from the Throat and Lung.
5. Blood and gastric contents are ejected by vomiting, and later there may be coughing.	5. Blood expelled first by coughing, but vomiting is common later.
6. Macroscopically, bloody fluid may contain clots of a dark brownish color ("coffee-ground" vomitus). In profuse hemorrhage bloody fluid shows no clotting and contains particles of food.	6. Fluid is bright red in color, and its surface is beaded with froth. Small blood-clots are occasionally seen.
7. There are no external evidences of hemorrhage between the attacks of vomiting.	7. The patient continues to expectorate blood-streaked sputum for some hours or days after the hemorrhage.

Clinical Course and Duration.—Not uncommonly young females suffer from one or more ulcers, their development being separated by wide intervals of apparent health.

Extensive ulceration near the pyloric end of the stomach often results in the formation of a large mass of cicatricial tissue that may interfere with the passing of food from the stomach, in which case gastric dilatation is likely to follow. We have seen cases in which gastric ulcer perforated into the general peritoneal cavity, and in one case, that of a private patient, general peritonitis with a fatal termination occurred one week after an operation. A fatal issue also followed in another case in which the ulcer perforated into the pleura.

Complications.—Stenosis of the pyloric orifice or of the cardiac orifice and perforation into the pleura (subdiaphragmatic pneumothorax), the colon, the gall-bladder, or the general peritoneal cavity are among the most serious complications of this malady.

HOUR-GLASS STOMACH.

Pathologic Definition.—A condition, either congenital or pathologic, characterized anatomically by a contraction of the stomach-wall near its center, and of sufficient degree to permit only a small opening through which the pyloric and cardiac portions of the stomach communicate.

Predisposing and Exciting Factors.—Hour-glass contraction of the stomach is a physiologic process occurring during the height of digestion, and at times when the stomach is being emptied of its contents; an attempt to outline the stomach by means of the *x*-rays, therefore, should be made only when the organ is approximately empty. Extensive gastric ulcer, followed by the formation of cicatricial tissue, may cause hour-glass contraction. Carcinoma situated near the center of the lesser curvature may be surrounded by adhesions and a general infiltration of the stomach-wall; this in turn may result in hour-glass contraction. Congenital hour-glass contraction seldom causes symptoms characteristic of the condition.

Principal Complaint.—In pathologic hour-glass contraction the patient gives a history of chronic dyspepsia, gastric ulcer, or gastric carcinoma. Rarely he states that the trouble followed the ingestion of highly irritating substances, caustics, or hot foods. The symptoms of hour-glass contraction are those of general dyspepsia unless ulcer or new-growth of the stomach-wall is present.

Physical Signs.—**Inspection** is, as a rule, negative, although there may be a distinct peristaltic wave over the cardiac portion of the stomach; this wave is, however, shorter than the normal wave. When the stomach is

distended by gas, the constriction may be apparent through a thin relaxed abdominal wall.

Percussion, and particularly **auscultatory percussion,** enables one to distinguish clearly the two expanded extremities of the stomach. When the stomach is distended, its contour is readily outlined by percussion.

Auscultation.—A succussion splash is extremely common in hour-glass contraction, and is likely to be limited to the cardiac expansion of the organ.

Laboratory Diagnosis.—A characteristic feature of hour-glass contraction is the fact that an attempt to remove fluid from the stomach by means of the stomach-tube is unsuccessful, for the reason that the fluid taken with the test-meal may have passed beyond the contracted portion of the stomach.

CARCINOMA OF THE STOMACH.

Pathologic Definition.—A carcinomatous infiltration of the stomach-wall involving the mucous, and later the submucous, muscular, and peritoneal coats. Gastric carcinoma is usually primary, although secondary involvement of the stomach is by no means uncommon. The variety of carcinoma involving this organ is, as a rule, composed of columnar epithelial cells. The entire wall of the stomach may be sclerotic, or there may, in other instances, be colloid degeneration or mere involvement of the glandular tissue, accompanied by softening. Both encephaloid and cirrhotic changes may follow the disease when it develops from the glandular structures.

Exciting and Predisposing Factors.—The exciting cause of carcinoma is, up to the present time, doubtful. Among the predisposing factors are:

Age.—In Welch's analysis of 2038 cases, 75 per cent. developed during the fourth, fifth, and sixth decads. Of 3257 cases reported in the literature, 2.5 per cent. developed before the age of thirty. Osler and McCrae,* in a review of the literature, found reports of 6 cases occurring in children under six years of age, and 13 cases that developed between the sixth and twentieth years. These authors, in their report of 150 cases of gastric carcinoma, gave 4 per cent. of cases under the age of thirty.

Heredity has been shown to figure in from 12 to 15 per cent. of cases.

Gastric ulcer materially predisposes to a later development of carcinoma, as was well exemplified in Hirschfeld's analysis of 900 cases, in which previous disease of the stomach was found to have existed in 5.6 per cent.

Chronic gastric catarrh is also regarded as a predisposing factor. Carcinoma of the stomach may be secondary to involvement of the liver, gall-bladder, bile-ducts, duodenum, pancreas, and intestines.

General Remarks.—The clinical manifestations of gastric carcinoma are varied, and the disease may exist without appreciable symptoms referable to disease of the stomach, in which case the true character of the condition is not revealed until autopsy. Again, cases of gastric carcinoma have been seen in which the leading symptoms were progressive emaciation, weakness, secondary anemia, and cachexia, without local manifestations.

Secondary carcinoma of the stomach, and even primary involvement of this organ, may be masked by peritoneal adhesions and by carcinoma of adjacent structures, *e. g.*, of the liver and pancreas. Typical cases of gastric carcinoma are readily diagnosed by the characteristic features of the disease.

Principal Complaint.—In the early stages of gastric carcinoma the patient complains of chronic gastritis. Not rarely the condition begins abruptly, although it may have developed insidiously with a gradual increase

* New York Med. Jour., April 21, 1900, p. 581.

in the severity of all the symptoms. The pain is progressive, becoming more and more intense from week to week, and is accompanied by increasing weakness and loss of flesh. The appetite varies from time to time, but, as a rule, there is a steady decline. In other cases there is a desire for food, but the patient does not eat for fear of epigastric pain.

The patient suffers from pain after the taking of food, and even when the stomach is empty. Many cases complain of a continual sense of oppression in the epigastrium, which amounts to true cardialgia, occurring in from two to four hours after eating. In typical advanced cases the patient suffers almost continually from pain, which is not greatly increased by the taking of food.

The character of the pain may vary somewhat in different cases, but, as a rule, the patient complains of a weight or of a dull boring pain in the epigastrium. Lancinating pains are also frequent, and may depend, in part, at least, upon peritoneal adhesions.

The distribution of the pain in carcinoma is by no means characteristic, and may be localized to the epigastrium or slightly to the left of the median line, and at times as low as the umbilicus. Occasionally we have observed associated gastroptosis where the pain was in the left inferior abdominal quadrant. The pain may radiate to the back, shoulders, loins, and, rarely indeed, it is reflected for some distance over the upper portion of the abdomen. Pressure over the epigastrium always excites acute pain, which is followed, for a period of some hours, by intense, dull pain.

Vomiting occurs late during the course of gastric carcinoma, and the patient may give a history of having vomited material presenting the appearance of "coffee-grounds," or, less often, there may be vomiting of pure blood. (Other conditions known to cause the vomiting of blood will be found under Gastric Ulcer, Differential Diagnosis, and Laboratory Diagnosis, pp. 494, 495. Early during gastric carcinoma vomiting may be dependent upon a catarrhal inflammation of the gastric mucosa, in which case it may take place shortly after eating. In advanced carcinoma the vomiting occurs in from two to four hours after food has been ingested, unless an associated gastric ulcer is present. In cases in which the carcinomatous growth has caused pyloric stenosis and there is associated dilatation of the stomach, vomiting occurs every three to six days, or even at longer intervals, at which time the vomitus presents the naked-eye appearance of gastric dilatation. (See Gastric Dilatation, p. 507.)

Constipation develops during the course of gastric carcinoma, and, indeed, in many cases it precedes disease of the stomach.

Vertigo, shortness of breath, headache, mental hebetude, and a general sense of nervousness are described by all who suffer from secondary anemia.

Physical Signs.—Inspection.—During the first few months inspection is negative, but after the disease has progressed, there is decided emaciation, the face is wrinkled, the skin cachectic, and there is a puffiness beneath the eyes. The conjunctivæ and mucous surfaces are pale, and the scleræ show yellowish deposits here and there. Inspection of the abdomen is negative, except when there is marked tumor formation, in which case the growth may be seen through the thin abdominal wall. (See Method of Inspection, p. 423.) The tumor may occupy the epigastrium, or may be found at almost any point between the epigastrium and the pubis, and to the left of the median line. Late during the course of gastric carcinoma edema of the ankles may be seen.

Palpation.—Pressure over the epigastrium, and particularly over the site of the tumor, is always followed by an increase of pain (see Fig. 201). When tumor is present, it may or may not change its position with respiration.

Percussion.—In cases complicated by pyloric stenosis and gastric dilatation, both percussion and auscultatory percussion are of great service in outlining the stomach. Percussion also gives valuable information regarding secondary involvement and consequent enlargement of the liver.

Auscultation.—A splashing sound is heard when carcinoma is complicated by dilatation.

Gastroscopy.—When the carcinomatous lesion involves the explorable portion of the stomach, the lesion may be inspected satisfactorily, and in this manner both its extent and the degree of ulceration, if any should be present, may be determined. It is important, in this class of cases, to ascertain the degree of stenosis of the pylorus if any should exist. (See Plates IX and X.)

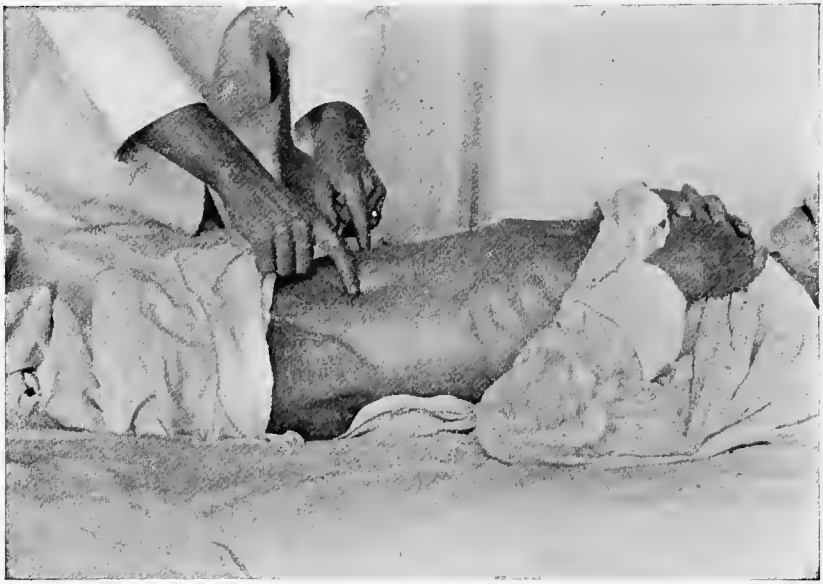


FIG. 201.—METHOD OF DETERMINING THE DEGREE OF ABDOMINAL TENSION OVER VARIOUS AREAS OF THE ABDOMINAL SURFACE.

The abdominal tension is increased above the umbilicus near the median line in gastric carcinoma. In new growths of the abdomen the abdominal tension is always increased immediately overlying the growth. A localized increase in tension is present in limited areas of peritonitis. (See Acute Appendicitis, p. 535.)

Laboratory Diagnosis.—The vomitus of gastric carcinoma contains particles of undigested food. It may be dark in color, and display a heavy brownish sediment, resembling coffee-grounds. This coffee-ground sediment is due to an admixture of blood that has escaped into the stomach, and been acted upon by the gastric juice. Shreds of carcinomatous tissue have been found in the vomitus.

Microscopically, the vomitus will be found to contain the food that has been ingested, blood-crystals, free blood-pigment, and scales of epithelial cells. Yeast fungi and sarcinæ are frequent findings. A large non-motile bacillus (Fig. 199), the Boas-Oppler bacillus, may be recovered from the liquid.

Chemically, gastric contents obtained after a test-meal is, as a rule, devoid of free hydrochloric acid, although in a small percentage of cases a trace is found. Rarely do we find the normal amount of free hydrochloric acid present. When hydrochloric acid is diminished or absent from the

gastric contents, lactic acid in large quantities will probably be present, and butyric acid is also common. Contents of the fasting stomach may show particles of mucous membrane, blood, and usually give a reaction for blood-pigments.

Secondary anemia is a constant feature of gastric carcinoma, the hemoglobin falling to between 70 and 30 per cent.; the red cells may be reduced to 2,000,000 in a cubic millimeter, whereas the leukocytes may be subnormal, or increased in number (see p. 351). Semenoff* experimented with the Salkowski and Kojo method of determining colloidal nitrogen in urine as an aid in the diagnosis of cancer of the internal organs. (1) To 100 c.c. of mixed twenty-four-hour urine freed from albumin, add zinc chlorid to saturation. (2) Let the saturated solution stand twenty-four hours, and pass through an ash-free filter. (3) Wash the precipitate five times with a saturated solution of zinc chlorid to remove all nitrogenous substances. (4) Incinerate the filter and precipitate by the Kjeldahl† method (see special works on Laboratory Diagnosis) and determine the amount of cyanogen. The total nitrogen in the urine is also determined by the Kjeldahl method, and the relation of the colloidal to the total nitrogen determined. (*a*) The coefficient of Salkowski and Kojo in healthy individuals is always low (maximum, 1.79). (*b*) There is always an increase of colloidal nitrogen in cancer of abdominal organs.

Neubauer and Fisher have suggested the *glycyltryptophan test*, and their observations have been repeated by Warfield and Friedman.‡ From a review of the literature upon this subject we are forced to conclude that while the test presents certain valuable possibilities, it is as yet too new to justify the authors in giving it complete description in this volume.

Late during the course of the disease, owing to the disintegration of the red blood-cells and the liberation of hemoglobin, the *urine* is high in color, and may contain the various compounds of iron. Traces of albumin, acetone, and diacetic acid are occasionally present, and indican is an almost constant finding.

Illustrative Case of Gastric Carcinoma.—Mr. E., aged fifty-six years; height, 5 feet 9½ inches; usual weight, 185 pounds; present weight, 157 pounds.

Family History.—One brother died of pneumonia and a sister of tuberculosis. A younger brother is living and in apparent health. Father died of carcinoma of the lip at the age of fifty, and a paternal uncle of carcinoma of the rectum.

Previous History.—Had the diseases of childhood, pneumonia at twenty-five, and typhoid fever at thirty-eight. Following the attack of typhoid he gained weight somewhat rapidly, and remained in perfect health until his present illness.

Social History.—Married, has three sons and two daughters living and in apparent health. He uses tobacco in moderation, but drinks no alcoholics.

Present Illness.—About six months ago he observed that he suffered from a variable amount of discomfort in the region of the stomach which increased in severity until, at the present time, epigastric pain exists during the greater portion of the twenty-four hours. Eating does not appear to aggravate the pain at the time such food is taken, but within the course of from two to four hours following a meal the pain is most intense, and has been so severe that it became necessary to administer opiates for its relief. He frequently vomits in from two to four hours after the taking of food, the vomitus containing undigested food; at times the liquid portion of the vomitus has been slightly stained with dark blood ("coffee-grounds"). He states that loss of strength has been progressive since the onset of the present illness, that he is extremely short of breath, and becomes fatigued after moderate exercise. There has been obstinate constipation until within the past month, during which time he has suffered from two mild attacks of diarrhea, both of which alternated with constipation.

* Clinical Significance of Determination of Colloidal Nitrogen in Urine by Salkowski and Kojo Method, in Diagnosis of Cancer of Internal Organs, Roussky Vratch, May 5, 1912.

† Zeit. f. Analyt. Chem., 1883, vol. xxii, p. 366.

‡ New York Med. Jour., August 17, 1912, p. 317.

Physical Examination.—*General.*—The skin and mucous surfaces are pale, there is a variable degree of yellowing of the face, neck, and hands, and the skin is decidedly wrinkled, hanging in folds about the face and neck. The skin of the arms and lower extremities also shows wrinkling the result of emaciation. The abdomen is scaphoid, and upon deep inspiration an undue movement is seen in the epigastrium, which appears to result from a small mass descending with inspiration. The muscles are extremely soft and flabby, the skin is somewhat dry, the tendon reflexes are decreased, and there is moderate trembling when he extends the hands.

Palpation.—A distinct, small nodular mass is detectable, approximately one-half inch above the umbilicus, and an equal distance to the left of the median line. Pressure over this mass produces extreme pain.

Percussion.—The lower border of stomach tympany extends fully 3½ inches below, and for a distance of one inch to the right of, the umbilicus.

Auscultatory percussion also outlines the area of the stomach.

Laboratory Findings.—The vomitus has at times been bloody—"coffee-grounds" in appearance. Gastric contents recovered after a test-meal did not show the presence of free hydrochloric acid. Lactic and butyric acids were present, but the total acidity was only 40. Chemic and microscopic analysis showed the presence of blood. Sarcinæ ventriculi and the Boas-Oppler bacillus were also present.

The urine was scanty, and had a specific gravity of from 1.014 to 1.022; a trace of albumin was present at times, as was also a decided reaction for indican. The hemic changes were those of secondary anemia with slight leukocytosis.

Diagnosis by Induction from Clinical Data.—The age of the patient, the progressive loss in weight, and the family history of carcinoma were considered sufficient in themselves to suggest the nature of the disease. The fact that the patient had a desire for food, and that eating did not increase the pain at the time the food was taken, were considered sufficient evidence to differentiate the condition in question from gastric ulcer. Again, epigastric pain, which was more or less continuous, was regarded as a strong feature in favor of gastric carcinoma. Vomiting, while not only a feature of carcinoma, suggested this disease from the fact that it occurred some hours after the taking of food; "coffee-ground" vomit was also occasionally present. An analysis of the gastric contents was of further importance, since it showed that free hydrochloric acid was absent and that lactic and butyric acids were present.

Course of the Disease.—Approximately seven months after the first definite signs had developed the patient complained of symptoms referable to pyloric stenosis and gastric dilatation. The physical signs of dilatation were now present, and the patient occasionally vomited large quantities of material that often contained particles of food ingested one or more days before the paroxysmal attack of vomiting. Treatment gave relief only in so far that the pain was mitigated and the patient enabled to take more nourishment. The disease progressed from bad to worse, terminating fatally in one and one-half years. Operation was contraindicated, as the patient did not come under observation early enough.

Summary of Diagnosis.—The diagnosis is usually based upon the age, the family history, and the history of preëxisting disease of the stomach or of carcinoma in other portions of the body. This evidence, together with progressive weakness, emaciation, and the characteristic pain (continuous and most intense some hours after eating), is of great importance in formulating a diagnosis. The occurrence of coffee-ground vomiting points to gastric carcinoma. Contents of the fasting stomach may contain blood.

The absence of free hydrochloric acid and the presence of lactic and fatty acids, together with the symptoms just outlined, are sufficient evidence upon which to base a diagnosis.

Differential Diagnosis.—The following table shows the distinctive features between chronic gastritis, gastric ulcer, and gastric carcinoma (elaborated from Anders).

Chronic Gastritis.	Gastric Ulcer.	Gastric Carcinoma.
1. Not confined to any age. More common in the middle-aged or in elderly persons.	1. May occur in middle-aged persons, but is more frequent in females from fifteen to thirty-five years of age.	1. Most common in elderly persons; rarely seen in persons under thirty years of age.

Chronic Gastritis.	Gastric Ulcer.	Gastric Carcinoma.
2. Pain in the epigastrium somewhat augmented by food; soreness is also present. Both are constant, although comparatively slight.	2. Pain in the epigastrium follows the taking of food; subsides when this is digested or vomited. Intermissions in the pain of considerable length are frequent.	2. Pain often paroxysmal, severe, and lancinating. Little or not at all affected by food. Pain rarely remits; never intermits for any considerable length of time.
3. Symptoms of indigestion marked.	3. Symptoms of indigestion may be but slight.	3. Symptoms of indigestion prominent. Anorexia the rule.
4. Vomiting may be present and may develop at any time during the day or night.	4. Vomiting may be present and always gives immediate relief from pain; as a rule, it takes place from a few minutes to one-half hour after eating.	4. Vomiting a very frequent symptom. Occurs from two to four hours after eating.
5. Hemorrhage rare.	5. Hemorrhage from the stomach common. Stools and vomitus may contain blood.	5. Hemorrhage not profuse, but frequently coffee-ground-like. Stools also give reaction for blood.
6. Upon making firm pressure over the stomach there may be slight diffuse tenderness, but pressure never excites actual pain.	6. Localized area of tenderness in the epigastrium, pressure over which excites extreme pain. Tenderness and pain are occasionally present at the lower dorsal vertebræ.	6. Epigastric tenderness not essential, although usually present. Firm pressure may or may not cause pain. The area of tenderness may be between the ensiform and the umbilicus, and to the left of the median line, or when there is gastroptosis, it may be found as low as the crest of the ilium.
7. Tumor of the epigastrium and abdomen absent.	7. In long-standing ulcer there may be a palpable mass at the pylorus.	7. The rule is to detect a mass in advanced cases.
8. Temperature normal.	8. Temperature normal	8. Intermittent attacks of slight fever may occur, but temperature is often subnormal.
9. Emaciation and cachexia absent.	9. Pallor and debility are extreme.	9. Progressive loss of flesh and of strength. Cachexia and hypertrophy of the peripheral lymphatic glands are prone to occur.
10. Contents of stomach contain free hydrochloric acid.	10. Hydrochloric acid in excess.	10. No free hydrochloric acid in stomach contents.
11. No lactic or fatty acids after the Boas test-meal.	11. No lactic or fatty acid after Boas test-meal.	11. Lactic acid present after Boas test-meal. Butyric acid common.
12. No dropsy.	12. No dropsy.	12. Edema of the ankles common.
13. Disease may be relieved or cured; is often of very prolonged duration.	13. Duration uncertain; may get well or may go on rapidly to perforation.	13. Average duration, two years.

Complications.—Chief among the complications should be mentioned: (*a*) Perforation of the stomach-wall, which, according to Brinton, occurs in 3.3 per cent. of all cases; (*b*) secondary involvement of the liver and of the gall-bladder; (*c*) involvement of the transverse colon by extension by contiguity; (*f*) pulmonary edema, effusion into the pleural sacs, myocarditis, or bronchopneumonia often end the scene; (*g*) nervous symptoms may be regarded as complicating conditions, and materially hasten a fatal termination, *e. g.*, the patient becomes somnolent and at times comatose.

Duration.—The course of gastric carcinoma is brief, death usually taking place within two years from the time the diagnosis is made. The average duration of the disease is from nine to fifteen months. "When it occurs in emaciated persons, it pursues a slower course than when occurring in fleshy individuals" (Anders). The disease tends to run a more rapid course in persons under the age of thirty.

DILATATION OF THE STOMACH.

Pathologic Definition.—An acute or chronic condition characterized by an increase in the size of the stomach. In the chronic form, and after dilatation has existed for some time, the gastric wall becomes thinned and atrophy of the glandular structures of the mucous membrane is generally present. A variable degree of pyloric obstruction exists in a large proportion of all cases, and downward displacement of the organ, with dragging down of the pylorus, invariably follows.

Exciting and Predisposing Factors.—Pyloric stenosis is the most frequent exciting factor, and may be due to:

(*a*) Carcinoma of the stomach, cicatrix resulting from gastric ulcer, excessive development of fibrous tissue of the pylorus, and cicatrices the result of the ingestion of corrosive poisons.

(*b*) Pressure upon the pylorus the result of hepatic or pancreatic disease or from abdominal tumor (enlarged lymphatic glands, enlarged gall-bladder, impaction from gall-stone).

(*c*) Chronic peritonitis may result in the formation of fibrous bands that encircle and constrict the lumen of the pyloric orifice.

(*d*) Congenital pyloric stenosis is also recognized as an exciting factor.

Gastric dilatation may exist without the presence of pyloric stenosis, but in the majority of such cases the degree of dilatation is not pronounced.

(1) Prolonged gastritis with atrophy of the muscular coat markedly predisposes to, and may even be the exciting cause of, gastric dilatation.

(2) Overdistention of the stomach, a condition, as a rule, excited by overeating or excessive drinking, resulting in the generation of gas and consequent distention of the organ. This type of dilatation is also seen in acute dyspepsia.

(3) Nutritional disturbances of the gastric wall in which there is a weakening of its muscular coat, as in wasting diseases (carcinoma, pernicious anemia, tuberculosis, chronic nephritis).

(4) Impaired muscular power of the stomach, in consequence of which an abnormal quantity of food is retained in the stomach.

(5) Displacement of the stomach (gastroptosis).

Acute Dilatation.—Acute gastric dilatation may develop during—(*a*) the course of infectious fevers, and is possibly dependent upon parenchymatous degeneration of the muscular coat of the stomach.

(*b*) Overloading the stomach with both liquid and solid food is followed by a variable degree of temporary dilatation.

(*c*) It may occur when the nervous energy of the body is at low ebb (during shock). Acute dilatation may also arise during the course of acute mania, dementia, melancholia, and monoplegia.

(*d*) The sudden development of an abdominal mass, an aneurism, abscess, or ulcer, may cause pyloric obstruction, and consequently acute dilatation.

Principal Complaint.—This varies widely in each particular case, since the patient first complains of the results of the initial or causative disease. The symptoms of gastric dilatation are consequently added to those of gastric ulcer, carcinoma, atrophic gastritis, and other affections.

The patient always complains of *intense thirst* throughout the entire course of dilatation, a symptom that, von Weinig claims, is due to the fact that the stomach is unable to take up liquids; extreme hunger probably results from the same cause. Soon after dilatation has occurred the patient notices a diminution in strength, exhaustion following slight exertion, and a progressive though moderate loss of weight.

There is a history of violent attacks of vomiting, during which one or more gallons of liquid material mixed with particles of undigested food are expelled. (See Laboratory Diagnosis, p. 507.) Eructation of gas is one of the most annoying symptoms.

The patient complains of cramps in the muscles of the calf, thigh, arms, and occasionally of the abdominal muscles. Tetany and a tetanic state of the muscles have been observed in those suffering from gastric dilatation. An almost constant symptom is a dragging or weight-like sensation over the upper portion of the abdomen.

As a result of impaired absorption of liquids from the stomach, and the fact that liquids taken by mouth do not reach the intestine, obstinate *constipation* is experienced. The patient voids but a small amount of *urine.* Late during the course of gastric dilatation horrible dreams, nightmare, and insomnia occur. Extreme distention of the stomach by gas may be followed by attacks of dyspnea, palpitation, and cardialgia.

Physical Signs.—Inspection.—That portion of the abdomen above the umbilicus is, as a rule, usually prominent, except immediately after a paroxysmal attack of vomiting, when it is likely to be scaphoid in shape. Prominence of the abdomen, the result of gastric dilatation does not involve the region of the epigastrium, but usually takes place at the umbilical line, and may be most marked in the left inferior abdominal quadrant. If there is a high grade of gastric dilatation, the left half of the abdomen presents two distinct grooves, the upper one of which corresponds to a line drawn from the umbilicus on a parallel with the left nipple, and is limited to the left superior abdominal quadrant (Fig. 164). Asymmetry of the abdomen is not conspicuous in either acute or chronic gastric dilatation unless gastroptosis is also present.

By distending the stomach with gas, either with the aid of the stomach-tube or by the administration of a seidlitz powder, the greater and lesser curvature of the stomach can be readily outlined. A peristaltic wave may also be detected over the left abdominal hemisphere. The skin and conjunctivæ are, as a rule, anemic, the tongue is heavily coated, and the mouth and throat are unusually dry.

Palpation.—In patients with a thin abdominal wall the stomach may

be outlined. The movements of the organ can be distinctly felt over almost the entire surface of the stomach.

Palpation combined with auscultation generally gives the most reliable signs of the presence of gastric dilatation, and is performed in the following manner:

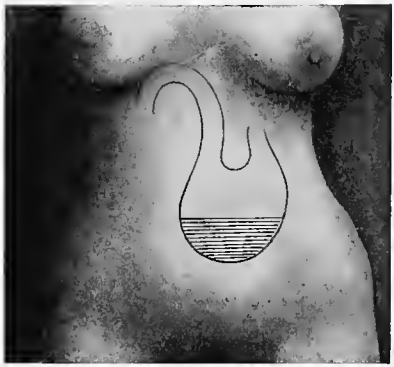


Fig. 202.—Displacement of Stomach, Illustrative of Private Case.

(*a*) Place the bell of the stethoscope over the center of the dilated stomach, and let it be held there by an assistant.

(*b*) Place the left hand over the pylorus and the right over the lower portion of the stomach, when, on making pressure alternately with the left and the right hand, a splashing sound is elicited.

Percussion.—By this means the area of stomach tympany may be outlined, but in order to be of clinical value, the stomach should be markedly distended when it is comparatively empty. In gastric dilatation stomach tympany may be elicited at any point to the left of the median line, and between the margin of the ribs and the pelvis. When the lower margin of stomach tympany with the organ empty is determined, the patient should be instructed to drink at least one quart of fluid, when, if dullness is found below the umbilicus (Fig. 202) and at the point where tympany was previously obtained, the stomach may be said to be dilated. (See also Fig. 203.)

Auscultatory percussion may also be of service in ascertaining the limitations of the stomach.

Auscultation.—All sounds heard over the stomach have a metallic quality. Placing the ear either on the patient's back or on his abdomen, and shaking him violently, a decided splashing sound will be heard. This splashing sound heard over the stomach is to be distinguished from a similar sound that may result from the presence of air and liquid in the pleural cavity (pneumothorax) and from a dilated colon that is partially filled with fluid.

Succussion Sounds Originating in the Abdomen.

1. Normal stomach (negro).
2. Dilatation of stomach.
3. Dilatation of sigmoid colon (rare).
4. Dilatation of ascending colon.
5. Pneumoperitoneum following:
   - (*a*) Perforating gastric ulcer,
   - (*b*) Perforating gastric cancer,
   - (*c*) Perforating duodenal ulcer,
   - (*d*) Perforating tuberculous ulcer,
   - (*e*) Perforating typhoid ulcer,
   - (*f*) Infection of ascetic fluid by bacillus coli communis.
6. Subdiaphragmatic abscess (communicating with the stomach).
7. Subphrenic abscess (hepatic, renal, or duodenal origin).
8. Intestinal obstruction (with enormous dilatation of bowel).
9. Ovarian cyst (infection by gas-producing bacteria).
10. Pneumocystitis (so-called pneumaturia), very rare. (See p. 72.)

Gastroscopy.—This method is of but limited value as a means of recognizing gastric dilatation, since the physical signs are fairly constant in this

condition. Gastroscopy may, however, enable one to detect malignancy or infiltration in the vicinity of the pylorus, and in this way reveal the actual cause for dilatation.

Laboratory Diagnosis.—The quantity of saliva secreted is diminished, and the *urine* is, as a rule, below the normal in amount; the latter is of high specific gravity and is often alkaline. Indicanuria is common. The blood presents the findings characteristic of secondary anemia.

Vomiting usually occurs every few days or weeks, and large quantities of fluid are ejected. A macroscopic examination of the vomitus discloses the presence of particles of food that may have been ingested several days be-

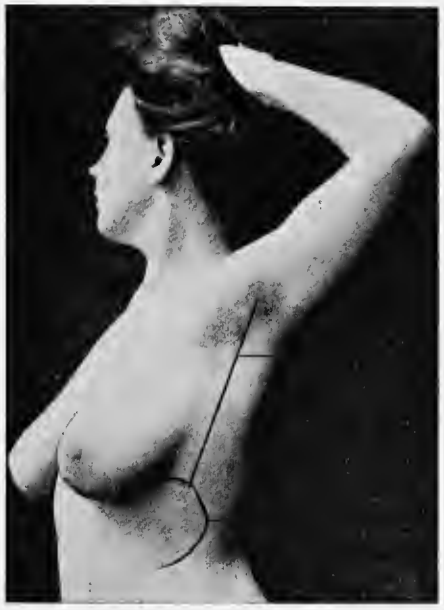


Fig. 203.—Area of Stomach Tympany when the Organ is Distended.

fore the paroxysms. A large amount of mucus is usually present, and rarely the vomitus is tinged with blood. Chemically, both the vomitus and the stomach contents obtained by means of the stomach-tube are found to be rich in lactic acid and in the other acids of fermentation. Hydrochloric acid is often absent, and but seldom, if ever, present in nomral amounts.

Gastric fluid obtained from the fasting stomach is of value, since normally the stomach at this time is nearly free from fluid. The recovery of 100 c.c. or more of stomach contents is strongly suggestive of dilation or pyloric stenosis. Recovery of only 50 c.c. points strongly to some pathologic condition. Particles of food are common in dilatation.

Summary of Diagnosis.—The preëxistence of a disease of the stomach (carcinoma, ulcer, chronic gastritis) that markedly predisposes to dilatation is of great diagnostic significance. The vomiting of large quantities of fluid, and the fact that this vomiting is not influenced by the taking of food, point strongly toward dilatation. Intense thirst and a ravenous appetite, together with an undue dryness of the mucous surface of the

mouth and throat, are features to be considered. The physical signs, and particularly those obtained by palpation and percussion, are also important in making a diagnosis of gastric dilatation. (See pp. 505, 506.)

Differential Diagnosis.—Among the conditions for which gastric dilatation may be mistaken are: (*a*) **Chronic tympanites,** the distinctive feature of which is a uniform prominence of the abdomen. Again, in gastric dilatation there is a distinct area of dullness below the umbilicus after the patient has taken a large quantity of liquid, a condition that is absent in chronic tympanites.

(*b*) **Megalogastria** (abnormally large stomach) is distinguished from gastric dilatation by the fact that the distinctive symptoms of dilatation are absent.

(*c*) **Gastroptosis** displays, as one of the points of diagnostic differentiation, the fact that both the greater and lesser curvatures are below their normal location (Fig. 204). Gastroptosis complicated by dilatation is not infrequently encountered, in which case the symptoms and signs of both conditions are present. (See Fig. 205.)

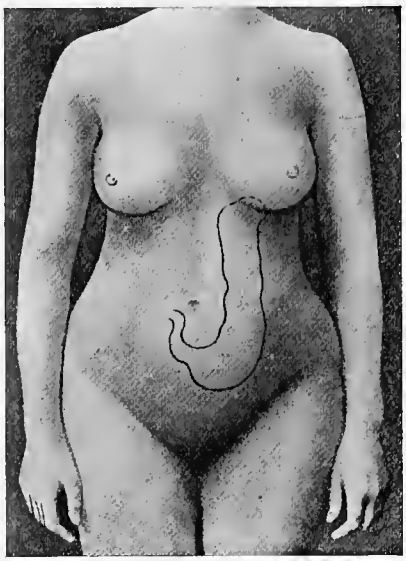


Fig. 204.—Gastroptosis. Tubular Outline of Stomach-note. Illustrative of Private Patient.

(*d*) **Ascites** may rarely be confused with dilatation of the stomach. The distinctive signs between these two conditions are elicited by percussion; *e. g.*, when the patient is in the recumbent posture, ascites gives a dull note in the flanks and tympany above the umbilicus, whereas gastric dilatation gives unilateral tympany to the left of the umbilicus and a dull note is not obtained in the flanks. The presence of a fluctuation wave is conclusive evidence of ascites, polyhydramnios, or ovarian cyst, and excludes gastric dilatation. The clinical history and the presence of long-standing heart, liver, or kidney disease also favor a diagnosis of ascites.

GASTROPTOSIS.

Pathologic Definition.—A condition characterized by displacement of both the lesser and the greater curvatures of the stomach, with or without enlargement or dilatation of the organ.

Remarks and Topography.—The *cardiac* end of the stomach is situated beneath the seventh costal cartilage, and one inch to the left of the sternum. The cardiac orifice is about opposite the upper border of the eleventh thoracic vertebra. The stomach is suspended by attachment to the diaphragm and also by the esophagus. From the foregoing remarks it is at once apparent that the cardiac extremity is practically fixed, and that it must remain so whether or not the stomach is displaced or dilated.

Normally, the *pylorus* is situated in the median line, but when the organ is filled, it will be found to be from one to two inches to the right of the center. Anatomically, the pylorus rests opposite the first lumbar vertebra, and midway between the tip of the xiphoid cartilage and the umbilicus. The

pylorus is almost completely surrounded by peritoneum, as is the upper portion of the duodenum, to which it is attached. The following six to eight inches of the duodenum are firmly attached to the spine and adjacent tissue, depending only upon the peritoneum for support anteriorly. Extensive displacement of the pylorus is improbable on account of the posterior attachments of the duodenum. Again, the gastrohepatic omentum, the so-called "suspensory ligament of the stomach," is directly attached to the lesser curvature. The gastrohepatic omentum, however, is found to lengthen with displacement of the stomach.

G. G. Davis, in making a series of sections of specimens hardened in formalin, found that the shape of the stomach varied greatly, and that the pyloric one-third was at times contracted to nearly the size of the duodenum. "This is the motor part, and the remaining two-thirds the reservoir, and in dilatation it is mainly this portion which is dilated and which prolapses down even to the pelvis." The pyloric portion of the stomach may be expanded, and this expansion may continue through the upper portion of the duodenum.

The fact that during health the greater curvature rises in the left hypochondrium to a point above the esophageal opening is of great clinical importance, and the greater curvature also descends in the median line to approximately a lower level than the pylorus. At or near the median line the lower edge of the greater curvature is found about two inches above, but may be on a level with the umbilicus anteriorly, and the third lumbar vertebra posteriorly.

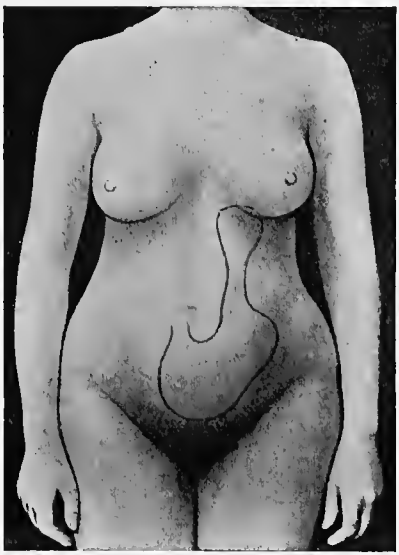


FIG. 205.—DILATATION WITH GASTROPTOSIS. ILLUSTRATIVE OF CASE FROM PRIVATE PRACTICE.

The questions that immediately suggest themselves are, what is the position of the stomach when displaced (Figs. 202, 204, 205, 209, 211) and what is its normal position? The cardiac end remains in place, but the greater and lesser curvatures are permitted to descend when the organ is dilated. There is some difference of opinion as to the degree of descent of the pylorus that occurs.

It has recently been claimed that the normal position of the stomach is more nearly vertical than was formerly taught. Repeated observations have shown that the cardiac end is opposite the eleventh thoracic vertebra, and that the pylorus is on a level with the first lumbar vertebra, the latter being from two and one-half to three inches below the former.

Predisposing Factors.—(1) **Age and Sex.**—Mienert found 80 per cent. of girls near the age of fourteen suffering from this condition, and 90 per cent. of all women examined at his clinic were similarly affected. It has also been found that in Continental Europe 5 per cent. of all males presented a variable degree of gastroptosis. Worden* reports that in a

* University of Penna. Med. Bulletin, August, 1906.

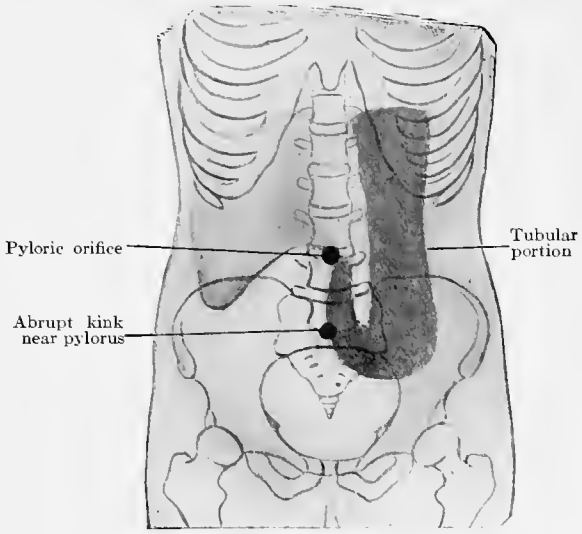


FIG. 206.—GASTROPTOSIS DESCRIBING THE TUBULAR SHAPE OF THE ORGAN, WITH RATHER UNIFORM DILATATION.
Note that the pylorus is far below its normal position. Patient standing.

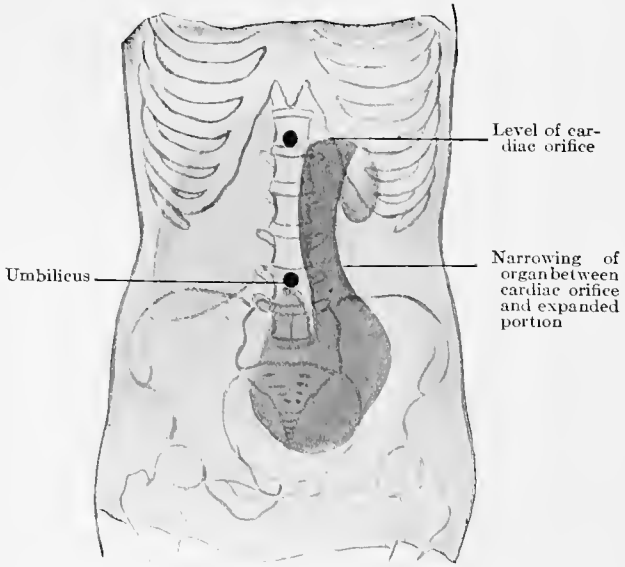


FIG. 207.—PATIENT IN ERECT POSTURE—ILLUSTRATIVE OF GASTROPTOSIS.
The greater curvature resting well in the pelvis.

skiagraphic study of 40 patients gastroptosis was present in 31. (2) **Lacing** is believed to be a prominent predisposing factor in the development of gastroptosis. (3) **Dislocation of the right kidney and of the hepatic flexure of the colon** is an etiologic factor. (4) **Relaxation of the abdominal wall** as the result of overdistention from whatever cause is among the predisposing features. (5) **Severe muscular strain** and (6) **deformities of the chest and spine** and either congenital or pathologic abnormalities of the trunk and abdomen are etiologic factors.

Principal Complaint.—The majority of cases go on for years without exhibiting symptoms, although certain functional disturbances are frequently encountered and are, as a rule, dependent upon the great difficulty with which the stomach is emptied. Sooner or later a variable degree of gastric atony develops, which is followed by impaired gastric

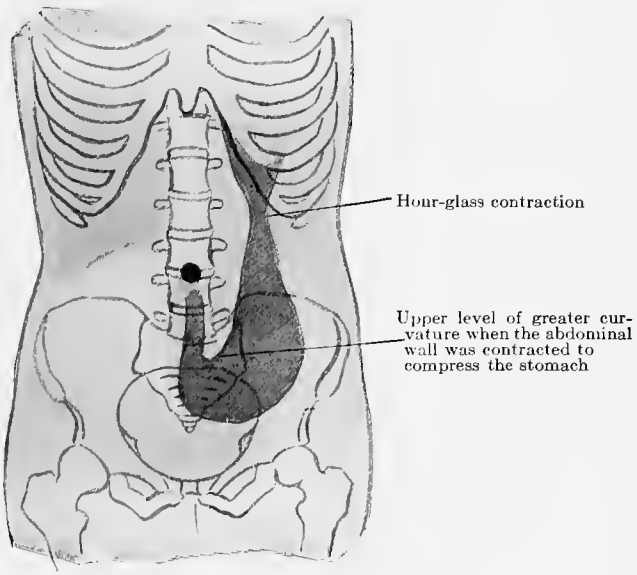


FIG. 208.—GASTROPTOSIS WITH HOUR-GLASS CONTRACTION NEAR THE CARDIAC ORIFICE. Note also the low level of the pylorus, confirmed by autopsy.

secretion, and the symptoms of chronic dyspepsia. (See Chronic Gastritis, p. 488.)

The patient complains of a sense of fullness, oppression, or weight in the abdomen. Her appetite becomes irregular, and may at times be perverted. Repeated attacks of headache, with or without vomiting, vertigo, cardiac palpitation, and dyspnea are among the commoner complaints. There is generally some evidence of malnutrition, and emaciation is not uncommon.

Constipation is the rule, and many patients complain of cramp-like pains in the epigastrium. Marked nervousness and insomnia are by no means uncommon.

Physical Signs.—Inspection.—When the patient is standing, there is usually undue prominence to the right of the median line and below the umbilicus, with distinct depression at the epigastrium.

Palpation discloses a lack of the normal resistance over the epigastrium,

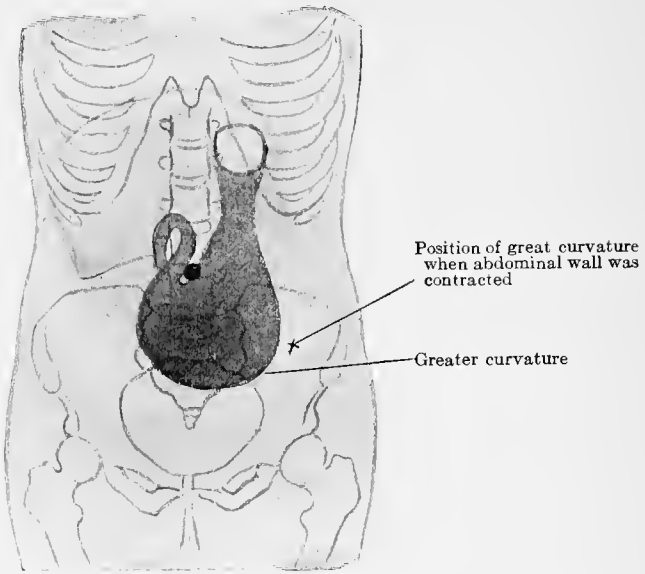


FIG. 209.—PATIENT STANDING, STOMACH CONTAINING FLUID.
In this case the stomach appeared to fill the brim of the pelvis. Note the peculiar looping of the pylorus.

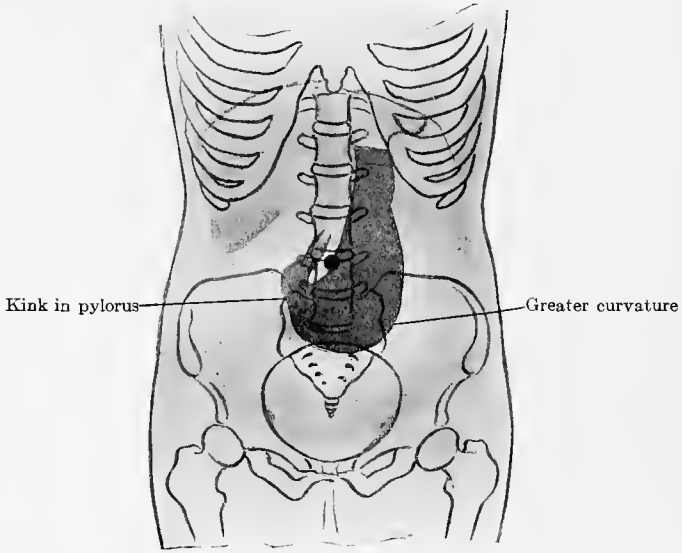


FIG. 210.—PATIENT STANDING.
Gastroptosis, with rather uniform dilatation. Note the low position of the pylorus, after taking solution of bismuth.

while that in the left inferior abdominal quadrant is slightly increased if the stomach is distended by gas or fluid.

By **percussion** over the distended stomach the organ may be clearly outlined, the tympanitic note being below the umbilicus, and perhaps extending even to the pelvis. The lower border of the greater curvature may be found well within the pelvis. The level of tympany outlining the lesser curvature will also be lower than the normal (Fig. 203).

A sign of great value in diagnosing gastroptosis is the fact that the stomach tympany is absent in the epigastrium and underneath the margin of the left ribs, a point where such tympanitic note should normally be present. Gastroptosis with gastric dilatation is by no means unusual, and for this reason the area of gastric tympany may be much greater than during health.

Auscultation.—A succussion splash (clapôtement) is audible over the

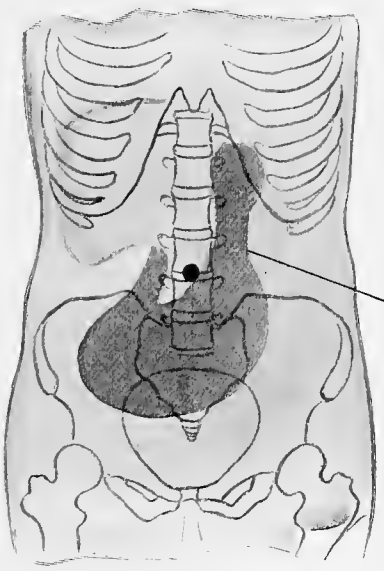


FIG. 211.—GASTROPTOSIS WITH UNUSUAL DILATATION.
The greater part of the dilated portion of the stomach resting to the right of the median line. Patient standing, stomach containing a solution of bismuth.

stomach when dilatation is associated, but this sign is not pronounced in simple gastroptosis.

X-Ray Diagnosis.—The *x*-rays are a most valuable aid in formulating a diagnosis. The stomach is first well filled with a solution of bismuth, which enables one to outline the entire organ and to ascertain the position of the pylorus. Worden, in his report, based upon a study of 40 cases, found a variable degree of displacement of the stomach 29 times. A study of his skiagraphs shows that the position of the stomach in gastroptosis may vary within wide limitations, either with or without dilatation. In our opinion, the most important finding obtained by a skiagraphic study of gastroptosis is the position of the pylorus, and in the series of 40 cases previously referred to the pylorus was to the right of the median line in 35, and "in general

was found prolapsed." Of 32 cases showing gastroptosis, in 28 the pylorus was below its normal level, and the orifice was situated at or near the level of the umbilicus. (See *x*-ray Diagnosis, p. 453.)

Figs. 168 to 173 are illustrative of individual cases when the stomach was outlined at our request by aid of the *x*-rays in the hands of the late Dr. Kassabian.

In gastroptosis there is generally an associated displacement of the colon. (See p. 457.)*

Summary of Diagnosis.—The general complaint of the patient, together with the physical signs, point strongly to gastroptosis. A skiagraphic study, however, furnishes the most positive evidence of the existence of this condition.

Prognosis.—This is favorable as to life, and guardedly favorable as to cure. Certain selected cases are amenable to surgical treatment, which in many instances gives absolute relief. In those cases in which surgical intervention is not possible the patient may continue to display the symptoms of chronic gastritis for an indefinite period.

THE INTESTINES.

METHODS OF EXAMINATION.

THE FECES.

Collection.—When searching for the Amœba coli (Amœba histolytica), it is of special importance that the feces be kept warm. Specimens collected at the patient's residence, or whenever a portion of the feces is desired for clinical study, should be passed into a warm, wide-mouthed bottle, corked tightly, and kept warm while being carried to the laboratory. As soon as the specimen is received in the laboratory the tightly corked bottle should be placed in an incubator at a temperature of 37° C., where it should be kept until the time for its examination.

Constipation and its Significance.—Obstinate constipation, although it is extremely troublesome and annoying, may not be of serious moment. It is an early sign of peritonitis and paralysis of the intestine. It is further suggestive of stenosis of the intestine, fecal impaction, or of obstruction of the bowel. Ribbon-like stools point definitely to a progressive narrowing of the lumen of the lower portion of the intestine.

Diarrhea.—One of the characteristic features in typhoid fever and dysentery are more or less frequent stools, the diarrhea being preceded by constipation. The quantity and character of the food ingested will also be found to influence materially the frequency and the quantity of the intestinal discharges. The rapidity of the peristaltic movements also affects the number of stools—rapid peristalsis exciting frequent bowel movements, whereas sluggish peristaltic waves favor constipation.

Odor.—The odor of the stools may or may not be characteristic of the disease affecting the alimentary tract, or it may be altered by the food or by the medicines ingested (onions, asafetida). As a rule, the odor is dependent upon the presence of indol and skatol in the feces, although at times a decided odor of sulphureted hydrogen may be detected.

* Pfahler, Proc. Phila. Co. Med. Soc., November, 1907.

Color.—Under normal conditions the color of the feces varies from a light yellow to a blackish-blue, depending upon the character of the food taken. Exposure to the air and light causes the stools to become darker. In health the ingestion of huckleberries produces blackish stools; chocolate gives the feces a dull-gray color; cocoa renders them of a light-gray shade; chlorophyl colors them green; starches effect a yellow color, and fats make them clay-colored.

Blood.—Following profuse hemorrhage from the bowels the feces are colored blood-red, but when the blood has been gradually extravasated into the bowel and retained there for a prolonged period, the stools become black. Hemorrhage from the stomach, from typhoid ulceration, or from other forms of intestinal lesions is followed by intensely black stools, the color being due to the formation of iron sulphid. When the hemorrhage is quite profuse,—say, when a few ounces of blood have been lost,—the stools are tar-like in color and consistence. The higher the seat of the hemorrhage is in the alimentary tract, the darker are the feces; thus, "coffee-ground" stools are seen after gastric and duodenal hemorrhages.

Tests for Blood.—If blood-cells in the feces cannot be detected by the microscope, special chemic tests for the presence of blood-pigments must be resorted to. Small quantities of blood in the feces may be detected by the method suggested by Müller and Weber.

Karczynski-Jaworski Test.—1. Place a small amount of the feces in a porcelain dish, and add a smaller amount of potassium chlorid, and one drop of chemically pure hydrochloric acid.

2. Heat carefully over the flame of a Bunsen burner until the mixture is decolorized (the addition of one or more drops of hydrochloric acid may be required to effect this). During this process chlorin escapes.

3. Add from one to five drops of a diluted solution of potassium ferrocyanid; the appearance of a distinct Prussian-blue color indicates the presence of blood-pigment.

Fallacies.—When pus is present in the feces in appreciable amount, the same reaction is produced with potassium ferrocyanid.

Occult Bleeding.—The tests employed for the detection, in the feces, of the blood from minute hemorrhages are the guaiac-hydrogen-dioxid and aloin-turpentine tests, and the findings are dependent upon a reaction obtained by the use of an acetic-acid-ethereal extract of the feces.

Technic of the Test.—If the stools are not in a semiliquid condition, they must be rendered so by mixing them thoroughly with distilled water. We usually employ 5 gm. of fecal matter for every test.

1. After the material has been thoroughly softened, the feces must be thoroughly mixed with at least its own bulk of ether, and the whole well shaken. Treatment with ether is a very necessary part of the procedure, as it removes the fat, which otherwise produces a thick emulsion when the stools are extracted with acetic acid and ether, and renders it almost impossible to obtain a satisfactory ethereal extract.

2. After being thoroughly shaken, the mixture of feces and ether should be allowed to stand for fifteen minutes or longer, the supernatant liquid being then poured off.

3. The remaining fecal matter is next mixed with one-third its volume of glacial acetic acid and 10 c.c. of ether. The mixture is again thoroughly shaken, and allowed to stand for at least fifteen minutes. The ethereal extract will rise to the top in a clear layer and can readily be separated.

4. The solution of aloin is made by dissolving a small quantity of the drug

—as much as will go on the end of a small spatula—in a test-tube one-third full of 70 per cent. alcohol.

5. Two or three cubic centimeters of the clear yellow aloin solution are then mixed in the test-tube with about the same quantity of ethereal acetic acid extract.

6. Two or three cubic centimeters of ozonized turpentine are then added, and the whole is gently shaken.

Reaction.—If blood is present, the reaction may occur in one of several ways: (*a*) The whole mixture may turn pink, the color gradually deepening to a cherry red; (*b*) the solution of aloin may sink to the bottom and form a layer beneath the mixture of ether and turpentine, and this lower layer of aloin in positive tests gradually becomes of a cherry-red color; (*c*) if the ether and turpentine are first mixed and the aloin is then allowed to flow gently down the side of the tube, the two sets of fluids may remain separate, and a deep-red ring will form at their junction.

Caution.—Not more than fifteen minutes should be allowed for the red color to develop, for after this the aloin will gradually turn red, *even if blood is not present.* It is extremely important to make up the solution of aloin as needed, for when it is allowed to stand exposed to light, it assumes the same color seen in the reaction when blood is present. When the test is negative, the color remains a light yellow, becoming red after standing for some time. Hydrogen dioxid does not work satisfactorily as a substitute for turpentine in the aloin test.

The ozonized oil of turpentine should be prepared by allowing a chemically pure oil of turpentine to stand exposed to the air for at least three weeks.

Guaiac Test.—The solution to be employed is made by shaking about a gram of gum guaiac in a test-tube half full of ether, allowing the mixture to stand until it becomes clear. About 2 c.c. of this solution are mixed with the same quantity of acetic acid, ethereal extract of the feces, and hydrogen dioxid. The hydrogen dioxid quickly settles at the bottom of the tube and the ethereal extract floats on top. The blue color that indicates a positive reaction appears very quickly in the supernatant layer, becoming a deep blue, if a decided reaction takes place, although the color may be somewhat masked by the brown tint of the urobilin in the ethereal extract. In such a case the blue is often converted into a purplish-brown, but even this reaction is not infallible. If the reaction is negative, no color change occurs.

The hydrogen dioxid to be used is a full-strength Oakland "Dioxygen." Ozonized turpentine makes an oxidizing agent equally as delicate as hydrogen dioxid, and may be used in its place. When this is done, the turpentine is added to the ethereal extract and guaiac mixture, and becomes intimately mixed with it, and the whole mixture then gradually turns blue. We have found that the guaiac test is easily made by the use of hydrogen dioxid. The solution of guaiac should be freshly prepared, but need not be quite so fresh as the aloin solution.*

Bilious Stools.—The so-called "bilious stools" vary in color from bright yellow to dark green, the color being dependent upon the presence of but slightly changed bile.

Clay-colored Stools.—Clay-colored stools are found in all forms of obstructive jaundice, but if the patient is kept upon a diet containing a minimum amount of fat, the feces are of a light-brown color.

It has been proved that the want of color in the feces is not always due

* J. Dutton Steele, Amer. Jour. Med. Sci., July, 1905.

to the presence of bile or of fats, and it is, therefore, reasonable to suppose that the decomposition of urobilin may result in the formation of decolorizing products.

Green stools may be due to the development of the Bacillus pyocyaneus or other bacteria that form a green chromogen.

Red Stools.—Carter and MacMunn have reported three cases in which the feces became red upon exposure to the light and air. The latter observer suggests that the chromogen here concerned is closely allied to stercobilin.

Reaction of the Stools.—The stools of an adult are alkaline in reaction, although occasionally they are neutral or even acid. The alkalinity of the feces is dependent upon ammoniacal fermentation. Acidity is due to the presence of lactic or of butyric acid, the formation of either of which may result from fermentation in the intestine. The feces of children on a milk diet should, under normal conditions, give a neutral or slightly acid reaction.

Fatty Acids.—All members of the group of fatty acids, from formic to stearic acid, may appear in the feces.

The detection of biliary acids in the feces is of certain clinical value in the various types of hepatic disease.

Macroscopic and Microscopic Study of the Feces.

The presence of many connective-tissue fibers in the feces is conclusive evidence that digestion has not been completed, this being due either to a pathologic condition of the gastro-intestinal tract or to dietetic errors.

Starch.—Starch-granules and chlorophyl are found in some of the undigested vegetable foods, but free starch-granules are not frequent in normal feces unless the patient has taken food composed principally of starch; for this reason, therefore, the feces of children fed upon prepared foods quite commonly contain starch-granules.

Mucoid Stools.—The definite recognition of mucus in the stools is indicative of a catarrhal inflammation of the mucous membrane of the intestine, yet this finding alone should not be regarded as of serious moment, since it may occur in conjunction with but slight intestinal inflammation. Large quantities of sago-like granules are suggestive of catarrh of the large intestine. The discharge of mucus is abundant in the feces in cases of acute intestinal catarrh in which the large intestine is involved.

Casts of the Bowel.—Mucus-cylinders are at times passed with the dejecta. These may be mere shreds of mucus-like material, or they may form a complete cast of the bowel.

Microscopic Study.—Microscopically, these mucous casts are found to consist principally of a faintly opalescent, hazy, homogeneous material (mucus). A number of epithelial cells, leukocytes, red blood-cells, mucus-corpuscles, and Charcot-Leyden crystals are rarely seen.

Blood.—It is unusual to find unaltered red blood-cells in the feces unless an ulceration involving either the lower portion of the bowel or the rectum exists. In acute catarrhal dysentery mucous bloody discharges are common, and in them the erythrocytes may be but slightly altered. It is impossible to recognize the red blood-cells as such in the feces when the hemorrhage has occurred high in the intestine, but instead of blood-cells, the feces will contain small, roundish, amorphous masses of a brown-red color. Crystals of hematoidin are occasionally encountered in the feces after intestinal hemorrhage. We have been unable to find red corpuscles in the feces after a hemorrhage occurring during the course of typhoid fever.

Pus.—Pure pus is seldom recovered from the feces, but when it is found, its presence indicates that an abscess probably communicates with the bowel.

In acute dysentery the feces contain blood, mucus, and a large quantity of pus. We have repeatedly seen mucopurulent and seropurulent stools occurring in this disease, and it is uncommon for the pus to form a large proportion of the entire stool.

Intestinal Sand.—The cases of enteric lithiasis reported probably number less than a dozen. Duckworth and Garrod, in addition to reporting a case, give an analysis of the cases furnished by the literature, and John K. Mitchell, of Philadelphia, has reported a case occurring in a male aged forty.

Fatty Feces.—Under normal conditions the feces contain a small amount of fat. Fat in pathologic amounts is usually recognized by the glistening, greasy appearance of the stool, which is often of a grayish-yellow color. When fat is present in large amounts, the stools become gray in color and at times nearly white. When placed under a one-sixth inch objective, fatty feces will show the presence of fat-globules and probably of fatty crystals; when a drop of solution of Sudan III is added to the specimen, the fat-globules are stained pink.

Clinical Significance.—(1) Fat in the stools is suggestive of the ingestion of large quantities of this substance; fat is present (2) during the course of diseases showing progressive emaciation, (3) during the essential anemias, (4) in obstructive jaundice, and (5) in nearly all forms of pancreatic disease.

Bacteria.—Many different forms of bacteria may be cultivated from the feces, but in the present state of our knowledge there are but few bacteria that are of positive diagnostic value.

Tubercle Bacilli.—The detection of the tubercle bacillus in the feces is readily accomplished whenever ulceration of the intestine, due to the development of this organism, exists. Collect a small portion of the mucoid or purulent material from the feces, smear it thinly upon a glass slide, and stain it in the usual way for the tubercle bacillus. When tubercle bacilli are present in the feces, they are oftenest seen in large clusters or dense aggregations, although they may be equally disseminated throughout the field. In tuberculous enteritis the tubercle bacilli are present in large numbers.

Pus-producing Organisms.—At times pus-producing organisms are present in the feces.

The **bacillus of Shiga** may be recovered from the dejecta of persons suffering from acute dysentery. The bacillus of Shiga, when isolated from the feces of acute dysentery, will be found to agglutinate with the patient's serum.

The *bacillus typhosus* may be obtained from the feces by cultural methods in cases of typhoid fever.

The *bacillus coli communis* is a normal inhabitant of the intestinal tract.

DISEASES OF THE INTESTINES.

DUODENAL ULCER.

Pathologic Definition.—A condition characterized by a rather clearly outlined ulcer of the wall of the duodenum, surrounding which there is a variable degree of inflammation. Duodenal ulcers vary greatly as to size and depth, displaying as they do most of the characteristics common to gastric ulcer. (See p. 491.) Adhesions to the liver, gall-bladder, stomach, and colon are often present.

Exciting and Predisposing Factors.—These are practically identical with those detailed under gastric ulcer (p. 491), except for the following exceptions: In the case of extensive burns of the skin, duodenal ulcer is rather frequent. Duodenal ulcer is also commonly encountered during the course of cholelithiasis and chronic nephritis. Males are effected more often than are females, and the disease is rather more common between the twentieth and fortieth years. Jejunal ulcer, a condition of extreme rarity, may result as a sequel of gastric or duodenal ulceration. The diagnosis of this last condition is rarely determined during life.

General Complaint.—The patient has usually suffered from what he describes as acute attacks of indigestion, certain of which are attended with severe pain in the epigastrium. The appetite may be poor, and there are usually eructations of acid substances into the throat and mouth. The patient has often observed that he has been losing weight, and that, hand in hand with emaciation, has developed languor and a sense of prostration. He has noted a distinctively tender spot just to the right of the median line in the epigastric region. Many patients experience a sense of hunger sometimes after the taking of a full meal and before the stomach has yet had time to empty itself. This hunger is often accompanied by pain, more or less boring in character, which is localized to the epigastrium. Pain, when severe, may radiate over the greater part of the superior abdominal hemisphere and to the back. Attacks of faintness are frequently experienced, and at times such attacks probably result from hemorrhage. The detection of blood in the feces may bear a direct relation to attacks of dyspnea and a sense of faintness.

Physical Signs.—After ulcer has persisted for weeks or months, there is likely to be emaciation and pallor with the usual signs of secondary anemia. (See p. 351.) Palpation reveals a localized area of tenderness over the region of the duodenum. In old cases, where there are extensive adhesions to the surrounding viscera, pain may be elicited by pressure over the entire epigastric region and along the right costal margin. Undue resistance is present over the duodenal region. In selected cases it is possible to outline a small tumor mass, which appears to be distinctly separated from the stomach.

X-Ray Signs.—Pfahler,* in a recent paper, has called special attention to the *x*-ray findings in connection with duodenal ulcer, and, in our opinion, an *x*-ray study is an essential adjunct to diagnosis. Barclay and Haudek have called attention to increased peristalsis, and regard the emptying of the stomach, within a period of less than six hours, suggestive of duodenal ulcer. Some observers have found that in selected cases the stomach may empty itself in less than one hour. This feature is in rather striking contrast with the *x*-ray findings in gastric ulcer.

"A remnant of bismuth outside of the duodenal outline, associated with resistance and not easily movable, points toward a penetrating duodenal ulcer."

Constrictions and secondary dilatation may result from contractions of a callous duodenal ulcer, and, when present, serve as confirmatory evidences only, since adhesions, from whatever cause, may produce a similar condition of the duodenum.

Laboratory Diagnosis.—Whenever the feces from a previously healthy individual are found to contain a large amount of blood, the exist-

* New York Med. Jour., May 10, 1913.

ence of duodenal ulcer is suggested. The vomiting of blood is an occasional feature, and suggests that the ulcer is located near the pylorus. Occult blood is found in the feces in acute or active ulceration of the duodenum.

Duodenal Tube.—Einhorn and Gross each devised a special instrument for the purpose of recovering the secretion of the duodenum in man. This instrument (Figs. 212, 213) is in reality an elongated stomach tube, containing a perforated metal bulb at one end.

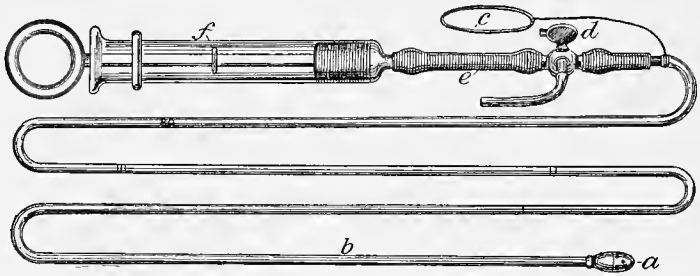


Fig. 212.—Einhorn's Duodenal Pump.

a, Metal capsule, lower half provided with numerous holes, the upper half communicating with tube *b*; I, II, III, marks of I=40, II=56, III=70 cm. from capsule; *c*, rubber band with silk attached to end of tubing, which can be placed over the ear of the patient; *d*, three-way stop-cock; *e*, collapsible connecting tube; *f*, aspirating syringe.

Use.—The patient is given a glass of milk and water, and an hour later the duodenal tube should be swallowed. Direct the patient to lie on the right side, and in course of time the end of the tube, at which is placed the metal ball, will be found to pass through the pyloric opening and well into the duodenum. The recovery of the secretion of the duo-

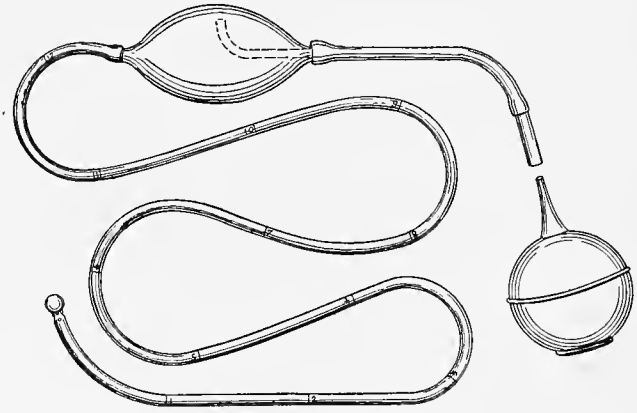


Fig. 213.—Gross Duodenal Tube.

denum is accomplished in practically the same way as is described for recovering the gastric contents. (See p. 462.) Doroff, in his study of 21 cases, estimates the duodenal fluid recoverable by this method to fluctuate between 10 and 25 c.c.

Time Needed.—Probably the leading objection to the employment of the duodenal tube in diagnosis is the time consumed in recovering the

duodenal fluid. Doroff found that from an hour to one and one-half hours was employed in this procedure, and he recommends placing the patient in the Trendelenburg position, stating that, through his experience, the duodenal fluid may thus be recovered in from twenty to thirty minutes.

Duodenal Fluid.—The quantity usually exceeds 10 c.c., in color ranging from a golden yellow to a greenish yellow, or at times a dark green. The color is influenced materially by the amount of bile present. Duodenal fluid may be clear, slightly cloudy, turbid, and occasionally milky. The reaction of the duodenal secretion is, as a rule, faintly alkaline or neutral, although it may be strongly alkaline or even strongly acid. Thus far we are not aware that the chemical reaction of the duodenal fluid has proved of great diagnostic value, although the possibilities for this line of clinical study are undoubtedly great. Gross has been able to recognize duodenal ulcer through the recovery of duodenal fluid that contained blood.

In the case of inflammatory processes of the gall-bladder and adjacent viscera, where, theoretically, a bacteriologic examination of the duodenal secretion should prove of clinical value, we can see but little advantage in obtaining the duodenal secretion through the duodenal tube, or duodenal bucket, over the method recommended by Louis Brinton. (See p. 751.)

Differential Diagnosis.—The distinctive differential features between duodenal ulcer and gastric ulcer are set forth in the accompanying table, which has been modified from "Ander's Practice."

Duodenal Ulcer.	Gastric Ulcer.
1. Usually occurs between twenty and forty years, except when due to external burns.	1. May occur at any age after childhood.
2. Males are more frequent sufferers than females, in the proportion of ten to one.	2. Females are the chief sufferers.
3. Onset often marked by intestinal hemorrhage, which may recur at intervals of varying duration.	3. Gastric hemorrhage frequently occurs and is preceded by other gastric symptoms.
4. The melena may rarely be preceded or accompanied by hematemesis.	4. Blood may appear in the stools, but usually after hematemesis.
5. Blood in the discharge is often bright red.	5. The blood in the dejections is dark and tarry from the action of the gastric juices.
6. Hunger pain, due to acid, may come on late, two to four hours after meals. It is localized a little above and to the right of the umbilicus. Pain relieved by eating.	6. Pain paroxysmal, greatly influenced by taking food. Pain sharply localized in the epigastric region, about 2 inches below the ensiform cartilage. Pain is aggravated by taking food, as a rule.
7. *X*-ray shows stomach empty in from one to six hours.	7. Retention of food beyond six hours.
8. Vomiting inconstant without relation to ingestion of food, and affords no relief.	8. Vomiting more common soon after food (during painful crisis) and affords relief.
9. Jaundice occasionally present from occlusion of bile-duct.	9. Jaundice absent.
10. No marked improvement after diet has been regulated.	10. Usually a marked improvement follows regulation of diet.
11. Dorsal pain-point absent.	11. Painful point (between the tenth and twelfth dorsal vertebræ on left side) usually present.
12. A remnant of bismuth outside shadow of duodenum strongly favors perforating ulcer.	12. Absent.

ENTEROPTOSIS.

Pathologic Definition.—A condition characterized by descent of the colon from its normal position.

General Remarks.—Displacement of the colon often develops coincidently with displacement of the stomach or of the kidneys. Glénard describes at length the symptoms resulting from a displacement of more than one of the abdominal viscera—a condition known as splanchnoptosis.

Exciting and Predisposing Factors.—(*a*) Gastroptosis serves as the most potent factor in the displacement of the transverse colon; (*b*) displacement of the right kidney often carries with it the hepatic flexure of the colon; (*c*) habits of dress figure prominently as causes of displacement of the colon; (*d*) tight lacing tends to lower the splenic and hepatic flexures, whereas the center of the transverse colon may remain at its normal height (Fig. 216).

Sex.—The majority of all cases occur in females. Repeated pregnancies, the use of violent cathartics, and rapid emaciation are likely to be followed by prolapse of the colon. Muscular strain, heavy lifting, etc., are also

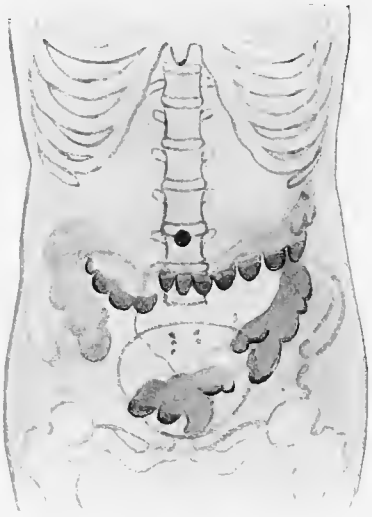


FIG. 214.—TRANSVERSE COLON DISPLACED BELOW UMBILICUS.
Dark areas indicate that portion occupied by bismuth solution.

FIG. 215.—LOWERING OF THE HEPATIC FLEXURE AND LOOPING OF THE DESCENDING COLON.

said to contribute toward this condition. Both the large and small intestines may take part in the displacement, although the former (coloptosis) is the more common.

Principal Complaint.—The colon may be greatly displaced without causing the patient any inconvenience (Fig. 215), and where there is but a moderate degree of displacement symptoms are uncommon. The patient usually complains of gastric or intestinal discomfort (indigestion), and does not gain flesh.

Flatulence is an almost constant symptom both before and after meals. There may be a history of repeated attacks of mucous colitis, especially

when the flexures are involved, but such a history is by no means constant. In addition to constipation, the patient complains of a dragging sensation in the abdomen and of attacks of colicky abdominal pains. In well-marked cases of enteroptosis constipation generally alternates with attacks of diarrhea during which many of the symptoms suggestive of gastroptosis are likely to be present. (See p. 511).

Among the **nervous manifestations** are headache, general irritability, sleeplessness, mental hebetude during the day, and well-marked hysteria.

Auscultation.—By injecting from 200 to 400 c.c. of water by the rectum it is possible, in many instances where there is ptosis of the transverse colon, to get a splashing sound over the lower portion of the abdomen (Figs. 217, 218, and 219).

Laboratory Diagnosis.—The urine is, as a rule, rich in indican, and is often high colored and of high specific gravity, except in persons of a hysteric temperament, in whom intermittent attacks of polyuria are prone to occur.

X-Ray Diagnosis.—By injecting a solution of bismuth into the colon it may be readily outlined by the aid of the *x*-rays, a means that gives the most positive evidence of displacement. (See Fig. 219.)

Summary of Diagnosis.—The only positive clinical evidences of prolapse of the colon are: (*a*) The picture revealed by the use of the *x*-rays; (*b*) the effect of distention of the colon by gas; and (*c*) the absence of colonic tympany in the upper portion of the abdomen. The history and general complaint should be taken into consideration, but they are in no way characteristic of displacement of the colon.

Clinical Course and Duration.—The prognosis as to life is favorable, the condition continuing for many years. Coloptosis may be cured in many instances by surgical measures, together with judicious medication.

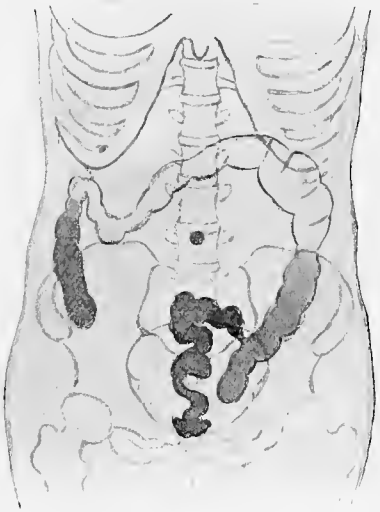


Fig. 216.—Position of the Colon and Rectum as Shown by *x*-Rays, When Filled with Bismuth Solution.

DILATATION OF THE COLON (Ectasia of the Colon).

Pathologic Definition.—A condition characterized by more or less general dilatation of the colon, most marked, in the majority of cases, at the sigmoid flexure. Hypertrophic dilatation of the colon is the rule, and although a marked catarrh is present there may also be ulceration of the mucous surface, and, sooner or later, atony of the muscular coat of the large bowel occurs.

Etiologic Factors.—Chief among the exciting factors are malformation (congenital elongation) of the colon, although dilatation may follow extensive ulceration, and is occasionally a sequel of typhoid fever, amebic dysentery, operations on the abdomen, etc.

Principal Complaint.—Constipation figures prominently in all cases, and the history, as a rule, dates from infancy, from an attack of typhoid fever, or following a laparotomy. The patient's abdomen has been unduly prominent for years, with a constant tendency to increasing disten-

tion. The degree of abdominal swelling is always lessened by an attack of diarrhea, but within the next two or three days the distention returns and continues until again relieved by diarrhea. In many instances the patient may pass an enormous quantity of feces.

The appetite is fair, sometimes even ravenous, and the patient is apparently well nourished.

Physical Signs.—Inspection.—There may be either localized or general distention of the abdomen, but, as a rule, the distention corresponds to the outline of the ascending colon.

Palpation discloses the presence of a soft, dough-like mass in the region of the cecum, whereas other portions of the abdomen are of nearly normal resistance. A reverse peristaltic wave is occasionally seen over the upper portion of the abdomen.

Percussion.—In cases of true typhlitis there is a variable area of impairment in the region of the cecum. Occasionally the impacted feces are discharged by the bowel, over which a tympanitic note is obtained, but deep

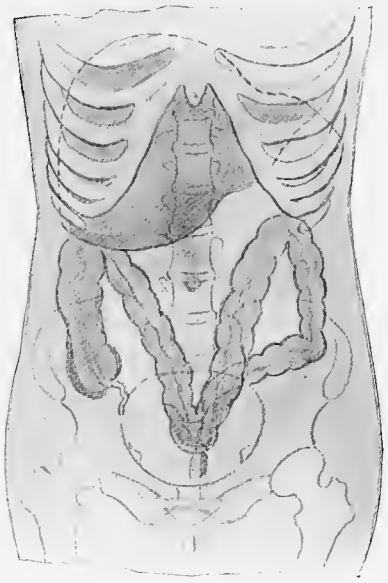


FIG. 217.—ILLUSTRATIVE OF AN EXTREME CASE OF COLOPTOSIS.

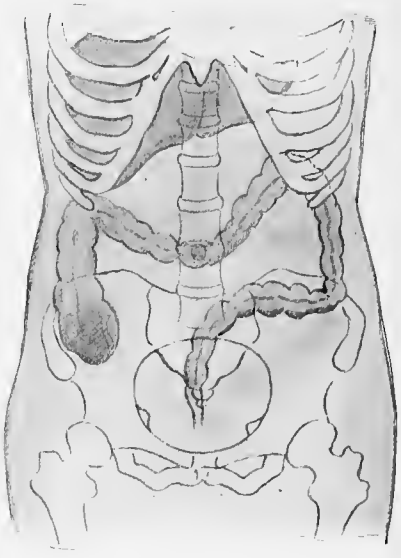


FIG. 218.—DECENSION OF THE BEGINNING OF THE ASCENDING COLON, WITH DISPLACEMENT OF THE TRANSVERSE COLON.

percussion will elicit dullness, and this may extend well into the loin. Over the expanded, but not impacted, portions of the colon there is an unusual degree of tympany, which may approach closely that obtained over the stomach.

Auscultation.—A splashing sound is often elicited over the dilated portion of the colon.

Laboratory Diagnosis.—During the attack of constipation the scybala passed are frequently covered with mucus or stained with blood.

The urine is highly colored and rich in indican.

Summary of Diagnosis.—This is founded largely upon the history of the case and the presence of an undue degree of tympany along the course of the colon, with a palpable, dough-like mass in the region of the cecum.

Differential Diagnosis.—Chronic dilatation of the colon is to be distinguished from ascites and intestinal obstruction. (See table of differential diagnosis, p. 527.)

INTESTINAL OBSTRUCTION.

Pathologic Definition.—Any pathologic or anatomic condition that causes either partial or complete occlusion of the lumen of the bowel, and consequently produces mechanic interference with the passage of the intestinal contents.

Varieties.—For convenience of study two classes of cases are recognized: (*a*) Acute obstruction and (*b*) chronic obstruction.

(*a*) The **acute form** is caused by pathologic conditions that suddenly narrow or obliterate the lumen of some portion of the intestine, usually above the ileocecal valve, and often, without warning, give rise to a group of characteristic symptoms.

(*b*) In **chronic obstruction** the large intestine is oftenest involved, and the symptoms usually develop slowly as the disease progresses. The intestinal wall above the seat of obstruction early undergoes compensatory hypertrophy, dilatation taking place very slowly unless chronic obstruction suddenly merges into the acute form—an event that is always announced by a fairly characteristic complex of symptoms.

In carcinoma of the colon below the sigmoid flexure the patient makes little or no complaint until the acute obstruction develops.

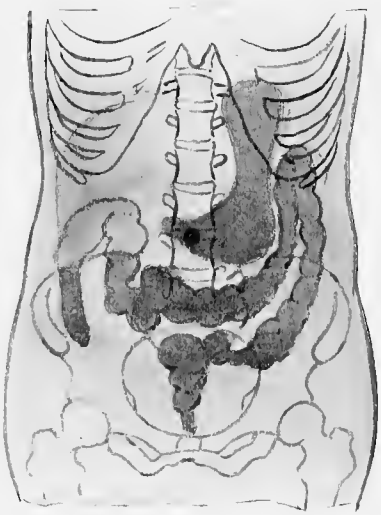


Fig. 219.—Displacement of Ascending and Transverse Colon; Gastroptosis also Present.

Exciting and Predisposing Factors.—(1) **Strangulation** figures as the commonest cause of *acute intestinal obstruction*. It is frequently the result of bands of adhesion that have formed from either recent or old peritonitis. The site at which adhesions are most likely to develop is the iliac fossa, although the formation of extensive adhesions may follow operations upon the abdomen.

Strangulation of an omental hernia is a not infrequent cause of acute obstruction, as are also diaphragmatic, inguinal, and femoral hernias.

(2) **Intussusception** is a condition in which there is an invagination of one portion of the bowel into an adjacent portion. It is believed to be caused by abnormal peristalsis. The site of invagination is oftenest the ileocecal valve, yet it may be encountered at different portions of the colon.

Age predisposes to the development of this form of acute obstruction, children and young adults being most often affected; volvulus, however, has been known to occur in the male after the age of forty. Ileus (paralysis of the intestine) permits of impaction of the colon.

Chronic obstruction results from fecal impaction of the colon, and is favored by obstinate constipation and by dilatation of the colon. Obstruc-

tion may be caused by the presence of foreign substances in the bowel, *e. g.*, hardened feces, gall-stones, intestinal parasites (ascaris and tape-worms), pins, fruit-stones, coins, and the like.

Congenital stricture of the intestine is an occasional cause of chronic obstruction.

New-growths formed within the intestinal wall or developing from adjacent structures and pressing upon the intestine may in turn narrow its lumen and produce chronic obstruction. Carcinoma of the bowel or of the peritoneum serves as one of the commonest causes of chronic obstruction. Sarcoma may attack the small bowel and be followed by obstruction, as may also sarcoma of the retroperitoneal glands (Löbstein's carcinoma). Polypoid tumors, glandular tumors, and all types of neoplasm in the abdomen are likely, sooner or later, to cause chronic obstruction.

Bands of adhesions may produce obstruction of the colon or of the small bowel, and chronic obstruction occasionally results from scar formation incident to extensive ulceration of the colon.

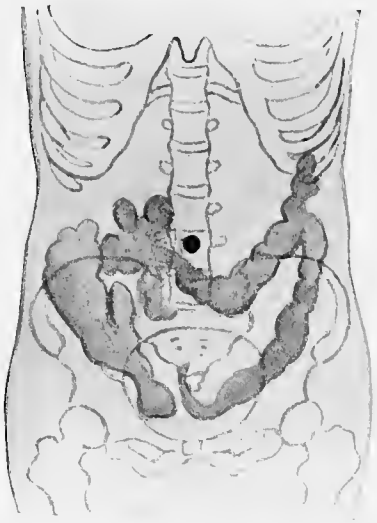


FIG. 220.—GENERAL DISPLACEMENT OF THE COLON INTO THE PELVIS.

Diminished Peristalsis.—This condition is occasionally due to paralysis of the muscular coats of the intestine (ileus). The degree of peristalsis may be greatly inhibited as the result of peritoneal adhesions and impaction of the colon.

Principal Complaint.—In acute obstruction the onset of the symptoms is sudden, the patient's appearance indicating acute suffering. *Pain* is usually present, but is sometimes general instead of being located at the site of obstruction. Intermittent attacks of colicky pain occur early, and later the pain may become agonizing and almost constant. If the obstruction is situated high, hiccough and eructations may precede the vomiting for some hours. *Vomiting* sets in early, and consists, first, of the contents of the stomach, and later of a fluid emitting the characteristic fecal odor.

Thermic Features.—The temperature soon becomes subnormal, and remains so until the obstruction is relieved.

Chronic Obstruction.—The patient describes a series of symptoms among which obstinate constipation and moderate abdominal distention are foremost. These symptoms have come on slowly or have been intermittent. *Constipation* is caused by the slowly advancing obstruction until the irritation causes an accumulation of fluid above the obstruction, when a watery diarrhea, followed by constipation, sets in. Blood is not uncommonly present in the stools, especially when the obstruction is due to an ulcerating carcinoma of the lower bowel. Dull, aching pelvic or sacral *pain* and bearing-down sensations are described. *Vomiting* is uncommon.

Physical Signs.—Inspection.—The expression is anxious, the cheeks are sunken, and the skin is cold and beaded with perspiration. According

to the location of the obstruction there is a variable amount of abdominal distention (the lower the obstruction, the more decided the abdominal enlargement).

Palpation.—The abdomen is at first only moderately distended and soft, but later the abdominal wall is tense, tender, and peristaltic waves may be felt and seen.

Percussion.—An increased amount of tympany is always present, and becomes extreme in those cases in which the site of obstruction is low. The *pulse* is rapid and weak.

Auscultation.—Borborygmi and a succussion splash are to be heard over the abdomen.

Laboratory Diagnosis.—The amount of feces expelled at each defecation is comparatively small, and if the obstruction is low, the stools are ribbon-like in character. Scybalous masses and blood are among the usual findings in carcinomatous obstruction.

Summary of Diagnosis.—Acute intestinal obstruction is diagnosed from the presence of pain, absolute constipation, marked, and later stercoraceous vomiting, subnormal temperature, and a tendency toward circulatory collapse. Tympany and abdominal tenderness, when present, are also of value in formulating a diagnosis.

Chronic obstruction is more difficult of diagnosis, since here the history of prolonged constipation, abdominal distress, and paroxysmal attacks of colic, followed by diarrhea and the passing of ribbon-like stools, is to be taken into consideration before it can be definitely determined that chronic obstruction exists.

Differential Diagnosis.—See below.

TABLE SHOWING THE DIFFERENCES BETWEEN DILATATION OF THE COLON, ASCITES, AND INTESTINAL OBSTRUCTION.

Dilatation of the Colon.	Ascites.	Intestinal Obstruction.
1. History of obstinate constipation alternating with attacks of diarrhea, and of the passing of scybala since childhood.	1. Chronic heart, liver, or kidney disease or some form of chronic anemia.	1. History of preëxisting hernia, peritonitis, or abdominal growths, and passage of ribbon-like stools.
2. Abdomen prominent since childhood, or dates to acute disease of bowel.	2. Enlargement has developed within a comparatively short period.	2. Enlargement recent (a few hours).
3. Left inferior abdominal quadrant is bulging, and where the ascending colon is enlarged, distention is fairly uniform.	3. Enlargement fairly uniform, except flanks bulging.	3. Irregular enlargement.
4. Reverse peristaltic wave may be seen.	4. Absent.	4. Reverse peristaltic wave common.
5. Soft, dough-like mass palpable in region of the sigmoid flexure.	5. Absent.	5. Absent.
6. Unusual degree of tympany along the course of the colon.	6. Marked tympany at the umbilicus when patient is in the recumbent posture. Location of tympany changes with position of the patient.	6. Tympany is localized and not affected by posture.

Dilatation of the Colon.—(*Continued.*)	Ascites.—(*Continued.*)	Intestinal Obstruction.—(*Continued.*)
7. Vomiting uncommon.	7. May have been vomiting of mucoid material.	7. Vomiting when the obstruction has become complete.
8. Dullness may be localized.	8. Dullness in the flanks when patient is recumbent (see Fig. 229) and over inferior abdominal hemisphere when in the erect posture.	8. Absent.
9. Aspiration of the abdomen negative.	9. Recovery of peritoneal fluid and disappearance of distention.	9. Negative.

TABLE SHOWING THE DIFFERENCES BETWEEN ACUTE GENERAL PERITONITIS AND ACUTE INTESTINAL OBSTRUCTION.—(*Modified from Anders.*)

Acute Peritonitis.	Acute Intestinal Obstruction.
Etiology.	
1. There is a history of causal conditions or diseases (ulcer, appendicitis, hepatic colic, pyosalpinx).	1. There is a history of previous chronic obstruction or of hernia.
2. Adults oftenest affected.	2. Intussusception is common in the young.
Symptoms.	
3. Considerable rise of temperature, may be absent later.	3. No early rise, except in volvulus.
4. Pain diffuse and continuous.	4. Pain paroxysmal and localized.
5. Vomiting and hiccough characteristic. No stercoraceous vomiting.	5. Vomiting becomes characteristically stercoraceous after stomach is emptied.
6. Collapse occurs later.	6. Collapse within a few hours.
Physical Signs.	
7. Marked general distention of the abdomen.	7. Less marked local distention, unless the obstruction is situated in the lower bowel.
8. No visible peristaltic waves.	8. Visible peristaltic wave when the seat of obstruction is low.
9. Tenderness marked and general.	9. Tenderness not extreme and always localized early.
10. Signs of effusion may appear.	10. Rare.
11. Auscultation negative.	11. Loud gurgling and splashing sound audible on auscultation over the abdomen.

Clinical Course.—Acute obstruction is of short duration, and, as a rule, demands prompt surgical treatment. Chronic obstruction extends over a period of weeks, months, or even years, and will in all probability terminate in complete obstruction.

MUCOUS COLITIS.

Pathologic Definition.—A condition characterized by changes in the mucous membrane of the colon, with the production of an excess of mucus. Large quantities of mucus may accumulate at certain points in the intestine and here excite violent irritation, hence the name, mucous colic. The mucus changes its location in the intestine from time to time, and is eventually discharged by the rectum.

Exciting and Predisposing Factors.—Direct irritation of the rectum, prolonged constipation, and new-growths of the pelvis, by exerting undue pressure upon the rectum and colon, may be exciting factors. Certain forms of exercise, *e. g.*, bicycle and horseback-riding, are said to be predisposing elements. The patients often display the hysterical temperament.

Age.—Mucous colic is extremely uncommon in children, usually attacking young adults.

Sex figures prominently, since 80 per cent. of all cases are seen in women.

Principal Complaint.—The patient gives a clear history of obstinate constipation, which may have continued over a prolonged period. Both long and short shreds of mucus are passed with the feces. In many instances the passing of mucus, with pains and tenesmus, occurs at various times during the year. In a case seen by us the pains lasted about two days, and recurred regularly at the end of every three months. The patient may have passed an almost complete cast of the lower bowel. Prior to the passing of mucous casts the constipation was more obstinate than usual, the intense pain subsiding after the passing of a quantity of mucus.

Attacks of constipation lasting from one to three days alternating with diarrhea are common. The patients declare that following attacks of diarrhea the constipation is more pronounced and colic more frequent. Equally as important as are the somewhat characteristic symptoms of mucous colic is the statement by the patient that she has not lost in weight, and that while the condition has existed for months, or probably years, there has been no decided evidence of weakness.

Laboratory Diagnosis.—Microscopically, the shreds of mucus are found to be composed of granular débris and cylindric and pavement epithelial cells.

Clinical Course.—The majority of cases run a course covering a period of from one to twenty years or even longer, although acute mucous colitis has been observed, this variety terminating favorably in from ten to thirty days.

DIMINISHED PERISTALSIS.

Definition.—A condition in which there is an abnormal lessening in the motor function of the intestine, with a loss of desire to go to stool and a tendency toward impaction of feces in the lower portion of the colon.

Principal Complaint.—Obstinate constipation may at times alternate with mucous or watery diarrhea.

ENTEROSPASM.

Definition.—A condition excited by an undue sensitiveness of the muscular coat of any portion of the intestine, which results in spasmodic contraction, and is characterized clinically by intestinal colic, constipation, and possibly the symptoms of intestinal obstruction.

General Remarks.—Clinically, there is a close resemblance between enterospasm and enteralgia (see p. 545), although abdominal pain may not be present in every case of enterospasm. Constipation is, as a rule, a temporary condition, and may even be absent.

Laboratory Diagnosis.—The stools consist of hard, rounded masses, or they may be thin and ribbon-like in form.

PLUMBISM

(LEAD COLIC; CHRONIC LEAD POISONING; SATURNISM).

Consideration.—A chronic intoxication due to the slow absorption of lead, and characterized pathologically by destructive changes in the blood (leukocytosis, loss in the percentage of hemoglobin, and basophilic degeneration of the red cells). Multiple neuritis is also seen in advanced cases, and general atheromatous changes in the arteries increase with the duration of the condition, Owing to a deposit of lead, a blue line is seen on the gum near its junction with the teeth.

Exciting and Predisposing Factors.—The exciting factor is the slow absorption of lead, the result of occupation or of accident.

Among the predisposing factors are:

(*a*) **Personal susceptibility** is quite prominent in a small percentage of all cases.

(*b*) **Sex.**—When subjected to the same environment and exposure, adult females are more susceptible than males.

(*c*) **Age.**—Infection takes place more often in adult males than in children or in women, but this is probably due to the greater exposure of men.

(*d*) **Occupation** figures prominently as an etiologic factor. Employees in lead or in paint works who handle either white lead or red lead are extremely likely to develop lead intoxication. Painters become affected in the same manner, and plumbers, owing to the fact that they use a large amount of lead in soldering, are also exposed. Diamond-cutters use an emery pencil that contains a large amount of lead; the tip of this pencil is repeatedly carried to the tongue of the operator while at work. Lead-miners, pottery glazers, type-setters, shot-makers, lace-makers, and dressmakers are likewise exposed.

(*e*) The **contamination of foods** and of drinking-water is occasionally the source of lead intoxication. Butter, candy, milk, cakes, and certain breads may be artificially colored by the addition of lead chromate, which in itself serves as the means of introducing lead into the system. "Tobacco wrapped in lead foil has less commonly resulted in symptoms of saturnism" (Anders).

The greater portion of lead enters the human system through the digestive tract, although it is said to enter also through the respiratory tract; moreover, a moderate amount is doubtless absorbed through the skin.

Principal Complaint.—The fact that the patient's occupation necessitates his handling lead is a guide to the diagnosis. There is a gradual loss of strength covering weeks or months, the appetite is irregular, and attacks of obstinate constipation occur. He complains further of extreme muscular weakness, and states that he is unable to grip certain tools or instruments as firmly in his hands as he did formerly.

Developing coincidentally with weakness of the hands there are vague pains in the muscles of the forearms and of the legs. He states that walking is difficult, and that he is continually stubbing his toes. At various times since his present illness he has had paroxysmal attacks of vomiting, and before and after such attacks frontal and occipital headache is severe. He is troubled with sleeplessness, and arises in the morning feeling unrefreshed.

Colic.—There may or may not be a history of previous attacks of intestinal colic. Lead colic is fairly characteristic; the pain is seated in the region of the umbilicus and is severe and griping; in extreme cases it radiates over the entire abdomen. The pain is paroxysmal at first, but later it is

continuous for a period of hours or even days. When it is severe and radiates to the epigastrium, paroxysmal vomiting develops. Pain is seldom absent, and there is almost always a sense of weight or discomfort in the abdomen after the initial cramp until treatment has been instituted.

Fig. 221.—Double Wrist-drop, as Seen in Chronic Lead Workers' Disease.

Nervous Phenomena.—Intestinal colic, muscular cramps, and tenderness among the course of the nerve-trunks are prominent among the nervous phenomena of chronic plumbism. Wrist-drop and foot-drop do not develop

Fig. 222.—Method of Detecting Tremor of the Hand.

until the case is well advanced, and when present, there may be some degree of anesthesia over the parts affected. Anesthesia may depend upon lead intoxication or upon an associated hysterical element, since hysteria is by no means uncommon. Paralysis is bilateral, a fact that serves to dis-

tinguish it from those forms that are excited by traumatism, uremia, etc. (See Differential Diagnosis.) Early during the course of lead intoxication both fine and coarse tremors are present, and are best detected by directing the patient to extend his hands and to then separate his fingers (Fig. 222). Tremor is markedly aggravated by emotional excitement and by exercise.

Sensation.—There may be localized areas of anesthesia, paresthesia, or hyperesthesia. (See Hysteria.)

The chief cerebral symptoms are usually referred to as "*lead encephalopathy*," which consists of a series of symptoms among which delirium, aphasia, convulsions, coma, hysteria, and insanity are included. Delirium and even coma are by no means uncommon, and may develop abruptly, although they are, as a rule, preceded by marked tremors and hallucinations. Both epileptiform and hysteric convulsions are among the nervous manifestations of this condition. We have not infrequently seen cases in which mania and melancholia developed during the course of chronic plumbism.

Headaches, joint pains, and stiffness of the joints are likely to be present during the entire course of the disease.

Cramp.—Cramps in the muscles of the arms, forearms, and legs are by no means unusual, and there may be cramp-like pain in the larger joints.

Physical Signs.—Inspection.—The skin is cachectic, and there is usually edema beneath the eyes and at the ankles. The abdomen is scaphoid in shape (Fig. 223) and the patient refrains from exerting himself in any way that will cause a movement of the abdominal muscles. The lips are dry and sometimes fissured, and the tongue is heavily coated.

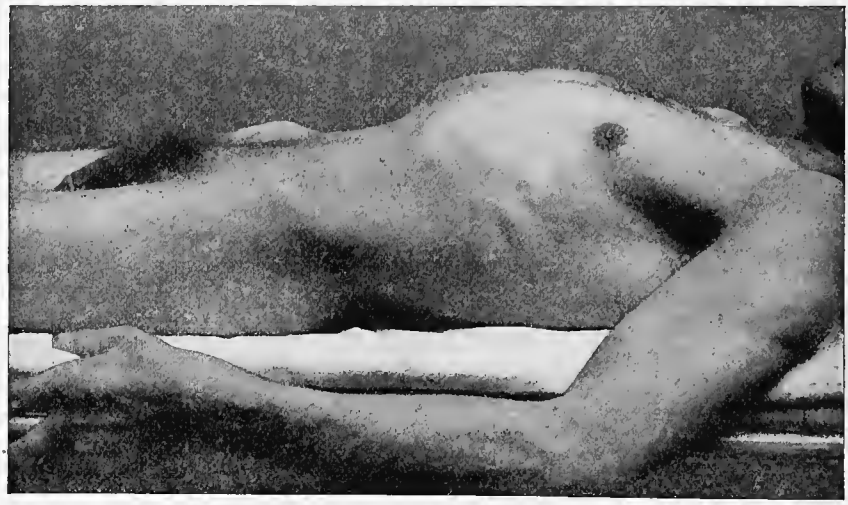


Fig. 223.—Scaphoid Abdomen Seen in a Case of Chronic Plumbism (Philadelphia Hospital).

The Lead Line.—A distinctly bluish line is seen upon the gums, located near the junction of the mucous membrane with the teeth. This blue line, which is characteristic of lead intoxication, is best demonstrated by carrying a thin white card on the point of a toothpick underneath the margin of the gum; in this way the lead line is distinctly discernible from any discoloration of the teeth, and is rendered more conspicuous. Wrist-drop and foot-

drop, deformities, and wasting of the muscles are also detected by inspection. (See Nervous Symptoms.)

Palpation.—There is decided tenderness along the course of the nerves of the forearms and of the legs. The flexor muscles of the hands are decidedly weakened, and the patient's grip is comparatively feeble. The abdominal muscles are in a state of spasm during an attack of colic, but are quite flaccid between such attacks. When the toxemia is extreme there may be tenderness about the larger joints. The skin of the ankles and of the hands may pit upon pressure, and in long-standing neuritis the muscles are flabby and there are other evidences of muscular wasting. There may be a hypersensitiveness or diminished sensation over the extremities. The reflexes may be inhibited, and the knee-jerks may even be absent.

Heart.—The pulse is normal or slower than normal, even in cases showing marked weakness. During an attack of colic the heart action is slow and forcible, and the pulse is strong, full, and of increased tension. Arteriosclerosis develops rapidly after lead intoxication.

Eyes.—Failing vision is an occasional symptom in those suffering from lead intoxication. Neuroretinitis, retinal hemorrhage, and hemianopsia have also been described.

Laboratory Diagnosis.—The urine is at first increased in quantity, but as the intoxication is prolonged, the quantity of urine gradually diminishes, is of high specific gravity and of high color, and chemically it contains lead and, at times, albumin.

Microscopically, the urine shows casts, many epithelial cells, leukocytes, and an occasional red blood-cell.

Elimination of lead is also said to take place by the bile and saliva, but such observations have not been confirmed.

The vomitus is usually profuse in quantity, white in color, and is found to contain much lead.

The Blood.—Secondary anemia develops early during the course of lead intoxication. The red cells are gradually decreased; but a more marked decrease in the hemoglobin is observed. There is an increase in the number of white blood-cells in a cubic millimeter. We have observed* that during the initial attack of lead colic the number of leukocytes ranges between 10,000 and 25,500 in a cubic millimeter. An equally important finding is that leukocytosis is less marked and may be absent during subsequent attacks of colic. From 1 to 4 per cent. of myelocytes are present, and nucleated red cells, both normoblasts and megaloblasts, are to be found. The red cells stain feebly, display an unequal distribution of their hemoglobin, and poikilocytosis is well marked. Punctate basic degeneration of the red cells is constantly present during lead intoxication, and is best seen after staining the blood with carbolthionin. The isolated areas of degeneration are disseminated throughout certain of the red cells, and stain heavily by the thionin.

Summary of Diagnosis.—In the majority of cases the diagnosis is strengthened by the patient's history of having worked in lead. The character of the vomitus, the fact that it contains lead, and the detection of lead in the urine are positive evidences of chronic plumbism. Foot-drop and wrist-drop associated with tenderness along the course of the nerve-trunks are also valuable data, but these may not develop until late. The blue line near the junction of the mucous membrane with the teeth is a positive sign. When sleeplessness, hysteria, and mania occur they should

* Philadelphia Med. Jour., September 27, 1902.

be distinguished from similar symptoms excited by lead intoxication. Leukocytosis and punctate basophilia are unusually common in chronic lead intoxication.

Differential Diagnosis.—Chronic lead poisoning is to be distinguished from **postdiphtheric paralysis,** the **shifting paralysis of uremia,** and **muscular rheumatism.** The first of these conditions is readily distinguished by a history of a recent attack of sore throat. A history of Bright's disease and the characteristic findings in the urine enable one to distinguish between uremia and lead intoxication, lead being present in the urine of lead workers only. In muscular rheumatism the tenderness is not distinctly localized along the course of the nerve-trunks, but is distributed over the muscles. Traumatic neuritis differs from the neuritis of lead intoxication in the fact that the former is always unilateral.

Duration and Sequelæ.—When patients are placed under treatment as soon as a colic develops, restoration to health follows in from six to twelve weeks, but a second attack is likely to develop when such patients return to their usual work.

After repeated attacks of lead colic the prognosis becomes more and more serious with each seizure, and the nervous symptoms are also more prominent, mania and permanent insanity sometimes ensuing. Death results from extensive paralysis, intercurrent complications, or the exhaustion of mania.

HABITUAL CONSTIPATION (COSTIVENESS).

Remarks.—A condition in which the feces are not evacuated with the normal frequency or in which they are inordinately hard and expelled with difficulty. Constipation is due to diminished activity of the muscular coat of the intestine or to lessened secretion from the mucous membrane, and often to both conditions.

Exciting and Predisposing Factors.—The quantity of intestinal fluid (succus entericus) secreted is influenced by such diseases as cirrhosis of the liver, valvular heart disease, etc., conditions that produce a chronic venous congestion of the intestines, and thereby interfere with the function of the intestinal glands. The action of the intestinal glands is also inhibited in wasting diseases, such as tuberculosis, carcinoma, and the anemias. In certain nervous conditions there appears to be an inhibiting action upon the secretory function of the intestine, consequently constipation is generally present in those suffering from melancholia, neurasthenia, mania, ataxia, and myelitis. Constipation the result of spasm of the bowel is exemplified by such diseases as tetanus and chronic plumbism.

Habit is of great importance as an exciting factor in this condition, since neglect to evacuate the bowel at stated intervals is sure to be followed by constipation.

In certain diseases of the nervous system characterized by diminished sensitiveness of the nerves of the intestine constipation prevails, and, indeed, a diminished sensation of the intestine is believed to figure prominently as an exciting cause of habitual constipation.

Local Causes.—Atony of the muscular coats of the intestine, peritonitis,—by reason of the fact that it inhibits peristaltic movements,—hemiplegia, and paralysis of the bowel. *Acute constipation* may result from a strangulated hernia, intussusception, impaction of the colon, and foreign bodies, and the condition may also follow operations upon the upper bowel and the rectum.

New-growths, either within the intestine or extending from adjacent structures, by lessening the lumen of the bowel from pressure, are also exciting causes of constipation.

Principal Complaint.—There is a feeling of fullness, weight, and pressure in the perineum and abdomen. Flatulence, colicky pains, and alternating diarrhea occur frequently. The hurried and often neglected performance of defecation gives rise to the so-called "cumulative constipation," in which the feces are but partially evacuated with each movement, and the rectum is not emptied. After such an action of the bowel a sense of fullness remains, and complete relief is not experienced.

Among the *reflex symptoms* are malaise, hebetude, irritability of temper, headache, flushing, palpitation, cold extremities, anorexia, paresthesia, vertigo, sleeplessness, and nightmare.

Pressure on the sacral and visceral nerves may cause neuralgia.

Physical Signs.—Inspection.—The tongue is coated and the lips and the mucous membrane of the mouth are often dry. The abdomen is, as a rule, distended unless there is gastroptosis, when there is a depression at the upper half of the abdomen, with a correspondingly conspicuous prominence of the lower abdominal hemisphere.

Palpation.—Upon palpation a peculiar dough-like feel is detected over the entire abdomen, particularly along the course of the colon. The flexures of the colon are readily outlined, and a tumor-mass at the sigmoid flexure is by no means uncommon.

Percussion.—There is an irregular distribution of abdominal tympany, and this changes at different times during the day.

Laboratory Diagnosis.—The urine is normal in quantity, of high color, and often displays a high specific gravity. It is rich in indican.

Complications.—Among the complications are ulcerative colitis, dilatation of the colon, hemorrhoids, typhlitis, intussusception, and the formation of intestinal sand.

AMYLOID DISEASE OF THE INTESTINE.

The cardinal symptom is diarrhea, which is persistent but mild in character, associated with symptoms of amyloid degeneration in other organs. Enlargement of the liver and spleen and an associated amyloid change in the kidneys are also present.

Laboratory Diagnosis.—The number of stools passed vary between two and six a day, and diarrhea may alternate with constipation.

The *urine* is usually of low specific gravity, nearly colorless, and may contain a moderate amount of albumin. Microscopically, the urine is usually rich in broad, pale casts (amyloid) (Fig. 268), and leukocytes and epithelial cells are common. The solid constituents of the urine are diminished.

The blood changes are those of a profound secondary anemia.

APPENDICITIS.

Pathologic Definition.—A catarrhal, ulcerative or gangrenous interstitial inflammation of the appendix, which may undergo resolution or progress to the formation of abscess. Perforation of the appendix is not uncommon, and following this accident there is localized and at times general peritonitis. In the catarrhal form of the disease the pathologic changes of the mucous surface are practically identical with similar grades of inflammation seen to involve other mucous surfaces. The inflammatory process tends

to subside, but there is a pronounced tendency toward relapses. In cases of long standing the appendix is frequently firmly adherent to adjacent structures.

General Remarks.—According to our present views appendicitis is a surgical rather than a medical affection. Since, however, general practitioners are constantly encountering cases of appendicitis, the prompt clinical recognition of the disease is not only a matter of interest, but also of great practical importance, in order that surgical treatment may be instituted at the proper moment, and furthermore because appendicitis ranks first in importance among the diseases of the intestinal tract.

The term "appendicitis" includes the affections known as *typhlitis* (inflammation of the cecum) and *perityphlitis* (a similar involvement of the connective tissue behind the cecum). When the symptoms of typhlitis or of perityphlitis are present, with but few exceptions the appendix was the part primarily affected.

Varieties.—Clinically, appendicitis may be divided into the following subclasses: (*a*) **Acute appendicitis,** in which the initial attack develops somewhat abruptly and without previous evidence of intestinal catarrh; (*b*) **chronic appendicitis,** characterized by a variable amount of discomfort localized in the right iliac fossa; (*c*) **recurrent appendicitis,** in which the patient experiences acute attacks at irregular intervals.

From a pathogenic standpoint we may further divide appendicitis into the following classes: *Catarrhal* or *obliterative* appendicitis, *ulcerative* appendicitis, *interstitial* or *parietal* appendicitis, and *perforating* appendicitis.

Exciting and Predisposing Factors.—The evidence at hand strongly suggests that no one organism is constant in exciting inflammation of the appendix. The bacillus coli communis, however, is present in from 70 to 80 per cent. of all cases. Various other pathogenic bacteria are also to be found.

(1) **Congenital structural deformities** contribute freely toward the production of appendicitis, and among these should be mentioned location, size, and length of the appendix. The length and position of the mesoappendix are also regarded as of importance, since these may tend toward an obliteration of the lumen of the appendix.

(2) **Stricture** near the cecum and **adhesive peritonitis** are also causative factors in appendicitis.

(3) **Fecal concretions, foreign bodies,** etc., are said to be contributing factors in about 7 per cent. of cases.

(4) **Ulceration** of the intestinal mucosa and of the appendix, *e. g.*, tuberculosis, typhoid fever, dysentery, and actinomycosis.

(5) **Heavy lifting** and straining are generally believed to be contributing factors.

(6) **Age.**—Appendicitis is especially common between the fifteenth and thirtieth years. It may, however, develop at any age, but very rarely occurs after the fourth decade or before the third year of life.

(7) **Sex.**—Males are attacked more often than females, the ratio being four to one.

(8) It has been conclusively shown that **gastrointestinal catarrh following influenza** is a potent factor in the production of appendicitis.

(9) **Heredity.**—Several members of the same family, as well as parent and offspring, have been reported as having appendicitis. Persons of a rheumatic and gouty diathesis are especially susceptible to this affection.

Principal Complaint.—Acute Appendicitis.—In the majority of

cases the *onset* is sudden, with *abdominal pain*, which is at first diffuse, but later becomes localized to the region of the appendix. Nausea, vomiting, and constipation are present. Occasionally, the disease is ushered in by a chill or a series of chilly sensations followed by fever; and there may be irritability of the bladder, and, later, retention of urine.

The pain is usually colicky in character, although it may be dull and aching. Not infrequently the pain is reflected over the entire abdomen, but the point of greatest intensity is, as a rule, in the right inferior abdominal quadrant and at McBurney's point. As the disease progresses the pain is usually localized to the site of the appendix, although it may radiate from that point in any direction. Patients not infrequently complain of pain in

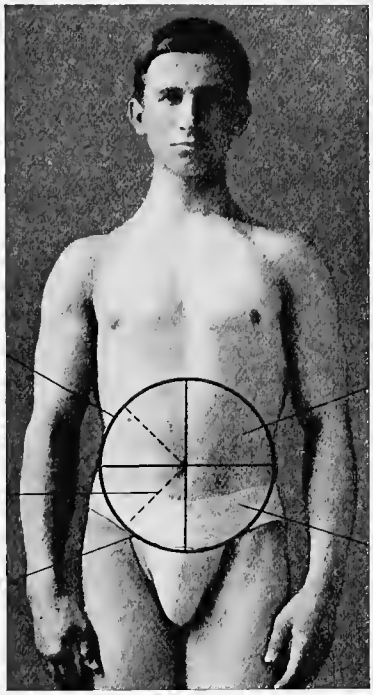


Fig. 224.—Arbitrary Regional Division of the Abdomen.

the right half of the abdomen, radiating to the right thigh and to the testicles. Dyspnea may be annoying.

In acute appendicitis the patient is loath to change his position, as the movement aggravates the pain. A symptom that is often overlooked is diarrhea, and it should be remembered that it may precede the initial pain in acute catarrhal appendicitis.

Thermic Features.—The patient is usually flushed, and the temperature rises somewhat abruptly to 102° to 104° F., mild cases fluctuating between 99° and 101° F. The degree of fever is no guide to the severity of the condition. In severe and fatal cases the temperature may be subnormal throughout the entire course of the disease, and if diffuse or gangrenous changes develop, the temperature may also be subnormal.

Physical Signs.—**Inspection.**—The patient sits or inclines toward the right side, and the right thigh is usually flexed upon the abdomen. Re-

traction of the right testicle is common. According to the degree of pain, the face will first be pinched or anxious, pale or blanched. When peritonitis develops as a complication the abdomen is distended.

The *tongue* is coated and moist, and in moderately severe cases the lips are often fissured. In advanced cases the tongue may be brown and deeply fissured, and the buccal mucous membrane dry and harsh.

Palpation.—The pulse is rapid (90 to 100), out of proportion to the fever, and in severe cases it may reach 120 or more a minute. It is strong and wiry at first, but later it frequently becomes weak, dicrotic, and compressible.

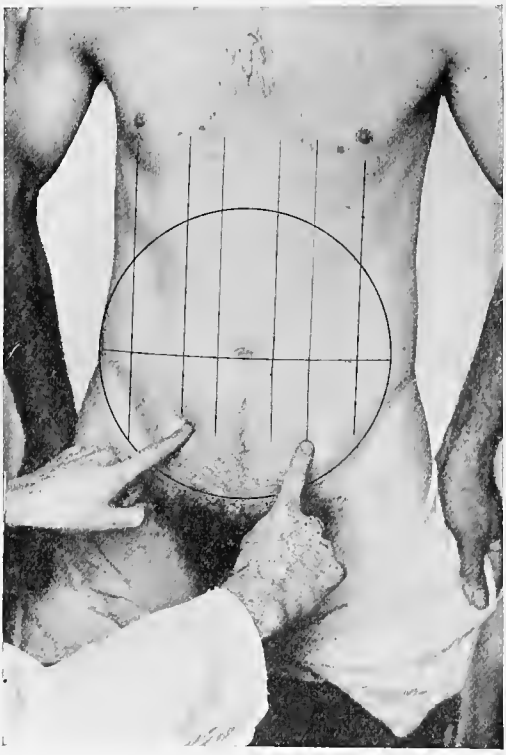


FIG. 225.—METHOD OF DETERMINING THE DEGREE OF ABDOMINAL TENSION, WHICH IS INCREASED OVER ENLARGED SOLID VISCI, PROLAPSED STOMACH, VARIOUS ABDOMINAL TUMORS, FLOATING KIDNEY, AND OVER THE MUSCLES OVERLYING ACUTE INFLAMMATORY PROCESSES.

Tension is universally increased in general peritonitis, ascites, and tympanitis.

Firm pressure over the site of the appendix (Fig. 224) will elicit a variable degree of tenderness, and often excite pain. Fixed tenderness is practically constant on pressure over a limited area at the center of a line between the anterior superior iliac spine and the umbilicus, and is a very valuable sign in appendiceal inflammation. Fixed tenderness at the right of the umbilicus is extremely unusual, although it has been observed. Tenderness may be distributed at different points of the abdomen (Fig. 224), but within a few hours after the onset it becomes localized at McBurney's point.

Palpation also elicits unusual rigidity over the right rectus muscle, a sign that is present early, even before actual tenderness is evident. The degree of tension of the two rectus muscles should be compared in every questionable

case of appendicitis, and although increased tension is not an infallible sign, it is to be regarded as one of great significance in formulating a careful diagnosis (Fig. 225). Circumscribed induration manifests itself about the second day, and is soon followed by swelling and obliteration of the normal curvatures about the iliac spine.

Percussion over any portion of the abdomen discloses the greatest amount of tenderness or pain to be present in the right inferior abdominal quadrant and at the site of the appendix.

Laboratory Diagnosis.—The urine is scanty, of high specific gravity, rich in indican, and in about 40 per cent. of cases contains a trace of serum-albumin. Frequent urination is by no means uncommon, but the twenty-four-hour quantity seldom equals 50 fluidounces. There may be an increase in the colloidal coefficient.

Leukocytosis is the rule—10,000 to 25,000 cells in a cubic millimeter. If a differential leukocyte count shows over 75 per cent. of polymorphonuclear elements, pus is probably present. We have observed both private and hospital cases when the number of leukocytes per cubic millimeter approximated normal. Pus when encapsulated may exist without exciting leukocytosis, therefore the blood findings are not constant in subacute or in chronic forms of appendicitis.

Illustrative Case of Acute Appendicitis.—J. R., female, aged nineteen years; height, 5 feet, 8½ inches; weight, 142 pounds.

Family History.—Father living at the age of forty; mother, at thirty-eight, and both enjoy good health. An older brother died of organic heart disease at the age of sixteen, and a younger sister of scarlet fever at the age of twelve. No history of tuberculosis, malignancy, or inherited disease in ancestors.

Previous History.—Had the diseases of childhood, including measles complicated by catarrhal pneumonia at the age of fourteen. Has enjoyed good health during the past five years.

Social History.—A pupil in the senior class at high school. For the past year parents have noticed that she finds her school work unusually difficult. Her appetite is good, and she takes a fair amount of outdoor exercise daily.

Present Illness.—While in the mountains during her summer vacation she ate more than usual, and upon one occasion suffered from a severe attack of abdominal pain, accompanied by nausea, vomiting, and later by obstinate constipation. She complained chiefly of soreness over the right inferior abdominal quadrant, the soreness being increased by exertion. The character of food taken did not appear to exercise any influence upon her general condition, although her discomfort was more pronounced when constipation existed. Ten weeks later, and following the original seizure, she experienced a severe attack of abdominal distress, accompanied by violent vomiting, which compelled her to take to her bed.

Paroxysmal *pain* was situated midway between the anterior-superior spine of the right ilium and the umbilicus, was cramp-like in character, and aggravated by movements and by pressure over the involved area. Pain radiated to the right thigh and down the right leg as far as the knee; there was also distress in the right lumbar region. Acute pain lasted for about twelve hours, and subsided after purging with citrate of magnesia.

During the first thirty-six hours the temperature fluctuated between 99° and 101.4° F., when it fell gradually to the normal.

Physical Examination.—*General.*—During the interval between the attacks the patient showed a decided tendency to incline toward the right side when standing, she also bent slightly forward, and there was moderate drooping of the right shoulder. The complexion was normal, the body well nourished, and the general appearance showed but little evidence of disease.

Local Examination.—*Palpation.*—The muscles of the right abdominal wall were distinctly rigid as compared with those of the left side of the body, and upon firm pressure over this area tenderness was elicited for several days following the paroxysmal attack.

Laboratory Findings.—The quantity of urine voided during the twenty-four hours approximated the normal, and was free from albumin and glucose, although it gave a decided reaction for indican. Microscopic and chemic examinations revealed nothing of pathologic interest.

Diagnosis by Induction from the Clinical Data. — The occurrence of an attack similar to the present one ten weeks previously was suggestive. The history of an acute onset of intestinal pain developing in a young and previously healthy individual, and the additional fact that such pain was localized to the inferior right abdominal quadrant, aroused a strong suspicion as to the nature of the disease. Extreme tenderness at McBurney's point and rigidity of the muscles of the right abdominal wall were confirmatory of the existence of appendicitis. The patient's attitude, flexing the right thigh upon the abdomen and inclining slightly toward the right side, was also significant. Again, the thermic changes pointed to appendicitis.

Differential Diagnosis.—The acute onset of the first attack, following dietetic error, strongly suggested the possibility of acute gastritis or intestinal colic, since vomiting and abdominal pain were among the conspicuous symptoms. The condition was distinguished from the two last-named maladies by the following: First, the pain was soon definitely referred to the region of the appendix; second, there was circumscribed tenderness at McBurney's point, and rigidity of the right abdominal muscles was present; third, localized tenderness existed for a period of three days.

Course of the Disease.—The patient came under our care during the second attack of abdominal pain; she was kept in bed for a period of eight days following the initial symptoms, and was then removed to the hospital for operation. The appendix was found considerably congested, and there were many adhesions about that organ, the colon, and the right ovary. She made an uneventful recovery, and returned to her home at the end of the third week, since which time she has been in good health.

Summary of Diagnosis.—This is based, first, upon the presence of abdominal pain, which eventually becomes more or less strictly localized to the region of the appendix, the history of previous attacks, the presence of persistent vomiting, and the position of the patient, *e. g.*, he inclines toward the affected side, and the right thigh is flexed upon the abdomen. Careful palpation is also invaluable in formulating a diagnosis, since rigidity of the right rectus muscle and localized tenderness at McBurney's point are among the important signs of this affection.

Moderate fever, when present, should be regarded as an important symptom, yet the absence of fever and the presence of the other essential features of the disease are at times observed.

The character of the attack, *e. g.*, sudden onset, gradual increase in pain, which eventually becomes localized, vomiting, and constipation, is to be considered in formulating a diagnosis of acute appendicitis.

Differential Diagnosis.—See differential table, as follows.

TABLE SHOWING THE DIFFERENCES BETWEEN ACUTE APPENDICITIS, RENAL COLIC, EXTRA-UTERINE PREGNANCY WITH RUPTURE, AND HEPATIC COLIC.

Appendicitis.	Renal Colic.	Extra-uterine Pregnancy with Rupture.	Hepatic Colic.
1. History of previous attacks common.	1. History of previous attacks common.	1. History of sterility.	1. History of previous attacks.
2. Pain over the right half of the abdomen, becoming localized later at McBurney's point.	2. Pain may be localized at either side of the abdomen, along the course of the ureter.	2. Pain low and at the center of the abdomen.	2. Pain in upper right abdominal quadrant.
3. As the disease advances the pain becomes circumscribed at McBurney's point.	3. Patient can feel the pain getting lower and lower until stone escapes into the bladder.	3. Pain disappears in a short time, and is followed by the symptoms of internal hemorrhage.	3. Pain radiates to right shoulder.

Appendicitis.—(*Continued.*)	Renal Colic.—(*Continued.*)	Extra-uterine Pregnancy with Rupture.—(*Continued.*)	Hepatic Colic.—(*Continued.*)
4. Vaginal hemorrhage absent.	4. Vaginal hemorrhage absent	4. Vaginal hemorrhage present.	4. Vaginal hemorrhage absent.
5. Temperature, 99° to 102° F. or, rarely, higher.	5. Temperature may be subnormal, followed by rapid rise, and then drop by crises.	5. Temperature subnormal for several hours.	5. Temperature may rise abruptly to 102° to 105° F., and fall by crisis.
6. Frequent micturition; urine rich in indican.	6. Frequent micturition; urine bloody.	6. Negative, urine blood-stained by vaginal flow.	6. Urine contains bile twelve to twenty-four hours after the initial pain.
7. Localized tenderness at McBurney's point, and pressure here intensifies the pain.	7. Localized tenderness not constant. Pressure exercises but slight influence.	7. Pressure negative.	7. Tenderness in the epigastrium and over the gall-bladder.
8. Distention of the right inferior abdominal quadrant within the first twenty-four hours.	8. No distention of the abdomen.	8. May be abdominal distention due to hemorrhage.	8. No abdominal distention.
9. Negative.	9. Negative.	9. Negative.	9. Jaundice twenty-four hours after the attack.

Among the conditions that may be mistaken for acute appendicitis, **acute indigestion** occupies a prominent place. Fixed pain in the region of the appendix and localized tenderness are two of the strong clinical points in favor of appendicitis, and in the absence of these symptoms, with intestinal derangement, acute indigestion with colic is highly probable.

Cholecystitis with Distention.—This gives rise to a superficial mobile, pear-shaped tumor, with or without jaundice—all of which features are not encountered in appendicitis. The tumor in appendicitis is generally below the umbilicus, but when the appendix extends upward, the tip may touch the gall-bladder, making an accurate diagnosis impossible.

Acute Peritonitis due to Pelvic Disease.—When the diseased appendix occupies the pelvic fossa, the differentiation between right-sided salpingitis and appendicitis is difficult. *Right ovaritis*, owing to the presence of pain, tenderness in the right iliac fossa, and fever, often closely simulates appendicitis. In ovaritis, however, tenderness is less pronounced, and the organs of generation show certain disturbances (menorrhagia, etc.). A complete history, coupled with a careful pelvic examination, will usually enable one to distinguish between these two conditions. The following table shows some of the distinctive features between pyosalpinx and appendicitis.

Pyosalpinx.	Appendicitis.
1. History of gonorrhea, puerperal sepsis, or of long-standing leukorrhea.	1. Negative.
2. Pain most marked at or near the menstrual period.	2. Not appreciably influenced by menstruation.
3. Escape of purulent discharge from the cervix uteri. Periodic discharge of a large quantity of pus from the vagina.	3. Absent.

Pyosalpinx.	Appendicitis.
4. Digital examination shows swelling in the region of the ovary and tube, and a soft, sausage-like tumor may be present.	4. Tenderness upon digital examination, but a tumor mass is not common.
5. Progressive secondary anemia the rule.	5. Less conspicuous.
6. Vomiting unusual.	6. Vomiting common.
7. Micturition undisturbed.	7. Frequent micturition early during an attack of appendicitis.
8. Temperature irregular (septic) in character.	8. Temperature 99° to 102° F., and of an irregular type.
9. Chills, followed by fever and sweats, quite common.	9. Unusual.
10. There may be constipation or diarrhea.	10. Obstinate constipation the rule.

Perinephritic Abscess.—A diagnosis is made either from the history of previous attacks of nephritic colic or by exploratory incision. Analysis of the urine may be of service if perinephritic abscess communicates with the pelvis of the kidney.

Acute Tuberculous Peritonitis.—In both appendicitis and tuberculous peritonitis there are pain, tenderness, and fever; in the latter condition, however, the onset is more gradual, and the signs of tumor and increased resistance in the ileocecal region are absent. Movable dullness may be present in a tuberculous affection of the peritoneum.

Acute hemorrhagic pancreatitis simulates appendicitis with generalized peritonitis. The deep-seated epigastric pain and shock present in pancreatic hemorrhage are absent in appendicitis.

Dietl's Crises.—In movable kidney all the symptoms may point toward appendicitis. A history of similar attacks, following which the patient voids large quantities of urine, renders the diagnosis clear. In patients who have suffered from Dietl's crises the kidney is readily palpable. (See differential table, p. 540.)

Acute Intestinal Obstruction.—Here tumor, if present, is not likely to be situated in the right inferior quadrant. The portion of the bowel below the obstruction is at times thoroughly emptied, and the discharges are frequently serous and bloody. Stercoraceous vomiting develops somewhat early in intestinal obstruction, and is an extremely uncommon symptom in appendicitis.

Neurasthenia may also simulate acute appendicitis. (See Fig. 226.)

Chronic Appendicitis.

Clinical Definition.—A condition in which repeated attacks occur at intervals of weeks or months, each relapse being characterized clinically by the symptoms and signs of acute appendicitis.

General Remarks.—During the intervals between attacks the patient may be free from symptoms of acute appendicitis, although many patients constantly complain of a sense of discomfort and of moderate soreness about the region of the appendix.

Relapses appear to be brought about by muscular effort, indiscretions in diet, acute gastritis, and constipation.

Nervous Manifestations.—When the patient has been told that he is suffering from appendicitis, and when two or more attacks have occurred,

he becomes extremely nervous. Neurasthenia and hysteria may develop, and the general health may become impaired.

Summary of Diagnosis.—The diagnosis of chronic, as of acute, appendicitis rests largely upon the following factors: (1) The history of localized tenderness; (2) pain at McBurney's point, (3) the existence of fever, which is often slight during each relapse.

Differential Diagnosis.—Chronic appendicitis must be distinguished from **carcinoma of the cecum** and from **tuberculosis of the cecum.** Typhlitis has been considered conjointly with and as resulting from chronic appendicitis.

Carcinoma of the cecum presents certain points of similarity to chronic appendicitis. The amelioration of all the symptoms for a period of weeks or months favors the existence of appendicitis, for in carcinoma there are no distinct intervals of remission. Fever is also a feature of chronic appendicitis, and is, as a rule, unknown in carcinomatous disease of the colon. Emaciation and progressive weakness are more pronounced in carcinoma than in appendicitis.

Appendicitis is a disease of youth and early adult and middle life, whereas carcinoma of the colon is rare before the fortieth year.

Tuberculosis of the cecum does not resemble chronic appendicitis until a variable degree of localized peritonitis is present. In tuberculosis there is no distinct intermission of symptoms, fever is usually absent or mild, and diarrhea is more common than constipation. The detection of tubercle bacilli in the stools makes the diagnosis of tuberculous enteritis positive.

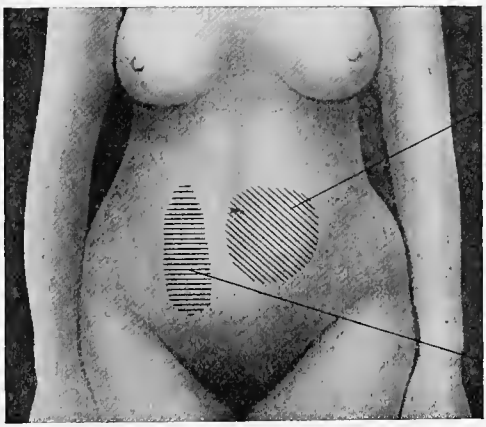


FIG. 226.—LOCALIZED AREA OF HYPERSENSITIVENESS SEEN IN NEURASTHENIC INDIVIDUALS AND LIABLE TO BE MISTAKEN FOR ABDOMINAL DISEASE.

RECURRENT APPENDICITIS.

Clinical Definition.—A condition in which successive attacks of acute appendicitis occur in the same individual at intervals of from several months to one or more years.

General Remarks.—Severe attacks may follow comparatively mild ones, or, on the other hand, each successive attack may be more and more mild for an indefinite time. The severity of the previous attack is no guide to the grade of the inflammation of the next recurrence.

Clinical Course.—Mild forms of catarrhal appendicitis tend to go on toward recovery in the majority of instances, although these comparatively mild cases may cause the formation of peritoneal adhesions. Severe cases of catarrhal appendicitis, and particularly those occurring after the patient has had one or more previous attacks, may terminate fatally unless surgical treatment is instituted. There is no means by which it is possible to estimate satisfactorily the danger in any case. The temperature and general condition

of the patient may be confusing, and, in fact, in selected cases of appendicitis, may be misleading. Irrespective of how mild the symptoms may be, the tendency is for an acute appendiceal process to become more and more general in its distribution, and such extension may take place without materially influencing the pulse, the temperature, or the degree of pain.

Suppuration may follow in those cases that apparently run an insidious course, and it must be remembered that fever is not a constant finding in appendiceal suppuration. It must also be emphasized that in this condition a subnormal or normal temperature may be present in cases that are as serious as those displaying a temperature of 101° or 102° F.

Complications.—Complications of any kind materially increase the danger in all types of appendicitis. Among the more frequent complications are abscess, perforation of the colon followed by general peritonitis, perforation of the colon with localized peritonitis, and localized peritonitis with the formation of adhesions and consequent lessening of the lumen of the intestine.

Mortality.—Collectively speaking, Fitz estimates the mortality for appendicitis at 14 per cent., but in our experience a much lower mortality rate has followed early surgical treatment. The more chronic the type of appendicitis, judging from the symptoms and frequency of attacks, the more uncertain is the prognosis. In our opinion, the mortality rate in all forms of appendicitis is greatly reduced by prompt surgical treatment, except in those cases in which other conditions make surgical interference impracticable.

CHOLERA INFANTUM.

General Remarks.—A type of diarrhea seen in young children, in which the general symptoms bear a striking resemblance to those of Asiatic cholera, but develop after the ingestion of impure milk or improper food.

Characteristic Features.—The onset is sudden, and is characterized by the passing of an unusually large quantity of feces. A distinctive feature between cholera infantum and the other types of diarrhea previously outlined is that no diarrhea or intestinal trouble antedates cholera infantum.

Gastric Symptoms.—Vomiting is an almost constant symptom, developing early and continuing throughout the attack. There is complete anorexia, and even ice is ejected almost immediately after it is taken. Intense thirst is present.

Thermic Features.—The temperature rises early to 103°, 104°, or 105° F., and may reach 106° or 108° F., but with the approach of the general symptoms of collapse the fever soon falls to normal. Eventually, the external temperature is subnormal in severe cases.

Nervous Manifestations.—The child may be extremely nervous at the onset, but later, as the vomiting subsides, he falls into a semicomatose state and coma finally develops.

Laboratory Diagnosis.—Within a few hours after the onset the *stools* become watery, and yellowish-green in color, and as the condition progresses the dejecta become watery. To the naked eye the stools contain shreds of mucus and small flocculi. The stools may not emit a distinct odor, although occasionally an odor of musk is present. The frequency of stools varies greatly, numbering from 12 to 50 a day.

The *vomitus* at first contains the contents of the stomach, but later it is often bile-stained and of the consistence of water.

Summary of Diagnosis.—The diagnosis is based upon: (1) The history of the ingestion of decomposed food; (2) the copious evacuation of the bowel and the character of the bowel movements; (3) the rapidity of the

heart's action and high temperature that prevail early during the attack; (4) the great tendency toward collapse; (5) the character of the vomitus.

Clinical Course and Duration.—In practically all cases of cholera infantum the prognosis is guardedly favorable. In those instances in which judicious treatment is instituted early, convalescence may be established in from one to four days, or later by the end of the first week. The intestinal symptoms commonly assume a subacute course between the first and fourth days, after which convalescence may not be established for several days or even for weeks.

NERVOUS DIARRHEA.

General Remarks.—A peculiar condition in which no true pathologic lesions are found, but in which there is an increased motor power of the bowel that results in diarrhea. Generally speaking, nervous diarrhea should be regarded as reflex, since it frequently develops after fright, attacks of hysteria, mental strain, and psychic influences. The number of stools may vary from three to twenty a day, but such stools do not present anything characteristic. Patients afflicted with this condition are, as a rule, well nourished, but of a neurasthenic temperament.

Differential Diagnosis.—Nervous diarrhea is to be distinguished from chronic dysentery, the latter condition being characterized by its prolonged duration and by the character of the stools. (See p. 734.) The fact that the diarrhea always follows nervous excitement differentiates nervous diarrhea from that due to dietetic errors. (See Summer Diarrhea of Children, p. 544.)

ENTERALGIA (INTESTINAL NEURALGIA).

General Remarks.—Neuralgic pain of the intestine may be localized or general, and in those cases in which there is associated enterospasm, the condition may be sufficiently severe to constitute intestinal colic.

Predisposing and Exciting Factors.—Enteralgia is usually the result of a neurosis, occurring in debilitated and neurasthenic individuals. At times it results from the ingestion of certain foods or from excitement. Enteralgia may be reflex in character, and follow the taking of indigestible or metallic substances into the stomach, infection by intestinal parasites, obstinate constipation, and the like.

Enteralgia the result of organic disease is best seen in the crises of locomotor ataxia, and may be due to toxic poisons, as evidenced by the intense abdominal pain of lead workers. (See Enterospasm.) There is unusual sensitiveness of the intestine in persons suffering from chronic appendicitis and from peritonitis of long standing.

Principal Complaint.—Enteralgia may develop suddenly, or it may set in less abruptly, and is then attended with eructations of gas and flatulence. In the fully developed attack the pain may be so violent as to cause the patient to faint. The pain is described as boring, tearing, or lancinating in character. It is not localized at any particular portion of the abdomen, but, on the contrary, is likely to be diffuse.

Attacks may be brief, or less often they are prolonged for hours or even weeks. Sudden subsidence of the pain is quite characteristic. Recurrences are the rule, but the intervals during which the patient is free from pain vary both in different cases and in the same patient.

Hypogastric Neuralgia.—When neuralgic pain is limited to the epigastrium, it is usually termed "epigastric neuralgia." Examples of this type are seen in locomotor ataxia and in rectal and ovarian diseases.

Clinical Course and Duration.—The prognosis depends entirely upon the etiologic factors. As a rule, when the patient's general condition is good, improvement follows.

CARCINOMA OF THE INTESTINE.

General Remarks.—Carcinoma of the bowel may be either primary or secondary in nature. Primary carcinoma of the intestine, however, is rare in comparison with the great frequency with which carcinoma occurs elsewhere. Carcinomatous involvement of either the large or the small intestine is a common cause of chronic intestinal obstruction. The occlusion is effected both by direct pressure and by intrusion of the growth upon the lumen of the gut.

Predisposing and Exciting Factors.—Age—after forty—and heredity stand as prominent predisposing factors. Gastro-intestinal catarrh and intestinal ulceration render the patient especially susceptible to carcinomatous disease.

Principal Complaint.—Clinically speaking, carcinoma of the lower portion of the intestine, especially when it involves the rectum, belongs to a special field of surgery, namely, proctology. The chief symptoms are *distress*, which increases progressively until there is *intense pain*, which may radiate from the rectum. The first discomfort experienced by the patient occurs during defecation, but with the progress of the disease the pain becomes almost constant, although it is always increased during and after defecation. *Diarrhea* usually alternates with constipation, and when a considerable portion of the rectal mucosa is involved, *blood* and *bloody mucus* are passed with the feces. As in carcinoma in other portions of the body, there are progressive loss of strength and emaciation, together with anxiety.

If the carcinoma is situated above the rectum, the patient's symptoms are often vague, and, in fact, there is no definite group of symptoms characteristic of carcinoma of this portion of the bowel. Progressive emaciation and weakness are, however, constantly present. Irregular attacks of lancinating abdominal pains usually occur, generally some hours after eating or after defecation. Nausea, vomiting, and anorexia are the rule. When carcinoma involves the duodenum, the vomiting of bile and jaundice is common.

Physical Signs.—**Inspection.**—The patient is emaciated and cachectic. When the neoplasm involves the sigmoid flexure of the colon, a peristaltic wave is often seen above the site of the obstruction. In selected cases it may be possible to detect an irregular enlargement of the abdomen.

Palpation.—By means of palpation it is often possible to outline a distinct mass in the abdomen, and the portion of the intestine involved is quite readily approximated by the situation of the tumor. A nodular abdominal growth is almost invariably carcinomatous in nature.

Location of the Growth.—To determine the site of a palpable, supposedly carcinomatous abdominal tumor, the following points may be of service:

(*a*) A hard, nodular mass situated near the median line, between the ensiform and the umbilicus, would suggest a gastric or duodenal lesion. If jaundice is present, the lesion is most probably duodenal.

(*b*) A tumor situated in the right iliac region, probably has its origin in the lower portion of the ascending colon or the cecum.

(*c*) A mass in the left iliac region, or even slightly above the brim of the pelvis, is suggestive of involvement of the sigmoid flexure or descending colon.

(*d*) In neoplasm of the splenic flexure the colon may be drawn from its

normal position and the tumor be detected to the left of the median line, and possibly as low as the brim of the pelvis.

Percussion is of value when the tumor mass is large, revealing, as it does, an area of dullness of variable size.

By distending the colon with air or water, it is possible, on making percussion over the distended portion, to determine the exact extent of the carcinomatous involvement, and this means of diagnosis may enable the clinician to distinguish between tumor of the colon and tumor extending from adjacent structures.

Laboratory Diagnosis.—The quantity of urine voided during the twenty-four hours is approximately normal, except during the attacks of diarrhea, when it is decidedly lessened. In those cases suffering from chronic obstruction the feces are passed in small nodular masses, or there may be the so-called thread-like or ribbon-like stools, which are quite characteristic.

Summary of Diagnosis.—In the majority of instances the diagnosis rests largely upon the age of the patient, the nature of the pain, and the fact that it increases progressively from day to day. The character of the bowel movements and the presence of blood and mucus in the stools are also of great importance in formulating a diagnosis.

Differential Diagnosis.—The following conditions may be mistaken for carcinoma of the intestine: (1) Early during its course carcinoma of the rectum is frequently confused with **chronic dysentery,** on account of the blood and mucus that are passed with the stools. Much stress should be laid upon the presence of an abdominal tumor and the detection of the peristaltic wave through the abdominal wall. Progressive emaciation and cachexia, although always present in carcinoma elsewhere, are also present, though in a lesser degree, in other diseases in which starvation is evident.

(2) **Fecal impaction** may be dependent upon carcinomatous stenosis of the colon, and therefore the distinction between these two conditions may be made largely upon the presence or absence of pain, cachexia, and emaciation, all three of which symptoms are less marked or entirely absent in simple fecal impaction.

(3) The tumor resulting from **intussusception** differs from carcinoma in that it develops abruptly, the pain being acute, instead of boring, as in carcinoma. Intussusception is of short duration, whereas carcinoma is chronic.

(4) **Carcinoma of the head of the pancreas** differs from carcinoma of the intestine in that the tumor mass is not movable. Jaundice is common.

(5) **Carcinoma of the gall-bladder** may result in stenosis of the common bile-duct, when distention of the gall-bladder may be confounded with tumor of the colon, and here the differentiation is at times possible after inflating the colon.

(6) **Hydronephrosis and floating kidney** are differentiated from neoplasm of the colon by the fact that these tumors are freely movable when the patient is turned from side to side. The tumor does not interfere with distention of the colon by either gas or water. A fact to be borne in mind is that neoplasm of the abdomen, developing outside the colon, may, by pressure or by the formation of adhesions, lessen the lumen of the colon, and in this way produce the signs and symptoms of carcinoma of the bowel.

Clinical Course.—As a rule, carcinoma of the intestine pursues a rapid course, terminating fatally in from a few weeks to six or even twelve months. When the pathologic changes in the colon are cirrhotic in nature, the disease may run a course of two or more years.

Complications.—Among the complications the most serious is perforation of the intestine, which is usually followed by acute suppurative peritonitis. Carcinoma of the rectum is likely to form fistulous openings into the vagina and bladder. Owing to stenosis of the rectum, instances have been reported in which overdistention of the colon was followed by general peritonitis.

CHOLERA MORBUS

(CHOLERA NOSTRAS; ACUTE DYSPEPTIC DIARRHEA).

Pathologic Definition.—An acute self-limited disease, excited by the eating of large quantities of indigestible food.

Predisposing and Exciting Factors.—Age.—Children and young adults are most prone to this disease. When it develops in infants the condition is usually referred to as cholera infantum. (See p. 544.)

Season.—During the *summer months* this disease is quite common, the greatest number of cases being seen during July, August, and September. *Unhygienic surroundings* without doubt contribute toward the production of cholera morbus, although the disease develops among the well-to-do classes quite as frequently as among the poorer. The theory has been advanced that this disease is probably microbic in origin. The variety of food taken certainly plays an important rôle in its production, since the eating of unripe fruits and of vegetables, such as string-beans, peas, egg-plant, cucumbers, etc., is often followed by an attack.

Principal Complaint.—A history of gastro-intestinal catarrh continuing for several days is quite common, and the patient will usually admit having eaten some indigestible substance or unripe fruit and vegetables.

The *onset* is, comparatively speaking, sudden, and is ushered in with acute abdominal pain, followed by violent vomiting, severe diarrhea, and a tendency toward faintness. In addition to *intestinal cramp* there are also cramp-like pains in the calves of the legs, thighs, and at times in the muscles of the forearms. In severe cases there is intense thirst, and the patient is unable to retain either liquids or solids taken by the mouth.

Thermic Features.—The temperature rises abruptly to from 100° to 104°, 105°, or even 106° F., remaining high during the acute stage of the disease; when the symptoms of collapse appear, the cutaneous temperature may be subnormal, whereas that of the rectum may be above 104° F.

Physical Signs.—Inspection.—The patient usually assumes a recumbent posture, with the legs well flexed upon the abdomen; the expression is anxious, the face is pale, the cheeks are sunken, the lips are cyanosed, and the abdomen is scaphoid in shape.

Palpation.—Tenderness upon deep pressure over the stomach and colon may be elicited. The skin soon becomes cold and clammy, and is often beaded with drops of cold perspiration. The hands, feet, and nose are extremely cold. The pulse soon becomes weak, the beats numbering from 110 to 140 a minute, and unless treatment is instituted early, it may become irregular and dicrotic.

Laboratory Diagnosis.—The number of stools voided during the early stages is extremely high. At first they contain only the contents of the bowel, generally partially digested food, each movement being accompanied by the passage of a large amount of flatus. Later the stool may be almost watery, and finally it becomes serous in character.

The vomitus at first contains the contents of the stomach, with some

of the food that has been previously taken, but later it may be made up of watery material.

A *bacteriologic study* of the vomitus and of the feces will reveal the presence of a number of bacteria, many of which are non-pathogenic. Finkler and Prior describe a spirillum which they found present in a large percentage of cases, but since streptococci and staphylococci are also occasionally present, it is quite difficult to estimate the pathogenicity of any one special organism cultivated from either the vomitus or the dejecta.

The *urine* is diminished in quantity, high in color, rich in indican, and often contains a trace of albumin.

Summary of Diagnosis.—A history of the ingestion of a large amount of indigestible food, and particularly of unripe fruits, is highly important. Violent vomiting, together with purging, intestinal colic, and cramps in the muscles of the extremities, are in themselves quite characteristic of cholera morbus. The appearance within the course of a few hours of extreme pallor, subnormal temperature, a weak, rapid pulse, and the fact that the patient's face is beaded with perspiration, all further substantiate the diagnosis.

Differential Diagnosis.—The general clinical picture of cholera morbus is quite similar to that of **Asiatic cholera,** but the geographic location and the absence of an epidemic, together with a clear history of the eating of certain unripe fruits, will be of great value in making a differentiation.

A bacteriologic study of the feces and of the vomitus will enable one to distinguish positively between these two conditions, since the comma bacillus is always present in those suffering from Asiatic cholera. Great care should be exercised in obtaining a careful clinical history, as this will serve to exclude the possibility of ptomain poisoning and of poisoning by drugs (*e. g.*, arsenic).

Clinical Course and Duration.—In the majority of cases of cholera morbus that tend to terminate favorably all the alarming symptoms subside in from three to six hours. In severe types of this condition the acute may be followed by a series of subacute symptoms, which may continue for from twenty-four to forty-eight hours. When cholera morbus develops in individuals who were previously healthy, a fatal termination is rare, and, in fact, the vast majority of all cases terminate favorably when judicious treatment is instituted early. The element of danger is that of profound collapse, and with this in mind, the physician is usually able to apply the treatment necessary to ward off this fatal symptom. Complications and sequelæ are rare.

DISEASES OF THE PERITONEUM.

ACUTE GENERAL PERITONITIS.

Pathologic Definition.—A disease characterized by an acute inflammatory process involving an extensive portion of the peritoneum.

Predisposing and Exciting Causes.—Age.—Acute general peritonitis occurs oftenest between the fifteenth and forty-fifth years, developing at a time in life when all the abdominal organs are most active. General peritonitis has been said to exist even in intra-uterine life, although it is quite uncommon in infants, Holt having found it but four times in 726 consecutive autopsies. In an analysis of 187 cases of acute general peritonitis occurring in children, Holt found 25 per cent. to have occurred in the new-born, 21 per cent. between the first and fifth years, and 54 per cent. between the fifth and

sixteenth years. The high percentage of cases found to affect the new-born is attributed to direct infection through the umbilical cord.

Sex serves as predisposing factor by reason of the great frequency of suppurative processes along the genital tract, and particularly in the uterus, ovaries, and Fallopian tubes. Again, young females are more likely to suffer from gastric perforation due to ulcer than are males. Gall-stones contribute toward the production of general peritonitis in older subjects, but this condition is usually followed by a local inflammation of the peritoneum, and unless rupture occurs, a general involvement of the serous sac is not to be expected.

It is generally believed that peritonitis may be a primary disease, but it is found to occur more often as a terminal complication of gout, general arteriosclerosis, rheumatism, and chronic Bright's disease. In the vast majority of all cases acute peritonitis develops as the result of infection from an adjacent viscus the surface of which is covered with peritoneum, or from rupture of one of the hollow abdominal viscera, *e. g.*, the colon, appendix, or stomach, tuberculous ulcer of the colon, or carcinoma of the intestine. Abscesses in this region may either rupture into the peritoneal sac or infect the peritoneum by the spread of their infection by contiguity. Acute peritonitis follows abscess, perinephritic abscess, empyema, or hepatic abscess. Peritonitis occasionally occurs as a sequel to septicemia and pyemia.

Bacteriologic Causes.—Many microörganisms are capable of producing acute general peritonitis; among these are Bacillus coli communis, Staphylococcus pyogenes, Streptococcus pyogenes, the bacillus of Friedländer, the pneumococcus, and Bacillus pyocyaneus. In one case seen by us acute general peritonitis was due to Micrococcus tetragenus. The peritoneal surface appears to be well adapted for the development of most pyogenic bacteria.

(*a*) The peritoneum is highly sensitive to the absorption of ptomains, a fact that serves to explain the high mortality rate in this affection.

(*b*) Peritonitis may result from the application of chemic irritants when these are placed directly upon the serous surface.

(*c*) Mechanical irritation, such as that produced by hernia, may give rise to a localized peritonitis that, in turn, may become diffuse. Secondary peritonitis may follow the introduction of certain toxic substances into the intestine, or may be the result of a slight inflammatory process involving the pleura.

(*d*) Rheumatic peritonitis is believed to follow exposure to cold and wet, and to be slightly influenced by season and by climate.

Principal Complaint.—This will be found to vary widely, such variations depending upon the character of the onset and the presence or absence of a preëxisting suppurative process covered by some portion of the peritoneum, with rupture of this abscess and the discharge of its purulent contents into the peritoneal sac. If general peritonitis follows a localized peritonitis due to an inflammatory process of one of the adjacent viscera, the onset is insidious, extending gradually over the entire peritoneal surface. If pus containing pyogenic bacteria is set free in the peritoneal cavity, the onset is sudden, and the height of the clinical phenomena is reached within a few hours. The early symptoms of peritonitis may be masked by those of the local inflammation that exists within the abdomen, and it is only by the exercise of great care that the clinician can arrive at an accurate interpretation of the clinical features.

Pain.—Pain constitutes the chief feature of diffuse peritonitis, and may

be of equal intensity over all parts of the abdomen, or, as is common, an acute pain may be localized. It is generally conceded that the area of intense pain corresponds to the site of the initial infection of the peritoneal surface—*e. g.*, intense pain in the region of the epigastrium and reflected toward the back and shoulder points somewhat strongly toward gastric ulcer. In one case seen by us in consultation there was rupture of the stomach, probably the result of an ulcer of long standing; in this case the extreme tenderness and intense pain were in the epigastrium, near the median line, and reflected over the right half of the abdomen, becoming most intense in the right inferior quadrant—McBurney's point. At operation, twenty-four hours after rupture, the peritoneal sac was found to be distended with the contents of the stomach, the appendix was normal, and there was no appreciable disease of the liver or of the intestine other than the diffuse peritonitis. Following rupture of a gastric ulcer the pain may be most intense in the lower half of the abdomen, and cases are recorded in which the reflected pain was limited to the bladder. After general peritonitis is well established it is practically impossible for the patient to inhale deeply, on account of the intense pain. The pain of peritonitis is continuous, although in asthenic patients it may be remittent or intermittent.

The *abdomen* becomes tympanitic within the first twenty-four hours, the pain appearing to be exaggerated by such distention. The abdominal muscles are spastic, and the movements of the abdomen are restricted. The patient complains that he is unable to take a deep breath, and that he must flex his thighs well upon the abdomen and lie upon his back in order to get relief.

Hiccough.—In patients whose strength has been well conserved prior to the onset of peritonitis, hiccough occurs early, and is apt to recur at intervals of but a few minutes, or at longest from one to two hours.

Between attacks of *vomiting* hiccough is an annoying symptom, and is suggestive of extensive peritoneal involvement. In the case previously cited vomiting was not an annoying symptom, whereas hiccough was more or less constant after the first twenty-four hours following gastric rupture.

Thirst is absent during the early stages of acute general peritonitis, but develops with the progress of the disease. The patient complains that his mouth and throat are dry and parched, and that his tongue appears to stick to the surface of his lips, teeth, and cheeks. In advanced peritonitis there may be deep fissures of the tongue and lips, from which blood-stained serum may ooze. Again, the tongue may be somewhat rolled together in the back of the oral cavity, and the patient be unable to protrude it.

Vomiting is produced by the taking of food and by increased peristalsis, although in many instances the vomiting is more or less continuous. At first the material ejected from the stomach may contain particles of food; later it is yellowish or greenish in color, and contains shreds of mucus. As the disease progresses the *vomitus* may be brownish in color and emit a fetid odor.

Constipation is a fairly constant feature of acute general peritonitis, and may be attributed either to spasm or to paralysis of the muscular coats of the intestine. Occasionally a case may be seen in which *diarrhea* is present throughout the entire course of the disease. Such diarrhea is attributable to a catarrhal infection (possibly septic) of the intestinal mucosa.

Thermic Features.—Peritonitis developing in the robust and previously healthy is likely to be ushered in with a decided rigor or a series of chills; even in the asthenic chilly sensations are experienced. Following

the chill, provided there has been a rupture of one of the hollow abdominal viscera, there may be shock, which is accompanied by all the nervous and circulatory phenomena of this condition, as, *e. g.*, a subnormal temperature, cold, clammy skin, anxious expression, and a weak, rapid pulse. A few hours later the symptoms of shock subside and the temperature rises steadily until it reaches 100° to 101° or 102° F., at which point it remains with moderate remissions. An intermittent temperature is occasionally seen.

Acute general peritonitis due to infection with the bacillus coli communis may be accompanied by but slight elevation of temperature, the average being from 99° to 101° F. Generally speaking, it should be stated that hyperpyrexia is unusual in general peritonitis, and when present, is probably due to an acute purulent process outside the peritoneum (puerperal sepsis, for example). In the last type of case, and occasionally in peritonitis affecting the previously healthy, the chill may be followed by a rapid rise in temperature, which may reach 104° or 105° F. within a few hours. In these septic cases the temperature assumes the continued type, reaching 104° or 106° F. by the third or fourth day of the disease. In sthenic subjects the temperature may rise abruptly, but during the first twenty-four hours following it may remit, reaching about 101°, and continuing at or near this point throughout the course of the disease. "Rectal temperature is often relatively high" (Anders).

Cardiovascular Peculiarities.—If the patient is seen during the stage of shock, the heart action will be extremely rapid, the pulse feeble and easily compressed. With the advance of general peritonitis the pulse becomes rapid, ranging between 120 and 160 beats a minute, and later it will be found to be intermittent, dicrotic, and compressible, the general evidences of circulatory collapse occurring, as a rule, between the fifth and twelfth days of the malady.

Owing to a general loss of cardiovascular tension and to extra muscular effort on the part of the heart there is often a decided pulsation of the vessels of the neck. The heart's apex impulse may be at the fourth and even the third interspace in the nipple-line. The heart displacement depends upon the elevation of the diaphragm due to abdominal distention. The area of relative cardiac dullness is seldom greatly increased, whereas the area of absolute cardiac dullness is increased and slightly elevated.

Respiratory Symptoms.—The respirations are rapid, varying in number from 25 to 45 a minute; the expansion is limited to the apex of the chest (superior thoracic type), although there is an apparent increased lateral expansion at the base. Owing to absence of the movements of the diaphragm, abdominal respiration does not occur. The patient is unable to take a full breath, and the acts of sneezing and coughing are accompanied by intense abdominal pain, which is likely to be followed by vomiting. The breath-sounds are decidedly altered and numerous râles are audible.

Nervous Manifestations.—In the majority of uncomplicated cases of general peritonitis these are not pronounced, and, in fact, it is customary for the mind to remain clear throughout the entire illness, except, say, for a few hours during the night. In certain instances delirium may be mild or extreme, and late in the disease it may terminate in coma. The fact that the patient states that he feels comparatively well except for the abdominal pain should be regarded as a grave symptom. There may be hypersensitiveness to light and sound, and the slightest jar of the bed causes agony.

Physical Signs.—**Inspection of the abdomen** reveals a general symmetric enlargement; the transverse diameter of the abdomen is increased

in proportion to the amount of fluid present. Abdominal distention is also influenced by the degree of relaxation of the abdominal muscles; consequently in the strong there may be but moderate enlargement. Generally speaking, however, the more decided the abdominal distention, the more virulent is the type of infection. The entire abdomen is immobile.

Palpation.—Rigidity of the abdominal wall is more or less pronounced, and is usually extreme after perforation of a hollow viscus. There is marked tenderness at every point overlying the peritoneal sac. After peritonitis has existed for some days, a friction fremitus may be detected over the abdomen. When there is great distention, the edge of the liver is not palpable, and at the upper portion of the abdomen there is a decided paramesial bulging between the tip of the sternum and the cartilages of the ribs.

Percussion over any portion of the abdomen causes great pain, and the note elicited is decidedly tympanitic. We have examined a number of cases in which liver dullness was absent, except in that portion occupying the posterior part of the superior right abdominal quadrant. This absence of liver dullness may be present irrespective of whether or not perforation of the stomach or colon has occurred. We have found at autopsy on subjects dead of acute general peritonitis that absence of liver dullness depended upon the fact that portions of the intestine had been forced between the surface of the liver and the abdominal wall.

It may be difficult, and at times impossible, to outline the area of splenic dullness in advanced peritonitis. Movable dullness due to the presence of free fluid in the peritoneal sac is at times detectable, yet in the majority of instances the fluid present does not change rapidly with the position of the patient, or the quantity may be too small to give rise to definite physical signs. Late in the course of acute general peritonitis adhesions may form that will materially inhibit the change of position of the abdominal fluid. We have seen cases at postmortem in which several pints of thick, tenacious fluid occupied the peritoneal sac.

The lung resonance does not extend as low as normal, and the margin of the lung is elevated in proportion to the degree of abdominal distention. As previously stated, the area of cardiac dullness is elevated (see Cardiac Features), as shown in a case reported by one of us.

Auscultation of the heart has been referred to under Cardiac Phenomena. Decided gurgling, due probably to intestinal fermentation as well as to peristalsis, is heard over the abdomen. When peritonitis has existed for a few days, it is possible to detect peculiar friction murmurs that resemble in character those heard over the pleura, but they are less likely to be rhythmic, although in a few instances we have found such murmurs to be synchronous with respiration.

Laboratory Diagnosis.—Fluid obtained from the peritoneum will be found to contain pathogenic bacteria, the bacillus coli communis, the streptococcus, and the staphylococcus being the organisms most commonly encountered; any pus-producing bacterium is, however, capable of causing acute peritonitis. The peritoneal fluid is rich in albumin, and microscopically is seen to contain many pus-cells, leukocytes, red blood-cells, and bacteria.

The *urine* is diminished in quantity during the entire course of the malady, but this diminution is commonly in direct proportion to the quantity of liquids taken. After the disease has progressed for forty-eight hours or more, albuminuria frequently develops, and the urine may contain hyaline and granular casts. In one of our cases the urine contained blood-casts and red blood-cells in large number. Indicanuria

develops early during the course of peritonitis and continues throughout, fluctuating in more or less direct relation to the degree of tympany present.

The *blood* displays decided evidence of suppurative infection, the number of leukocytes rising abruptly to from 10,000 to 20,000 in a cubic millimeter, and a much higher count may be observed. A differential count of the leukocytes shows the increase to affect chiefly the polymorphonuclear elements (which normally comprise 65 to 72 per cent. of the total number of white cells), which may equal 85 to 95 per cent. of the total number of leukocytes present. In selected cases leukocytosis is absent.

The number of red blood-cells in a cubic millimeter is but slightly, if at all, altered until peritonitis has advanced for several days, when there may be a corresponding decrease in the number of red cells and in the percentage of hemoglobin (secondary anemia). If the patient has been greatly depleted from hemorrhage or purging, the number of red cells in a cubic millimeter and the percentage of hemoglobin may both be above the normal limit, owing to concentration of the blood.

Summary of Diagnosis.—The presence of diffuse abdominal tenderness, which results in extreme pain when the patient moves or when pressure is made over the abdomen; marked tympanites, with rigidity; anxious expression; together with an elevation of temperature (which is governed by the type of infection), and leukocytosis with an abnormally high percentage of the polymorphonuclear cells are the cardinal symptoms of acute general peritonitis.

Clinical Course and Duration.—When acute general peritonitis develops in young subjects who have previously been in good health, the duration will vary from four to eight days, according to the predominant microörganism causing the infection. Infection by the bacillus coli communis is somewhat longer in duration, ending fatally between the eighth and fourteenth days. If the type of infection is highly virulent and the amount of purulent material that has escaped into the peritoneal cavity is large, the patient seldom survives such infection longer than forty-eight hours.

The asthenic forms of diffuse peritonitis, while they display less pronounced local symptoms at the onset, are equally as fatal as those of the sthenic type. Acute general peritonitis resulting from intestinal perforation, appendicitis, gastric ulcer, duodenal ulcer, etc., is severe from the onset, and tends rapidly toward a fatal issue. Rheumatic peritonitis may occur during the course of, or during convalescence from, acute articular rheumatism, and is the only type of acute general peritonitis that is amenable to medicinal treatment.

ACUTE LOCALIZED PERITONITIS.

Pathologic Definition.—An acute, circumscribed inflammation of some portion of the peritoneum that overlies an organ known to be the seat of an inflammatory process.

Varieties.—Localized peritonitis is often referred to as pelvic, in which case it arises from extension of an inflammatory process affecting the uterus (perimetritis), ovaries (peri-ovaritis), bladder (pericystitis), or appendix. If the circumscribed inflammation is located in the superior right abdominal quadrant, it is designated as perihepatitis or subdiaphragmatic peritonitis; when the peritoneum covering the spleen is involved, the condition is known as perisplenitis. Circumscribed areas of inflammation may arise at any

portion of the peritoneum as the result of carcinoma, tuberculosis, or rheumatism.

Predisposing and Exciting Factors.—Puerperal sepsis, gonorrhea, pyosalpinx, appendicitis, gall-stone, abscess of the liver, ovarian abscess, tuberculosis of one of the glands of the abdomen or of either the hollow or the solid abdominal viscera, primary carcinoma of the peritoneum or secondary carcinoma.

Principal Complaint.—Localized tenderness of the abdomen with acute lancinating pains upon movement or upon deep pressure over the tender area, constitute the chief complaint in connection with localized peritonitis. The degree of pain and of discomfort varies in direct correlation with the area of peritoneal surface involved and with the amount of movement common to the abdominal wall overlying the inflamed peritoneum.

These symptoms are milder than those seen in acute general peritonitis, and are often entirely concealed for a time by the symptoms referable to the organ known to be the primary seat of the infection. If the acute localized peritonitis becomes general, all the symptoms of acute diffuse peritonitis previously described (p. 550) appear within the first twenty-four hours. There is often a tendency for acute localized peritonitis to subside gradually between the third and tenth days, and to continue for an indefinite period, running a subacute course. Constitutional symptoms, as seen in acute general peritonitis, vary in direct proportion to the extent of the peritoneal inflammation present.

Thermic Features.—The temperature in localized peritonitis varies with the character of infection from which it has arisen—*e. g.*, in pelvic peritonitis following puerperal sepsis the temperature may be high—102° to 104° F.—and of the continuous type, although we have seen cases in which the temperature did not exceed 101° F. In perihepatic and localized peritonitis the temperature is more likely to be lower (100° to 101° F.) than when the peritoneum of the pelvis is involved, this peculiarity probably depending upon the fact that pelvic peritonitis is likely to be excited by virulent pus-producing organisms—*e. g.*, streptococcus and staphylococcus. There is no appreciable elevation of the temperature in acute localized peritonitis resulting from carcinomatous or tuberculous infection, and, in fact, these forms of peritonitis are frequently accompanied by a subnormal temperature during the morning hours.

Laboratory Diagnosis.—Usually, when acute localized peritonitis follows suppuration of some of the pelvic organs, leukocytosis is present with the other evidences of secondary anemia. (See Laboratory Diagnosis of Acute Peritonitis.) The urine generally contains indican, even when the area of peritonitic involvement is small.

Summary of Diagnosis.—The presence of localized abdominal tenderness and pain, which are increased by deep inspirations and by pressure over the abdomen, serves as the cardinal symptom of acute localized peritonitis.

The temperature is also an important factor, but is in no way characteristic, being controlled largely by the degree of fever resulting from the initial infection.

Duration.—Acute localized peritonitis runs a course of varied duration. It usually begins abruptly, and continues until the inflammatory process that has extended to the peritoneum from one of the adjacent tissues has abated. In a great many instances localized peritonitis is amenable to surgical treatment.

SUBPHRENIC PERITONITIS (SUBDIAPHRAGMATIC ABSCESS).

Pathologic Definition.—An acute inflammation of the peritoneum, which may or may not be suppurative in nature, limited to the right or left lobe of the liver, or at times to the lesser peritoneal cavity and the adjacent peritoneal covering of the diaphragm. Subphrenic abscess may contain air. Rarely, abscess of the liver due to infection with the bacillus coli communis may extend to the subphrenic peritoneum and cause a similar abscess, the walls of which are distended by gas. The Bacillus aërogenes capsulatus is also capable of producing abscess formation and of generating gas.

Predisposing and Exciting Factors.—Extension of inflammation from: (*a*) Perforating gastric ulcer; (*b*) the appendix; (*c*) perforation of the intestine; (*d*) perforating duodenal ulcer; and (*e*) from extension by contiguity from abscess of the liver or the pancreas.

Principal Complaint.—The patient complains of symptoms referable to the preëxisting pathologic condition of the abdomen—*e. g.*, gastric ulcer, appendicitis, hepatic abscess, duodenal ulcer, etc. He usually states that the condition occurred somewhat abruptly, with extreme pain and the vomiting of a large quantity of bile-stained or of bloody material. He declares that his breathing was difficult at the time this pain occurred and that there was a tendency toward faintness. Within the first forty-eight hours the patient experiences a chill or a series of chilly sensations, which are followed by profuse drenching sweats; night-sweats are likely to continue. The appetite is poor, there is great prostration, and at times the fever is that characteristic of suppuration.

Instances are recorded in which subdiaphragmatic abscess has ruptured through the diaphragm and communicated with the bronchi; in such cases the patient expectorates a portion of the contents of the abscess. One of us has reported a similar case, in which abscess of the liver following amebic dysentery communicated with the lung, and the patient expectorated large numbers of Amœba histolytica. Pneumothorax may be produced by rupture of an abscess into the pleural cavity, and it also occasionally results from a gastric ulcer perforating the diaphragm.

Cardiovascular Peculiarities.—If subdiaphragmatic abscess follows rupture of the stomach or duodenum, the pulse will become weak, rapid, and irregular at the onset; but after the symptoms of shock subside, the pulse remains full and strong, with but moderate acceleration, until the symptoms of sepsis—*e. g.*, chill, fever, sweats, etc.—appear. Following the appearance of these last-named symptoms the pulse increases in frequency and later becomes weak, rapid, compressible, and dicrotic. In one instance, seen at the Philadelphia Hospital, the pulse remained at 140 for a period of twenty-eight days, when a fatal termination occurred. If a large subdiaphragmatic abscess causes pressure upon the thoracic sympathetic system, unilateral flushing, unilateral sweating of the face, and inequality of the pupils are prone to occur.

Physical Signs.—Inspection.—There is usually bulging of the upper portion of the abdomen, and if the abscess is situated between the liver and the diaphragm, there is a bulging below the costal margin, due to the liver having been pushed downward. In one case, studied at the Philadelphia Hospital, the outline of the margin of the liver could be readily seen through the abdominal wall, and reached a point almost on a level with the umbilicus. The epigastric angle may be bulging in subdiaphragmatic abscess, and there may be prominence of the chest on the affected side. Movements of the

affected portion of the abdomen are limited, and if there is extensive peritonitis, the respiratory movements of the abdomen are decreased.

Palpation elicits more or less extensive tenderness in the region of the diaphragm, its extent depending upon the degree of involvement of the peritoneum. The upper portion of the abdomen is tense, owing to distention from the abscess and to peritonitis (muscular spasm). The liver or the spleen may extend for some distance below the costal margin.

Percussion.—In the presence of an abscess that does not contain air, the upper margin of the liver dullness will be found to extend to the fourth rib, above which there will be pulmonary resonance in the nipple line. If the abscess contains air or gas, there is a distinct area of tympany between the upper margin of the liver and the lower border of pulmonary resonance. Again, the area of liver dullness will be found to change slightly with the position of the patient, and its upper border will be far below the lower margin of the fifth rib.

If the abscess is situated at the left of the median line, a zone of tympany will be found between the splenic dullness and the lower border of lung resonance. In one instance in which a large subphrenic abscess was present, the area of cardiac dullness was displaced upward. Rarely, the lesser cavity of the peritoneum becomes filled with pus, when there is dullness in the left superior quadrant of the abdomen. If the lesser peritoneal sac is distended by gas, percussion will elicit a tympanitic note in the particular area affected. It is highly important to differentiate between the tympanitic note due to gas in the peritoneum or in the abscess, and the note obtained over the transverse colon. Tympany or dullness depending upon subdiaphragmatic abscess involving the lesser peritoneal cavity is always above the area of the colonic note. When the abscess is well distended by air, tympany may be elicited as high as the fourth rib in the left mammary line.

Auscultation.—There is an absence of breath-sounds over the affected area, whereas the normal respiratory murmur is heard above the abscess. Vocal resonance is also absent over the involved area, unless adhesions to the pleura have been formed and there is a direct communication with the lung, in which case voice-sounds are feebly heard. In the case of a large abscess, the lung may be decidedly compressed, and the respiratory sounds and voice-sounds over such lung are markedly intensified. Friction murmurs due to pleuritic or to peritoneal adhesions are not uncommon.

In two cases of subphrenic abscess studied by us the diaphragm on the right side of the thorax had been elevated to the lower border of the third rib, and in both of these cases the physical signs closely simulated those of pyopneumothorax, the distinctive feature being that voice-sounds were not well transmitted to the lower level of the chest.

It is usually stated that subdiaphragmatic abscess of the left side simulates pyopneumothorax more closely than does the same condition when situated to the right of the median line. Further, the distinction between subdiaphragmatic abscess and pyopneumothorax can be made only after a guarded analysis of the symptoms obtained and the physical signs elicited has been made.

Laboratory Diagnosis.—Aspiration of the abscess may be of great value in the diagnosis, since the fluid recovered will usually contain albumin, many pus-cells, few red blood-cells, and various forms of bacteria, among which the colon bacillus, the streptococcus, and the staphylococcus deserve special mention. (For complete laboratory diagnosis see Empyema, p. 160.)

Summary of Diagnosis.—A history of the existence of disease of the stomach, duodenum, or liver, with the sudden development of acute pain in the upper abdomen, followed by the general symptoms of shock. Gradual onset may terminate in a decided tenderness along the diaphragmatic margin and beneath the diaphragm. Later, characteristic features appear—*e. g.*, temperature (septic in type) and the general symptoms of pyemia. Leukocytosis, indicanuria, and, less commonly, albuminuria are present.

The physical signs of subphrenic abscess are of great importance in formulating the diagnosis. (See Physical Signs.)

The table below sets forth the most prominent features that will enable the clinician to distinguish between subdiaphragmatic abscess and pyopneumothorax.

Differential Diagnosis.—The accompanying table is designed to show by comparison the distinctive clinical features of pyopneumothorax and subphrenic abscess.

Pyopneumothorax.	Subdiaphragmatic Abscess.
1. History of pulmonary tuberculosis or of traumatism to the chest.	1. History of gastric or hepatic disease or traumatism to the abdomen or base of the chest.
2. Develops suddenly with severe pain in the side, followed by the symptoms of shock.	2. Develops insidiously.
3. Cough accompanied by free expectoration.	3. Dyspnea with cough, and when expectoration is copious, it is apt to be bloody and to emit a fetid odor. The breath may also be foul.
4. Practically no expansion on the affected side of the chest.	4. The expansion limited at base of chest on affected side.
5. Clavicle elevated and neck apparently shortened on affected side.	5. Not a conspicuous feature.
6. The voice and breath-sounds may show a metallic quality over the affected side.	6. Absent.
7. Normal breath-sounds absent over affected side.	7. Absent at base of thorax on affected side. Exaggerated at apex of same side.
8. Succussion splash audible when the ear is placed over any portion of the chest on the affected side.	8. Heard at base.
9. High tympanitic note is heard over the entire pleura on affected side.	9. Tympanitic note over base of affected side seldom extending above the level of the nipple.
10. Bell tympany (coin test) present over entire pleura on affected side.	10. Present over base of chest on affected side and not infrequently indistinct.

Clinical Course and Duration.—The duration is usually from ten days to twelve weeks, and this condition should be regarded as a purely surgical one.

CHRONIC DIFFUSE PERITONITIS.

Pathologic Definition.—An extensive chronic inflammation of the peritoneum involving both the lesser and the greater sac.

Predisposing and Exciting Factors.—Infection with the tubercle bacillus is the most common cause, although general chronic peritonitis may result from infection extending from a previously localized peritonitis, as, *e. g.*, in carcinoma or inflammatory disease of the liver, uterus, intestine, kidney, or retroperitoneal glands.

Principal Complaint.—In addition to that volunteered by the patient, there are general abdominal soreness and tenderness, which are increased upon movement of the abdominal muscles and upon deep inspiration. The patient always complains of a variable amount of discomfort that he cannot well describe—a sensation of fullness, or as though something were pulling in his abdomen.

After general peritonitis has existed for some months or even years, numerous adhesions of the peritoneum have formed, and the intestines are everywhere bound together by both fine and coarse filamentous bands. In many instances the parietal and visceral layers of the peritoneum are united, and small sacculations filled with serous fluid may be formed. During the early stage of chronic diffuse peritonitis ascites may be present, but later there is little if any abdominal fluid.

Physical Signs.—Palpation.—Rigidity of the abdominal muscles is an early symptom, but becomes less decided with the progress of the disease. A tumor-like mass is often palpable in the region of the umbilicus, and is due to a rolled-up condition of the omentum. In other cases a similar mass is felt between the umbilicus and the transverse colon. There are no definite physical signs known to chronic peritonitis, and the exact significance of dullness and tympany is often misleading, on account of the decided misplacement of the viscera by peritoneal adhesions. From the last-named cause marked irregularity in the contour of the abdomen is likewise misleading, and demands most judicious consideration.

Clinical Course and Duration.—The disease usually lasts over a period of one or more years, although at no time does the patient enjoy perfect health.

CARCINOMA OF THE PERITONEUM.

Remarks.—Primary carcinoma of the peritoneum is not common, but secondary involvement of the peritoneum by extension from contiguous structures is often seen—*e. g.*, extension from carcinoma of the liver, stomach, uterus, gall-bladder, ovaries, and rectum.

During the course of carcinoma of the peritoneum the patient, in addition to carcinoma of the organ primarily affected, develops ascites. We have found it possible, in a few instances, before ascites develops, to palpate small nodules through the abdominal wall, and after ascitic fluid has been removed, these carcinomatous nodule masses are readily palpable. The general features of carcinomatous peritonitis are very similar to those described under Chronic Diffuse Peritonitis (p. 558). Metastatic involvement of the other glands, inguinal and axillary, generally occurs.

Laboratory Diagnosis.—Fluid obtained from the peritoneal sac is likely to be blood-stained and to contain the usual amount of albumin present in sanguineous serous fluids.

Microscopically, the fluid shows red blood-cells, white blood-cells, and occasionally small sheets of desquamated, peritoneal endothelium—the so-called "carcinoma cells." Since microscopic sheets of cells are to be found in the peritoneal fluid in tuberculosis of the peritoneum, it is not wise to formulate a diagnosis of carcinoma of the peritoneum upon a microscopic study of the sediment alone.

Summary and Differential Diagnosis.—Carcinoma of the peritoneum is readily diagnosed when it is known that carcinoma of some one of the adjacent structures existed. It is extremely difficult on many occasions

to distinguish between carcinoma and tuberculosis of the peritoneum. Age serves as the most distinctive feature, tuberculosis being more likely to attack the young and carcinoma the aged. A nodular condition of the abdomen may be due to the presence of small cysts, and in two cases of chronic peritonitis believed to be due to tuberculosis it was found that the condition depended upon a number of hydatids that occupied the omentum. (See table, p. 562.)

Clinical Course.—Carcinoma of the peritoneum goes from bad to worse, terminating fatally in from a few weeks to two years.

TUBERCULOSIS OF THE PERITONEUM.

Pathologic Definition.—A subacute or chronic affection excited by the bacillus tuberculosis, and characterized by miliary tubercles of the peritoneum.

Varieties.—(*a*) Primary miliary involvement of the peritoneum with ascites; (*b*) tuberculosis of the peritoneum with the formation of a fibrous exudate and numerous adhesions; (*c*) ulceration of the peritoneum, tuberculous in character; (*d*) a localized tuberculosis of the peritoneum due to direct extension from the mesenteric glands; and (*e*) localized tuberculosis of the peritoneum from direct extension from a tuberculous focus in the liver, kidney, ovary, or a retroperitoneal gland.

Predisposing and Exciting Factors.—Among the predisposing factors should be mentioned pulmonary tuberculosis. Phillips' analysis of 107 cases showed 99 of them to be secondary to tuberculosis of the lung; in 60 of these cases he found the pleura involved, and 80 of them showed tuberculosis of the kidney. Tuberculosis of any one or more of the abdominal viscera materially increases the danger of infection. The ingestion of uncooked beef or pork and the drinking of large quantities of milk that has not been sterilized favor general infection.

Age figures prominently in tuberculosis of the peritoneum, as is shown by Holt's collection of 156 cases, 71 of which were under three years, 26 between the third and eighth years, and 23 between the eighth and tenth years. In 119 autopsies upon the bodies of children under three years of age this author found that 8.5 per cent. of them showed tuberculosis of the peritoneum. The frequency with which tuberculosis attacks children after the third year is further substantiated by the statistics of Ashby, who found that in 105 autopsies on children dead of tuberculosis 36 per cent. showed the peritoneum to be involved.

Biedert analyzed the reports of 883 autopsies made upon children, and found that 18.3 per cent. of them showed tuberculosis of the peritoneum. The greatest number of cases of the condition are to be found between the tenth and fortieth years, whereas after the forty-fifth year it is decidedly uncommon.

Sex appears to exert little, if any, influence on children, whereas adult females are more prone to be attacked than are adult males. The ratio based upon sex is as 3 is to 2, in favor of the female. The condition often extends from the Fallopian tubes and ovaries.

Race and Nationality.—The American Indian is more susceptible to tuberculosis of the peritoneum than members of any other race, yet the negro and the Mongolian (Chinese and Japanese) are far more susceptible than the Caucasian.

Close contact with persons known to be suffering from pulmonary tuberculosis occupies the front rank in the etiology of tuberculosis. We have in mind the case of a cook suffering from pulmonary tuberculosis who was employed in a hospital, and in a period of two years five physicians in this hospital developed pulmonary tuberculosis. This cook's sputum was found to contain great numbers of tubercle bacilli, yet he had comparatively few general symptoms or signs of pulmonary tuberculosis. Not infrequently it is found that the mother of a large family is suffering from a chronic type of pulmonary tuberculosis; her sons and daughters in turn contract the disease, while she survives her family for many years. As an illustration may be cited the case of a family who came to Philadelphia about ten years ago; the mother was suffering from a cough with profuse expectoration, and her sputum was found to contain many tubercle bacilli; at that time three daughters and two sons were healthy. Within a period of eight years all five children had succumbed to tuberculosis, whereas the mother was but slightly, if at all, worse than when she first came under our care. There is no more rapid method of spreading tuberculosis than for persons infected with the disease to be occupied in either the kitchen or the dining-room.

Principal Complaint.—The chief complaint of patients suffering from general miliary tuberculosis of the peritoneum will be found to vary with the virulence of the type of infection, and will depend upon whether or not the patient has suffered from tuberculosis of some other portion of the body for an indefinite time. If the onset is sudden, the patient complains of such severe symptoms and constitutional disturbances as *nausea, vomiting, pain* and *tenderness* over the abdomen, and either *diarrhea* or *constipation.* As tuberculosis of the peritoneum progresses the pulse quickens and the general symptoms of secondary *anemia* appear—*i. e.,* palpitation, shortness of breath, and vertigo. The condition progresses rapidly until the patient assumes the so-called *typhoid state,* when the tongue is parched, the lips are fissured, there is intense thirst, and there may or may not be ascites.

Pus in the peritoneal sac is uncommon, yet it may be found, and at this time or even when there is tuberculosis of the lung with cavity formation night-sweats are prone to occur.

In those cases that do not display ascites, tuberculous nodules may be felt through the emaciated abdominal wall, and the other symptoms and signs of general peritonitis (see p. 553) are present.

A child may complain of nothing that would lead one to suspect that there is any involvement of the peritoneum, and ascites is unusual.

Cutaneous Manifestations.—Pigmentation of the skin of the abdomen is not unusual, and may appear in the form of blotches localized near the median line, or in the form of a general pigmentation of the skin of the abdomen and back.

Thermic Features.—The patient complains of feeling feverish, and the temperature will be found to vary between 101° and 104°, although we have seen cases in which the temperature did not exceed 100° F.

Laboratory Diagnosis.—The blood changes of secondary anemia (p. 356) are found after the disease has progressed for but a short time; thus the hemoglobin falls to below 70 per cent., with a corresponding decrease in the number of red cells in a cubic millimeter. Leukocytosis is commonly found in those suffering from tuberculous peritonitis, although leukopenia is the rule before suppuration is present in some other portion of the body.

The urine gives a reaction for indican, and may contain albumin (febrile albuminuria, p. 643). In three cases studied at the Philadelphia Hospital

in which tuberculosis of the peritoneum followed an initial tuberculosis of the kidney tubercle bacilli were found in the urine throughout the entire course of the illness.

Summary of Diagnosis.—The recognition of tuberculosis of the lung or of some one of the abdominal viscera, together with the characteristic symptoms described above, points somewhat conclusively toward tuberculosis of the peritoneum. In many instances it is practically impossible to determine the exact nature of the peritonitis without opening the abdominal cavity. In a number of cases following operation upon the uterus and ovaries we have seen tuberculosis of the peritoneum develop in from three to five years, but in none of the cases observed was a careful pathologic study of the tissues removed from the uterus or ovaries carried out. In two cases diagnosticated as uterine fibroid, tuberculosis of the peritoneum developed five years later. In children it is often very difficult to recognize tuberculosis of the peritoneum, and when the condition is only a portion of a general involvement, the peritoneal surface shows but little evidence of tuberculous infection at autopsy.

Differential Diagnosis.—See table below.

TABLE SHOWING THE DIFFERENCES BETWEEN CHRONIC GENERAL PERITONITIS, GENERAL TUBERCULOSIS OF THE PERITONEUM, AND CARCINOMATOUS PERITONITIS.

Chronic Generalized Peritonitis.	Generalized Tuberculosis of the Peritoneum.	Carcinomatous Peritonitis.
1. History of rheumatism or of tonsillitis.	1. Family history of tuberculosis or of some chronic infection of the lungs common.	1. Family history points toward carcinoma, and there is usually evidence favoring carcinoma of the stomach liver, or rectum.
2. Social history negative.	2. In many instances the patient has been more or less intimately associated with a person suffering from pulmonary tuberculosis.	2. Social history negative.
3. Occurs most often in the obese and in those displaying a gouty tendency.	3. Develops in slender and ill-nourished subjects.	3. Seldom develops until the patient shows decided emaciation.
4. May develop at any age, but is oftenest seen between the thirtieth and fiftieth years.	4. Common in children and during early adult life. The greatest number of cases appear between the sixteenth and thirty-fifth years.	4. Rare before the fortieth year, although we have seen one case in a female aged thirty-three years.
5. Sex appears to exercise but little influence.	5. More common in adult females. In children both sexes are equally affected.	5. Slightly more common in the female, due to extension from carcinoma of the uterus.
6. Ascites absent.	6. Ascites the rule.	6. Develops after a fairly large surface of the peritoneum has become involved.

Chronic Generalized Peritonitis.—(*Continued.*)	Generalized Tuberculosis of the Peritoneum.—(*Continued.*)	Carcinomatous Peritonitis.—(*Continued.*)
7. Abdominal tenderness appears to be uninfluenced by climatic changes. Symptoms are materially lessened by medication.	7. Tenderness constant, and not influenced by treatment.	7. Unaffected by treatment.
8. Disease advances slowly, and may remain stationary for years.	8. Progresses rapidly from bad to worse, a fatal termination ensuing within from a few weeks to one year.	8. Terminates fatally in from three to eighteen months.

Clinical Course.—Tuberculosis of the peritoneum runs a somewhat rapid course, progressing steadily from bad to worse, and terminating fatally, as a rule, in from three months to one year.

SARCOMA OF THE RETROPERITONEAL GLANDS (Löbstein's Cancer).

Pathologic Definition.—A sarcomatous growth in the retroperitoneal glands near the attachment of the mesentery. It develops somewhat rapidly from a growth the size of a goose egg to that of a human head. There is comparatively little pain, but slight constitutional symptoms, and no fever is present.

Predisposing and Exciting Factors.—Sex.—So far as we know J. Dutton Steele's analysis of 65 reported cases furnishes the only tangible data regarding the etiology of retroperitoneal sarcoma, and shows that males are more susceptible than females, in the ratio of 12 to 8. A pathologic analysis of the 65 cases showed that 39 per cent. were of the spindle-celled variety; 34 per cent. of the round-cell variety; and 14 per cent. were lymphosarcomata. A history of traumatism to the abdomen and spine is occasionally obtained.

Age.—In Steele's analysis retroperitoneal sarcoma was shown to develop more commonly during the first decade, and between the fourth and fifth decades, of life.

Principal Complaint.—The onset is insidious, and the patient first becomes conscious of his condition when he detects a *hard, ball-like mass* in the abdomen. The tumor is not tender, and he often states that he is able to move the mass to various portions of the abdomen. Again, he may state that the tumor falls from side to side of the abdomen with change of posture.

After the tumor has attained sufficient size to interfere with the circulation of the abdominal viscera, the following symptoms may be described: nausea, anorexia, constipation, and intermittent cramp-like pains. According to the size of the tumor, the patient will suffer from a decided drawing or pulling sensation in the back and loins, and a sensation as of a weight in the abdomen. As the tumor increases in size it is likely to interfere materially with the venous return blood-currents, and edema results.

Pain.—As the result of pressure upon the nerve-filaments and nerve-trunks, the patient suffers from intense neuralgic pains in the lower extremities, abdominal wall, lumbar region, and genitalia.

After retroperitoneal sarcoma has continued for several months, the patient becomes anemic, and then complains of palpitation, marked prostration upon exertion, and a general feeling of malaise.

Physical Signs.—**Inspection.**—In patients in whom the abdominal wall is thin a peculiar irregularity, due to the presence of the tumor, is at once apparent. In selected cases the tumor mass may be seen to rise and fall slightly with the respiratory movements. In a case now under our observation the tumor is not affected by respiration, and in a series of cases studied by us the tumor was firmly adherent to the peritoneal tissues.

Palpation.—Early, the tumor is not tender to the touch, and it may be apparently moved by forcibly pressing over the mass. The growth is always hard, more or less irregular in outline, and situated at one or the other side of the median line, a portion of the mass commonly resting over the spine.

After there has been extreme pressure upon the abdominal nerve-trunks, there may be marked tenderness over the lower extremities, and in one case seen by us hypersensitiveness of the left testicle was an early and annoying feature. When there is decided interference with the circulation through the spermatic veins, the testes will be found swollen and soft to the feel, with the general signs of varicocele.

Percussion tends to confirm palpation. The size and shape of the tumor are readily outlined, and surrounding it the normal tympanitic note due to the presence of the intestine may be demonstrated. Ascites frequently develops late during the course of retroperitoneal sarcoma. (See Ascites, below.)

Laboratory Diagnosis.—The blood changes are those of secondary anemia. The urine becomes pale, and later, after the development of ascites, it may contain indican and albumin.

Summary of Diagnosis.—A nodular growth in the abdomen, of less than two years' duration, which is not decidedly painful, and may be palpated without any inconvenience to the patient, is highly suggestive of retroperitoneal sarcoma.

Duration.—The disease terminates fatally in from six to eighteen months after the patient discovers the presence of the tumor.

ASCITES.

Pathologic Definition.—An accumulation of fluid (serous, sanguineous, purulent, or chylous) in the peritoneal sac, with consequent distention of the abdomen.

General Remarks.—This symptom is considered separately, since it displays so wide a variety of physical signs and aggravates an equally large number of other annoying symptoms. The conditions capable of causing an accumulation of liquid in the peritoneal cavity are: (1) Cirrhosis of the liver. (2) Pulsating liver. (3) Echinococcus cyst of the liver. (4) Perihepatitis. (5) Peritoneal adhesions binding down the portal vein as it enters the liver, and general peritoneal adhesions, both of which conditions interfere with the return blood-current from the mesentery. (6) Tuberculosis of the peritoneum. (7) Carcinoma of the peritoneum. (8) Retroperitoneal sarcoma or Löbstein's cancer. (9) Sclerosis of the mesentery. (10) Tumors of the uterus—fibroma, myoma, and carcinoma. (11) Tumors of the ovaries—sarcoma, tuberculosis, and abscess. (12) Tumors of the kidneys—sarcoma, hypernephroma, and hydronephrosis. (13) Enlargement of the spleen (malarial, leukemic). (14) Perisplenitis. (15) Essential anemias, leukemia, and less often pernicious anemia and chlorosis. (16) Secondary anemia, as is seen in ankylostomiasis, pernicious malaria, and chronic mineral poisoning. (17) Chronic parenchymatous nephritis and late in

chronic interstitial nephritis. (18) Valvular heart lesions with tricuspid insufficiency. (19) Cirrhosis of the lung. (20) A large pleural effusion. (21) Tumors of the abdomen pressing on the ascending vena cava or portal vein.

The following table may serve in classifying the causes of ascites under their respective subheadings:

a. Diseases of the Peritoneum:

Tuberculous peritonitis.
Carcinomatous peritonitis.
Non-suppurative acute peritonitis.
Peritoneal adhesions.
"Simple" chronic peritonitis.
Hydatid cysts in the peritoneal cavity.

b. Obstruction to the Main Portal Vein:

Non-suppurative thrombosis.
Peritoneal adhesions.
Aneurysm.

c. Tumors and Enlargements of Adjacent Organs:

Liver.
Pancreas.
Kidney.
Stomach.
Duodenum.
Colon.
Suprarenal capsule.
Retroperitoneal sarcoma.

d. Hepatic Causes:

Atrophic cirrhosis.
Hypertrophic cirrhosis.
Perihepatitis.
Syphilis.
Hydatid disease.
Carcinoma.
Sarcoma.
Cyanotic liver with enlargement.
Pulsating liver (chronic).

Any condition accompanied by extensive enlargement of the liver may, from obstruction by tortion or pressure of the portal vein, be accompanied by ascites. (See causes of hepatic enlargement, p. 586.)

e. Obstruction of the Inferior Vena Cava:

Thrombosis.
Obstruction of thoracic duct.
Rupture thoracic duct.
Rupture of the receptaculum chyli (chylous ascites).
Chronic adhesive pleurisy.
Congenital cysts.
Filariasis.
Stenosis by chronic mediastinal adhesions.
Occlusion by mediastinal growth.

f. Chronic Valvular Heart Conditions Usually Accompanied by:

Tricuspid regurgitation.
Mitral stenosis.
Mitral regurgitation.
Aortic stenosis.
Aortic regurgitation.
Myocarditis { Fatty degeneration. / Fatty infiltration. / Fatty superposition.
Fibroid heart.
Primary alcoholic heart.
Adherent pericardium.

g. Nephritis:

In Bright's disease ascites may be caused in different ways:
Part of a general dropsy.
Secondary to hypertrophy and dilatation of the heart, followed by failure of compensation and tricuspid regurgitation.

h. Essential Anemias:

Splenomedullary leukemia.
Lymphatic leukemia.
Hodgkin's disease (rare).
Aplastic anemia.
Splenic anemia.
Pernicious anemia.

Varieties.—In the vast majority of all cases fluid obtained from the peritoneal sac will be found to be **serous** in character; and when studied microscopically, it will be seen to contain white blood-cells, endothelial cells, and an occasional red cell. This type of ascites is usually considered under the head of **serous** or **true ascites**, and results from mechanical causes or a mild inflammation of the peritoneum.

In still other cases we encounter a more dense exudate—*e. g.*, in tuber-

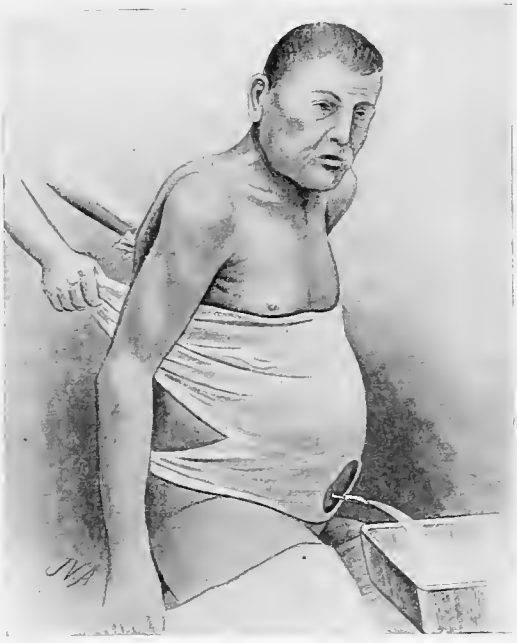


Fig. 227.—Position of the Patient and Method of Employing a Binder in Aspiration of the Abdomen for Ascitic Fluid.

culosis of the peritoneum and carcinoma of the peritoneum, while a *sanguineous* exudate is by no means uncommon. Ruptured ectopic gestation also causes the peritoneal fluid to be bloody. In certain instances the fluid is merely a transudate, *e. g.*, following valvular heart disease, renal insufficiency, and leukemia.

Purulent ascites may follow a chronic suppurative process involving a portion of the peritoneum, and occasionally there is a purulent infection of the entire peritoneal surface. Pus from caries of bony structures rarely enters the peritoneum.

Chylous ascites is the name applied to two rare conditions in both of which milky fluid collects in the peritoneal cavity.

Principal Complaint.—The patient states that his abdomen has been enlarged, and that his clothing has been uncomfortably tight for a period of weeks or months. He also complains of a sense of weight in the lower part of the abdomen, and usually suffers from such gastro-intestinal disturbances as nausea, anorexia, vomiting, and hemorrhoids. Since the majority of symptoms due to ascites are more or less common to many of the pathologic conditions from which it arises, it is considered unnecessary at this point to discuss at great length the symptomatology of this condition.

Physical Signs.—Inspection.—When the patient is resting in a supine position, the abdomen bulges laterally between the ribs and the crests of the ilia, and there is a variable degree of flattening at the umbilicus. When there is a large amount of fluid and the distention is extreme, there is but little change in shape as the result of position, except when in the standing posture, when the anterior portion of the abdomen below the umbilicus is somewhat pendulous. The skin overlying the abdomen becomes shiny, and the veins are prominent. There is often seen at the umbilicus a network of dilated veins—the so-called "caput medusæ." There is swelling of the lower extremities and the genitalia, and edema of the prepuce and scrotum may be most annoying.

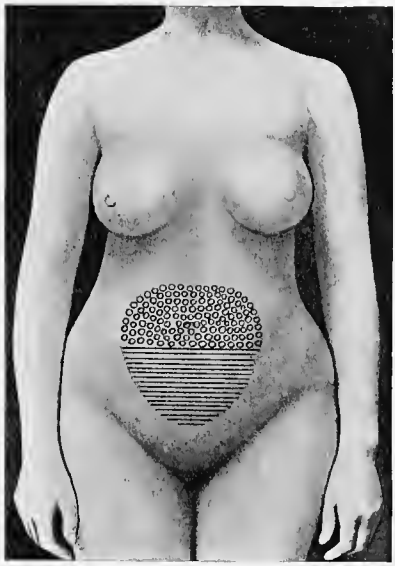


Fig. 228.—Lines Indicate Area of Flatness Due to Distention of the Bladder or the Uterus.

Dotted area indicates that portion of the solid mass where intestine is interposed between it and the abdominal wall, and over which area modified tympanitic note is obtained.

Palpation.—If the amount of peritoneal fluid is small, palpation is negative; but when there is a liberal quantity of liquid in the sac, it is possible to obtain a wave of fluctuation, which is elicited by placing the palm of the left hand against one side of the abdomen, and then tapping the opposite side of the abdomen with the right hand while an assistant holds his hand upon the abdomen at the median line, in order to break any jar that may be conveyed through the abdominal wall.

The abdominal wall may be hard and lusterless, owing to the general effusion of the serum into the alveolar tissue, and a characteristic sensation is detected by palpating over the "caput medusæ." The lower portion of the abdominal wall, particularly in the region of the hips and loins, pits upon pressure and is rarely sensitive.

Percussion.—When the patient is resting upon his back, there is a varying area of flatness in both flanks, and above and anterior to this area a tympanitic note is obtained (Fig. 229). The area of liver dullness is, as a rule, approximately that of the normal, but a large collection of ascitic fluid may force a portion of the bowel between the surface of the liver and the anterior abdominal wall, making it impossible for the examiner to outline the lower border of hepatic dullness anteriorly. Posteriorly, the level of liver dullness will be found to be from one-half to one and one-half inches higher than normal. The area of splenic dullness is changed by the presence of a large accumulation of ascitic fluid. The area of cardiac dullness is higher in cases in which a large amount of peritoneal fluid is present, and after ascites has existed for some time, the area of cardiac dullness is often found to be increased as the result of an effusion into the pericardium. (See Hydropericardium, p. 243; also Fig. 112.)

Upon change of posture—*e. g.*, on sitting or standing—the area of flatness due to the presence of fluid will occupy the lower portion of the abdo-

men, whereas the area of tympany will be transferred to the upper portion. Again, on turning the patient from side to side the area of flatness disappears from the superior side, to be replaced by a corresponding area of tympany.

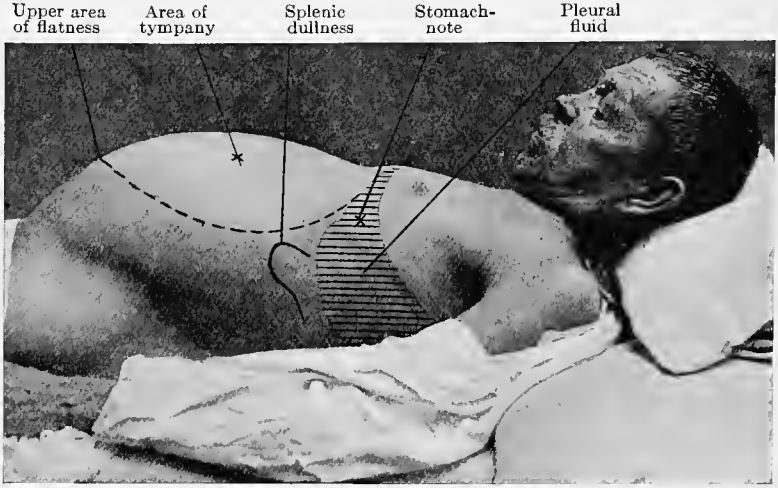


Fig. 229.—Ascites and Pleural Effusion, Areas of Tympany, Etc.

Lastly, the level of flatness will be found to change perceptibly while the patient is sitting merely by tilting him backward or forward.

Summary and Differential Diagnosis.—Distention of the abdomen with the presence of an area of flatness that changes its position with

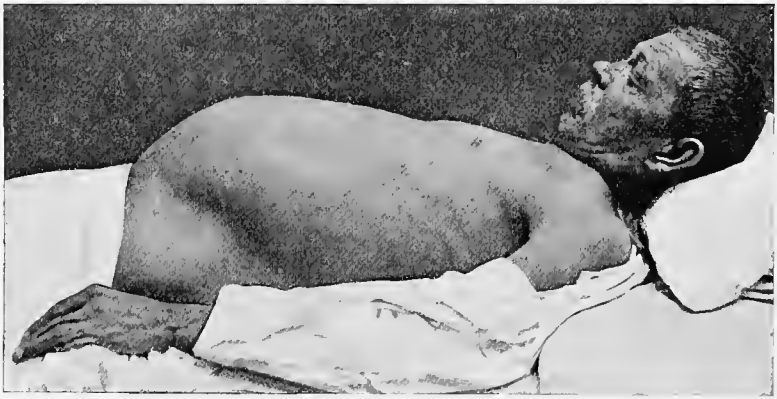


Fig. 230.—Ascites.

the change of posture of the patient is positive evidence of the presence of free fluid in the peritoneal cavity. The detection of a wave of fluctuation is further confirmatory, but by no means infallible, evidence of the existence of ascites.

The accompanying table is designed to set forth the definitive features of ascites, etc. (See also Fig. 229.)

Ascites.	Polyhydramnios.	Overdistended Bladder.	Ovarian Cyst.	Chronic Tympanites.
1. History of chronic heart, liver, or lung disease, one of the conditions known to cause ascites.	1. History of pregnancy.	1. History of urethral obstruction or of some disease of the spinal cord from which the muscular power of the bladder has become impaired.	1. A history of a long-standing tumor of the abdomen which is continually progressing in size.	1. History of abdominal fullness, which varies in degree, and is somewhat influenced by the taking of certain foods and by constipation.
2. Contour of the abdomen shows flattening at the umbilicus and bulging in the flanks when in the recumbent posture.	2. Less decided flattening.	2. Bulging in the flanks moderate.	2. More marked in one than in the other flank.	2. Position of the patient does not change the contour of the abdomen.
3. In the erect posture there is a pouting of the abdomen below the umbilicus.	3. Distention appears to occupy the center of the abdomen and the area below the umbilicus.	3. Distention occupies the lower portion of the abdomen, seldom extending above the umbilicus.	3. Distention over the pelvis, seldom rising high in abdomen.	3. Distention general, and at one or other side or at the top. The abdomen may be unevenly distended.
4. Fluctuation wave is present.	4. Fetal movements present. There may be a distinct wave of fluctuation.	4. Fluctuation rare, yet possible, but when present is limited to the lower part of abdomen.	4. Fluctuation present, but is usually limited to certain portions of the abdomen, giving evidence of a loculated cyst.	4. Absent.
5. The abdominal wall overlying the fluid has lost its normal feel.	5. Less marked change.	5. Seldom is the degree of distention sufficient to have destroyed the ordinary feel of the abdominal wall.	5. Abdominal wall resembles closely that found in ascites if the cyst is large.	5. Abdominal wall normal.
6. When resting on the back there are flatness in the flanks and tympany in the region of the umbilicus and at the top of the abdomen.	6. Less pronounced tympany in the region of the umbilicus, but markedly tympanitic above the uterus.	6. No tympanitic note between the bladder and the anterior abdominal wall.	6. There may or may not be tympany anterior to the cyst.	6. Tympany over all portions of the abdomen, and particularly below the umbilicus.
7. Normal gurgling heard over the abdomen.	7. Fetal heart-sounds heard after the seventh month.	7. Negative.	7. Negative.	7. Increased gurgling over all portions of the abdomen.
8. Aspiration recovers either serous or chylous fluid, the latter being readily distinguished from any other substance found in the peritoneal sac. Serous ascitic fluid has a specific gravity of between 1.005 and 1.010.	8. May recover amniotic fluid, which is usually rich in crystals of cholesterin.	8. Specific gravity of fluid varies between 1.015 and 1.025.	8. Specific gravity of fluid usually 1.020 to 1.025, pale and cloudy, may contain cholesterin crystals and rarely blood.	8. No fluid recovered by aspiration.
9. Fluid contains serum-albumin, and if chylous, is rich in well-emulsified fats.	9. Fluid contains some albumin and emits a characteristic heavy odor.	9. Fluid contains urea in liberal amounts, and emits an odor of urine.	9. Fluid contains albumin.	9. Negative.
10. Microscopically, fluid contains few leukocytes and epithelial cells from the peritoneal wall of the abdomen. Should the fluid be chylous, fat-globules and epithelial débris are present	10. Microscopically, the fluid may contain crystals of cholesterin and granular débris.	10. Microscopically, the fluid contains epithelium from the bladder or the kidney, and, rarely, renal casts, white blood-cells, and erythrocytes.	10. Fluid is usually rich in crystals of cholesterin and contains much granular matter.	10. Negative.

CHYLOUS ASCITES.

Remarks.—For convenience of study, two types of chylous ascites are described—true and false. The former arises from a rupture of the thoracic duct, or from some pathologic condition that interferes with the passage of chyle through the lymphatics into the receptaculum chyli. In 128 cases of milky ascites analyzed by one of us* many were chylous, others pseudochylous,

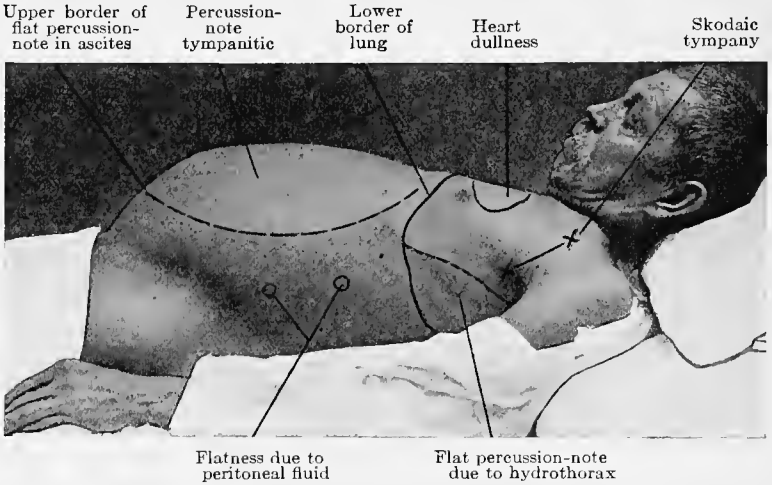


Fig. 231.—Case of Advanced Cardiac Disease Complicated by Neprritis with Effusion into the Peritoneal and Pleural Sacs. (Studied at Philadelphia Hospital.)

and in probably 20 per cent. the records as to what caused the turbidity of the peritoneal fluid were incomplete.

Pseudochylous ascites is a condition in which the peritoneal fluid is milky (chyloid), its turbidity and color being due to the presence of degenerated epithelial cells, microscopic particles of fat, and pigment resulting from such degeneration.

Predisposing and Exciting Factors.—In the series of cases previously cited, chylous ascites was found to complicate carcinoma of the peritoneum in 24; tuberculosis in 17; cardiovascular conditions in 11; rupture of the thoracic duct in 11; disease of the liver in 8; puerperal sepsis in 7; rupture of the receptaculum chyli in 7; congenital cysts in 4; and infection with the Filaria bancrofti in 3.

Age.—A further analysis as to the number of cases according to the various periods in life gave 6 under one year of age; 3 between one and five years; 13 between five and ten years; 9 between ten and twenty years; 12 between twenty and thirty years; 34 between thirty and fifty years; and 24 after the age of fifty.

Sex.—Sixty of the cases analyzed were females, 50 were males, and in the remainder the sex was not given.

Laboratory Diagnosis.—True chylous peritoneal fluid always results from an admixture of chyle, yet pseudochylous fluid is by far the more com-

* Jour. Amer. Med. Assoc., February 18, 1905.

mon finding. It was first pointed out by Pagenstecher that the milky appearance of ascitic fluid depended upon fatty degeneration of the cells derived from the serous membrane, or upon an alteration in an inflammatory exudate—possibly an admixture of mucoid substance.

Chylous ascitic fluid will be found to have a specific gravity of about 1.013, and according to Pagenstecher it contains 3.06 per cent. of albumin, 2.087 per cent. of fat, and 0.32 per cent. of sugar. Chylous fluid, when allowed to stand in the cold, will show a heavy precipitate upon the top and bottom of the fluid. If the fluid is truly chylous, the heavier precipitate will be on the surface of the liquid, whereas if the fluid is pseudochylous, a heavy precipitate will be at the bottom of the liquid, a few oil-globules floating upon the surface.

Microscopically, chylous fluid is found to consist, for the most part, of well-emulsified fats (small globules), a variable amount of pigment, large fat-globules, and granular débris. In both varieties red blood-cells may be present. (See Method of Aspiration, Fig. 227, p. 566.)

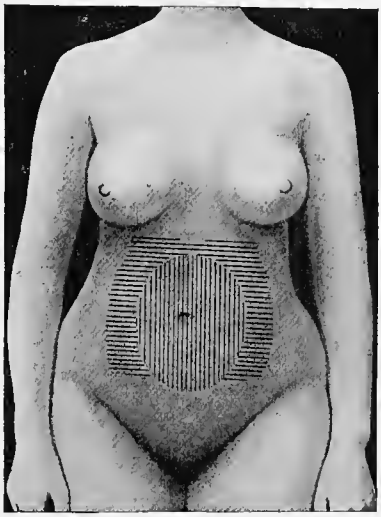


FIG. 232.—ASCITES.

Transverse lines indicate area of flatness when patient is in the recumbent posture, while the vertical lines transcribe the area of tympany.

DISTINCTIVE FEATURES OF CHYLOUS AND PSEUDOCHYLOUS FLUIDS.

CHYLOUS EFFUSION.	PSEUDOCHYLOUS EFFUSION.
1. The fluid consists of a fine emulsion containing but few cellular elements.	1. A less perfect emulsion, containing large numbers of epithelial cells, which are seen to display the various stages of degeneration and to contain few fat-droplets and granular débris.
2. It accumulates rapidly after removal by tapping.	2. Collects more slowly, varying with the exciting pathologic causes.
3. It contains sugar.	3. Sugar is absent, as a rule.
4. The melting-point of the fat present in the fluid will be found to vary with the melting-point of the fat taken as food.	4. Fatty foods have no effect on the melting-point of fats contained in the fluid.
5. The fat-droplets are small and uniform in size.	5. Great variation in size of fat-droplets.
6. The amount of fat present is in direct relation to the amount of fats ingested.	6. The amount of fat present is not affected by diet.

Clinical Course of Ascites.—An accumulation of fluid may remain in the peritoneal sac for long periods of time, and we have seen cases in which the quantity of fluid in the sac varied greatly at different examinations covering a period of several years. In the majority of instances ascites, when pronounced, is a precursor of a fatal termination, which may occur within a few months or, at the longest, within one year. Rest, stimulation, tapping, and surgical interference materially modify the duration and general course

of ascites and prolong life. There is at present under our care a patient whom we have aspirated nineteen times, a large quantity of peritoneal fluid having been removed at each tapping. He was first brought to our attention six years ago, and while his health to-day is much better than it was upon his first visit, there is still some free fluid in the peritoneal cavity. The benefit to be derived from repeated aspirations of the peritoneal sac is dependent entirely upon the factors that have caused the accumulation of the fluid.

We have seen several cases in which surgical intervention has effected a cure, and one instance in which chronic peritonitis following aspiration resulted in the formation of extensive adhesions and the disappearance of fluid from the peritoneal cavity.

If the ascites is cardiac in origin, the peritoneal sac will be found to fill with fluid when the patient overexercises; and, on the other hand, as the result of rest and medication, such fluid will be absorbed. Although ascites, irrespective of its cause, is a somewhat unfavorable symptom, it is by no means a positive sign of an early fatal termination in all cases, and it is, therefore, to be regarded as a symptom amenable to treatment whenever it is possible to combat the exciting factors.

THE LIVER.

METHODS OF EXAMINATION.

TOPOGRAPHIC ANATOMY OF THE NORMAL LIVER.

In considering the liver, it must be borne in mind that the organ is a somewhat wedged-shaped, solid viscus, suspended from the upper portion of the abdomen, and that, on account of its peculiar contour, it is forced to occupy a position chiefly to the right of the spine, immediately beneath the right lung. The thinner portion of the organ crosses the spinal column, and extends to the left as far as the left parasternal line, at the lower border of the fifth rib. Sometimes, indeed, the left lobe of the liver reaches to the spleen.

The superior surface of the right lobe is decidedly convex (dome-like), and is covered by the diaphragm; it fits snugly into a concavity extending well up into the base of the right lung (Fig. 233). The convexity of the right lobe elevates the diaphragm to the lower border of the fourth rib in the right midclavicular line, from which point it descends somewhat abruptly toward the sternum and the right axillary line. (See Figs. 233 and 235.) Consequently, the greater area of parietal dullness (uncovered liver) is found in the right midclavicular line. In the right scapular and midaxillary lines the liver is found as low as the eleventh rib. (See Figs. 235 and 236.)

The inferior surface of the right lobe of the liver is adjacent to the pylorie end of the stomach, duodenum, hepatic flexure of the colon, and top of the right kidney. The inferior surface of the left lobe is in contact with the stomach, and its lower rounded edge sometimes rests upon the transverse colon, whereas the extreme left superior surface is immediately beneath the heart (Fig. 237).

The antero-inferior margin (rounded edge) of the liver extends from the right body wall obliquely between the ninth and tenth costal cartilages, and thence across the median line at the junction of the upper one-third with

the lower two-thirds of a line extending between the tip of the ensiform cartilage and the umbilicus, to the left eighth costal cartilage. The peculiarly curved outline of the rounded edge of the liver is shown in Figs. 236 and 237. The interlobular notch is in the median line.

The gall-bladder projects from beneath the liver at the point at which the right linea semilunaris crosses the ninth right costal cartilage.

Age and Posture.—The liver is relatively larger in children than in young adults, and the rounded edge (antero-inferior margin) extends from one to one and one-half inches below the costal margin in the midclavicular and midaxillary lines (Fig. 235). After the fiftieth year the size of the liver is reduced (due to atrophic and sclerotic changes), the lower border being one inch above the margin of the ribs, in the right midclavicular line. It is important to estimate accurately the topography of the liver in the aged, in order to eliminate the possible existence of hepatic cirrhosis.

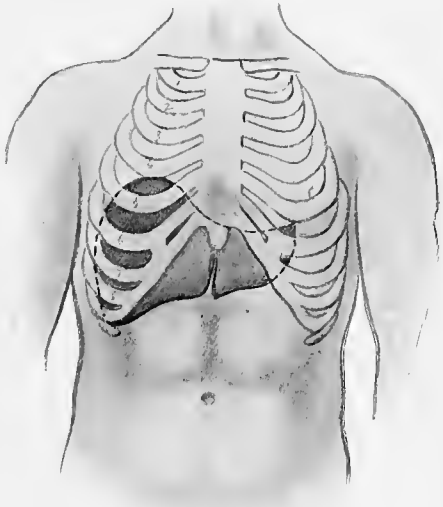


FIG. 233.—NORMAL AREA OF LIVER AND ITS RELATION TO THE ABDOMEN AND THORAX.

Owing to the function of the suspensory ligament, the position of the liver changes slightly with the position of the patient—*e. g.*, when lying upon the left side, the right lobe descends for a distance of an inch or more, whereas the left lobe displays a corresponding rise (Fig. 239; see also Fig. 238) and is seen well up underneath the heart.

Deep inspiration forces the liver downward, so that, in subjects with thin or relaxed abdominal walls, its rounded edge can be distinctly palpated immediately below the costal margin.

DISEASES OF THE LIVER.

ANOMALIES OF POSITION.

Anomalies of position are occasionally encountered, the organ being displaced downward or laterally. The common cause of displacement is an unusual lengthening of the suspensory ligament, which may permit the viscus to occupy any portion of the abdomen. A relaxed abdominal wall and ptosis of other abdominal viscera, *e. g.*, colon and stomach, are usually found to accompany hepatoptosis. In 1908 Savelieff collected from the literature 118 reports of cases, and Andresen* reported five (5) cases in 1911.

Clinical Varieties.—(*a*) Hepatoptosis, without any annoying symptoms; (*b*) wherein the symptoms of chronic gastritis are prominent; (*c*) hepatalgic types, where hepatic and scapular pains are annoying; (*d*) cases where hepatic colic is the leading complaint, and (*e*) cases showing dyspnea and at times asthmatic seizures.

* New York Med. Jour., Dec. 23, 1911.

Diagnosis.—Hepatic displacement is more easily recognized by employing oscultatory percussion (patient standing, see Fig. 240, also topography, p. 572.

Recognition.—Anomalies in position are recognized by the fact that the liver dullness is absent in the area in which it is normally present, and that dullness and a palpable mass are present elsewhere in the abdomen. Upon firm palpation and change of posture the liver may be returned to its normal position, except in cases of transposition of the viscera.

Abnormalities in contour are extremely rare, and can be definitely demonstrated only upon those persons in whom the abdominal wall is thin and flaccid.

Abnormalities of the liver do not concern us clinically, except in so far

Fig. 234.—Arched Portion of Liver Shaded to Show Relation to the Nipples in the Female and Extent to Right of Median Line.

as they are associated with annoying symptoms, *e. g.*, a weight in the abdomen, discomfort, or pain that may radiate to the right shoulder and to the back. Intense acute pain may follow a sudden jar with marked traction or with torsion of the suspensory ligament.

Clinical Considerations.—Clinically speaking, we have found it practical, for teaching purposes, to divide diseases of the liver into four classes: (*a*) Those conditions in which there is a permanent, abnormal enlargement of the organ; (*b*) acute temporary enlargement; moderate but temporary enlargement is a feature of most of the acute fevers, *e. g.*, typhoid fever, scarlet fever, yellow fever, etc.; in this type of enlargement the liver usually assumes its normal size during convalescence; (*c*) pathologic states

resulting in an undue contraction of the viscus; and (*d*) conditions in which jaundice and the many symptoms associated with it are present.

(*a*) Permanent enlargement of the liver is found in the following conditions: (1) Hypertrophic cirrhosis. (2) Hepatic carcinoma. (3) Sarcoma. (4) Cyanotic congestion (due to organic heart lesions). (5) Abscess. (6) Echinococcus cysts. (7) Distended gall-bladder. (8) Fatty degeneration. (9) Fatty infiltration. (10) Amyloid disease. (11) Leukemia. (12) Early stage of atrophic cirrhosis. (13) Perihepatitis (early stage). (14) Tuberculosis. (15) Obstruction to the cystic duct.

Fig. 235.—Relation of Liver to Axillary and Scapular Regions.

(*c*) Among the pathologic conditions known to produce an abnormally small liver are: (1) Syphilis (inherited). (2) Syphilis (chronic). (3) Atrophic cirrhosis (late stage). (4) Acute yellow atrophy. (5) Phosphorus-poisoning. (6) Capsular (Glissonian) cirrhosis.

HYPERTROPHIC CIRRHOSIS.

Pathologic Definition.—The organ is increased in size, and the lower margin is several inches below the border of the ribs. The inferior margin of the liver is somewhat rounded and thicker than normal. The organ cuts with unusual resistance, and the cut surface presents a mottled, yellowish, slightly green appearance.

Fig. 236.—Effect of Respiration upon the Position of the Liver.

Microscopically, there are round-cell infiltration of the peripheral zone of the hepatic acini, with the formation of embryonal tissue, and hyperplasia of the interlobular connective tissue. Late during the course of the disease the interlobular connective tissue undergoes hyperplasia and produces obstruction of the bile-ducts, with retention of bile.

French writers refer to biliary cirrhosis in which there is also obstruction of the bile-

ducts with retention of bile and swelling of the organ. The microscopic changes in this form of cirrhosis simulate in many respects those just described for the hypertrophic variety of the disease, except that the hepatic cells are more deeply bile-stained, and that isolated areas of necrosis are present in the peripheral zone of the acini. The formation of new ducts and liver-cells may also be observed.

Varieties.—(*a*) Hypertrophic cirrhosis of Hanot; (*b*) biliary cirrhosis, which is probably precipitated by obstruction of the bile-ducts. The action of the bile, when under high tension, upon the liver tends to produce sclerotic changes later, but clinically the majority of cases of this type are quite identical with those of hypertrophic cirrhosis, although pathologically they are not the same.

Predisposing Factors.—**Sex.**—Males are most frequently affected, the ratio being six to one. **Age** exerts some influence, the majority of cases occurring during early adult and middle life. Hypertrophic cirrhosis in children is extremely uncommon.

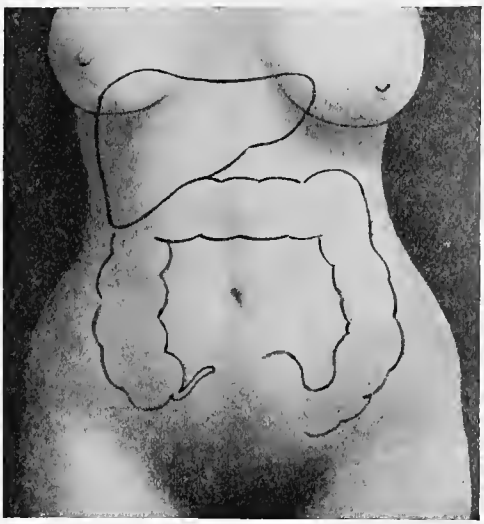


Fig. 237.—Relation of Liver and Colon to the General Anterior Area of the Abdomen.

Alcohol is believed to play an unimportant rôle, although in two cases occurring in brothers both were alcoholics, and three other patients recently studied at the Philadelphia Hospital used alcohol to excess.

Principal Complaint.—The patient grows progressively weaker, and, in addition to being easily exhausted, he is unable to concentrate his mind. With the progress of the condition there is moderate delirium at night, which may become so severe as to demand restraint. Maniacal delirium, carphologia, and jactitation are seen late.

The *appetite* is spasmodic, and later there is but little desire for food. *Nausea* is not uncommon, and the vomiting of bile-stained material is an occasional complaint. *Itching* may not be an annoying symptom, although the patient is almost continuously unconsciously irritating the skin.

Hand in hand with increasing weakness there is a gradual enlargement of the abdomen. *Paroxysmal cramp-like pains* are often experienced, although they are, as a rule, less severe than are the pains of hepatic colic. Hemorrhoids and ascites may or may not develop.

Thermic Features.—Early the temperature may be normal, and there are occasional instances in which fever is absent throughout the entire course of the disease. As a rule, however, the temperature ranges between 99° and 100° F. at the time when there is but slight jaundice, but later, and when there are other severe symptoms, *e. g.*, profound jaundice, delirium, ecchymoses, and obstipation, the temperature fluctuates between 101° and 104° F.

Physical Signs.—Inspection.—The skin is slightly jaundiced even

during the early stage, the jaundice becoming more and more intense as the disease advances. The tongue is glazed at first, but later becomes heavily coated, fissured, and there may be sordes upon the teeth. Small hemorrhages into both the skin and mucous membranes usually develop, and the veins over the chest and abdomen are distended. The abdomen is promi-

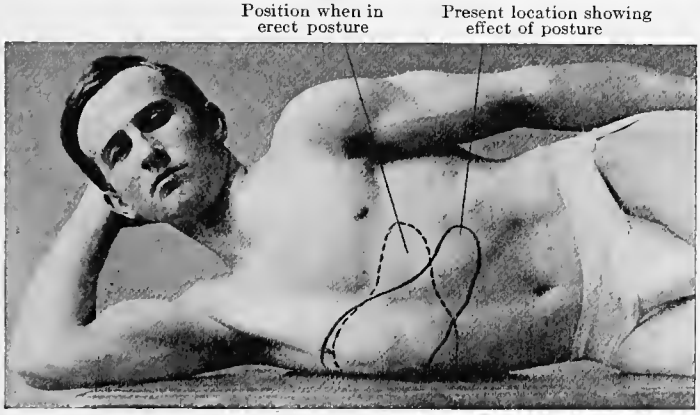


Fig. 238.—Effect of Posture upon the Position of the Normal Liver.

nent early, and increases in size until death. The conjunctivæ are jaundiced, and profuse sweating in the axillary and inguinal regions is the rule. (See Jaundice, p. 604.)

Palpation.—The skin is somewhat roughened to the touch, and may at

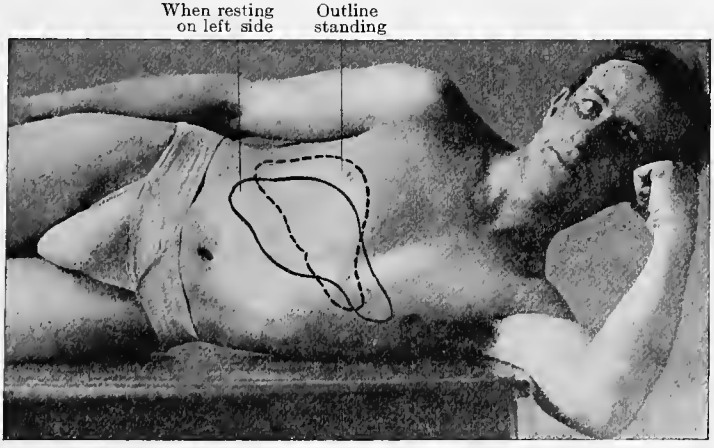


Fig. 239.—Effect of Posture upon the Position of the Normal Liver.

times display a slightly elevated eruption. The pulse is at first nearly normal, but with increasing prostration it becomes weak, rapid (100 to 120), compressible, and dicrotic.

There is increased resistance over the superior right abdominal quadrant,

and the entire upper portion of the abdominal wall displays undue tension. The rounded edge of the liver is readily palpable, smooth, and may extend below the level of the umbilicus and well to the left side of the abdomen, as was observed in the patient shown in Figs. 241 and 242. In cases in which hepatic enlargement is extreme, ascites occasionally develops. (See Physical Signs, p. 576.)

Percussion confirms palpation as regards the area of hepatic enlargement. In those cases in which ascites develops there is flatness in the flanks, which is replaced by tympany when the position of the patient is changed. Splenic enlargement with increased area of splenic dullness may be present.

Auscultation.—The heart-sounds become weak, and hemic murmurs are audible in the advanced stage.

FIG. 240.—OUTLINING THE LIVER BY AUSCULTORY PERCUSSION.
Begin area from the liver and strike the abdominal wall every one-half inch, approaching the liver until the note produced shows a decided change. This marks the outer margin of the liver.

Laboratory Diagnosis.—In those cases in which vomiting is present the vomitus is usually bile-stained, and contains particles of undigested food and, rarely, blood and shreds of mucus.

The stools are, as a rule, dark—the so-called "bilious stools." There may be sufficient hemorrhage from the intestinal mucous membrane to produce free blood in the stool. The bleeding may, however, be so slight as to be detected only on applying the test for occult blood. Bleeding from hemorrhoids must always be excluded when the occult blood test is positive.

The *urine* is at first slightly colored by bile-pigment, but later it becomes deeply stained; its specific gravity varies between 1.025 and 1.030; upon

shaking it displays a heavy froth, and upon standing a decided sediment. *Microscopically,* the leukocytes and epithelial cells are bile-stained, and casts, when present, are likewise stained a pale yellow. Albuminuria is common, but is of moderate consequence, unless tube-casts are present. Crystals of leucin and tyrosin (Fig. 263) are occasionally found in the urine in cases of hypertrophic cirrhosis. Leukocytosis may be present.

Summary of Diagnosis.—A diagnosis is based upon the presence of persistent jaundice, hepatic enlargement, progressive weakness, hemorrhages into the skin and mucous surfaces, decided nervous symptoms, and fever. In those cases in which fever and nervous symptoms are absent, the diagnosis is made with great difficulty.

Differential Diagnosis.—Hypertrophic or biliary cirrhosis is to be distinguished from *hepatic abscess,* from which it is differentiated by the fact that in abscess the symptoms develop abruptly, the temperature is septic in type, and leukocytosis is of a high grade—15,000 to 30,000 in a cubic millimeter. Obstruction to the common duct, either from conditions within or without the liver, may cause a distinct enlargement of the organ with jaundice, but in such cases prostration, nervous symptoms, and fever are less conspicuous features than in hypertrophic cirrhosis. The fever of gallstones is usually characteristic. (See p. 611.)

Clinical Course.—The majority of cases terminate fatally in from a few months to one and one-half years.

Fig. 241.—Extreme Enlargement of the Chest and Abdomen Accompanying Enlargement of the Liver and Spleen.

Note increase in anteroposterior diameter. (Photograph obtained through courtesy of Dr. David Riesman.)

CARCINOMA OF THE LIVER.

Pathologic Definition.—A disease characterized by malignant growths situated in various portions of the liver, which differ in no way from carcinomatous changes found elsewhere. These growths are altered greatly in shape as the result of pressure. The entire organ is markedly enlarged, and whitish or yellowish tumor masses may be seen over its surface, and at times these elevate the hepatic capsule. The cut surface of the organ displays similar growths more or less equally disseminated throughout.

Microscopically, giant-cells and areas of pigment—"brown granules"—are to be found within the carcinomatous masses. The so-called colloid degeneration is also present, and immediately surrounding the carcinoma nests both hyaline and myxomatous degeneration is common. Carcinoma may involve but one lobe of the liver, although a more or less general involvement is somewhat characteristic. Eggels has collected 133 cases of primary hepatic carcinoma, and makes special reference to the frequency with which carcinoma is associated with atrophic cirrhosis.

Varieties.—Primary carcinomatous involvement of the liver is conceded to be extremely rare. Hepatic carcinoma secondary to carcinoma of the stomach, duodenum, rectum, and bile-ducts comprises by far the majority of all cases of carcinomatous involvement of this viscus.

Exciting Factors.—In primary hepatic carcinoma this is unknown, whereas in secondary carcinoma it is either due to direct extension or to the disease traveling through the blood-vessels or the lymphatics and lodging in the liver, which is best exemplified by hepatic carcinoma following epithelioma of the rectum.

Predisposing Factors.—These are somewhat numerous, and among those known to exercise a decided influence should be mentioned:

1. **Age.**—The disease is uncommon before the thirtieth year, but common during the fourth and fifth decades.
2. **Sex.**—Males are more often affected than females.
3. **Heredity** has long been known to play an important part in the etiol-

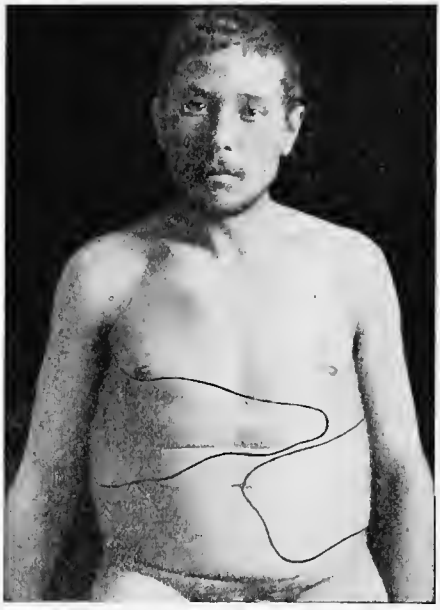


Fig. 242.—Same Case Shown in Fig. 241.
Position of liver and spleen as shown by auscultatory percussion. (Photograph obtained through courtesy of Dr. David Riesman.)

ogy of carcinoma. Lichtein analyzed 1137 cases, and found heredity to be a factor in 17 per cent. of them.

4. **Cholelithiasis.**—Carcinoma of the liver not infrequently follows cholelithiasis, yet it is possible that in these cases the carcinomatous growth originated either in the gall-bladder or in the common duct.

Principal Complaint and Symptoms.—Cases are occasionally encountered in which there are but few, if any, symptoms referable to the liver. We have seen cases at postmortem in which well-advanced carcinoma of the liver existed without having produced any symptoms dis-

tinctly referable to that organ. The location of the carcinomatous nodules influences the chief complaint in this disease, viz., pain.

Pain, when present, is more or less constant, dull, boring, and sometimes lancinating, and, as a rule, localized to the superior right abdominal quadrants. When there is considerable peritoneal involvement, or when the bile-ducts are likewise attacked by the carcinomatous process, the pain will be found to radiate to the right scapular region. Pain becomes more annoying with the advance of the disease, and is not relieved by local applications.

Loss of Strength and Emaciation.—Irrespective of the character of the principal complaint, there is always a decided and progressive loss in strength during the entire course of the disease, and the patient always observes that he is becoming markedly emaciated. The face may not show evidences of emaciation as early as do the muscular regions.

The *appetite* is fair at first, but anorexia, constipation, and intermittent attacks of diarrhea soon occur. An annoying feature in many cases is a peculiarly parched condition of the tongue and buccal mucous membrane. The patient frequently complains of *acid eructations*. Vomiting is by no means a common complaint, yet there are certain cases of hepatic carcinoma that closely resemble carcinoma of the pylorus, and in these the general complaint is similar to that described for gastric carcinoma (see p. 499). The carcinomatous process may extend to the peritoneum, in which case an effusion may accumulate in the peritoneal sac; in this class of cases there are added to the foregoing general complaint those symptoms characteristic of ascites. At least two-thirds of all cases terminate without the development of ascites.

Nervous Phenomena.—There may or may not be nervous symptoms, but in the advanced stage they often appear—*e. g.*, mental hebetude, headache (possibly due to anemia), delirium (rarely), and coma may end the scene.

Thermic Features.—Fever is not a feature early during the course of hepatic carcinoma, but late in the disease the temperature will be found to fluctuate between 99° and 104° F., moderate temperature, however, being the rule. The fever is decidedly irregular in type.

Physical Signs.—Inspection.—Slight jaundice may be observed, and becomes profound whenever the bile-ducts are involved. Harley's studies of 100 cases showed jaundice to be a common occurrence at some time during the disease. In our experience mild jaundice has been present in a larger percentage of cases than is shown by Harley's statistics. It is readily understood that jaundice is not an essential feature of hepatic carcinoma unless the growth obstructs the bile-ducts or there is associated involvement of the gall-bladder or of the common duct. Jaundice, when present, will be accompanied by a series of symptoms described under Jaundice (see p. 604). Cachexia is the rule.

There is distinct emaciation, the upper portion of the abdomen is unduly prominent, and there may be prominence of the superficial abdominal veins. The inferior border of the liver may be seen to rise and fall with respiration through a thin and relaxed abdominal wall. In advanced cases there are slight puffing beneath the eyes and at the ankles. The cardiac impulse is feeble but diffuse, and there is throbbing of the vessels of the neck.

Palpation.—The lower margin of the liver is felt distinctly through the abdominal wall (Fig. 243), and when the patient is directed to inspire deeply, it is often possible to outline the nodules upon the surface of the

liver. The margin of the liver may extend but a short distance below the right costal border, but in advanced cases it has been found at or even some distance below the level of the umbilicus.

Carcinomatous hepatic enlargement appears to affect mostly the right lobe, consequently the degree of enlargement may, comparatively speaking, be moderate. We have examined patients in whom the lower margin of the

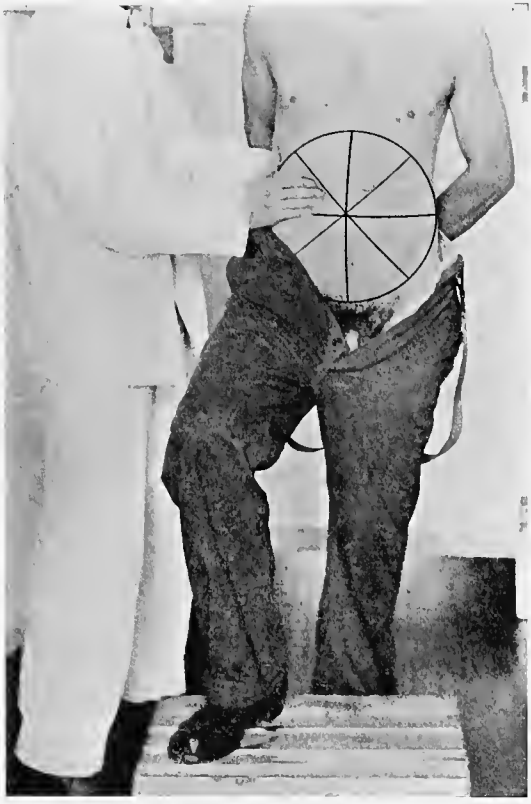


FIG. 243.—HERE THE PATIENT RESTS THE RIGHT FOOT UPON A SMALL BOX, WHICH GIVES IDENTICALLY THE SAME RESULT AS STANDING WITH BUT A SMALL PORTION OF HIS WEIGHT ON THE RIGHT TOE (Boston, New York Med. Jour., Nov. 1, 1913).

liver extended below the umbilicus, and yet no nodules were present upon the surface of the organ.

The spleen is usually palpable in those cases in which ascites develops as the result of extension of the carcinomatous process to the peritoneum, and the physical signs of ascites are also to be expected. (See p. 567.)

The heart impulse is extremely weak, and a distinct pulsation is felt over the right carotid. Edema of the feet and of the hands is a late finding.

Percussion gives positive findings as to the degree of enlargement of the liver and the spleen, and regarding the presence or absence of peritoneal fluid (movable abdominal flatness). It is not uncommon to find the degree of liver dullness extending from the lower border of the fourth rib to the crest

of the ilium in the anterior axillary line, although, as a rule, the superior border of dullness is found at the fifth rib or the fifth interspace.

Laboratory Diagnosis.—The blood changes are those of secondary anemia, the red blood-cells falling to 3,000,000 or below, while the hemoglobin displays a corresponding decrease, except in those cases in which jaundice is present, when the estimation of hemoglobin is difficult. The presence or absence of leukocytosis is of but little importance in connection with hepatic carcinoma, since it is impossible to estimate to what degree the peritoneal surface is involved, and also what other modifying conditions coexist.

The character of the *urine* is influenced largely by the nature of the food taken and by the condition of the digestive tract; evidence of kidney disease is rare. If jaundice is present, the urine is highly colored and its organic sediments are also colored by bile-pigment. It is to be remembered that jaundice, when continued over a long period, produces the inflammatory renal changes indicated by the presence of albumin and renal casts in the urine.

Illustrative Case of Carcinoma of the Liver.—M. J., female, aged fifty-six; height, 5 feet 6 inches; normal weight, 155 pounds; present weight, 120 pounds.

Family History.—Father died of epithelioma of the lip at the age of fifty-two; mother died of pneumonia at the age of sixty-four. Two young brothers and a sister are living and are in good health. One paternal uncle died of carcinoma of the stomach, and a half sister of pulmonary tuberculosis.

Previous History.—Thinks she had the diseases of childhood. Had influenza at the age of forty-five, and another attack at fifty. Has had four children, and all are in perfect health.

Social History.—She was married at the age of twenty-two. She has been compelled to attend to her own household duties since she married, but she has also been permitted to get a fair amount of outdoor exercise. Menopause at fifty-two; uses no intoxicants, but tea and coffee in moderation.

Present Illness.—States that during the past year she has had considerable annoyance from what she termed stomach trouble, which included occasional attacks of nausea, moderate anorexia, some abdominal pain, and obstinate constipation. Three months ago the pain in her abdomen became appreciably increased, and she then consulted her physician with reference to this particular symptom and obtained considerable relief. When first seen by us she was markedly emaciated and weak, unable to take nutriment, and complained of a constant sense of weight in the region of the liver. She also stated that she became readily exhausted upon slight exertion, and that on ascending a single flight of stairs she was compelled to rest for a few minutes. She experienced a variable degree of vertigo after exercise, and was constantly annoyed by eructations of gas.

She slept poorly, especially during the night hours, although there was mental hebetude during the day. Delirium was not present early, but later low muttering delirium was more or less continuous. The pain was constant, and was always situated in the right hypochondrium, but occasionally, when it became severe, it radiated to the back and right shoulder, and less often was reflected over the anterior surface of the abdomen. A peculiar dull headache was annoying early during the illness, but later this symptom disappeared.

The temperature was approximately normal until the last three weeks of the disease, when it rose during the evening to 101° to 103° F., whereas the morning temperature was at or near the normal level.

Physical Examination.—*General.*—The skin was markedly jaundiced, the expression was dull, there was slight puffing beneath the eyes, and evidences of emaciation were general. At first the patient's gait was feeble and tottering, but later she was unable to leave her bed, and she rested in the supine position, complaining when asked to turn from side to side.

Local Examination.—Inspection.—The conjunctivæ were markedly jaundiced, the tongue was heavily coated, and the breath was foul. The abdomen showed distinct bulging in the region of the liver, and there appeared to be some restriction in the expansion at the right base of the chest.

Palpation.—Even moderate pressure over the liver at the costal margin excited

severe pain, and it was possible to palpate the lower edge of the liver at a distance of 2½ to 3 inches below the costal border. Careful though fairly firm palpation also revealed the existence of nodular masses upon the palpable surface of the liver. The pulse was full at first and fairly strong, the beats numbering 50 to 60 a minute, a clinical feature probably explained by the early development of jaundice, but late during the disease the pulse became weak, rapid, and thready, and the beats numbered 120 to 140 a minute.

Percussion.—This confirmed palpation with reference to the size of the liver.

Laboratory Findings.—The quantity of urine voided during the twenty-four hours varied between 20 and 35 ounces a day; its specific gravity was 1.025 to 1.030. Chemically, albumin was present, and microscopically numerous hyaline and granular casts were seen, both of which were more or less deeply stained by bile. An examination of the blood showed the red cells to number 2,380,000, whereas the white cells numbered 7400 in a cubic millimeter. Owing to the existence of extreme jaundice, an accurate estimation of the percentage of hemoglobin was impracticable.

Diagnosis by Induction from Clinical Data.—The duration of the present illness, the fact that it was accompanied by increasing weakness and rapid and progressive emaciation, and the presence of a sense of weight and pain in the region of the liver were considered to be clinical points decidedly favoring the existence of carcinoma. The family history and the age of the patient also supported this view. The later development of jaundice suggested that either the gall-bladder or some of the larger bile-ducts were involved in the process. Pressure over the liver always excited severe pain, and the detection of nodular irregularities on the surface of the liver was considered to be of great importance in establishing the diagnosis.

Differential Diagnosis.—Certain features in the case somewhat resembled hypertrophic cirrhosis, from which disease it was differentiated by—(*a*) The presence of nodular enlargements on the surface of the liver; (*b*) the occurrence of continuous pain in the region of the liver; and (*c*) the fact that the pain was intensified by making firm palpation over the organ.

Course of the Disease.—In approximately one year from the time the patient consulted a physician with reference to pain in the region of the liver the case terminated fatally, catarrhal pneumonia complicating the condition in its last stages. Autopsy revealed the presence of multiple carcinomatous growths disseminated throughout the substance of the liver; a fistulous communication between the gall-bladder and the transverse colon, which was completely surrounded by dense peritoneal adhesions and had doubtless resulted from a softening of the carcinomatous mass, was also found. The common duct was patulous, but there was extensive evidence of inflammation of most of the surface of the gall-bladder.

Summary of Diagnosis.—The age of the patient (after forty years), the history of carcinoma existing elsewhere in the body, the character of the pain, the presence of cachexia, and the fact that the patient has lost from twenty to sixty pounds in weight, point strongly to hepatic carcinoma. The detection of nodular enlargement of the liver makes the diagnosis practically certain.

Differential Diagnosis.—**Carcinoma of the pylorus** may be mistaken for hepatic carcinoma, yet a point ever to be borne in mind is that the latter is frequently secondary to pyloric involvement; consequently by the time the patient consults the physician, and when the symptoms are well marked, there is likely to be a primary carcinomatous process in the stomach, with secondary involvement of the liver. Carcinomatous tumor limited to the pylorus displays but a single nodule, which nodule differs from those of hepatic carcinoma in that it is depressed by deep inspiration, but is not elevated by forced expiration. If firm adhesions bind the stomach to the liver, palpation is negative. Gastrectasis, if present, points to involvement of the pylorus, and the diagnosis can often be confirmed by associated gastric symptoms.

Carcinoma of the Colon and Omentum.—Here carcinoma is, as a rule, secondary. Carcinoma of the intestine involves most often the sigmoid flexure, and is characterized by the symptoms of chronic intestinal obstruction (p. 525) without jaundice or ascites. In carcinoma of the liver the pain is

higher in the abdomen, there is hepatic enlargement, and there may be jaundice. Carcinomatous growths developing from the colon and omentum can, as a rule, be shown to be differentiated from those involving the liver by auscultatory percussion—an invaluable sign.

Carcinoma of the Suprarenal Body.—Rarely, carcinoma involves primarily the suprarenal body, and spreads by direct extension to the inferior surface of the liver. We have observed two such cases, but in both the symptoms of hepatic carcinoma were obscure, and symptoms referable to adrenal disease were present early—*e. g.*, pain, tenderness, a mass in the region of the right kidney, and bronzing of the skin.

Hypertrophic cirrhosis can be mistaken for that unusual form of carcinoma only when the carcinomatous nodules are equally disseminated throughout the liver, with uniform enlargement of the organ, thereby giving a rounded edge to that portion of the liver projecting beneath the costal margin. The age of the patient, the character of the pain, and the late development of fever, together with extreme emaciation, will indicate carcinoma.

The distinctive features between hepatic carcinoma and **hepatic abscess** are shown in the accompanying table (modified from Anders):

Hepatic Carcinoma.	Hepatic Abscess.
1. May be hereditary. History of carcinoma elsewhere in the body—rectum, stomach, etc.	1. History of dysentery, traumatism, gall-stones, or suppuration elsewhere.
2. Occurs after middle life.	2. Commonest in early adult life.
3. Fever present only during the latter stage of the disease.	3. Septic fever throughout.
4. Cachexia a constant feature.	4. Absent.
5. Pain dull and constant.	5. Pain sharp, boring, and paroxysmal.
6. Chills unusual.	6. Chills and profuse sweating prominent symptoms.
7. Increased area of hepatic dullness extends downward.	7. Extends upward and may reach above the lower margin of the fifth rib.
8. Aspiration negative.	8. Aspiration may recover pus containing liver-cells, pus-cells, bacteria, or amebæ.

The distinctive features between *hepatic carcinoma* and *sarcoma* will be found under consideration of the latter condition (below), as will also that of *hydatids* (p. 980) and *amyloid disease* (p. 593).

Duration.—All cases tend to terminate fatally in from a few months to one year.

Complications.—Most serious of these are perforation of the colon or of the stomach. There is also likely to be carcinomatous peritonitis and carcinoma of certain of the other abdominal viscera.

HEPATIC SARCOMA.

Pathologic Definition.—A disease characterized by infiltration of the hepatic tissue by a sarcomatous growth, together with enlargement of the organ. Sarcoma may be primary, but in the majority of instances it is secondary to a similar lesion elsewhere. Melanotic sarcoma commonly attacks the liver after sarcoma of the choroid has been present. The enlargement is fairly uniform, and the organ may occupy the greater part of the abdomen, extending below the brim of the pelvis, and as far to the left as the left midclavicular line.

Predisposing and Exciting Factors.—The liver is often attacked by sarcoma following the removal or incision of a sarcomatous growth elsewhere in the body. Following the removal of sarcoma of the choroid, melanotic sarcoma of the liver may develop in from one to two and one-half years. Sarcomatous growths removed from the scalp are especially likely to recur in the liver, as are also those situated upon the lower extremities. Generally speaking, the liver is the viscus most likely to be secondarily attacked after the removal of a sarcoma.

Principal Complaint and Symptoms.—In general, the symptoms of hepatic sarcoma are those of mechanical obstruction, *e. g.*, gastritis, ascites, and, in certain cases, hemorrhoids. There are distinct anemia and progressive emaciation, both of which appear to result from malnutrition. The liver is uniformly enlarged, extending below the costal margin.

Laboratory Diagnosis.—There is a somewhat progressive secondary anemia. When there is extensive metastasis to other organs and the sarcoma is of the melanotic type, the urine may be brownish or blackish in color (melanuria).

Differential Diagnosis.—Hepatic sarcoma is differentiated from *hepatic carcinoma* by the following diagnostic features: Sarcoma is more common in the young, and often follows in the wake of a tumor of the eye. Pain is less frequent in sarcoma and cachexia is not so common. The hepatic surface is not nodular.

Duration.—All cases tend rapidly toward a fatal termination.

ACUTE HYPEREMIA.

Pathologic Definition.—A condition characterized by slight enlargement of the liver, with acute arterial congestion of the hepatic vessels.

Predisposing Factors.—Active hepatic congestion is seen to follow dietetic errors, chronic or acute alcoholism, and traumatism to the organ. It is also encountered in unfavorable cases of typhoid fever, typhus fever, pernicious malaria, and other infections. Hyperemia may be vicarious in character, *e. g.*, following sudden cessation of hemorrhage from the bowel and of the menstrual flow; rarely it may complicate hepatic cirrhosis (early stage).

Clinical Course.—Those cases due to dietetic error terminate favorably within from a few hours to one week. Hyperemia occurring during the course of hepatic cirrhosis is of grave prognostic significance, and when acute congestion follows traumatism over the liver, subsequent abscess-formation is to be feared.

PASSIVE HYPEREMIA.

Pathologic Definition.—A secondary condition excited by cardiac insufficiency (tricuspid regurgitation) and characterized by dilatation of the hepatic veins, with uniform enlargement of the liver.

Exciting Factors.—This is clearly tricuspid regurgitation, since the blood regurgitating from the right ventricle to the right auricle continues its backward current through the inferior vena cava until it reaches the liver—the first organ capable of being expanded.

Predisposing Factors.—Among the predisposing factors are: All diseases of the heart and lungs, or even those of the blood itself, in which there is a tendency toward the formation of blood-clots. Any condition that favors or excites tricuspid insufficiency predisposes to venous hepatic

congestion—*e. g.*, disease of the left heart, myocarditis, increased blood tension in the lung, anemias, and thoracic tumors of whatever nature pressing upon the ascending vena cava.

Such local conditions as pressure over the portal area by new-growths, cysts, etc., and abnormalities of the walls of the veins, as is seen in syphilitics and in thrombosis of the hepatic vessels, are among the predisposing factors.

Principal Complaint and Symptoms.—The patient complains of an undue *sense of fullness* in the upper portion of the abdomen that often increases to a distinct distress. Such gastric symptoms as nausea, vomiting, anorexia, and constipation are likely to be present. *Dyspnea* is a common symptom, and the patient frequently complains of an annoying *cough*, which may be accompanied by expectoration.

Physical Signs.—Inspection.—The upper right abdominal quadrant is unduly prominent, and in advanced cases edema of the feet and of the hands is seen. Jaundice, while not common, is occasionally observed, and when present in a marked degree, the patient also displays the clinical features usually associated with jaundice. (See p. 605.)

Palpation confirms inspection, and in addition enables one to outline the liver, the anterior inferior margin of which will usually be found some distance below the costal cartilages, and may even extend below the umbilicus. When the veins of the liver are greatly distended and tricuspid regurgitation is pronounced, the entire organ is found to pulsate.

Percussion and Auscultatory Percussion.—Percussion confirms palpation as to the size of the liver, and is of further service in determining the outline of the heart (cardiac dullness usually blending with liver dullness).

Auscultation.—In pronounced cases a distinct murmur is heard over the liver. The heart-sounds are weakened, as a rule, and murmurs are present in those cases that have reached the stage of cardiac dilatation.

Laboratory Diagnosis.—The blood-findings are those of secondary anemia. In cases showing jaundice the stools are clay colored, and the urine is of high color, of high specific gravity, and often contains a trace of albumin, which may be the result of passive renal congestion.

Duration.—This is governed entirely by the cause. In those cases resulting from valvular heart disease the liver may return to its normal size after the institution of judicious treatment. The rule is for the hyperemic condition of the liver to return as soon as treatment of the cardiac condition is stopped or the patient exercises beyond a limited degree.

ACUTE HEPATIC ABSCESS (SUPPURATIVE HEPATITIS).

Pathologic Definition.—A circumscribed accumulation of pus in the liver substance, with destruction of the hepatic tissue. The organ is, as a rule, enlarged, and such enlargement is often symmetric, whereas to the feel one or more areas of softening are detected. A single abscess is usually located in the right lobe near the superior surface, yet multiple abscesses are by no means uncommon, and may involve the left lobe. The tissues immediately surrounding the abscess are deeply congested, and in decidedly acute cases no well-marked abscess-wall is present, but in the subacute variety of abscess a distinct wall of demarcation is produced. Upon cutting through the abscess it is found to be filled with a liquid which contains pus-cells, necrotic tissue (liver-cells), and a variable amount of serous exudate. (See

also Amebic Abscess.) The amount of fluid contained in a hepatic abscess may vary from a few ounces to two or three quarts.

Microscopically, many of the hepatic cells are distorted in shape and devoid of nuclei. Round-cell infiltration is seen in the vicinity of the blood-vessels, and certain of the smaller vessels are plugged by emboli. Streptococci and staphylococci are usually present.

Varieties.—Clinically speaking, abscess may be acute, subacute, or chronic in character. A special heading is usually employed to designate amebic abscess, which is not in reality a true abscess, since it does not of necessity contain pus-producing bacteria.

Exciting and Predisposing Factors.—The introduction of pathogenic bacteria (cocci and bacilli) into the liver substance excites acute abscess.

Climate is the most potent predisposing influence; the majority of cases developing in tropical or subtropical districts or in those persons who have recently lived in the tropics. Occlusion of the bile-ducts, including that resulting from gall-stones, antedates nearly 50 per cent. of cases. *Disease of the gall-bladder* and other hepatic conditions were found by Kobler to be present in nearly 25 per cent. of cases; he also found that 13 per cent. of his cases followed pyemia. *Gastric ulcer* is frequently followed by hepatic abscess, as is also ulceration of the colon and appendix. Kelsch, in his analysis of 500 cases, found that 85 per cent. of them followed *dysentery*. Manson refers to 3680 fatal cases of dysentery on which autopsies were made (collected by Woodward), in 21 per cent. of which abscess of the liver existed.

Suppurating wounds of the scalp are not infrequently followed by hepatic abscess, as are also operations upon the rectum. Intestinal parasites may find their way into the gall-bladder and excite acute cholecystitis, which may terminate in abscess, as is shown by Leik's report of 19 cases following migration of the Ascaris lumbricoides to the gall-bladder. Liver-flukes and hydatids may excite the initial inflammation which terminates in hepatic suppuration. Foreign bodies taken into the stomach occasionally reach the liver, and there produce abscess; among such mechanic irritants are pins, fish-bones, needles, and buttons.

Principal Complaint and Symptoms.—**Pain,** a constant feature, is in the hepatic region, and radiates to the shoulder. The more superficially the abscess is located, the more severe is the pain, which is due to involvement of the peritoneum. The patient describes his pain as dull and boring in character. Pleuritic pain is experienced when the abscess is situated near the superior surface of the liver, and has excited an inflammation that involves the diaphragm and the pleura. Relief is afforded when the patient is in certain positions, and his agony is intensified by pressure over the right costal margin and by lying on the left side.

Chills, and even distinct rigors, are frequently experienced, and these are followed by profuse sweating.

In chronic hepatic abscess there are progressive weakness, emaciation, and the general symptoms of chronic sepsis.

Gastro-intestinal symptoms are constant, but are of such nature as to be of but limited service in formulating a diagnosis; they consist of flatulence, epigastric uneasiness, nausea, and vomiting, all of which symptoms become intensified as the disease progresses.

Nervous Symptoms.—The rule is for the patient to remain rational during the first thirty-six hours, after which period the nervous manifestations depend upon the degree of hepatic destruction and the grade of intoxication. Cephalalgia, muttering delirium, tremor of the tongue and

hands, mental hebetude, and finally coma are seen in cases that terminate fatally.

Thermic Features.—In those acute cases in which there is rapid, extensive destruction of liver tissue the temperature rises somewhat abruptly, reaching 103° or 104° F. during the first thirty-six hours. The character of the fever is distinctly irregular, and, as a rule, intermittent, resembling that of malaria. (See p. 948.) In those cases in which hepatic abscess is about to terminate fatally the temperature becomes subnormal, and the general condition is that of collapse.

Physical Signs.—Inspection.—The face is flushed at first, but later becomes pale, and jaundice may develop, depending upon the degree of hepatic destruction and the location of the abscess, as well as upon an associated involvement of the gall-bladder and the bile-ducts. Bulging over the region of the liver is present only when there is extensive abscess formation. Chest expansion is often limited upon the right side, due to an abscess resting near the surface of the diaphragm, and consequently exciting diaphragmatic and possibly pleural irritation. The tongue is coated, the lips are dry and fissured, and the teeth are often covered with sordes.

Palpation.—By deep palpation it is usually possible to elicit distinct tenderness along the costal margin. The liver is often felt below the margin of the ribs, and when the patient is directed to inspire deeply, the lower hepatic outline presents a smooth surface. In unusually large abscesses fluctuation may be elicited. Abscess of the left lobe of the liver is decidedly uncommon, yet when present, is quite easy of recognition. In those cases in which the adjacent peritoneum is involved, a friction fremitus may be felt. The abdominal muscles overlying the liver are at times unusually tense, which materially interferes with palpation.

The *pulse* becomes rapid and bounding during the first twenty-four hours, but as the disease advances, its frequency is increased, whereas its volume and force are diminished. In subacute and chronic cases the pulse is that of general sepsis, *e. g.*, weak, rapid, dicrotic, compressible, and irregular.

Percussion.—An increased area of liver dullness may or may not be present, although in typical cases the liver note will be found above the lower border of the fourth rib, and rises high (fifth rib) in the axillary line. Posteriorly, hepatic dullness usually extends to the angle of the scapula. In outlining the area of hepatic dullness by auscultatory percussion it will be found that the liver extends for a slight distance below the normal level.

Pulmonary Symptoms and Signs.—There is *cough*, which may be severe and non-productive, although in characteristic cases there is moderate expectoration of a reddish-brown, mucosanguineous material.

Auscultation reveals numerous fine and coarse râles over the base of the right lung, and rarely a friction murmur is detected.

Laboratory Diagnosis.—The blood changes are those of sepsis—*e. g.*, leukocytosis with a proportionate increase in the number of polymorphonuclear cells. As the disease progresses the hemoglobin and red cells are gradually decreased. During the height of the fever albuminuria is present, and casts and leukocytes may be found. In those cases developing jaundice the urinary sediment is colored, and in other respects the urine is that of jaundice. (See p. 606.) Indicanuria is to be expected.

Summary of Diagnosis.—A history of dysentery, gastric ulcer, or operation upon the intestinal tract (rectum) is of great help in formulating a diagnosis. The character of the pain, the presence of tenderness over the liver, an increased area of hepatic dullness when associated with leukocytosis,

and an increase in the polymorphonuclear elements of the blood strongly favor hepatic abscess.

Fever, while not characteristic, is highly suggestive of this condition, its main features being that it is decidedly irregular at first, but assumes the intermittent type as the disease advances. Chills and profuse sweating are likewise of clinical importance.

Differential Diagnosis.—**Malaria.**—Abscess, when developing in those residing in malarial districts, is likely to be mistaken for malaria on account of the periodicity with which the chill, fever, and sweating recur. Again, hepatic tenderness and splenic enlargement are characteristic of both conditions. Although there are many slight differences between acute hepatic abscess and malaria, in the light of our present knowledge there appear to be but three actually distinctive features: (*a*) Finding of the plasmodium of malaria in the blood; (*b*) the recovery of pus by aspiration of the abscess; and (*c*) leukocytosis is a feature of hepatic abscess and leukopenia is a characteristic feature of malaria.

Hepatic Colic.—There is usually a history of previous attacks that were followed by jaundice. The paroxysms (chills, fever, and sweat) of hepatic colic do not occur with such regularity as do those of hepatic abscess. Extreme pain with each seizure is characteristic of gall-stone colic, whereas in abscess a deep burning pain may be almost constant. In those cases in which a gall-bladder is impacted with stones there may be persistent jaundice, with practically all the symptoms known to accompany this condition, whereas in uncomplicated abscess of the liver jaundice is by no means constant.

Hepatic carcinoma can scarcely be confounded with hepatic abscess. (See Carcinoma, p. 581.) *Echinococcus cyst*, while it may present enlargement of the liver resembling that of abscess, is characterized by a normal or subnormal temperature, unless the cyst becomes infected with pathogenic bacteria, in which case it becomes a true hepatic abscess. The prolonged existence of hepatic enlargement, the absence of tenderness, the possible intimate association with dogs, and a residence in a country in which echinococcus disease is indigenous, all taken together, go far to support the diagnosis of hydatid cysts.

DISTENTION OF THE LIVER AND OF THE GALL-BLADDER.

Pathologic Definition.—A condition produced by obstruction to some one or more of the hepatic ducts, and characterized further by an abnormal distention of the ducts of the liver, with enlargement of the organ. In certain cases, as the result of undue pressure, there may be destructive changes in the parenchyma of the organ.

Exciting Factors.—Any condition that obstructs any one or more of the bile-ducts must of necessity be followed by a variable degree of hepatic enlargement.

Intrahepatic Conditions.—(1) Gall-stones, by blocking the common duct or the cystic duct; (2) carcinoma of the common duct; (3) nodular carcinomata of the liver pressing upon one of the ducts; (4) catarrhal inflammation of the common duct with obstruction by edema of the mucous lining (excited by cholecystitis, liver flukes, and extension of catarrh from the duodenum); (5) perihepatitis (syphilitic), by constriction and eventual interference with the flow of bile through the common duct.

Extrahepatic Causes.—(1) Carcinoma of the head of the pancreas, by pressure and obstruction of the common duct; (2) epithelioma of the duo-

denum, by extension to the duodenal orifice of the common duct; (3) gastric ulcer with extensive peritoneal adhesions, resulting in obstruction to the common duct; (4) abdominal tumors pressing upon the common duct or upon the inferior surface of the liver; (5) displacement of the liver (floating liver), with a variable amount of torsion; (6) blocking of the common duct by migrating intestinal parasites.

Principal Complaint.—There is a sense of fullness or discomfort in the superior right abdominal quadrant. There may or may not be pain, this symptom depending upon the cause of the enlargement.

Physical Signs.—Inspection.—The patient is, as a rule, jaundiced, although jaundice is not an essential feature of cystic distention. There is prominence of the right superior abdominal quadrant.

Palpation.—The pulse is unusually slow in those cases displaying jaundice. When enlargement follows carcinoma, the pulse becomes weak, thready, and almost imperceptible as the disease advances. One is able to outline the peculiar sausage-shaped mass projecting from the lower hepatic margin in those persons in whom the abdominal wall is not unusually thick. The enlarged gall-bladder may attain an enormous size, extending to a point on a level with or below the umbilicus. The tumor is usually dough-like in feel and may display fluctuation, and firm pressure does not excite pain.

Percussion.—Both percussion and auscultatory percussion confirm palpation with reference to the size of the gall-bladder and the liver.

Auscultation.—In those cases in which there have been quite extensive peritoneal adhesions, a friction murmur may be heard by placing the stethoscope over the lower margin of the liver, and then directing the patient to inspire deeply. If this sound is dependent upon obstruction of the common duct and impaction of the gall-bladder with calculi, then auscultation combined with firm palpation over the liver may result in producing a grating sound caused by friction of the calculi.

Laboratory Diagnosis.—In those displaying jaundice, both the blood and the urine are tinted yellow. The urinary sediment is likewise colored by the bile. If jaundice has existed for more than three weeks, the urine is especially likely to show albumin and casts. The froth of the urine is excessive, and of a yellowish tint, which is in itself characteristic of jaundice.

The feces are clay colored, and when studied *microscopically*, are found to be rich in globules of fat which are responsible for the peculiar color.

Summary of Diagnosis.—The diagnosis is based almost entirely upon the peculiar outline of the abdominal mass, which appears to be continuous with the liver. Persistent jaundice, the detection of fat in the stools, and the absence of bile-pigment from the feces are also of diagnostic value.

Clinical Course.—We have studied cases in both hospital and private practice in which the gall-bladder has remained decidedly enlarged for several weeks and even for months. In all such patients there were progressive emaciation, weakness, and a tendency toward the development of nephritis.

FATTY INFILTRATION AND FATTY DEGENERATION WITH HEPATIC ENLARGEMENT.

Fatty Infiltration.—Definition.—Fat infiltration may be found to involve localized areas of the organ in which the deposit is so intense as to give the cut surface of the liver a shiny or oily appearance. If fatty infiltration is general, the liver is enlarged, and the edge of the organ is rounded.

A cut section of the infiltrated hepatic tissue does not sink in water to the same degree as does that of the normal organ.

Microscopically, the normal hepatic cells are encroached upon by the deposit of fat, and fatty infiltration appears to favor the later development of fatty degeneration. Consequently, these two conditions may be found in the same case.

Predisposing Factors.—General obesity is most common, although fatty infiltration may be the result of interference with oxidation of the blood; consequently it is occasionally seen during the course of chronic organic maladies, *e. g.*, tuberculosis, and also in chronic conditions characterized by a severe type of anemia (chronic malaria, carcinoma, syphilis).

Principal Complaint.—There are no symptoms known to be characteristic or even highly suggestive of fatty infiltration, although in the majority of cases the patient states that he has not felt in perfect health for some months. There is a continuous sense of weight in the abdomen, while pain is absent. Clinically speaking, hepatic enlargement without symptoms is, to say the least, suggestive of fatty infiltration.

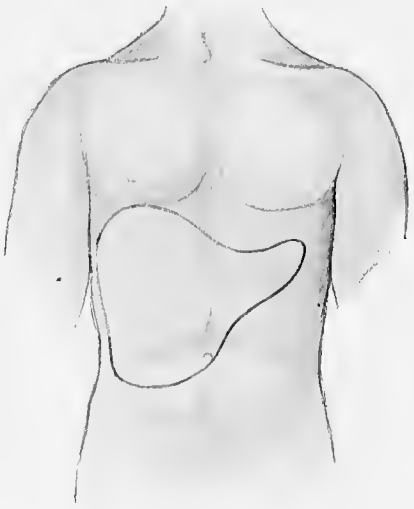


Fig. 244.—Fatty Liver Illustrative of Private Case.

Physical Signs.—Inspection.—The expression of the patient and the degree of emaciation will depend entirely upon the conditions that antedated hepatic enlargement. In most cases the right superior abdominal quadrant is unduly prominent.

Palpation discloses the lower border of the liver at some distance below the costal margin, and we have occasionally found it below the umbilicus. The edge of the liver is decidedly thickened and smooth, and firm pressure upon it does not produce pain. When the liver is extremely large and rests upon the descending aorta, distinct pulsation is felt over the hepatic area, which pulsation must be distinguished from that arising from aneurism of the abdominal aorta, the chief differential feature being that pulsation transmitted through liver substance is not expansile, as is the pulsation of an aneurism.

Percussion.—Both percussion and auscultatory percussion usually show the outline of liver dullness to be markedly increased. The liver note is seldom found above the fifth rib in the nipple-line, and is usually at about the same level in the axillary line, a point where, normally, the liver is more than an inch lower than in the mammary line (Fig. 244).

The left lobe of the liver will be found to extend well across the median line, and to be from two to four inches in breadth at the left border of the sternum, and between the ensiform and umbilicus. The spleen remains nearly of the normal size, although exceptions to this rule are encountered.

Differential Diagnosis.— In **amyloid disease** of the liver the distinctive features are that the spleen is also greatly enlarged, the urine is of

low specific gravity, contains a trace of serum-albumin, and may display casts.

Leukemia causes an enlargement of the liver that closely resembles that resulting from fatty infiltration, but in leukemia, as in amyloid disease, the spleen is enlarged. The history of repeated hemorrhages from the mucous membranes and of submucous hemorrhage points strongly toward the existence of leukemia. An examination of the blood gives findings (see p. 368) that distinguish positively between the large liver of leukemia and that of fatty infiltration.

FATTY DEGENERATION OF THE LIVER.

Pathologic Definition.—Any condition dependent upon fatty degeneration of the hepatic cells. On examining the organ, its size is not materially altered, but the cut surface presents a yellow color, and is soft and friable. The normal relation between the interlobular connective tissue and the acini is lost, the latter being replaced by fat. Equally disseminated throughout the substance of the organ small areas of pigmentation are to be seen.

The liver-cells have lost their normal shape, and their nuclei are indistinct or absent. Crystals, shreds, granular débris, cholesterin, and tyrosin are at times seen in the degenerated tissue.

General Remarks.—Fatty degeneration seldom results in hepatic enlargement, although such examples are occasionally encountered. After fatty degeneration is well established, there are usually present certain atrophic changes in the hepatic substance which keep the liver at or near its normal size, and, indeed, the dimensions of the organ may be far below the normal. Acute poisoning is often followed by fatty degeneration—*e. g.*, phosphorus (see p. 596), chloroform, and arsenic. Fatty degeneration is also present in acute yellow atrophy and in certain acute fevers.

Symptoms.—These are in no way characteristic, and the diagnosis is based largely upon the clinical history, which is often that of profound anemia resulting from some acute infection, such as dysentery, or from toxic poisoning.

The **physical signs** are of distinct clinical value only when hepatic enlargement is present.

AMYLOID DISEASE OF THE LIVER.

Pathologic Definition.— A condition characterized by amyloid (waxy) degeneration of the hepatic tissue, the walls of the blood-vessels being first affected, with uniform enlargement of the organ. Amyloid change in the liver is usually but one feature of a more or less general amyloid disease, the same pathologic changes affecting the kidneys, and less often the intestines and other portions of the body.

Predisposing Factors.—All chronic suppurative processes markedly predispose to the development of amyloid change in the viscera, and it must be remembered that when it affects the liver, it is but a portion of a more or less general amyloid degeneration. A similar change is also found, as a rule, affecting the kidneys and the spleen.

Tuberculosis or syphilis of the bones and, in fact, all types of chronic suppurative bone disease, are likely to be followed by amyloid degeneration. Syphilitic ulceration of the soft tissues (*e. g.*, rectum) also predisposes to amyloid change, and the condition occasionally follows carcinoma.

Principal Complaint.—The patient's general complaint is that of one suffering from a somewhat high grade of secondary anemia, *e. g.*, weakness, shortness of breath, palpitation, occasional attacks of vertigo, and gastro-intestinal disturbances. When the colon is also affected, there may be diarrhea.

Physical Signs.—Inspection.—The skin of the face and extremities is of a peculiar milk-white tint, although in some cases there is a slight bronzing. Puffiness beneath the eyes and at the ankles is common. The upper portion of the abdomen is usually prominent, and in thin subjects, when the patient inspires deeply, the outline of both the liver and the spleen may be seen through the abdominal wall (Fig. 245).

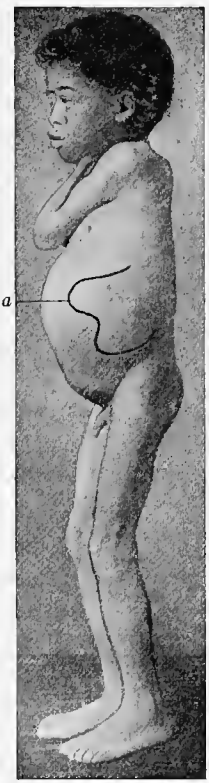


Fig. 245.—(*a*) Outline of Spleen and Splenic Dullness in Amyloid Disease (courtesy of Dr. Edwin E. Graham, of Philadelphia).

Palpation.—Both the liver and the spleen (Fig. 245) are found to be distinctly enlarged, although their surfaces are smooth and pressure does not elicit either pain or tenderness. As the disease advances, the pulse becomes weak and more and more rapid, and the apex-beat may be feeble and wavy.

Percussion confirms palpation in the detection of an abnormally large area of hepatic and of splenic dullness.

Laboratory Diagnosis.—There is usually coexisting amyloid change in the kidneys, in which case the quantity of urine voided during the twenty-four hours is normal (40 to 50 fluidounces) or may be markedly increased. It is pale, usually of low specific gravity, and contains a trace of serum-albumin and at times of serum-globulin. *Microscopically*, if the condition affects the renal tubules, hyaline and waxy casts are present, but amyloid disease of the kidneys may exist and the urine contain no waxy casts. (See p. 680.)

The *feces* are often pale or milk-white in color, but bile-pigment is present. Digestion of fats may be impaired, but not to the degree seen in pancreatic disease. (See p. 616.)

Summary of Diagnosis.—A history of prolonged suppuration, emaciation, weakness, and anemia, together with enlargement of the liver, strongly suggests amyloid disease. The diagnosis is confirmed, however, by the fairly characteristic urinary findings when there is an associated amyloid change in the kidneys. Pale, watery stools, when seen during the latter stages of the disease, are of great diagnostic significance.

Differential Diagnosis.—Amyloid enlargement of the liver is to be distinguished from fatty infiltration (see p. 591) and from sarcoma (see p. 586).

Clinical Course.—This depends entirely upon the nature of the predisposing and exciting conditions, *e. g.*, should the case in question be one of syphilitic origin, the course is modified by antisyphilitic treatment. In those depending upon pulmonary disease with cavity formation the course is of short duration.

ATROPHIC CIRRHOSIS.

General Remarks.—During the early stage of atrophic hepatic cirrhosis the liver may be moderately enlarged, although such increase in size is but temporary, and is followed, within a few months or a year, by a diminution in the size of the organ. At the beginning the symptoms and signs are often vague and indistinct. (For further symptomatology of this malady see p. 598.)

ACUTE PERIHEPATITIS (ACUTE SYPHILITIC PERIHEPATITIS).

Pathologic Definition.—A syphilitic involvement of the capsule of Glisson, characterized clinically by hepatic tenderness, hepatic enlargement, jaundice, ascites, and fever.

General Remarks.—This type of perihepatitis is extremely rare, but one case having been seen by us at the Philadelphia Hospital. This condition has been described by White and Martin and by other writers upon genito-urinary diseases.

Clinical Features.—Late during the second or at the beginning of the third stage of syphilis the patient develops a more or less continuous type of temperature, which ranges between 101° and 105° F. There is intense jaundice, which is accompanied by the other symptoms characteristic of this condition (slow pulse, mental hebetude, biliuria, etc.).

The liver is distinctly enlarged, and may extend for two or more inches below the costal margin, and the gall-bladder may be distended. The liver is usually somewhat tender, but this symptom rapidly disappears following the administration of antisyphilitic treatment. Ascites, when pronounced, makes the detection of hepatic enlargement difficult, but this may usually be overcome by turning the patient from side to side while making the physical examination.

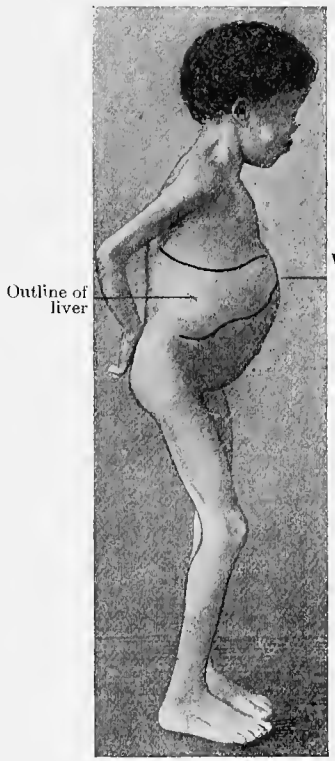


FIG. 246.—OUTLINE OF LIVER IN AMYLOID DISEASE (courtesy of Dr. Edwin E. Graham, of Philadelphia).

TUBERCULOSIS OF THE LIVER.

Pathologic Definition.—A condition usually secondary to tuberculosis elsewhere in the body, characterized by the presence of multiple tuberculous foci in the hepatic tissue, hepatic enlargement, and in many

instances localized and at times diffuse tuberculosis of the peritoneum. The disease may accompany any form of tuberculosis.

Exciting Cause.—Infection with the bacillus of tuberculosis.

Predisposing Factors.—Tuberculous lesions situated along the course of the mesenteric veins are most likely to be followed by general tuberculosis of the liver. Tuberculous lesions, wherever located, may antedate hepatic tuberculosis. In general miliary tuberculosis the liver is also involved.

Physical examination shows the liver to be enlarged, its edge rounded, and, as a rule, smooth, although tubercles palpable upon the superior surface of the liver have been reported.

Clinical Course.—All cases terminate fatally in from a few weeks to several months.

THE LIVER IN PHOSPHORUS-POISONING.

Pathologic Definition.—A condition excited by the entrance of phosphorus into the system, and characterized by fatty degeneration of the hepatic parenchyma. The liver is enlarged, light brown or yellowish in color, and presents a more or less mottled appearance. The substance of the organ is soft and friable. The cut surface shows decided mottling, and is streaked with areas of fat, the acini standing out more prominently than the interlobular tissue. Selected areas of the organ are deeply bile-stained, and the substance obtained by scraping the cut surface is composed mainly of bile and fat-globules. There is extensive disintegration of the liver-cells, and spheres of leucin, as well as crystals of tyrosin and cholesterin, are present. In this condition the destructive pathologic changes are not limited to the liver alone, but the gastric mucous membrane is thickened, opaque, and yellowish in appearance, and ulcerative gastritis may be present. Degenerative changes are also found in the kidneys, and are characterized by extensive fatty changes.

The exciting factor is the introduction, in various ways, of phosphorus into the system.

Predisposing Factors.—(1) **Occupation.**—Those working in match factories are necessarily exposed. Large amounts of phosphorus may also be taken in with certain foods, and poisonous doses may be accidentally swallowed.

Remarks.—This condition will be found to vary greatly, depending upon the rapidity with which phosphorus has been introduced into the system; *e. g.*, where the condition attacks a worker in a match factory or one exposed to the handling of phosphorus, the symptoms often develop quite insidiously, whereas in those having recently taken a single lethal dose of the drug the symptoms develop acutely.

Principal Complaint.—From three to twelve or even twenty-four hours after the system has become charged with phosphorus the patient complains of a general sense of uneasiness, nausea, and oppression over the epigastrium, which may develop into a dull pain, accompanied by vomiting. In favorable cases vomiting subsides within two or three days.

Nervous Features.—Among the nervous phenomena are headache, wakefulness, vertigo, and at times maniacal delirium. The patient may become comatose, from which state he seldom rallies.

Constipation is present at the onset, but may alternate with attacks of diarrhea. (See Laboratory Diagnosis.)

Thermic Features.—The temperature is by no means characteristic, but is generally between 99° and 101° F. A subnormal temperature is of unfavorable prognostic omen.

In chronic poisoning the mucous surface of the mouth may display ulceration, and destruction of the maxillary bones is also seen.

Physical Signs. —**Inspection.**—In acute phosphorus-poisoning inspection reveals distinct jaundice of the skin and mucous surfaces.

Palpation.—On making deep palpation it is possible to detect the lower border of the liver two or three inches below the costal margin. In those cases in which there has been a chronic poisoning of the system with phosphorus, the liver is rarely found to be appreciably smaller than normal. Tenderness is elicited upon making deep pressure over the hepatic region.

Laboratory Diagnosis.—The vomitus at first contains the contents of the stomach, but later it becomes bile-stained. The characteristic feature of the vomitus is that, when placed in a dark room, the liquid is phosphorescent, and that the presence of phosphorus may be demonstrated chemically. After a high grade of irritation of the gastric mucous membrane is produced or after marked alteration has taken place in the blood, hemorrhage into the stomach follows, and the vomiting of blood that has been acted upon by the gastric fluid (black vomit) occurs.

The quantity of urine voided during the twenty-four hours is diminished; it is bile-stained, its specific gravity is high (above 1.025), and chemically there are to be found bile-pigment, sarcolactic acid, and albumin. *Microscopically*, crystals of leucin and tyrosin (Fig. 263) may be seen, and the detection of casts, fat-globules, and renal epithelial cells is quite common.

Summary of Diagnosis.—The diagnosis is based upon the following salient points: (*a*) A history of having taken phosphorus or of following an occupation that necessitates the handling of phosphorus; (*b*) the vomiting of material that becomes phosphorescent when placed in the dark; (*c*) the development of jaundice; and (*d*) the fact that the urine is bile-stained and albuminous, and also contains sarcolactic acid. Lesions of the mucous surface of the mouth and of the maxillary bones also aid in the diagnosis.

Differential Diagnosis.—The condition with which acute phosphorus-poisoning is most likely to be confounded is **acute (hepatic) yellow atrophy,** and the differential features between these two conditions are set forth in the following table (modified from Anders):

Acute Phosphorus-Poisoning.	Acute Yellow Atrophy of the Liver.
1. There is a history of accidental taking of poison (match-heads, rat poison), or of occupation with exposure to phosphorus.	1. Indefinite.
2. The onset is sudden, with violent nausea, vomiting, and pain over the region of the liver.	2. A slow onset, with malaise, nausea, and vomiting.
3. Jaundice appears on the second or third day.	3. Jaundice is a beginning symptom.
4. Nervous symptoms appear late in the disease, and are always preceded by jaundice.	4. Nervous symptoms may appear early —even before the occurrence of jaundice.
5. The vomit and stools are phosphorescent. Black vomit precedes death.	5. Black vomit occurs early, and persists throughout. It is never phosphorescent.
6. Temporary arrest of symptoms between the occurrence of jaundice and black vomit.	6. Progressive increase of symptoms with no remission.
7. Sarcolactic acid is present in the urine, and leucin and tyrosin but rarely present.	7. Leucin and tyrosin are common in the urine.

Clinical Course.—This is short—from a few days to six weeks—and, in the majority of cases, fatal. In chronic poisoning the condition may tide over weeks or months and recovery follow.

DISEASES IN WHICH THE SIZE OF THE LIVER IS DIMINISHED.

ATROPHIC CIRRHOSIS.

Pathologic Definition.—A chronic condition characterized by extensive pathologic fibrous changes in the liver substance or in its capsule. The organ is somewhat enlarged during the early stage of the disease, and, in fact, a more or less permanent enlargement is possible, although in typical cases the size of the liver is reduced and the capsule thickened. The organ feels firm, and may at times be decidedly altered in shape. It cuts with resistance, and displays grayish-white bands of connective tissue coursing through the cut surface. The liver may be mottled and show yellowish areas, which at times project above the surface. The organ may display a roughened surface, the so-called "hob-nailed" appearance.

Microscopically, the pathologic changes are seen to begin as an increase in the connective tissue surrounding the terminal branches of the portal vein. Later, the liver-cells are compressed, and the portal veins are also encroached upon by fibrous tissue. Early during the disease atrophic changes involving the hepatic cells are apparent, and the biliary canaliculi may be appreciably increased in number. In atypical cases of alcoholic cirrhosis the liver is enlarged, smooth, faintly granular, and somewhat soft.

Varieties.—(1) Atrophic cirrhosis (Laennec's cirrhosis, alcohol or gin-drinker's liver); (2) Glissonian or capsular cirrhosis, in which the fibrous changes are limited to the capsule of Glisson; and (3) syphilitic cirrhosis.

Remarks.—Foxwell has recently shown that during the early stage of alcoholic cirrhosis the liver commonly shows moderate enlargement, although the organ becomes decreased in size as the disease progresses. The investigations of Morse throw a somewhat confusing light upon this subject, since his examination of the records of 37 cases of hepatic cirrhosis discloses the fact that 13 of them showed enlargement of the liver, in 11 the size of the organ was normal, and in 12 the liver was abnormally small. In Glissonian cirrhosis the diminution in the size of the organ is due to the capsule of Glisson being greatly thickened, and ofttimes the organ is surrounded by dense bands of fibrous tissue that, by their contraction, compress the liver at various points, giving it an extremely roughened surface, which has given to it the designation of "hob-nailed" liver.

Predisposing and Exciting Factors.—(*a*) Practically all writers are agreed that the excessive use of **alcohol** materially predisposes to, and is probably the exciting factor of, atrophic cirrhosis in a large percentage of all cases. The stronger the beverage used, *e. g.*, whisky, gin, etc., and the larger the quantity of this substance consumed, the more likely is the patient to develop cirrhotic changes in his liver. "Doubtless by the side of alcoholism all other causes combined are comparatively insignificant" (Anders).

(*b*) **Sex.**—Males are affected more often than females.

(*c*) **Age.**—Early adult and middle life is a prominent predisposing factor, more than 60 per cent. of all cases developing during the third and fourth decades.

(*d*) **Congenital syphilis, gout, chronic malaria,** and **chronic tuber-**

culosis, either of the lungs or of the bones, are believed to act as predisposing factors to the development of atrophic hepatic cirrhosis.

(*e*) **Fatty cirrhosis** is said to result from the excessive use of malt liquors, and is also seen to occur in persons suffering from obesity.

(*f*) **Chronic passive hyperemia** of the liver, the result of cardiac incompetency (tricuspid regurgitation) or of interference with the circulation through the lungs, may in time lead to the development of cirrhosis of the liver.

(*g*) **Acute Infections.**—Rarely, hepatic cirrhosis is seen to follow certain acute infections.

Principal Complaint.—The symptoms of atrophic cirrhosis are often vague until there is interference with the portal circulation; therefore, characteristic symptoms appear late in those cases in which the superficial abdominal veins are expanded to compensate for the impaired circulation through the liver.

The patient complains early of having lost flesh and of *morning nausea;* his tongue and teeth are frequently covered with a peculiar gummy substance upon rising after a night's sleep. Constipation develops early, and has often existed for years before the physician is consulted. As the disease advances the patient, in addition to being nauseated, may *vomit* once or twice during the twenty-four hours, and the vomitus may be blood-stained. As interference with the portal circulation becomes more marked the nausea and vomiting gradually increase, until the patient vomits nearly every morning. One of the earliest complaints of business men suffering from atrophic cirrhosis is of mental hebetude and inability to concentrate the mind, such patients often declaring that it tires them to think.

The continuous expectoration of blood-streaked sputum is an occasional late symptom, and is caused by *minute hemorrhages* from dilated esophageal veins. In conjunction with hemorrhages from the stomach and esophagus the patient develops hemorrhoids, and now complains of hemorrhage from the rectum. The severity of the hemorrhage from the rectum may vary greatly: at first but a small quantity—a few drops—is passed with each movement of the bowels, but the quantity increases until one or two ounces of blood may be expelled at each evacuation. Throughout the entire course of the malady there is a gradual but progressive *loss* in both *strength* and *flesh.*

Nervous Phenomena.—Toxemia may develop at any time during the course of the disease, but is most often seen in advanced cases, and is believed to be due to some toxic substance having entered the blood as the result of imperfect hepatic function. The nervous symptoms of toxemia usually develop at a time when there is inactivity of the kidneys, and, indeed, they closely resemble those of uremia. Severe headache may be continuous for days or even weeks, and periodic outbreaks of headache are not unusual. Confusion of ideas and even maniacal delirium may be seen, whereas convulsions and coma are occasionally observed.

Thermic Features.—Fever may be present at any time during the course of the disease, and ranges between 99° and 102° F. It is to be borne in mind, however, that atrophic cirrhosis may run its long course without showing an elevation of temperature.

Physical Signs.—**Inspection.**—*Skin.*—The complexion is somewhat sallow, and there is slight evidence of jaundice in 25 per cent. of cases, although distinct jaundice may not appear at any time during the disease. Bronzing of the skin (hemochromatosis), as described at length by Opie and others, is rarely observed.

The expression is anxious, the face is pinched, and there is emaciation of the chest and of the extremities. The abdominal veins are usually enlarged, and even the veins of the chest may attain an enormous size. Immediately surrounding the umbilicus there is often seen a plexus of dilated veins—the "caput medusæ." A nodular dilatation of the hemorrhoidal veins is seen at the rectum. After ascites sets in the abdomen becomes enormously distended. As the disease advances, the lower extremities become more and more edematous; edema of the scrotum, prepuce, and labia is a late annoyance.

Palpation.—The skin of the ankles and often of the lower limbs pits upon pressure, and later there is pitting over all dependent portions of the trunk and chest. The pulse is of good tension, but becomes weak and more rapid with the advance of the disease. In those uncommon instances in which jaundice develops the pulse-rate is somewhat slower. Following the accumulation of fluid in the peritoneal cavity all the physical signs of ascites are present. (See p. 567.) Prior to the development of ascites or after the peritoneal fluid has been removed by aspiration the spleen is readily palpable, whereas the liver *is not* palpable.

Percussion.—The area of liver dullness in the midclavicular line is greatly diminished, and is usually found to extend to a level of one or more inches above the costal margin, whereas under normal conditions hepatic dullness extends to the margin of the ribs. Posteriorly, hepatic dullness is found to begin at a lower level than it does under normal conditions.

In cases exhibiting typical symptoms of atrophic cirrhosis the area of liver dullness may be increased or normal; it has been our experience to find such cases, when followed to autopsy, display other associated pathologic abdominal conditions.

Auscultatory percussion often enables one to ascertain the actual size of the liver (p. 59) in those cases in which ascites is present and where percussion is of little value.

Laboratory Diagnosis.—The *vomitus* displays nothing characteristic, although the morning vomiting of bloody material is highly suggestive of atrophic cirrhosis. The sputum may be blood-streaked. Late in the disease the urine is often highly colored and of high specific gravity. In those cases in which jaundice develops the urine is rich in bile. Albumin was found in but a single instance in 28 cases studied by Henry; in our experience albuminuria is common late in the disease, but urines rich in serum-albumin are not always found to contain renal casts. The quantity of urine and the amount of urea excreted during the twenty-four hours are below normal. Indicanuria is a common feature of atrophic cirrhosis.

The *hemic changes* are those of chronic secondary anemia, *e. g.*, the hemoglobin and red cells are decidedly diminished, and these changes may or may not be accompanied by alteration in the number of leukocytes.

Illustrative Case of Atrophic Cirrhosis.—H. H. C., male, aged forty years; height, 5 feet 9¾ inches; usual weight, 168 pounds; present weight, 147 pounds.

Family History.—Father died of heart disease at the age of fifty-one years; mother, of carcinoma of the uterus at sixty-four. Two younger brothers living and reported to be in good health. A younger sister has suffered from kidney disease for the past two years, and another sister died of pneumonia at the age of fourteen. No further history of malignancy in family.

Previous History.—The patient had scarlet fever, measles, and whooping-cough before puberty; fracture of the left arm at the age of twenty-three; and typhoid fever at about the age of thirty. He had influenza six years ago, and since that time has been subject to repeated "colds." Two years ago he consulted his physician in regard to a

condition of the rectum, and since that time he has been treated repeatedly for hemorrhoids. He also states that he refused operation for this affliction, and that he frequently loses considerable blood during the act of defecation.

Social History.—Married at the age of twenty-three; three sons and a daughter are living, and apparently in perfect health. He states that he has not taken a vacation during the past twenty years. Since the age of twenty-five he has always eaten heavily, partaking of a large amount of rich food and particularly of meats. He also has used alcohol in its various forms, and states that until within the past year he has taken from two to six glasses of whisky or gin and probably two to four bottles of beer daily. He has taken but little outdoor exercise for several years, declaring that such exercise tired him, and that he felt best when resting quiet. He does not use tobacco.

Present Illness.—The patient has not felt perfectly well for the past year, and has become progressively weaker and has lost in weight. He complains of being easily exhausted, and that he does not become rested as the result of sleep, although he has been able to sleep fairly well until within the past two weeks, when he could obtain sleep only by sitting in bed. For the past two months he has noticed a progressive enlargement of his abdomen, and at present the circumference of the abdomen is 4¼ inches greater than normal. The appetite is poor, and he is unable to eat breakfast. Upon several occasions he has vomited a large quantity of watery material. During the past two months there have been two attacks of mild nose-bleed, and when he clears his throat he not unusually expectorates blood-streaked material. Constipation is obstinate, and the use of laxatives has been necessary for the past two years. At present a subconjunctival hemorrhage involving the right eye exists, and the patient states that during the past year he has had three or more similar hemorrhages.

He has suffered at times from rectal pain, probably as the result of hemorrhoids. Headache is quite constant, and at times is a most annoying feature. He describes it as a dull pain that is commonly present upon rising after a night's sleep, but becomes less distressing after midday.

The patient has become extremely irritable during the past two years. Slight exercise induces dyspnea, which is followed by cardiac palpitation.

The temperature is normal during the morning hours, but reaches 99° to 100° F. during the afternoon of each day. Fever was not present until the past two weeks.

Physical Examination.—*General.*—The skin and mucous surfaces are pale, there is some puffing beneath the eyes, and the evidences of emaciation are present. The veins of the lower extremities are also enlarged and tortuous, and there is swelling of the feet and of the ankles. The finger-nails are clubbed, rough, and extremely brittle.

Local Examination.—*Inspection.*—The cheeks are sunken, and the features are sharp. The abdomen is unusually prominent, and the superior abdominal veins are markedly enlarged. There is a distinct network arrangement of enlarged veins about the umbilicus (caput medusæ). There is an extensive hemorrhage beneath the conjunctiva of the right eye.

Palpation.—The lower portion of the anterior abdominal wall shows evidences of slight edema upon pressure, and the skin about the ankles and feet also pits upon pressure; the scrotum is edematous. Fluctuation is distinctly felt over the peritoneum. The pulse is small, fairly strong, and of moderate tension, the beats numbering 90 a minute. The skin is rough and somewhat dry, and over the abdomen and lower extremities it is covered with fine scales.

Percussion.—When the patient is resting upon his back, there is flatness in both flanks, rising as far anteriorly as the crests of the ilia, and above these flat areas there is tympany. By turning the patient on his left side the flat area of the right side and flanks disappears, and is replaced by tympany, and a similar result is obtained when the patient is turned on his right side, the left flank and left side becoming tympanitic. By placing the patient upon his left side the area of hepatic dullness is found to be appreciably decreased, the upper margin of the liver dullness being found 1½ inches above the lower border of the costal margin. On making auscultatory percussion the spleen is found to be enlarged, although, owing to the presence of peritoneal fluid, this evidence could not be elicited by ordinary percussion.

Auscultation.—The heart-sounds are clear at the mitral area, the second pulmonic sound being distinctly accentuated; and a systolic murmur is heard at the aortic cartilage, which is transmitted for some distance upward through the vessels of the neck.

Laboratory Findings.—The patient gives a history of having passed large quantities of urine for several years, but since his abdomen became enlarged, he has observed that he voids urine less often than formerly; at present he is passing but from 18 to 24 ounces during the twenty-four hours. The urine is highly colored, has a specific

gravity of 1.026, is rich in solids, and contains a trace of albumin. A decided reaction for indican is present, and a microscopic study showed the presence of a few narrow hyaline casts and many leukocytes.

A blood examination showed hemoglobin, 67 per cent.; red cells, 3,100,000; white cells, 5400. Stained blood gave the evidence of secondary anemia.

Diagnosis by Induction from Clinical Data.—The age of the patient, as well as the protracted course of the illness, was considered as characteristic evidence, and when, later, ascites developed, and the area of hepatic dullness was found to be appreciably diminished, cirrhosis of the liver was strongly suggested. Great importance was also attached to the early development of hemorrhoids.

Course of the Disease.—When the diagnosis was established, it was deemed advisable to aspirate the peritoneal cavity for the purpose of removing the ascitic fluid. Two and one-half quarts of serous fluid were removed from the abdominal cavity, following which the patient's general condition improved greatly, but fluid subsequently reaccumulated within the peritoneum, and six weeks later aspiration was again performed. Four months after he was first seen at the hospital it was found that there was an accumulation of fluid in both pleuræ, and at this time the area of cardiac dullness was such as to raise the question whether or not a moderate pericardial effusion existed. The peritoneum was aspirated several times, but the patient's general condition went from bad to worse, until general anasarca supervened, and he finally died of exhaustion, twelve months after his first visit to the hospital, and approximately two and one-half years after he consulted his physician for hemorrhoids.

Summary of Diagnosis.—Progressive emaciation, gastric catarrh with morning vomiting, hematemesis, and blood-streaked expectoration which is cleared from the throat and not expelled by coughing, all support the diagnosis of hepatic cirrhosis. Hemorrhoids, ascites, edema of the lower extremities, secondary anemia, splenic enlargement, and diminution in the size of the liver are also among the cardinal features of atrophic cirrhosis.

Differential Diagnosis.—Late during its course atrophic cirrhosis is to be distinguished from both acute and chronic conditions that are known to exhibit ascites as one of their features.

Renal Disease.—Hepatic cirrhosis is distinguished from *renal disease* by an examination of the urine, which in the latter condition is rich in albumin and contains many casts. (See Chronic Parenchymatous Nephritis, p. 671.)

Valvular heart disease, when accompanied by ascites, is distinguished from atrophic cirrhosis by the fact that the liver is enlarged in cardiac conditions and small in cirrhosis, whereas the spleen is enlarged in both of these conditions. The exception to the foregoing statement is seen in those cases in which atrophic cirrhosis follows prolonged cyanotic congestion of the liver resulting from cardiac insufficiency.

Clinical Course.—Nearly all cases of atrophic hepatic cirrhosis are rapid in their course, and terminate fatally in from a few months to one year after symptoms of portal obstruction develop. Rarely, indeed, compensatory circulation is established through the superficial veins, and the patient lives for years.

GLISSONIAN CIRRHOSIS.

Pathologic Definition.—A disease characterized by an abnormal fibrous thickening of the capsule of Glisson, with contraction of the liver, destruction of the hepatic cells, and a diminution in the size of the organ, with the symptoms of atrophic cirrhosis.

General Remarks and Etiology.—Perihepatitis is believed to be syphilitic in origin in the majority of instances, although certain other conditions are named as predisposing factors, as, *e. g.*, prolonged tight lacing, the following of occupations that make undue pressure over the liver, and suppurative processes of the liver and adjacent structures.

Glissonian cirrhosis may assume a decidedly chronic course, without giving evidence of interference with the flow of the return blood from the stomach and from the intestine.

Summary of Diagnosis.—An antemortem diagnosis is quite impossible. The features that point most strongly toward Glissonian cirrhosis are a history of syphilitic infection, a diminution in the size of the liver, pain over the right superior abdominal quadrant, and the amelioration of the symptoms after the application of antisyphilitic treatment.

ACUTE YELLOW ATROPHY (Malignant Jaundice).

Pathologic Definition.—An acute, possibly infectious disease, characterized pathologically by extensive destruction of the hepatic parenchyma, diminution in the size of the liver, multiple cutaneous hemorrhages, and jaundice.

Predisposing Factors.—Age and Sex.—The majority of cases develop between the age of fifteen and thirty-five years; females are affected more often than males, and the condition is said to follow childbirth.

Acute fevers, general sepsis, and syphilis are also regarded as exerting slight predisposing influence, whereas alcoholism and chronic atrophy of the liver are possible predisposing causes. The disease is certainly of toxic nature, and the weight of opinion seems to point to a microbic origin.

Principal Complaint.—The patient complains early of indisposition and gastro-intestinal disturbance, neither of which, however, is characteristic of the disease. When the condition is well established, there are more or less continuous *headache*, periodic attacks of *nausea* accompanied by *vomiting*, and complete *anorexia*.

Within the course of one or two weeks the acme is reached, and *grave nervous symptoms* develop, as, *e. g.*, mental hebetude, followed by restlessness, excruciating pains in the head, and, later, there is generally either low muttering delirium or maniacal outbreaks. *Convulsions* may develop late, and are commonly followed by stupor, which gradually merges into coma. The development of nervous symptoms is of unfavorable prognostic omen, as the patient usually becomes *comatose* in from twenty-four to forty-eight hours thereafter.

Thermic Features.—At the onset there may be moderate fever, although it is in no way characteristic. The temperature may remain practically at the normal until near the close, reaching 101° to 103° F. a few hours before death.

Physical Signs.—Inspection.—Within a few days after the initial symptoms the skin becomes jaundiced, and with the progress of the disease this symptom becomes intensified until the entire body is of a greenish-yellow hue. Petechial hemorrhages into the skin are a feature of acute yellow atrophy, and larger ecchymotic areas are not uncommon. The lips become dry and fissured, and the tongue is at first moderately coated, but may assume the so-called typhoid appearance late in the disease.

A coarse tremor of the hands and of the tongue is present. The mucous membrane of the mouth may show minute hemorrhages, and bleeding from the oral and nasal mucous surfaces may occur.

Palpation.—The liver is not readily palpable except during the initial or first stage of the disease, when the lower margin may be felt. As the disease progresses there is a distinct recession of the anterior inferior border

of the liver, and this is most readily detected over the left lobe. Firm pressure over the liver elicits tenderness and may excite pain.

The *pulse* is not greatly accelerated at first, but the volume and tension are both diminished. As the disease progresses to the typhoid state the pulse becomes weak, rapid, and dicrotic.

Percussion.—At the onset the area of hepatic dullness is normal, or possibly slightly increased, but after the patient has entered into the typhoid state the area of liver dullness is markedly diminished. A characteristic feature is that this area lessens appreciably from day to day. Cases are recorded in which the liver was increased in size.

Laboratory Diagnosis.—Vomiting during the premonitory state is of common occurrence, and the vomitus contains, first, the contents of the stomach, and later, as a rule, blood is vomited; blood may also be extravasated into the mouth.

Constipation usually obtains, and the stools are often clay colored. When the disease reaches its acme, hemorrhage from the bowel occurs.

The urine voided during the twenty-four hours is smaller in quantity than normal, is of high color (stained with bile), and the specific gravity ranges between 1.025 and 1.035. *Chemically,* serum-albumin is present, there is a diminished excretion of urea, and hemorrhage from the bladder is not unusual.

Microscopically, the urine contains casts, leukocytes, and epithelial cells, all of which are bile-stained. Crystals of leucin and tyrosin (Fig. 263, see p. 658) are frequently seen. Red blood-cells are an almost constant finding.

Summary of Diagnosis.—The clinical factors—gradually increasing jaundice, moderate rise in temperature, and progressive increase in severity of the nervous symptoms—and the urinary findings, *e. g.,* choluria, hematuria, and the presence of leucin and tyrosin, are sufficient to warrant a diagnosis.

Differential Diagnosis.—Acute yellow atrophy is distinguished from **hypertrophic cirrhosis**—(*a*) By its gradual onset; (*b*) by the fact that leucin and tyrosin are seldom present in the urine; and (*c*) by the presence of enlargement of the liver in the latter condition.

Acute yellow atrophy closely simulates **acute phosphorus-poisoning,** and at times it is difficult to distinguish between these conditions.

Clinical Course.—Acute toxemia develops early, the cases usually progressing from bad to worse, until grave nervous symptoms develop. A fatal termination invariably follows.

JAUNDICE AND THE PATHOLOGIC CONDITIONS OF THE LIVER, GALL-BLADDER, AND BILE-DUCTS IN WHICH JAUNDICE FORMS A PROMINENT SYMPTOM.

JAUNDICE.

Pathologic and Clinical Consideration.—A symptom of any condition characterized by obstruction of the passage of bile through the biliary ducts or from the hepatic cells, and by staining of all the body-tissues with bile. Choluria, intense itching, urticaria, furunculosis, anorexia, constipation, clay-colored stools, headache, vertigo, insomnia, and a slow pulse comprise the other features of jaundice.

General Remarks.—It must be remembered that jaundice is but an expression of some underlying pathologic condition. In dealing with jaun-

dice under a special heading we are simply considering at length the conditions capable of producing it, and, clinically, the many symptoms and signs that are invariably present. In other words, if the statement is allowable, we are here dealing with the "symptomatology and signs of a symptom," and not with a disease itself.

Varieties.—Two types of jaundice are recognized: (*a*) Hepatogenous (obstructive jaundice), and (*b*) hematogenous or jaundicing of the skin the result of degeneration of the blood-cells and liberation of their pigments. At present the latter condition is extremely rare, and some authors go so far as to refuse to recognize this type of jaundice; for these reasons we shall consider only that type which is of hepatic origin, and which certainly includes the vast majority of cases.

Exciting Factors.—(1) Obstruction to the common duct (as, *e. g.*, by gall-stones); (2) invasion of the common duct by round-worms (rare); (3) the existence of liver flukes in the bile-ducts (rare); (4) carcinomatous growths within the common duct; (5) acute catarrh of the common duct; (6) acute catarrh of the gall-bladder; (7) purulent inflammation of the gall-bladder; (8) empyema of the gall-bladder; (9) stricture of the common duct following ulceration or traumatism from gall-stone; (10) reduced blood-pressure in the liver, which is followed by the absorption of bile by the blood; (11) rapid destruction of the liver tissue, as is seen in phosphorus-poisoning, and also interference with the escape of bile from the hepatic cells, as is seen in hypertrophic cirrhosis and acute yellow atrophy; and (12) pressure upon the common duct from without, which may result from tumor of the omentum, pancreas, liver, stomach, floating kidney, pregnancy, abdominal aneurism, etc.

Clinical Picture.—Cutaneous Manifestations.—Yellowing and pigmentation of the skin are among the earliest signs of jaundice, and there is also discoloration of the conjunctivæ. In this condition it is important that the patient be examined by sunlight, since the cutaneous pigment of jaundice is not discernible with artificial light. After jaundice has persisted for several weeks, the skin is tinted a greenish hue, and may even assume a greenish bronze color.

Pruritus and *itching* are troublesome, and the more chronic the condition, the more pronounced are these symptoms. *Urticaria* may develop at any time during an attack of jaundice, and is often persistent. Furunculosis is common, and infection of the skin by pus-producing organisms probably takes place from attempts to relieve the intense itching. Profuse *sweating* is the rule, and may be either general or localized. The palms of the hands, soles of the feet, and the skin overlying the abdomen are favorite sites for profuse sweating, and the axillary and inguinal regions may be similarly affected.

Xanthelasma, the development of bright yellow, slightly elevated spots upon the skin, may follow in those cases in which jaundice is protracted, and the favorite site is about the eyelids. Cutaneous ecchymoses (see Acute Yellow Atrophy) and ecchymoses of the mucous surface are also an occasional feature in grave types of jaundice.

Gastro-intestinal Features.—The symptoms of gastro-intestinal catarrh are usually present, *e. g.*, anorexia, nausea, hiccough, a sense of fullness over the epigastrium, and flatulency. In the more serious types of the condition vomiting, intestinal pain, pain over the liver, and hepatic tenderness are quite common. In jaundice following a chill (severe rigor) vomiting is the rule. Constipation is the rule, the stools being of a glazed slate color,

and having a pronounced odor. Diarrhea may develop at any time during an attack.

Thermic Features.—Fever is not an essential feature of jaundice, and when present, will be found to vary in direct accordance with the exciting cause of the jaundice, or some other associated febrile malady.

Nervous Phenomena.—Mental dullness, inability to concentrate the mind, forgetfulness, irritability of temper, insomnia, headache, and vertigo develop early, and increase in severity as the condition progresses. Maniacal or low muttering delirium and coma may develop in those cases in which auto-intoxication is profound. When marked nervous symptoms are present, the condition is generally referred to as acholia or cholemia, although the true nature of the toxic agent responsible for such symptoms is doubtful.

Ocular Peculiarities.—In cases of deep jaundice the vision is sometimes yellow—a condition known as *xanthopsia*. In very rare instances the tears are tinged with bile.

Secretions and Excretions.—The *urine* is colored by the bile, and displays a distinctly yellowish or yellowish-green hue. When shaken, the urine of jaundice displays a heavy froth, which is also bile-stained—a feature that serves to distinguish the froth of choluria from that of other conditions. The organic sediments of the urine—*e. g.*, casts, leukocytes, red blood-cells, and epithelial cells—are also bile-stained.

Milk.—When jaundice develops while the mammary glands are functionating, the milk is stained by bile-pigment; and when jaundice has persisted for a long time, there is slight yellowing of the saliva. A fact to be borne in mind in this connection is that the lacrimal secretion which is colored by the bile escapes into the nose and may find its way into the mouth.

Clinical Course.—The intensity of discoloration of the skin, secretions, and excretions depends entirely upon the character of the causal factor in each case, and upon the length of time jaundice has existed. In those cases due to simple catarrhal inflammation of the bile-ducts that has probably extended from the duodenum, the icteroid tint disappears in from ten days to three weeks, but where there is obstruction of the duct, the condition may persist for weeks and even for months. When jaundice is the result of infection of the gall-bladder by pyogenic bacteria (cholecystitis), the already prominent symptoms of the preëxisting or underlying affection are simply aggravated, as is exemplified by the jaundice occasionally seen to complicate lobar pneumonia and other acute infections.

CATARRHAL JAUNDICE.

Pathologic Definition.—A condition excited by the extension of a simple gastro-duodenal catarrh to the common bile-duct, with temporary obstruction, as the result of swelling of the lining mucous surface of the duct. There is also jaundice of the skin and viscera.

The **exciting factor** is, as a rule, extension of a catarrhal process from the duodenum.

Among the **predisposing factors** are: (1) Dietetic errors—(*a*) Overeating and the eating of rich and improperly cooked foods; (*b*) rapid eating, which necessitates imperfect mastication; (*c*) the excessive and prolonged use of such irritating beverages and stimulants as alcohol, strong coffee, and tea. (2) Exposure to cold and wet. (3) Overwork, mental anxiety, and undue mental strain. (4) The condition may develop during the course of acute infections, *e. g.*, typhoid fever, pneumonia, etc., but the

jaundice of malaria seldom belongs to this particular type. (5) Rarely, indeed, epidemic outbreaks of catarrhal jaundice have been reported; and (6) interference with the portal circulation, such as is seen in chronic heart, lung, and kidney conditions, may contribute toward the development of catarrhal jaundice.

Principal Complaint.—*Gastric disturbance* is present for two or more days prior to the development of jaundice, and consists of anorexia, inordinate thirst, fullness over the abdomen, slight discomfort in the region of the liver, and flatulence. Vomiting may also be present.

Diarrhea, lasting from a few hours to as many days, may appear early. *Constipation*, however, is the rule. Following the initial symptoms the patient exhibits malaise, general weakness, stupor, and a tendency to sleep during the day, whereas at night he is often wakeful.

Thermic Features.—The temperature may be normal at the time jaundice is detected, although prior to its development the fever is that of catarrhal gastritis. (See p. 483.) Until the pigmentation begins to disappear, there may be a temperature ranging between 99° and 101° F. A high temperature is suggestive of approaching complications.

Physical Examination.—When the patient is examined by daylight the first evidence of the presence of jaundice is a slight yellowing of the conjunctivæ, which discoloration rapidly deepens to a bright lemon yellow; when jaundice persists for two or more weeks, the skin and conjunctivæ may display a greenish tint.

The signs, symptoms, and general characteristics of the conditions accompanying jaundice are described under the general consideration of jaundice (see p. 605), and are, as a rule, present in simple catarrhal jaundice. The essential fact to be borne in mind is that the characteristic features of jaundice vary in severity, depending entirely upon the duration of the attack.

Illustrative Case of Catarrhal Jaundice.—Jacob G., male, aged twenty-two years; height, 5 feet 5½ inches; weight, 138 pounds.

Family History.—Father and mother living at the ages of fifty and fifty-two years respectively. An older brother and two younger sisters living and in good health. One maternal uncle died of tuberculosis at the age of twenty-eight, and a younger sister was afflicted with chorea at the age of nine, from which she made a perfect recovery.

Previous History.—The patient had the usual diseases of childhood, also diphtheria at the age of nine, and for some years following this he was subject to attacks of sore throat which lasted for from one to three days. He does not recall having consulted a physician since the age of seventeen.

Social History.—Single, a clerk by occupation, but gets abundant outdoor exercise. Has an exceptionally well-trained voice, and frequently sings at concerts and entertainments. Never finds his work irksome, and states that he frequently dines late at night, and at such time partakes liberally of rich foods and of wines. Uses tobacco in moderation.

Present illness began two days ago, and followed a meal which included salads and more alcoholic beverages than usual. When seen the next morning, his symptoms pointed to acute gastritis, and he stated that he had vomited several times during the night. There were also present epigastric distress, mental dullness, headache, and hebetude. The following day he returned to his work, although he complained of feeling unusually dull during the day, and was compelled to return to his room. The next morning he again attempted to work, but was unable to complete the day, and upon the morning of the third day he again consulted his physician, who pronounced his illness catarrhal jaundice secondary to acute gastritis.

There was a mild degree of pain in the epigastrium during the acute attack of gastritis, which disappeared during the first day of his illness. From the second to the sixth days there was a sense of oppression over the right hypochondrium.

The patient was unable to sleep well during the night although during the day,

and particularly in the afternoon, he became drowsy. There was mental hebetude, and this was especially noticeable when he attempted to read, and he stated that he was unable to concentrate his thoughts even when reading the daily papers. He was more or less irritable, and worried continually about his work.

When first seen during the acute attack of gastritis, the temperature was 102° F., but fell to 99° F. during the next twenty-four hours, following which it continued to fluctuate between 99° and 100° F. for a period of four days, when it gradually fell to normal.

Physical Examination.—*General.*—On the fourth day the skin showed slight yellowing, which pigmentation increased rapidly for a period of three days. The patient perspired freely, and at times sweating was so profuse during the night that he found it necessary to change his clothing. The bed-linen and clothing were also bile-stained by the perspiration.

Local Examination.—Inspection.—When first seen, the patient's eyes were markedly congested, the cheeks were slightly flushed, and the face was somewhat drawn. The body was well nourished. When seen on the morning of the fourth day following the acute attack of gastritis, the conjunctivæ were jaundiced—almost of a golden yellow hue. The tongue was heavily coated, and the buccal cavity contained an abnormal quantity of saliva.

Palpation.—During the attack of acute gastritis there was distinct tenderness on making pressure over the epigastrium. From the third to the sixth day of his illness he complained of tenderness over the liver, although this organ could not be felt below the costal margin. The pulse was full and of moderate tension, the beats numbering 55 to 65 a minute; the patient stated that during health his pulse was 75 to 85 a minute.

Percussion.—The area of hepatic dullness was apparently normal, except that portion occupied by what appeared to be a distended gall-bladder, where the dull note was obtained for a distance of from 1½ to 2 inches below the costal margin.

Laboratory Findings.—The urine was bile-stained and gave a questionable reaction for albumin. Microscopically, the epithelial cells and leukocytes were bile-stained. The feces were clay colored, emitted an unusually offensive odor, and microscopically many fat-cells were seen.

Course of the Disease.—The prominent symptoms of acute gastritis disappeared by the second day following the institution of treatment. Jaundice, which was first detected on the fourth day, continued to increase for a period of approximately four days, and during this time the patient's general symptoms, referable to the jaundice, continued to increase. With the disappearance of the pigment from the skin and mucous surfaces the patient recovered and was able to resume his work by the end of the second week.

Duration.—The majority of all cases tend toward recovery by the end of the second week, although the condition may rarely continue for six to eight weeks. "If symptoms continue longer than two months, grave doubts may be entertained as to the case being one of simple jaundice" (Anders).

SUPPURATIVE CATARRH OF THE BILE-DUCTS.

Pathologic Definition.—A condition characterized by a secondary septic process involving the mucous surface of the bile-ducts and resulting in temporary obstruction. The skin, mucous surface, and viscera are also jaundiced.

Etiologic Factors.—(1) Direct extension of a suppurative process from one of the adjacent structures. (2) The entrance into the common duct of animal parasites such as the liver fluke is also regarded as an exciting cause. (3) The passage of gall-stones may have occasioned an initial inflammation that renders this a favorable site for infection. (4) Carcinoma of the common duct is a predisposing factor. (5) The condition may also follow operations upon the rectum, and acute sepsis and infections.

Summary of Diagnosis.—It is with great difficulty that one is able to make a positive diagnosis of suppuration of the bile-ducts, since in the majority of such cases there is likely to be a suppurative process of the gall-bladder (purulent cholecystitis). The symptoms and signs displayed by

suppuration of the bile-passages resemble closely those occurring in other forms of disease of the liver. A clinical history of sepsis elsewhere, or of an attack of gall-stone colic, when jaundice is present, together with high, irregular fever and marked prostration, are suggestive of the existence of a purulent inflammation of the mucous surface of the bile-ducts.

CHRONIC CATARRH OF THE BILE-DUCTS.

Pathologic Definition.—A condition characterized by chronic obstruction to the common bile-duct, with chronic inflammation of the mucous surface of the bile-passages. The mucosa of the ducts and the gall-bladder is covered by a heavy, mucoid exudate, and the epithelial cells are destroyed.

Predisposing Factors.—The predisposing factors are the passage of gall-stones, pressure from without, malignant disease, and stricture of the common duct.

Varieties and Characteristics of Each.—(1) In complete obstruction of the common bile-duct there is usually a history of repeated attacks of hepatic colic, and during some of these attacks there may have been distinct temporary enlargement of the gall-bladder. With distention of the gall-bladder there is increasing jaundice. The temperature remains approximately normal. Persistent enlargement of the gall-bladder is strongly suggestive of the extension of malignant disease, while intermittent enlargement indicates that gall-stones are the exciting factors.

(2) If incomplete or temporary obstruction to the common bile-duct is due to gall-stones, there are recurrences of paroxysmal pain, accompanied by chills, fever that rises abruptly to from 103° to 105° F., and falls rapidly to the normal, and profuse sweats. The paroxysms of incomplete obstruction to the common bile-duct resemble closely the paroxysms of malaria (quotidian, tertian, or quartan fever).

In certain selected cases jaundice is persistent, but in the majority of them it is intermittent, and its intensity is influenced by the length of time it has existed. Again, the patient becomes more and more jaundiced after each attack. The general clinical picture of incomplete obstruction to the common bile-duct is that described under Cholelithiasis.

CHOLELITHIASIS

(GALL-STONES; BILIARY CALCULI; CALCULOUS CHOLECYSTITIS).

Pathologic Definition.—A condition resulting from the pathologic precipitation of salts from the bile and the formation of calculi within the gall-bladder or the bile-ducts. There may also be a subacute or chronic inflammatory process involving the mucous surface of the gall-bladder and of the larger bile-ducts. At times pronounced inflammation results in an appreciable diminution in the size of the gall-bladder, the organ becoming thickened and shriveled.

Varieties.—Clinically, there are two great classes: (1) That in which there is no hepatic colic, and which probably comprises more than 50 per cent. of all cases; (2) that in which hepatic colic forms a most prominent feature.

Exciting Factors.—(*a*) Concentration of the bile, which results in a precipitation of its salts. (*b*) The belief is now quite general that the initial excitant is an infection of the gall-bladder with microörganisms, *e. g.*, members of the "colon group" of bacteria.

Predisposing Factors.—Age and Sex.—Although biliary calculi may form at almost any age, they usually occur during the fourth and fifth decades. Women are far more frequently subject to this condition than men, the ratio being approximately four to one.

Sedentary habits greatly predispose to the formation of gall-stones, as do also overeating of rich foods and excessive indulgence in alcoholic beverages. Inflammation of the inner surface of the gall-bladder may result from infection by the liver fluke in some of the adjacent gall-ducts. Again, disease of the gall-bladder may complicate typhoid fever, pneumonia, or other of the acute infections, and in such cases there is probably a specific infection of the gall-bladder which precedes the formation of an excess of mucus and favors precipitation of bile salts.

Constipation, chronic duodenal catarrh with partial obstruction of the common duct, pancreatic disease, and extrahepatic conditions (tumors, causing constriction of or pressure upon the common bile-duct) favor the development of gall-stones.

General Remarks.—Brockbank, in an analysis of over 13,000 post-mortem records, found gall-stones in 7.4 per cent. Probably the majority of those having gall-stones never display symptoms referable to this condition. Gall-stones may be unusually small, and escape through the common bile-duct without causing any irritation either of the common bile-duct or of the surrounding tissues. On the other hand, calculi may be too large to enter the common duct, and thus the symptoms of colic are absent. In those cases in which the gall-bladder is filled with one or more calculi the organ itself becomes greatly thickened and is often actually diminished in size.

Principal Complaint.—In the absence of hepatic colic the diagnosis is made largely from the clinical history and the physical signs. Given an individual over forty who complains of slight discomfort over the region of the liver and displays the following signs, one should always suspect the presence of cholelithiasis.

Physical Signs.—Inspection.—As a rule, the skin presents a slightly icteroid hue, with somewhat brownish circles beneath the eyes.

During the attack the patient usually writhes in pain, and lies with his chest bent forward and his thighs flexed well upon the trunk. His general appearance suggests agonizing intestinal colic, so that it becomes necessary to differentiate carefully between hepatic, renal, and intestinal colic. (See Differential Diagnosis, p. 612.)

Jaundice develops within forty-eight hours after an attack, when occlusion of the common bile-duct occurs. In those cases in which repeated attacks take place, jaundice will be intensified after each paroxysm. Slight prominence of the hepatic area may be present. The symptoms and signs characteristic of jaundice (p. 605) are also present in hepatic colic, and Fitz claims that jaundice is present in about 50 per cent. of all cases.

Palpation.—It is usually possible to elicit slight tenderness over the region of the gall-bladder, and by combined auscultation and palpation, when more than one stone is present, a distinct crepitus is often audible. Place the stethoscope over the margin of the gall-bladder, and while auscultating, carry the palpating fingers well underneath the costal margin, and endeavor to compress the gall-bladder. Gall-stone fremitus is a positive finding, and enables one to make the diagnosis irrespective of the presence of colic.

If the attack is mild, the pulse is stimulated, but during a severe attack of gall-stone colic the pulse becomes weak, rapid, small, and dicrotic. After

jaundice develops the pulse is practically that of jaundice, and will be found in uncomplicated cases to number between forty and sixty beats a minute.

The edge of the liver is felt beneath the costal margin, and distention of the gall-bladder is quite common.

Biliary Colic.—*Pain.*—When calculi become impacted or lodged within the hepatic duct, the cystic duct, or common bile-duct, there is agonizing pain, usually described as cutting, tearing, or boring in character. Pain always begins in the superior right abdominal quadrant, and when it becomes severe, radiates to the back, particularly to the right scapula and shoulder. In the majority of cases the patients are able to localize the seat of the pain, and in these it is two or three inches to the right of the median line, and probably two inches from the costal cartilages. Pain is occasionally localized to the region of the gall-bladder, and is then said to be a symptom of occlusion of the cystic duct; acute cholecystitis, however, will also give rise to pain in this region.

Hepatic colic often develops some hours after a meal, and it may rarely appear with such suddenness and severity as to produce a state of collapse within a comparatively short period. When the calculus escapes from the common bile-duct, pain immediately subsides. In those cases in which the common bile-duct is occluded for a prolonged period, the pain may abate for a time, but does not completely disappear. When the pain is severe, vomiting is especially likely to occur.

Chills.—An attack of gall-stone colic is frequently ushered in with a severe rigor, and during and after the chill the temperature will be found to have risen abruptly to 101° or even 102° F. It should be remembered that hepatic calculi may pass through the common bile-duct and enter the duodenum without giving rise to any symptoms, unless, perhaps, they set up sufficient irritation to cause cholecystitis.

Repeated attacks are the rule, but a history of previous attacks is in no way suggestive of the severity of the present attack.

Sweating.—Following the chill and fever there is a drenching sweat, during which the patient is likely to fall asleep.

Laboratory Diagnosis.—The *feces* are clay colored, and emit a very offensive odor. Gall-stones may escape through the common bile-duct into the intestine, or through fistulous communications between the intestine and the gall-bladder. Hepatic calculi, varying in size from that of a kernel of corn to that of a walnut, have been discovered in the feces.

The *urine* is bile-stained, and frequently contains serum-albumin and casts. Glucose may be present in small amounts; Exner asserts that he found it present in 39 of 40 cases examined. It may here be stated that the copper or Fehling test is unreliable when bile acids are present in the urine. Personally, we have never seen a case of cholelithiasis in which glycosuria was present, and the observations of Fausch, which include a study of 85 cases, showed glycosuria to be present in but one of them. The *gastric contents* may show an excess of free hydrochloric acid.

Summary of Diagnosis.—Owing to the obscure clinical features displayed by those cases in which hepatic colic is not present, a diagnosis is made with extreme difficulty. The presence of tenderness over the region of the gall-bladder and the eliciting of gall-stone crepitus are most valuable in attaining a diagnosis.

When the calculus becomes impacted in the duct and the general clinical picture is that of biliary colic, the diagnosis is based on—(*a*) the presence of colic-like pain in the epigastrium, radiating to the shoulder; (*b*) the develop-

ment of jaundice within seventy-two hours; (*c*) the characteristic fever; and (*d*) the recovery of hepatic calculi from the feces.

Differential Diagnosis. — Hepatic colic may be confused with practically any condition in which paroxysmal pain in the abdomen occurs. It is readily distinguished from **uterine colic,** since the later recurs with distinct periodicity and always antedates or develops during menstruation.

The **colic of lead-workers** can readily be distinguished from hepatic colic by a history of employment that necessitates the handling of lead. The lead line on the gums, the presence of lead in the urine, marked anemia (with basophilic degeneration of the red cells), and obstinate constipation are pathognomonic of chronic plumbism.

Gastralgia is seen only in neurasthenic individuals, and its characteristic features are: (*a*) Paroxysmal pain in the epigastrium, encircling the base of the chest, and extending to the back; (*b*) the pain begins when the stomach is empty; (*c*) it is relieved by the taking of food and by making firm pressure over the epigastrium; (*d*) it is an afebrile condition, and (*e*) there is an absence of jaundice. These features are all widely different from those found in hepatic colic.

Intestinal Colic.—Here there is usually a history of dietetic errors, as, *e. g.*, of overindulgence in unripe or decomposing fruits. The pain at first extends over the entire abdomen, but soon becomes localized to the umbilicus (not to the hepatic region), and in contradistinction to that of hepatic colic, it does not radiate to the right shoulder. Abdominal distention is quite common; vomiting is profuse, and the vomitus contains undigested food. Diarrhea may develop during the attack, and the general clinical picture may be that of cholera morbus (p. 548). Characteristic fever is absent.

Course.—As a rule, the attacks recur. Cardiac palpitation is present, and may be followed by circulatory collapse. If infection of the parts follows an attack, the case becomes one of purulent cholecystitis.

Complications.—**Rupture of the common bile-duct** is a rare but serious complication, and is usually followed by extensive peritonitis. **Localized peritonitis** may result from direct extension of the infectious process, but is seldom fatal.

Intestinal obstruction may result from impaction of the cecum with hepatic calculi, and fistulous communications between the gall-bladder and the colon are also among the rare findings. **Hepatic abscess,** when complicating cholelithiasis, immediately becomes of serious moment.

TABLE SHOWING THE DISTINCTIVE FEATURES BETWEEN HEPATIC COLIC, RENAL COLIC, AND APPENDICITIS.

Hepatic Colic.	Renal Colic.	Appendicitis.
1. A history of having recovered gall-stones from the feces at previous attacks.	1. Possible history of having passed calculi with the urine.	1. Negative.
2. Attack begins some hours (two to four) after a full meal.	2. Not influenced by eating.	2. Pain may develop at any time.
3. Paroxysmal pain, radiating to the scapula or right shoulder.	3. Pain radiates along the course of the ureter, and patient may feel the site of pain getting lower and lower as the stone passes toward the bladder.	3. Pain at first over entire abdomen, but later becomes localized at McBurney's point.

Hepatic Colic.—(*Continued.*)	Renal Colic.—(*Continued.*)	Appendicitis.—(*Continued.*)
4. Pain disappears abruptly.	4. Disappears abruptly.	4. Lessens gradually.
5. Tenderness in the region of the gall-bladder and in the epigastrium at right of the median line.	5. Tenderness may be elicited along the course of the ureter, but is often absent.	5. Tenderness at McBurney's point, and rigidity of the right abdominal wall.
6. Temperature rises abruptly to 100° to 102° F., and falls by crisis.	6. Temperature resembles closely that of hepatic colic, except in severe cases, when it becomes subnormal.	6. Temperature 99° to 101° F.; irregular in type.
7. Chill antedates the pain.	7. Chill less common.	7. Absent.
8. Jaundice usually develops within the first twenty-four to forty-eight hours.	8. Absent.	8. Absent.
9. Urine bile-stained.	9. May be bloody or contain calculi.	9. Indicanuria.
10. Calculi may be passed with the feces.	10. Absent.	10. Absent. Constipation the rule.

CARCINOMA OF THE BILE-DUCT.

Pathologic Definition.—An epitheliomatous process, with primary involvement of the mucous membrane of the common bile-duct. There is a tendency toward metastasis to the liver and adjacent viscera, and the disease may spread by direct extension to the gall-bladder.

Predisposing Factors.—Among the predisposing influences are **mechanical irritation,** such as is produced by the passing of gall-stones through the duct. Confirmatory of this are the researches of Osler, who found that seven of every eight persons dead of carcinoma of the gall-duct had suffered from cholelithiasis.

Heredity is not without effect, and the disease is seldom seen in persons before the forty-sixth year.

Sex.—Carcinoma of the gall-duct is more common in females than in males, the ratio being as four is to one.

Principal Complaint.—A clear history of previous attacks of hepatic colic is usually given. These attacks may have continued over a period of years. When there is only partial obstruction of the common bile-duct, the patient may display some of the features of *jaundice,* although distinct pigmentation of the skin is not an essential factor at this stage. Epigastric distress, a sense of fullness in the region of the liver, and vague pains in the hepatic region are generally experienced, although none of these symptoms is characteristic.

Physical Signs.—Inspection.—Evidences of emaciation and cachexia are seen early. Jaundice, with its characteristic cutaneous and scleral pigmentation, develops early, and persists throughout the disease. Abdominal distention occurs as a late symptom in those cases in which ascites develops.

Palpation usually reveals the fact that the gall-bladder is somewhat enlarged, but not tender, and the peculiar doughy mass felt to protrude beyond the costal margin is quite characteristic of obstruction of the common bile-duct. The surface of the liver may become nodular from metastatic growths.

Laboratory Diagnosis.—The urine is early colored with bile-pigment,

as are all its organic sediments. When obstruction becomes complete, there is an absence of bile-pigment in the feces.

Clinical Course.—This disease runs a less rapid course than does carcinoma developing elsewhere, although after metastasis to the liver has taken place it assumes a more rapid course.

OBSTRUCTION AND STENOSIS OF THE BILE-DUCT.

Obstruction and stenosis of the bile-duct have been considered at length on p. 605; both conditions are characterized by jaundice.

DISEASES OF THE PORTAL VEIN.

THROMBOSIS.

Pathologic Definition.—A condition resulting in occlusion of the lumen of the portal vein by an organized blood-clot. Ascites, general venous stasis, hemorrhage into the mucous surfaces, and jaundice are present.

Predisposing Factors.—This rare condition may have an obscure etiology, although its development is favored by traumatism to the abdomen, hepatic cirrhosis, carcinoma, and pressure from tumors within the liver or other abdominal growths (enlarged glands, abscess, carcinoma, etc.). Rarely does thrombosis follow ulcerative processes of the intestine, as, *e. g.*, appendicitis, tuberculous enteritis, and catarrhal dysentery. Impaired circulation through the liver, as is seen in valvular heart disease, and those blood conditions characterized by splenic enlargement, are also believed to favor the formation of a clot in the portal vein.

Principal Complaint.—In the majority of instances the symptomatology of thrombosis is indefinite, and a diagnosis is made only with extreme difficulty. In many cases the symptoms are slight, the patient complaining only of general weakness and of slight gastric and intestinal disturbances.

Complete Occlusion of the Vein.—Here the patient early manifests *hemorrhage* from the mucous surfaces; thus the vomiting of blood is quite common, as is also hemorrhage from the bowel. *Jaundice* and the many symptoms that accompany it develop early, and continue through the course of the disease. There are anorexia, general weakness, and, possibly, diarrhea. *Nervous symptoms*, as, *e. g.*, delirium, stupor, and coma, appear when the disease is at its height.

Physical Signs.—Inspection.—The abdomen may be somewhat distended; the skin and conjunctivæ are jaundiced, and the expression is anxious.

Palpation.—The liver is found to extend perceptibly below the costal margin, and firm pressure over the organ elicits tenderness. There is also splenic enlargement. In those cases complicated by ascites, movable dullness and fluctuation are present.

Sequelæ.—When thrombosis is due to the presence of septic emboli, abscess-formation generally occurs.

PURULENT PHLEBITIS.

Pathologic Definition.—A purulent inflammation of the portal vein or of its tributaries, following ulcerative processes in the abdomen or infection through the umbilical cord of the new-born.

Etiology.—Phlebitis of the portal vein follows purulent appendicitis

probably oftener than any one other preëxisting condition. It has also been known to develop during the course of pyemia, tuberculosis of the intestine, typhoid fever, dysentery, and ovarian abscess, and to follow infection through the umbilical cord.

Principal Complaint.—The symptoms are, as a rule, obscure, the diagnosis being based largely upon the existence of an actively septic process. *Pain* is usually present, but is in no way characteristic. In a case recently under observation at the Philadelphia Hospital intense pain was experienced in the epigastrium, and was reflected over the upper portion of the abdomen.

Chills are likely to occur every twenty-four hours, and are always followed by a rise of temperature and by profuse sweating. *Nausea* and *vomiting* develop early, and may continue throughout the attack. *Diarrhea* is not uncommon.

Nervous Symptoms.—Stupor and low muttering delirium are present from the first day, and with the advance of the disease the delirium becomes maniacal. Coma is a grave symptom, and continues until death.

Thermic Features.—The temperature is irregular, ranging between 99° and 104° F., but as the disease advances, it commonly assumes the continued type.

Physical Signs.—Inspection.—The skin is jaundiced, but assumes more of a muddy hue than that of distinct icterus. The upper portion of the abdomen is slightly prominent. The tongue is heavily coated, the teeth are covered with sordes, and the lips are parched and often fissured.

Palpation.—The pulse soon becomes thready, numbering 120 to 140 beats a minute, and as the disease advances, it grows, weak, dicrotic, and compressible. The liver may or may not extend below the costal margin. In those cases in which abscess exists the liver is distinctly enlarged. There is also some tenderness over the upper portion of the abdomen, and splenic enlargement is the rule.

Percussion shows an increased area of splenic dullness, which is best detected in the axillary line. The area of liver dullness may be increased.

Laboratory Diagnosis.—Examination of the blood shows the number of leukocytes to be between 12,000 and 30,000 in a cubic millimeter, and this increase concerns chiefly the polymorphonuclear cells. Cultures from the blood may show the presence of pathogenic bacteria, as was recently observed in two cases under observation at the Philadelphia Hospital.

Course.—All cases terminate in death in from seven to twenty-eight days.

ICTERUS NEONATORUM.

Pathologic Definition.—A condition brought about by alteration in the blood-pressure in the hepatic vessels, by destruction of red blood-corpuscles, or by congenital obstruction of the common bile-duct.

Varieties.—(1) The so-called physiologic variety is often referred to as the simple type, and follows ligation of the umbilical cord. (2) The pathologic variety is usually dependent upon a congenital deformity in the hepatic ducts or upon syphilitic hepatitis, and under this heading should also be considered septic phlebitis following infection of the umbilical cord.

General Remarks.—By some a variable degree of icterus following ligation of the umbilical cord in the new-born is regarded as a physiologic process. Jaundice develops by the end of the third day after birth, and the cutaneous phenomena resemble those of jaundice in the adult. The urine is not bile-stained unless the degree of jaundice is extreme, and the feces are of

the normal color, since there is no obstruction to the escape of the bile into the duodenum.

The severe pathologic and congenital types are of grave prognostic moment. Jaundice becomes extreme by the third to the fifth day after birth. The stools are clay colored, and a fatal termination is the rule.

THE PANCREAS.

METHODS OF EXAMINATION.

Clinical Remarks.—The great difficulty with which disease of the pancreas is recognized antemortem is doubtless responsible for the small space allotted to this subject in works on diagnosis and general medicine. Recently, the efforts to establish a method for the recognition of pancreatic disease have been limited to laboratory research, and for this reason the various laboratory methods employed to further the diagnosis of pancreatic disease, and the clinical significance of such findings, will be described here.

Fatty Stools.—Either constipation or diarrhea may be present in disease of the pancreas. The stools often contain fat-globules, which give to the mass of dejecta a glazed or greasy appearance. Microscopically ($\frac{1}{6}$ to $\frac{1}{8}$ inch objective), the field shows many oil-globules. If further proof is desired, a drop of a saturated alcoholic solution of Sudan III is allowed to pass underneath the edge of the cover-glass, and as it mingles with the fat-globules contained in the stool, each globule coming in contact with the solution is stained a brilliant pink. Staining with a weak solution of osmic acid renders the oil-globules black. For practical purposes, we have found Sudan III the more reliable and convenient fat stain.

Undigested Fats.—The quantity of fat present in the stool in pancreatic disease is in direct relation to the amount of fatty food ingested, the inability to digest fats being one of the characteristic features of a pancreatic lesion.

Clinical Significance.—*Fat* in the stools occurs clinically in acute pancreatic hemorrhage, acute pancreatitis, pancreatic carcinoma, pancreatic cyst, when the organ is firmly compressed, pancreatic calculi, and acute and chronic pancreatitis.

Meat-fibers (beef, pork, etc.) are imperfectly digested when any condition, either mechanical or functional, that interferes with the escape of the pancreatic secretion into the duodenum exists. In certain cases of pancreatic disease we have found that a macroscopic study of the stool showed the presence of many particles of meat-fiber. Meat-fiber may be detected only microscopically, and there may be but few fibers present in the stool, even though the patient has ingested a meal rich in lean meats, therefore the following test is of great value:

Nucleus Test.—Schmidt's test, which was first described in 1904, is conducted as follows:

(1) Harden a particle of lean beef not larger than 0.5 cm. in diameter (or the size of a pea) in absolute alcohol for a few days.

(2) Then tie it in a small bag of Brussels net.

(3) Return to the alcohol until required for use.

(4) After washing in water for three hours permit the patient to swallow several of these small net bags, each containing a meat-ball.

(5) Eighteen hours later the net bags and their contained meat-balls are recovered from the feces.

(6) Wash the Brussels net thoroughly, open the bag, and transfer the meat-ball to clean water, where it is to be thoroughly washed and made ready for microscopic study.

(7) The outer layer of the meat-ball will, in nearly all instances, be found to be digested. Microscopically, the nuclei of the cells will be broken in health, but if pancreatic digestion is imperfect, the nuclei of the fibers occupying the interior of the meat-ball will not be destroyed.

(8) The meat-ball may be cut in half, and tissue obtained from the center of the ball teased thoroughly upon a slide and examined immediately, to detect the state of the nuclei present, or, as is possibly still better, though less practical, the entire meat-ball may be hardened and sectioned and a careful microscopic study made of the sections. Whatever course is pursued, the meat-ball must be removed from the Brussels net and studied within a short time after it is passed by the bowel, otherwise the test is valueless.

Caution.—Should the meat-ball pass through the intestinal tract too soon, the test will be valueless. In a healthy person digestion may be complete in from twelve to fourteen hours, but from eighteen to twenty hours are required to complete the process when testing for disease.

Clinical Significance.—The nucleus test is but fairly reliable as an aid in making a diagnosis of disease of the pancreas. If the nucleus test is positive, disease of the pancreas is likely to be present, and although, as stated, it is by no means infallible, it is probably one of the best tests at hand.

The Cammidge Reaction.—In 1904 P. J. Cammidge described a test for the detection of pancreatic disease, which is commonly known as the Cammidge reaction. The test consists in boiling the filtered urine with hydrochloric acid, and, after treatment with lead salts and the removal of these salts, heating with phenylhydrazin hydrochlorid, and examining the sediment for the occurrence of rosets of yellow, needle-like crystals. Cammidge originally described two reactions, which he called the A and B tests; but later modified his method and proposed a reaction which he calls the C test, which is now generally used. Cammidge originally thought that glycerose was the substance which produced the osazone crystals; but he abandoned this view, and later advanced the opinion that a pentose formed from some "antecedent substance" in the urine by boiling with hydrochloric acid gave rise to the crystals.

Method.—The urine is first shown to be free from albumin and glucose. In determining the absence of the latter ingredient the test with Fehling's solution is not accurate enough, and the phenylhydrazin hydrochlorid test should be employed. If albumin is present, it should be removed by the addition of acetic acid, by boiling, and filtering. If the phenylhydrazin hydrochlorid test gives a positive reaction, the glucose should be removed by fermentation.

Forty cubic centimeters of filtered urine known to be free from albumin and glucose, and 2 cubic centimeters of strong hydrochloric acid (sp. gr. 1.16), are placed in a small Erlenmeyer flask, in the mouth of which a small funnel is placed to act as a condenser, and boiled on a sand-bath for ten minutes from the time the first bubbles are observed. The flask is then cooled in running water and 8 grams of lead carbonate are slowly added to neutralize the acidity. After standing for a few minutes so as to allow the reaction to

be completed, the flask is again cooled in running water and its contents filtered through a well-moistened, close-grained filter-paper, until a perfectly clear filtrate is obtained. The filtrate is then treated with 8 grams of tubaric lead acetate and the resulting precipitate is removed by filtering through a moistened filter-paper. Repeated filtration should be done if the first filtrate is cloudy. The lead now in solution in the fluid is next removed by adding 4 grams of powdered sodium sulphate, bringing the contents of the flask to the boiling-point, and filtering after cooling in running water. The lead may also be removed by passing a stream of sulphuretted hydrogen through the fluid and filtering. The former method precipitates the lead as a sulphate, the latter as a sulphid. Ten cubic centimeters of the filtrate resulting from the last operation are made up to 17 cubic centimeters with distilled water and 0.8 gram of phenylhydrazin hydrochlorid, 2 grams of sodium acetate, and 1 cubic centimeter of 50 per cent. solution of acetic acid are added. The mixture is placed in a small Erlenmeyer flask fitted with a funnel condenser and the whole boiled on the sand-bath for ten minutes from the time of the appearance of the first ebullition. The hot fluid is filtered through a filter-paper previously moistened with hot water into a test-tube provided with a 15 c.c. mark. If the filtrate falls short of the 15 c.c. mark, it is made up to that point with distilled water and the two fluids carefully mixed. A stopper is then put in the tube and the tube placed in an upright position in a test-tube rack overnight, or until the solution is cool, preferably the former. The sediment is then pipetted off and examined with the $\frac{1}{6}$-inch dry lens. A positive reaction is indicated by the presence of rosets and sheaves of delicate, long, yellowish, needle-shaped crystals. These crystals curl up at the ends and have a tendency to branch.

Clinical Significance.—The Cammidge crystals have been found in the urine of patients suffering from diseases of the pancreas by numerous observers; they have been found in the urine of dogs in which pancreatitis has been induced by experimental methods; they have been found in numerous other diseases. In a recent clinical study of the subject by Swan and Gilbride, based on 120 examinations in 53 cases, the conclusion was reached that it was a mistake to interpret every positive Cammidge reaction as indicative of organic disease of the pancreas. That it was not to be depended upon as pointing to the necessity for an operation. If the patient complained of upper abdominal symptoms, particularly pain, and the reaction was positive, it might be considered a link in the chain of evidence pointing to pancreatic disease, disease of the gall-bladder, or of some neighboring organ. In the absence of upper abdominal symptoms a positive reaction was looked upon as merely an interesting discovery.

DISEASES OF THE PANCREAS.

ACUTE HEMORRHAGIC PANCREATITIS.

Pathologic Definition.—A disease probably excited by infection of the pancreas with bacteria, and characterized by both circumscribed and diffuse hemorrhagic infiltration into the substance of the organ. There may also be thrombosis of the pancreatic veins and round-celled infiltration involving the interlobular tissue. Hemorrhage may extend to the adjacent structures, and hyperemia and ecchymoses are at times seen upon the gastric mucous membrane. In selected cases there is evidence of localized peritonitis, and disseminated fat necrosis is commonly associated with it.

Exciting Factors.—The exciting factor in man is not definitely determined. Experimentally, Flexner found that by injecting acids and alkalis into the duct of Wirsung, acute pancreatitis resulted, and the same investigator found, that by injecting cultures of pathogenic bacteria, a similar result ensued. These experiments strongly suggest that the disease is probably of microbic origin.

Predisposing Factors.—Age figures prominently—nearly all cases have been seen in persons over forty. Those having suffered from gastroduodenal catarrh, from chronic dyspepsia, or from diabetes, appear to be most susceptible to this type of disease. Traumatism over the epigastrium is not only a predisposing but a probable exciting factor in certain cases. Pancreatic hemorrhage may give rise to pancreatitis, and acute infectious fevers also predispose to the development of acute pancreatitis.

Principal Complaint.—There is usually a history of chronic gastrointestinal disturbances or of traumatism to the abdomen. The development of symptoms is *sudden*, and among them should be mentioned *violent deep-seated pain*, oftenest limited to the epigastrium, but which may be as low as the umbilicus. Accompanying the extreme pain are *nausea*, *violent retching*, and *vomiting*. Intermittent pains, when present, are generally conceded to be the result of associated gall-stone colic. Hiccough may develop early, and be a prominent and annoying symptom, although in other cases it may be absent. The patient may be conscious, or there may be a variable degree of delirium. Labored respiration is an early feature, and, in fact, dyspnea may persist throughout the attack. The patient may faint while occupied with his usual duties, and there is also a rapid tendency toward collapse. Constipation is the rule, although diarrhea may be present.

Thermic Features.—A high temperature is seldom seen, whereas moderate fever is the rule; the temperature may, however, reach 103° or 104° F. During the stage of collapse the temperature may become subnormal.

Physical Signs.—**Inspection.**—The patient is usually found in the recumbent posture, or the chest may be tilted slightly forward and the legs flexed upon the abdomen. The expression is that of agony, and within a few hours the cheeks become sunken, the features distorted, and the expression anxious. In those cases in which dyspnea and cardiac failure are prominent, there is cyanosis, which is most apparent in the lips, ears, and fingertips, and, in fact, the face may display a peculiar dusky hue. Abdominal distention is occasionally present. Jaundice, when present, is usually dependent upon the presence of gall-stones.

Palpation.—Localized tenderness is often elicited in the epigastrium, and slight rigidity of the muscles overlying this area is not uncommon. The entire abdomen may be somewhat tense as the result of tympanites. The pulse becomes rapid early, and as the condition progresses, it becomes small, weak, dicrotic, and compressible.

Auscultation.—As the disease progresses, the heart-sounds, in addition to being rapid, may lose their muscular quality.

Laboratory Diagnosis.—The *vomitus* at first contains the contents of the stomach, and later may be made up almost entirely of mucus. Blood-stained material is often ejected.

Diarrhea may be present, the stools being watery in consistency and containing particles of fat.

The quantity of *urine* excreted may be normal or slightly decreased. Glycosuria is an occasional feature, but in such cases it is necessary to elim-

inate the existence of this condition prior to the development of the present attack. Albuminuria is the rule, although it is not a constant feature. Cammidge's reaction is positive.

Summary of Diagnosis.—In all cases the diagnosis is made with difficulty, the symptoms of hemorrhagic pancreatitis closely resembling those found in other pathologic abdominal conditions. The age of the patient (after middle life), the history of previous dyspepsia or of diabetes, and a possible history of traumatism to the abdomen are of considerable importance. Most characteristic, however, is the sudden onset, the deep-seated epigastric pain followed by nausea, vomiting, and circulatory collapse. Cammidge's reaction and the presence of fat in the feces are also to be considered.

Differential Diagnosis.—Obstruction of the Bowel.—When the obstruction occurs in an aged person, the distinction is made with difficulty. (*a*) Acute intestinal obstruction is more common in the young than in the aged, and the pain is less definitely localized than that of pancreatitis. (*b*) Abdominal distention is more marked in intestinal obstruction. (*c*) Fecal vomiting, a characteristic feature of obstruction, is absent in acute hemorrhagic pancreatitis. (*d*) The temperature is normal at the onset, but soon becomes subnormal in obstruction; (*e*) indicanuria is a somewhat constant symptom in obstruction.

Acute Gastroduodenal Catarrh.—(1) In this condition there is a distinct rise in the temperature at the onset. (2) The symptoms are not sudden and the pain is of a different character—not deep-seated or localized, as is the case in acute inflammation of the pancreas. Cammidge's reaction is negative, as is also an examination for fat in the feces. There is not the same degree of prostration and as marked a tendency toward circulatory collapse as are characteristic of acute pancreatitis.

Clinical Course.—This is, as a rule, rapid, the condition going on from bad to worse. Although surgical treatment is said to be of value, the majority of cases terminate fatally between the second and the fourth days.

SUPPURATIVE PANCREATITIS.

Pathologic Definition.—A disease characterized by acute or subacute inflammation of the pancreatic substance, terminating in suppuration. Suppuration may be diffuse, with the formation of numerous minute abscesses, although it is more common to find a single abscess within the head or body of the organ. There is generally extensive destruction of the pancreatic tissue, and the inflammatory process may extend to adjacent structures. Disseminated fat necrosis is occasionally present, and both hepatic abscess and pylephlebitis are seen at times.

Predisposing Factors.—The causal factor may be acute hemorrhagic pancreatitis that has gone on to suppuration. Extreme prostration sets in early, and there is progressive loss of flesh, together with a peculiar dusky pallor or slight jaundice.

Thermic Features.—The fever is irregular, ranging between 99° and 102° to 104° F.

Physical Signs.—Inspection.—The expression is anxious, the skin is jaundiced, and the abdomen is slightly distended.

Palpation.—Tenderness over the epigastrium is commonly present, and pressure usually excites pain. A distinct mass may occupy the epigastric region. Both tenderness and pain are usually limited to the left side of the median line, and abnormal tension of the abdominal muscles is the rule. The spleen is often palpable.

Summary of Diagnosis.—The character and localization of the pain, the irregular temperature, the presence of emaciation and prostration, together with hiccough and vomiting, point strongly toward the existence of pancreatic abscess. Fatty stools at times constitute the only symptom that points directly toward pancreatic disease, and in many cases it is impossible to formulate a diagnosis without knowledge upon this clinical point; indeed, the diagnosis of suppurative pancreatitis is, as a rule, founded upon general, rather than upon accurate scientific, principles.

Clinical Course.—In a small percentage of cases the disease assumes a chronic course, the patients living for weeks or even months; if, perchance, the pus should make its escape into the stomach or intestine, recovery may follow. Most cases, however, terminate in death by the end of the first week.

GANGRENOUS PANCREATITIS.

Pathologic Definition.—This condition is usually the terminal stage of the affection, and resembles that described for acute hemorrhagic pancreatitis. The majority of, if not all, cases terminate in death. There are no known clinical methods by which we are able to recognize the existence of gangrenous pancreatitis antemortem.

CHRONIC PANCREATITIS.

Pathologic Definition.—A chronic inflammatory process characterized by involvement of the substance of the pancreas, and resulting in an abnormal formation of fibrous tissue. The glandular substance may be almost obliterated. Owing to pressure upon the small ducts, small cysts may form. Interstitial hemorrhages are occasionally present, and extensive adhesions may surround the organ. In the chronic suppurative type of the disease small circumscribed abscesses exist.

Principal Complaint.—This is in no way characteristic of the disease in question, and the early symptoms of chronic pancreatitis resemble those of chronic dyspepsia. The patient gives a history of chronic gastric catarrh, with more or less periodic outbreaks of diarrhea. After the condition has become well advanced, he may complain of deep-seated epigastric pain, which is often paroxysmal. During these paroxysms the patient becomes unusually anxious; he may have attacks of faintness, and express a sense of great fear. Emaciation is rather rapid, and weakness is progressive.

Thermic Features.—During the attacks of pain moderate fever may be present.

Physical Signs.—**Inspection.**—Cachexia develops within the course of a few months, and continues throughout the disease. There is evidence of emaciation, and the skin overlying the abdomen may be peculiarly wrinkled.

Palpation.—Deep pressure elicits the fact that there is moderate tenderness over the epigastrium, although a distinct mass is seldom palpable.

Laboratory Diagnosis.—The feces contain fat, and the nucleus test may be positive.

The urine is often bile-stained, and may contain fat. Cammidge's reaction is positive. Glycosuria is fairly common, and, in fact, true diabetes mellitus may be a feature of chronic pancreatitis.

Summary of Diagnosis.—The general features of chronic pancreatitis, with intermittent attacks of diarrhea, following which periodic attacks of

deep-seated epigastric pain develop and are accompanied by fever, faintness, and anxiety, form a group of symptoms that strengthens the clinical picture. The presence of fat in the stools and of a positive Cammidge reaction in the urine, when taken in connection with the foregoing symptoms, points strongly toward the existence of chronic pancreatitis.

Clinical Course and Duration.—The condition runs a decidedly unfavorable course. The duration will be found to vary greatly, depending upon the portions of the pancreas affected. Autopsy has revealed the fact that extensive areas of sclerotic change may be present in the pancreas and the patient during life display no symptoms referable to pancreatitis.

PANCREATIC HEMORRHAGE (APOPLEXY).

Pathologic Definition.—A condition characterized by the presence of circumscribed areas of hemorrhage into the gland and adjacent tissues. The pancreas is often enlarged, although it may be of the normal size. In the region of the hemorrhage the organ is soft. Extensive hemorrhage may infiltrate the omentum and transverse colon, and invade the retroperitoneal fatty tissue and mucous membrane. Late, secondary reactive inflammation and necrosis are prone to occur.

Predisposing Factors.—Age serves as the most prominent predisposing factor, since the condition is seldom seen in persons before the fourth decade. About 75 per cent. of cases have occurred in males. Traumatism may be a direct cause. A history of chronic alcoholism is often obtainable. Some local vascular weakness or lesion (*e. g.*, necrosis) may operate as a cause. Severe *symptomatic* pancreatic hemorrhage may be secondary to acute pancreatitis and carcinoma.* Singularly, those apparently in perfect health may be attacked.

The **diagnosis** is based upon the suddennses of onset, with colicky pains in the upper portion of the abdomen, and, later, the occurrence of nausea and obstinate vomiting. Depression and, indeed, prostration become profound within a few minutes, and the patient expresses great fear of impending death.

Thermic Features.—Immediately following the attack, the temperature becomes subnormal, and remains at this point until death occurs; in some cases, however, the temperature approximates the normal during the greater portion of the attack.

Physical Signs.—Inspection.—The face is pinched, the expression is anxious, and the skin is pale and bathed in perspiration.

Palpation.—The pulse is weak, small, rapid, and compressible, and becomes imperceptible shortly after the onset of the attack.

Summary of Diagnosis.—A careful consideration of the clinical history, physical signs, and symptoms renders it possible for one to make a probable diagnosis of hemorrhage into the pancreas, although the clinical course of this condition is so rapid that the diagnosis is seldom made except at autopsy.

Clinical Course.—All cases terminate in death in from one-half to twenty-four hours. One of us, from a study of twenty-four cases, concluded that death was the immediate result of shock, or was caused by pressure upon the solar plexus.

* "Pancreatic Hemorrhage," Jour. Amer. Med. Assoc., December 2, 1899, by J. M. Anders.

PANCREATIC CARCINOMA.

Pathologic Definition.—A disease characterized by the presence of a primary carcinoma of the head of the pancreas. In the majority of cases sclerotic changes are present, with enlargement of the head of the organ. The carcinomatous process may extend to the surrounding tissues. Occlusion of the pancreatic duct may result in cystic formation. Carcinoma of the liver, stomach, and duodenum is occasionally found to complicate pancreatic carcinoma.

Predisposing Factors.—**Age.**—Aged men are affected more often than are aged women, but carcinoma of the pancreas is also occasionally found to occur during early adult life.

Principal Complaint.—This is in no way characteristic, and, generally speaking, the patient's complaint is practically the same in this disease as in pyloric carcinoma. As a rule, the patient gives a clear history of having suffered from chronic dyspepsia, accompanied by an appreciable loss in both strength and flesh. *Pain* in the region of the epigastrium is the rule, and may be deep, boring, and continuous in character, or it may be intermittent; the former, however, is the more common. Paroxysms of epigastric pain may develop at any stage of the disease, and during these attacks the patient often enters into a state of *collapse*. *Vomiting* may result either from acute pain or from an associated gastric catarrh, but is in no way characteristic.

Physical Signs.—**Inspection.**—Emaciation is pronounced, and there is usually slight distention of the upper portion of the epigastrium. If the tumor is situated well anteriorly, a distinct prominence is seen. Jaundice, due to obstruction of the common duct, is not unusual. The tongue is heavily coated.

Palpation.—Firm pressure over the region of the pancreas elicits pain if the head of the organ is involved, but in those cases in which the tail of the pancreas is the portion chiefly involved, pressure causes but slight pain, and possibly only discomfort. It is at times possible to palpate distinctly a mass in the epigastrium that is decidedly tender.

Results of Pressure.—(*a*) If pancreatic carcinoma causes sufficient infiltration to result in obstruction of the common bile-duct, jaundice results. (*b*) Pressure upon the portal vein, either from the mass itself or from surrounding adhesions, is likely to be followed by ascites. (See Signs of Ascites, p. 567.) (*c*) Because of the anatomic relation of the pancreas, pressure is often exerted upon the thoracic duct and results in the development of chylous ascites (p. 570). (*d*) From pressure upon the inferior vena cava dropsy of the lower half of the body and of the duodenum results, and as a consequence of the latter, the patient may display the symptoms of acute intestinal obstruction.

Laboratory Diagnosis.—The blood presents the changes of secondary anemia, *e. g.*, the red cells and hemoglobin are greatly reduced, and stained specimens show evidences of pallor and degeneration of the erythrocytes. (See Blood, p. 355.) Leukocytosis is often present.

Constipation is the rule, although intermittent attacks of diarrhea are present in nearly every case of pancreatic carcinoma; during these attacks the feces have a greasy appearance and contain a large amount of fat. If the pancreatic function is greatly interfered with, muscular fibers pass through the digestive tract without undergoing complete dissolution, and the nucleus test (p. 616) is positive.

In a large proportion of all cases the urine contains glucose, and a portion of the broken fat molecules is often present.

Summary of Diagnosis.—Rapid and progressive anemia, a deep, boring pain in the epigastrium, undigested muscle-fibers, and fat in the feces are among the early symptoms upon which the diagnosis of carcinoma of the pancreas is founded.

Later *jaundice*, with enlargement of the gall-bladder, the presence of a mass in the epigastrium, together with the absence of the gastric findings characteristic of pyloric carcinoma, further support a diagnosis of pancreatic carcinoma.

Differential Diagnosis.—At times it is extremely difficult to distinguish between carcinoma of the head of the pancreas and *carcinoma involving the transverse colon, the omentum,* or the *pyloric end of the stomach.* The accompanying differential table (modified from Anders) sets forth the clinical differences between carcinoma of the pancreas and carcinoma of the pylorus:

Carcinoma of the Pancreas.	Carcinoma of the Pylorus.
1. The tumor is deep-seated and fixed; later it becomes slightly movable. It is not associated with gastric dilatation.	1. Tumor is more freely movable, and is usually associated with dilatation of the stomach.
2. Symptoms of chronic dyspepsia are mild.	2. There are more marked gastric symptoms.
3. The vomitus is bilious in character and rarely contains blood.	3. "Coffee-ground" vomitus is the rule.
4. Free hydrochloric acid is present in the gastric fluid after a test-meal; lactic acid is absent.	4. Free hydrochloric acid is greatly decreased or absent. A decided reaction for lactic acid is the rule.
5. Oppler-Boas bacilli are absent from the gastric fluid.	5. Oppler-Boas bacilli are present in the gastric fluid, and blood-cells are common.
6. The stools contain undigested muscle-fibers and sometimes fat.	6. Tarry stools, when present, indicate hemorrhage.
7. Metastasis to the liver is unusual.	7. Common.

The distinction between *carcinoma of the pancreas* and *carcinoma of the colon* is made by inflating the colon, when, if the lesion is localized in the colon, the tumor is brought anteriorly and is readily palpable, moving with respiration.

Clinical Course.—The course of pancreatic carcinoma is extremely rapid, and may terminate fatally in from a few weeks to as many months. Removal of a portion of the gland is said to prolong life.

PANCREATIC CALCULI.

Pathologic Definition.—A condition believed to be caused by an inflammation or other morbid process that may give rise to an alteration in the secretory function of the pancreas, with the formation of calculi.

Summary of Diagnosis.—A colic-like pain is localized along the left costal margin, and usually radiates to the back and left shoulder. If the colic is severe, the pain may radiate over the upper half of the abdomen and to the right side of the body, and in such instances it is practically impossible to distinguish between this condition and that of hepatic colic. The detection of fat in the stools and of undigested meat-fibers and undestroyed nuclei, when considered in conjunction with the foregoing symptoms of colic, justify the presumption that pancreatic calculi exist.

Course.—The case usually terminates as one of chronic pancreatitis. (See p. 621.)

PANCREATIC CYST.

Varieties.—In classifying cysts of the pancreas from the standpoint of their etiology we have: (*a*) Those resulting from traumatism to the organ. Körte, in his analysis of 121 cases, found that 33 of them were dependent upon trauma. (*b*) Cysts following prolonged chronic inflammation. (*c*) Retention cysts, which depend upon occlusion of the duct of Wirsung by calculi or as the result of pressure from new-growths.

Predisposing Factors.—Age.—In Körte's analysis of 116 cases, 66 of them were between the ages of thirty and fifty. Cysts have been found in children during the first year of life.

Symptoms.—These are in no way characteristic of the condition in question. In the majority of instances pain is absent, although paroxysms of *colic* may be experienced. The *pain*, as in other forms of pancreatic disease, radiates to the left shoulder, in striking distinction to pain in hepatic disease, which radiates to the right shoulder. *Jaundice* and *ascites* commonly result from pressure upon the liver and upon the inferior vena cava. The *stools* may contain fat, and occasionally the patient complains of salivation. *Glycosuria* and *albuminuria* may be present, but neither of these is characteristic of the existence of pancreatic cysts.

Physical examination may disclose the presence of a smooth, fluctuating tumor in the epigastrium. Pancreatic cyst is oftenest located either above the stomach or immediately below the colon. The tumor moves but slightly with respiration, and there are recorded instances in which cysts of extreme size have occupied the upper portion of the abdomen. The development of pancreatic cyst is, as a rule, insidious, although a rapidly forming cyst is occasionally encountered.

Summary of Diagnosis.—The diagnosis is based almost entirely upon the physical signs. The presence of fat in the stools goes to support the findings obtained by physical examination.

Course.—Surgical interference materially modifies the course of the disease and may effect a cure.

THE SPLEEN.

TOPOGRAPHY.

Under normal conditions the spleen measures about three by five inches. Anatomically, it extends posteriorly to a point one and one-half inches to the left of the spine, and anteriorly to the axillary line. It is bounded above by the lower margin of the eighth rib, below by the eleventh rib, and its long axis is practically parallel with the ribs (Figs. 250 and 253). Posteriorly, the organ is in direct apposition to the top of the left kidney. Its superior surface is in contact with the diaphragm, whereas anteriorly and inferiorly it is in touch with a portion of the stomach, transverse colon, small intestine, and diaphragm (Fig. 249). The notch situated about the center of

the anterior border of the spleen is of great clinical significance in determining the size of the organ when this indentation is recognized by palpation.

Abnormalities in Position.—A floating spleen is the result of congenital lengthening of the ligaments suspending this organ, or of undue overstretching of such structures either by abnormal weight of the organ itself or by violence. This condition is found more commonly in women than in men, and frequently accompanies general visceroptosis. Floating spleen is detectable by palpation, and, unless the abdominal wall is unusually thick and tense, it is possible for one to outline the shape of the spleen and to return it to its normal position. (See Fig. 247.)

Clinical Significance.—In those cases in which there is a general displacement of the abdominal viscera, the prognosis is unfavorable as to cure, but favorable as to life. Surgical treatment may be of assistance in certain cases.

DISEASES OF THE SPLEEN.

DISPLACEMENT OF THE SPLEEN.

The spleen is often forced downward as the result of a left pleural effusion, tumor of the thorax, pyopneumothorax, and bilateral emphysema. In any of these conditions the edge of the spleen may be felt below the costal margin.

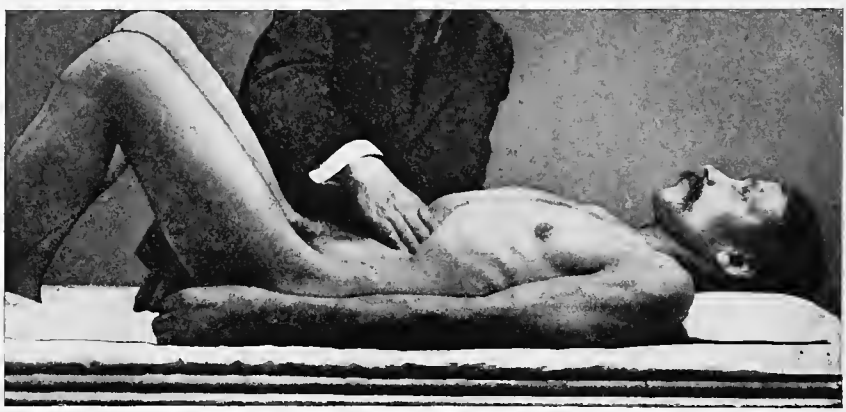


FIG. 247.—METHOD OF PALPATING THE SPLEEN.
When the operator's hand is in this position, the patient is directed to inspire deeply.

A diagnosis is readily attained from an analysis of the physical signs present over the left lung. The clinical importance to be attached to displacement of the spleen from pulmonary conditions is but slight.

In well-marked tympanites the spleen may be elevated above its normal position, and the same condition may result from extensive adhesive pleuritis or a fibrous tuberculous process involving the left lung.

ENLARGEMENT OF THE SPLEEN.

Causes for Chronic Enlargement of the Spleen.—The following classification, while it may in certain respects appear wanting, it will under average conditions serve as a guide for the determination of the character of enlargement present in the individual cases under consideration.

Pronounced enlargement of the spleen:

Splenomedullary leukemia.
Lymphatic leukemia (rare).
Mixed leukemia.
Chronic malaria.
Kala-azar.
Splenomegalic polycythæmia.
Splenomegalic cirrhosis.
Splenic anemia.
Pseudoleukemia infantum.
Amyloid disease.
Tricuspid regurgitation.

Moderate enlargement may be encountered in the following conditions:

Rickets.
Congenital syphilis.
Hodgkin's disease.
Cirrhosis of the liver.
Thrombosis of the portal vein.
Pressure on the portal vein by enlarged lymphatic glands or by adjacent tumor.

Acute enlargement of the spleen is observed during the course of the following acute infections:

Typhoid fever.
Paratyphoid fever.
Relapsing fever.
Ulcerative endocarditis.
Malaria.
Erysipelas.
Septicemia.
Puerperal sepsis.

Less often seen in:

Pneumonia.
Diphtheria.
Scarlet fever.
Small-pox.
Typhus fever.
Influenza.
General acute tuberculosis.

Among the pathologic conditions characterized by enlargement of the spleen are:

Amyloid Degeneration.—Diagnosis.—This is made from the physical signs of splenic enlargement, with or without decided increase in the size of the liver, although the latter condition is usually present. The history of conditions that lead to amyloid degeneration of the viscera, *e. g.*, chronic suppuration and syphilis, together with the symptoms and signs of amyloid disease of the kidney (p. 680) and the liver (p. 593), goes far to support the diagnosis and, in fact, often amply confirms it.

Clinical Course.—This, owing to the associated amyloid changes of the other viscera, is decidedly unfavorable. Occasionally, surgical treatment may remove the disease upon which the enlargement depends, after which the patient is greatly improved. In selected cases antisyphilitic treatment is also followed by improvement.

Leukemia.—Here there is enlargement of both the spleen and the liver, and not uncommonly the spleen is enlarged to a greater degree than the liver, although these viscera may increase in size simultaneously.

Diagnosis.—This is based upon an examination of the blood (see Leukemia, p. 368), although the physical signs, together with the general symptoms and signs of leukemia, must be taken into consideration in order to distinguish between splenic enlargement of leukemia and that of chronic sepsis.

Cyanotic Congestion Resulting from Valvular Heart Disease.—In valvular heart disease in which there is both tricuspid regurgitation and myocarditis, there is, first, a venous stasis of the liver, which results in an enlargement of this organ and materially interferes with the return circulation from the spleen. After hepatic enlargement has existed for some

time the spleen becomes enlarged, although to a much less marked degree than the liver.

Diagnosis.—This is based upon the general signs and symptoms of organic heart disease, together with the physical signs of enlargement of the liver and of the spleen. A feature of great importance in cyanotic congestion of the liver and of the spleen is that these organs are not tender upon firm pressure, and also that they decrease in size when the patient is placed in the recumbent posture and cardiac stimulants are administered.

Clinical Course.—This is practically the same as that of tricuspid regurgitation with myocarditis. (See p. 286.) Temporary improvement

FIG. 248.—TOPOGRAPHY OF LEFT LUNG, STOMACH, PLEURA, AND SPLEEN.

follows rest and the administration of cardiac stimulants. All cases grow worse gradually until general anasarca results.

Cirrhosis of the Liver with Splenomegaly (Banti's Disease).—This symptom-complex is regarded by the majority of writers as the terminal stage of splenic anemia. (See p. 632.)

Certain acute infections, as, *e. g.*, malaria, acute sepsis, subacute suppuration, typhoid fever, typhus fever, scarlet fever, etc., manifest splenic enlargement as a clinical characteristic.

Malaria.—In all forms of malaria splenic enlargement develops early and continues, becoming more marked as the disease progresses. In chronic malaria the spleen may attain an enormous size, extending as low as the umbilicus, and occupying the greater portion of the left superior abdominal quadrant. There is, as a rule, an associated enlargement of the liver. Hepatic tumor, however, disappears after the application of treatment, but

the spleen may remain enlarged for months or even years after an attack of pernicious malaria. We have observed that a large proportion of Italians admitted to the Philadelphia hospitals show a variable degree of splenic enlargement, and in practically all such cases there is a history of the patient having had malaria in his native country. Enlargement of the spleen in those who have lived in the tropics should always be regarded as possibly of malarial origin, until proof of the non-existence of malarial infection has been established.

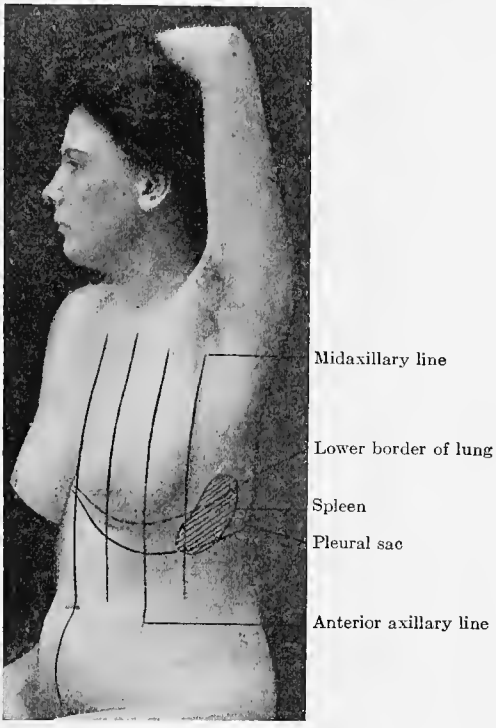


FIG. 249.—RELATION OF SPLEEN TO THE LUNGS, PLEURÆ, AND ANTERIOR LATERAL SURFACE OF THE CHEST.

Clinical Course.—The spleen may remain large for months, but seldom causes annoying symptoms.

New-growths of the spleen, while extremely rare, may result in enlargement of the organ, and chief among these are echinococcus cysts and sarcoma, more commonly of the melanotic variety. Tuberculosis and gummata may exist, but are seldom, if ever, detected during life. Secondary carcinoma of the spleen is often found at autopsy, but is of no clinical significance. Gummata of the spleen and amyloid degeneration may be present in the same organ, although there is no known means by which these conditions could be diagnosed antemortem.

Acute enlargement of the spleen is characteristic of the **acute infections,** but is more common in typhoid than in other types of continued fever.

Enlargement of the spleen following **subacute suppuration** is easily

diagnosed, since there is always a history of a subacute process that most often involves the bony structures.

SPLENITIS.

Pathologic Definition.—Either an acute or a chronic proliferative or suppurative process, involving the substance of the spleen. This condition is commonly secondary to metastatic inflammation and to embolic infarction. Benign splenic embolus is quite common during the course of valvular heart disease, and such infarcts are usually surrounded by sero-

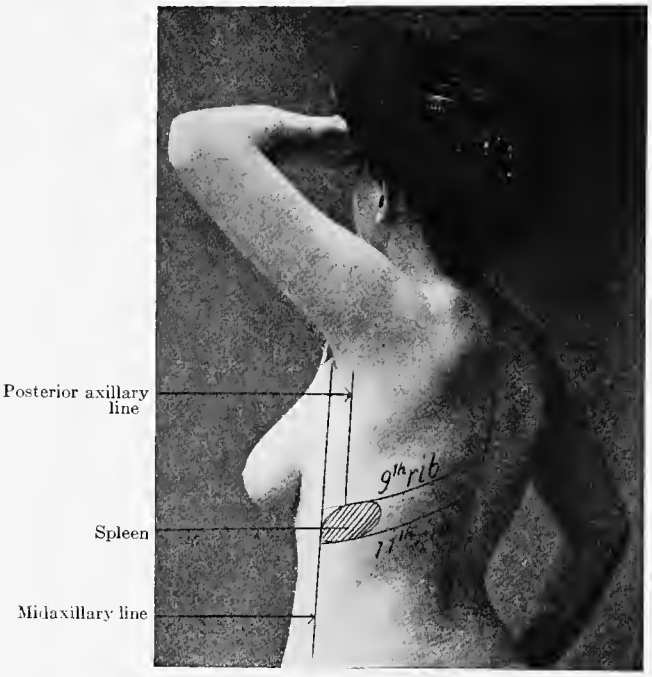


Fig. 250.—Normal Position of the Spleen in the Adult Female.

hemorrhagic infiltration; at the site of such infiltration there may later be necrotic softening, calcareous changes, or a fibrous cicatrix. Splenic infarcts, when infected by pus-producing microörganisms, go on to abscess formation, with more or less extensive destruction of the splenic tissue. Perisplenitis usually results in adhesion of the organ to the stomach and colon. In acute splenic tumor with active congestion there are round-celled infiltration and moderate proliferation of the splenic cells. The organ is also enlarged, soft, and friable.

Varieties.—**Acute hyperplastic splenitis (acute splenic tumor)** is a feature of acute infections, as, *e. g.*, typhoid and typhus fever, malaria, etc., whereas chronic splenic enlargement is associated with chronic infections or repeated attacks of acute infections and passive congestion of the spleen. (See Causes of Enlargement of the Spleen, p. 626.)

General Symptomatology.—In more than 80 per cent. of all cases

the symptoms are indefinite or absent. Among the characteristic features are: (1) Absence of pain and tenderness, the latter being present only when perisplenitis exists. (2) A sense of weight in the abdomen when the organ is markedly enlarged. (3) Occasionally shortness of breath and cyanosis upon exertion, these being most common in cases in which profound anemia is associated. Further physical examination reveals those features characteristic of splenic enlargement.

Summary and Differential Diagnosis.—**Acute suppurative splenitis** is extremely difficult to diagnose, since it often resembles acute gastric and pancreatic conditions. A careful analysis of the symptoms,

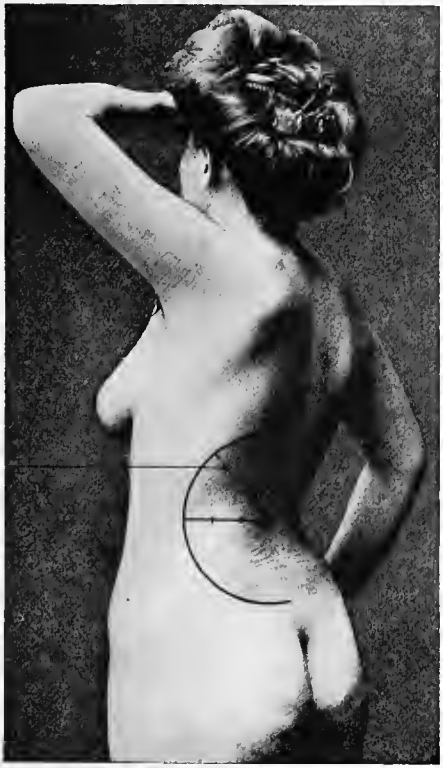


FIG. 251.—LEFT HEMISPHERIC DIVISION OF BACK, SHOWING METHOD OF LOCATING THE SPLEEN.

together with the presence of an abdominal tumor, is usually sufficient to enable one to discriminate between acute splenic suppuration and *gastric* or *pancreatic carcinoma.* A laboratory study of the secretions and excretions is of great importance in this connection, the feces being laden with fat-globules in pancreatic disease, whereas a negative result is obtained in acute suppurative splenitis. Pancreatic disease shows the leukocytes to be normal or subnormal in number, while in acute splenitis leukocytosis obtains. An analysis of the gastric contents is usually sufficient to enable one to determine the existence or non-existence of *gastric carcinoma.*

Splenic enlargement is to be distinguished from **hepatic tumor,** but in this there is seldom, if ever, any difficulty, the rule being that firm abdominal

palpation results in distinctly separating these two organs. Of still greater value is auscultatory percussion (page 59). The characteristic change of note elicited when percussion is made over a solid viscus is incapable of misinterpretation, and is a most valuable method for outlining the viscera. The other methods employed for distinguishing between hepatic and splenic tumor will be found of service in differentiating between splenic and **renal tumor,** splenic and **omental growth,** and splenic and **ovarian tumors** or **cysts.** The examiner should ascertain definitely that a pleural effusion has not forced the spleen below the costal margin, and that the organ itself is not enlarged, but merely displaced.

Fecal impaction of the splenic flexure and of that portion of the colon between the splenic flexure and the rectum differs from splenic tumor in the following respects: (*a*) Fecal impaction lends to the palpating finger a peculiar "doughy feel," whereas splenic tumor is always solid and clearly outlined; (*b*) distention of the colon by feces is found to give irregular areas of dullness and tympany; (*c*) by passing a rectal tube into the colon and injecting from one-half to one and one-half pints of olive oil, distention due to impaction usually disappears following a copious evacuation of the bowel.

RUPTURE OF THE SPLEEN.

Remarks.—Rupture may result from extreme hyperemic engorgement of the organ, such as is seen in cardiac conditions and in abscess. This accident rarely occurs in typhoid fever, and is said to have followed malaria.

Clinical Picture.—The initial symptoms resemble those of intestinal or of gastric perforation, and, indeed, one of these conditions is usually present when actual splenic rupture has taken place. Rupture of the spleen is seldom diagnosed except at autopsy.

Clinical Course.—This will vary with the character of the pathologic changes that have taken place in the spleen prior to rupture. If merely the capsule has ruptured and hemorrhage into the peritoneal sac is not profuse, recovery is possible. In the event of splenic abscess rupturing into the general peritoneal cavity, purulent peritonitis results.

SPLENOMEGALY (TYPE OF SO-CALLED SPLENIC ANEMIA).

Pathologic Definition.—A chronic affection of unknown origin, characterized by the presence of enlargement of the spleen, anemia of a chlorotic type, leukopenia, hemorrhages into the skin, from the nose, and from the gastric mucous membrane, and a terminal stage in which hepatic cirrhosis, ascites, and persistent jaundice of the skin, mucous surfaces, and viscera develop (Banti's disease).

Predisposing and Exciting Causes.—Sex.—Males are affected more often than females, the ratio being approximately as 5 is to 3. It is not uncommon among persons who have resided in malarial districts, although all cases do not give a previous history of malaria. (See also Histoplasmosis, p. 943.) Several members of the same family are at times affected.

General Remarks.—It is reasonable to presume that many of the cases reported in the literature as cirrhosis of the liver with splenomegaly, Banti's disease, splenic anemia, and simple splenomegaly are in reality different stages of one and the same condition, and that the slight variations in the

clinical pictures accompanying each report are due entirely to the stage of the disease present at the time the report was made.

Principal Complaint.—There is but slight loss of weight. The patient is languid, however, and does not feel rested after a night's sleep. During the early stages the appetite is somewhat fickle, although the patient takes sufficient food for nourishment; in the terminal stage, however, when there is venous stasis of the gastric mucosa due to changes in the liver, he complains of dyspepsia, constipation, and a continual sense of weight in the abdomen. When splenic enlargement is conspicuous, there is a constant dragging sense of distress in the upper portion of the abdomen. Weakness is progressive, and becomes extreme during the later stages, at which time the patient continually complains of cold, particularly affecting the extremities.

Laboratory Diagnosis.—The red blood-cells are moderately reduced in number, and there is a decided reduction in the hemoglobin. The leukocytes are usually reduced in number. We have seen two cases in which the leukocyte count was below 4500.

Clinical Course and Duration.—Osler,* in an analysis of 25 cases of chronic splenomegaly, gives the following figures: Over five years, 9 cases; between one and five years, 9 cases; in 7 the duration was unknown, and the longest period during which any case suffered from chronic splenic enlargement was eleven years.

* Amer. Jour. Med. Sci., August, 1902, p. 751.

DISEASES OF THE URINARY SYSTEM.

TOPOGRAPHY OF THE KIDNEYS.

These two glandular organs are symmetrically located on the back wall of the abdomen, one occupying each lumbar region.

The right kidney is placed immediately below the liver, and rests alongside of the lower thoracic and upper three lumbar vertebræ (Fig. 253). The right twelfth rib crosses the right kidney on a level with the junction of its upper third with the lower two-thirds of the organ (Fig. 255). The outer border of the right kidney is located by drawing a transverse line about 4 inches (10 cm.) from the spines of the vertebra. (See Fig. 255.)

In front of the right kidney are placed the ascending portion of the colon and the descending portion of the duodenum. Immediately above the right kidney is the suprarenal capsule. The kidneys are posterior to the peritoneum. (See arbitrary division of the abdomen, p. 127, and arbitrary division of the posterior abdominal wall, Fig. 254.)

The left kidney is located slightly higher than the right and usually corresponds to the eleventh thoracic vertebra or the top of the twelfth rib. (See Fig. 253.) In front of the left kidney are: the tail of the pancreas, the fundus of the stomach, and the descending portion of the colon. Above is the left suprarenal capsule, and the spleen also overlaps the kidney (Fig. 253).

X-RAY EVIDENCE IN DISEASES OF THE KIDNEYS, URETERS, AND BLADDER.

By George E. Pfahler, M.D.

At present the rays are used to discover and locate stone in the kidney, ureter, or bladder; to determine the presence, size, and shape of the kidney; to outline the pelvis of the kidney; to determine the course of the ureter; and to outline the bladder.

Renal calculi are at times the cause of very obscure symptoms, such as dull pains in the back (lumbago), pains in the appendiceal or hepatic region, or septic symptoms (pyonephrosis). The classic symptoms are often absent, and equally often the classic symptoms of calculi are present without the presence of stone. While other methods of examination will lead one to suspect stone, in the determination of either the presence or absence of stone we are dependent upon the Roentgen rays.

Preparation of the Patient.—Every patient should have an active purgative on the night preceding the examination. I prefer a bottle of magnesium citrate, which is more reliable than pills, and gives good, full,

watery evacuations. Every practitioner should bear this in mind, because it serves to eliminate fecal concreta, pills, compressed tablets, fragments of bismuth, and other foreign bodies in the intestines, which are liable to lead to error, or to cause unnecessary delay, annoyance to the patient, and expense in repeated examinations.

The shadows cast by renal calculi vary much in density, shape, location, and number, and they can be confused with at least twenty other conditions. The differentiation, technic, etc., must be left to special works on Roentgenology.

Ureteral calculi are even more obscure in their symptoms and more difficult to diagnose by any method than renal calculi, but with careful technic can be demonstrated by means of the Roentgen rays. At times it is necessary to pass ureteral sounds into the ureters and then repeat the *x*-ray examination, in order to make differentiations. As an example of the obscurity of symptoms in ureteral calculi, I will mention the recent case of a young woman of twenty-six years, who has had paroxysmal pains in the left labia majora for nineteen years, with no other symptoms. I found a

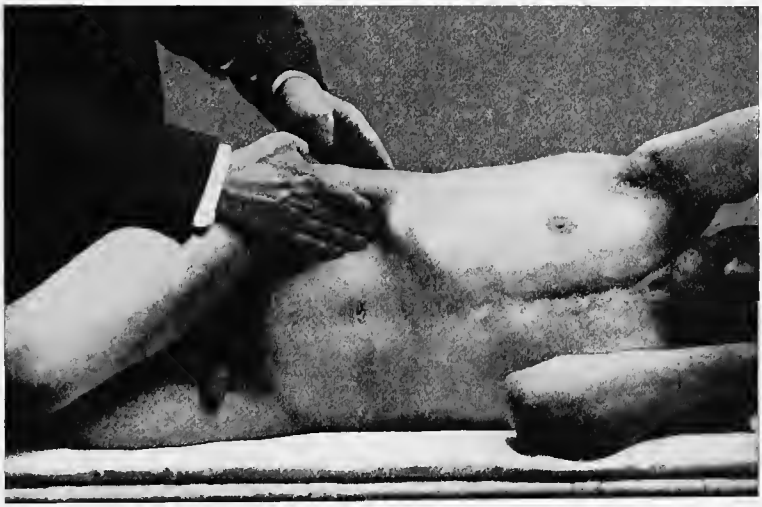


FIG. 252.—PALPATION OF THE RIGHT RENAL REGION, RIGHT THIGH FLEXED UPON ABDOMEN.

stone ⅜ inch in diameter in the lower end of the left ureter. This condition had been diagnosed by a number of physicians as a neurosis.

The Presence, Size, Shape, and Position of the Kidney.—Before the removal of a kidney is decided upon, one should determine the presence of the opposite kidney. One can determine the presence of two ureters by ureteral catheterization, but it is possible for both to come from one kidney. By means of modern technic one can demonstrate, in the great majority of patients, the outline of the kidney, thus determining the *presence*, *size*, and *shape* at once. Its size and shape will at times give a clue to such diagnosis as tumor, hydronephrosis, or pyonephrosis.

A definite statement as to the position of a kidney, and especially as to its mobility, is more difficult to make, since when the patient is in a recum-

bent posture (the usual position of a kidney examination), even a floating kidney may drop into its normal position. However, if one finds a difference in the relative position of the two kidneys, even in the recumbent posture, it is evidence of displacement, and may suggest movable kidney, or a displacement due to new-growth.

To demonstrate mobility of the kidney, one must demonstrate the shadow in the recumbent, and again in the upright or semi-recumbent position. It is much more difficult to show the shadow of the kidney in the upright position, especially in stout or muscular subjects.

The outline of the pelvis of the kidney and the ureter can be demonstrated by injecting an opaque substance into the ureter (collargol, colloidal

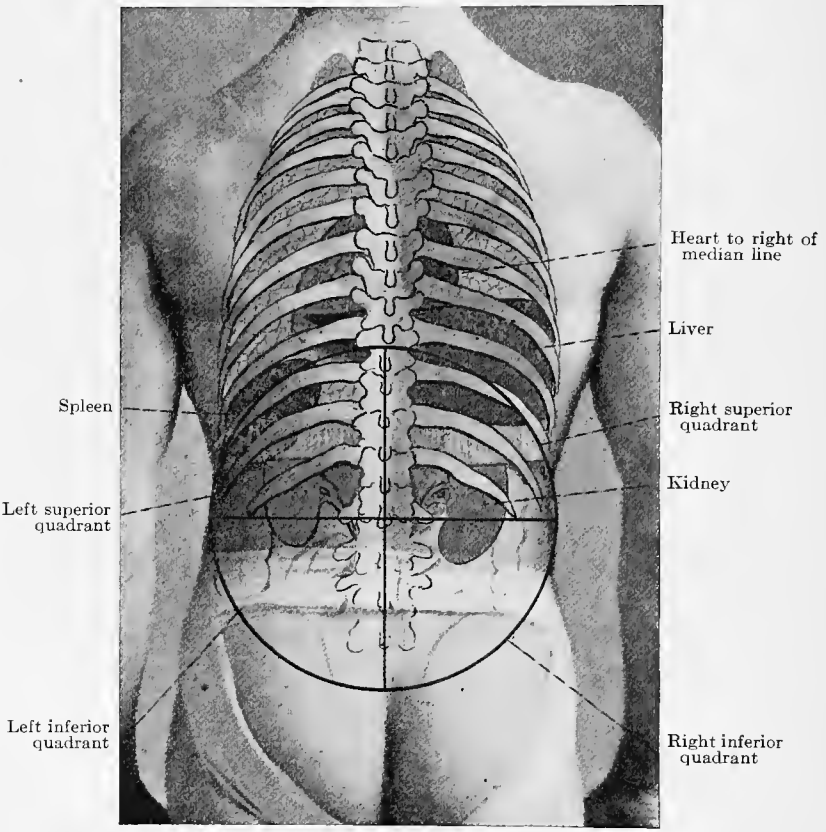


FIG. 253.—POSTERIOR VIEW OF ABDOMEN AND THORAX; MUSCULAR TISSUE REMOVED.

silver), or the ureter can be outlined by passing a leaded catheter into it, and then making an *x*-ray examination. Such combined cystoscopic and *x*-ray examinations are tedious, but will give valuable information, in determining an elongated ureter, or an anomalous position, or in diagnosing a dilated kidney pelvis (pyonephrosis).

The bladder may show calculi by means of the rays, or by distending with air a tumor can at times be outlined. A diverticulum is best located by filling the bladder with an opaque solution, such as colloidal silver or collargol.

SWEATING.

"The perspiratory function of the skin is of the utmost importance, as a means by which effete materials are removed, and the temperature prevented from rising above the normal."

Whenever the quantity of perspiration is altered beyond the physiologic normal for the individual in question, then this action of the skin becomes

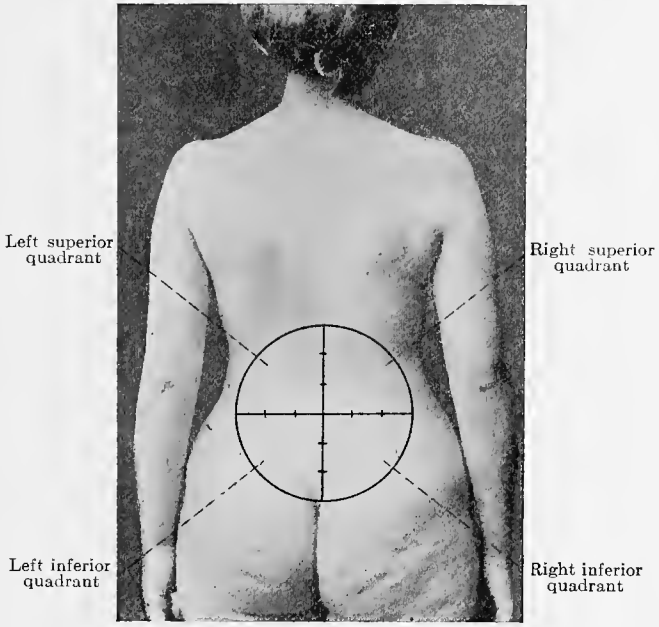


FIG. 254.—ARBITRARY REGIONAL DIVISION OF BACK.

an important feature in diagnosis. In certain maladies there may be not only alterations in the quantity of perspiration, but also in its color and odor.

Hemidrosis is characterized by the escape of bloody fluid from the skin, and is a clinical feature rarely observed in neurotic women, and may occur in such subjects with rather distinct periodicity.

Chromidrosis describes a condition where sweat may be either of a yellowish, greenish, reddish, or blackish color.

Coloration of the sweat may be found to affect certain portions of the body, *e. g.*, face, abdomen, hands, feet, arm-pits, and genitalia. The varying color of the sweat may at times depend upon the prolonged use of iron, and to the presence of indican and other pigments that are present. Chro-

midrosis is far more common in females than in males, as is shown by Dr. Foot's analysis of 46 cases, in which 40 developed in women. A reddish, brown, and blue color of the sweat may depend upon the development of certain bacteria upon the skin. The sweat occasionally displays a greenish tinge after the continued use of copper, iodin, phosphorus, turpentine, and a diet rich in fish. It is also a rare finding in connection with pulmonary tuberculosis and malaria. In jaundice, from whatever causes, the sweat is yellow and discolors the patient's clothing.

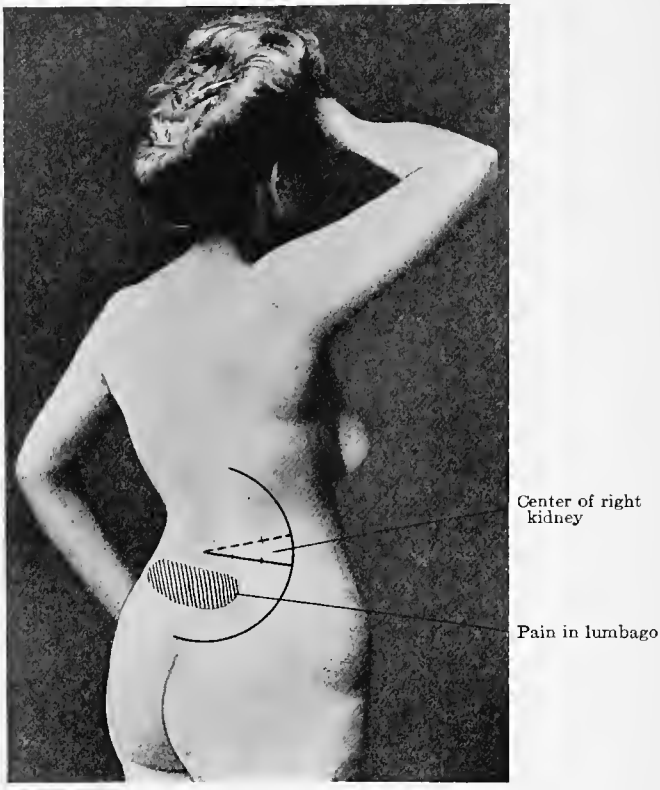


Fig. 255.—Right Hemispheric Division of Back to Locate Kidney
Transverse lines on level with spine of second lumbar vertebra.

Bromidrosis is commonly a functional disorder of the sweat-glands characterized by a production of sweat which emits a disagreeable odor. Ordinarily the quantity of sweat is increased. Offensive odor of the sweat may result from the ingestion of certain drugs and foods, and also from the development of the bacillus fœtidus in the perspiration. It is also a clinical feature in connection with neurotic and psychic individuals. This condition usually affects the feet, axillary, inguinal, mammary, and perineal regions.

Hyperidrosis.—Collectively speaking, this term includes any condition wherein there is an increased amount of sweat. Over-production of sweat

may be more or less continuous, remittent, intermittent, or periodical. This condition may also be either acute or chronic. Again it may be general or local, and at times we find it of clinical importance where sweating is unilateral. (See Aneurysm, p. 311.) Profuse sweating is a clinical manifestation in practically all infectious conditions that are accompanied by a chill at the onset, *e. g.*, malaria, scarlet fever, acute sepsis, tonsillitis, smallpox, and puerperal sepsis.

Profuse sweating and also sweating of the head and neck are common features among the continued fevers, *e. g.*, typhoid fever, pneumonia, and ulcerative endocarditis. Profuse drenching sweats may occur during sleep as a result of profound exhaustion; pulmonary tuberculosis with cavity being a typical example of disease in which this type of hyperidrosis is seen.

Lastly the skin may be beaded by drops of perspiration, and yet the cutaneous temperature be normal or subnormal, a clinical feature characteristic of all conditions accompanied by shock.

Anidrosis (decreased or complete cessation of sweat) is a clinical feature in connection with certain cutaneous maladies involving the sweat-glands; and it is also a conspicuous feature in diabetics, nephritis, scurvy, and such constitutional conditions as cretinism, exophthalmic goiter, and myxedema. In all patients displaying anidrosis the skin is dry and harsh to the feel, and if the condition persists for weeks or months, decided roughening develops.

EXAMINATION OF THE URINE.

We shall not attempt to discuss at great length the examination of the urine, but shall endeavor to explain certain practical methods that will enable the physician to collect specimens for examination, and, at the same time, we will describe certain approved methods by which albumin, glucose, acetone, and other pathologic urinary constituents are to be distinguished.

Collection of the Urine.—Whenever possible, the specimen for examination should be taken from the urine collected during the twenty-four hours, the quantity being measured. We have found it possible to obtain satisfactory results by collecting, in separate bottles, from two to four ounces of the urine voided three hours after a full meal and after the day's toil (evening urine), and the same quantity of urine voided in the morning after a night's sleep. The bottles should be thoroughly cleansed before using, and after placing the urine in them, they should be corked tightly and kept in a cool place.

Under normal conditions the quantity of urine excreted should measure about 1500 c.c. (50 fluidounces), but owing to various conditions that cause decided fluctuation in the quantity of the twenty-four hours' product, this standard is not essential.

Conditions that Cause Excessive Excretion of Urine.—Barometric and thermometric conditions (humidity, temperature) are responsible for marked fluctuation in the quantity excreted daily. An increase in the quantity of urine excreted occurs during the early part of the night and early morning hours. Exposure to cold, certain conditions of the skin in which free perspiration is impossible, and the too free imbibition of liquors are also responsible for an increase. In such pathologic conditions as diabetes insipidus, diabetes mellitus, chronic interstitial nephritis, and amyloid disease

an increase in the urine voided during the twenty-four hours occurs. Hydronephrosis, floating kidney, and hysteria may cause a temporary increase, but such increase is intermittent, lasting for but a few hours. True polyuria is seen in diabetes and during the absorption of serous exudates. It is to be remembered, further, that between the paroxysms of intermittent fever the flow of urine is usually increased—a feature well exemplified by relapsing fever. A moderate increase in the urine will be seen after the administration of certain drugs.

Conditions that Cause a Decrease in the Excretion of Urine.—Diminution in the quantity of urine excreted occurs during deep sleep, when persons are taking a dry diet. Pathologically, we find the urine diminished in acute nephritis, acute exacerbations of chronic nephritis, chronic parenchymatous nephritis, during the fastigia of acute fevers, and in robust and gouty individuals in whom the quantity of liquid taken during the day is extremely small. Oliguria may also develop as a result of interference with the renal circulation, as occurs, *e. g.*, in chronic heart disease, cirrhosis of the liver, ascites, cardiac embarrassment, anemias, abdominal tumors, pleural effusion, and other conditions.

Suppression of the urine is a common feature of uremia and of conditions in which a large quantity of fluid has been excreted from the body-tissues, as, *e. g.*, in hemorrhage, Asiatic cholera, and dysentery. Suppression, toxic in origin, may follow the administration of such drugs as oxalic acid, arsenic, turpentine, and others, although some of these drugs are capable of causing a moderate increase in the renal secretion when administered in medicinal doses.

CHYLURIA.

Chyluria, or milky urine, as a rule results from the rupture of lacteals into the urinary tract.

Clinical Significance.—Chylous urine is seen in those cases in which infection with the Filaria bancrofti has occurred. (See p. 960.) It is also seen in disease of the bladder and following surgical operations along the genito-urinary tract (traumatic chyluria), and a few cases are reported that followed severe injury to the kidney. In one instance chyluria followed septic infection of the pelvic organs, and in two cases it occurred after operations upon the pelvic viscera. The detection of embryo filariæ in the urine will serve to separate parasitic from traumatic chyluria.

HEMATURIA AND HEMOGLOBINURIA.

Definition.—The former term is applied to a symptom the result of blood entering the genito-urinary tract, whereas the latter term designates a state in which merely the coloring-matter of the blood escapes with the urine.

Remarks.—Hematuria occurs in acute nephritis, and may follow the administration of drugs (renal irritants) or traumatism of the kidney. The condition may develop during the course of severe anemia, malarial cachexia, purpura, etc. Hematuria is a symptom of pyelitis, renal calculi, cystic calculi, cystitis, enlarged prostate, vesical polypi, tuberculosis of the bladder, tuberculosis of the kidney, carcinoma of the bladder or of the kidney, and urethral stricture. It is also a characteristic feature of Bilharz's disease (see p. 961) and of infection of the bladder with other animal parasites.

Causes.—The following is a list of the chief causes for blood in the urine:

RENAL CAUSES.

Pronounced hematuria.	Hydronephrosis.
Sarcoma.	Polycystic disease.
Carcinoma.	Nephritis (acute).
Papilloma of pelvis.	Drug-poisoning, turpentine, carbolic acid, cantharides, quinin.
Calculus.	
Tuberculosis of pelvis.	Calculus (renal).
Injury to the loins.	Traumatism.
Eustrongylus gigas.	Oxaluria.
Hematuria may be slight.	Tuberculosis.
	Floating kidney.

VESICAL CAUSES.

Papilloma.	Acute cystitis.
Villus-covered carcinoma.	Epithelioma.
Prostatic enlargement (adenoma or carcinoma).	Tuberculosis.
	Calculus.
Traumatism, usually instrumental.	Traumatism.
Bilharzia hematobia.	Filariasis.
	Strongyloidis stercoralis.

DISEASE OF THE ADJACENT TISSUES INVOLVING THE URINARY TRACT.

Carcinoma of the vagina.	Acute salpingitis.
Carcinoma of the rectum.	Pelvic abscess.
Carcinoma of the uterus.	Tuberculosis of intestine (rare).

GENERAL MALADIES AND INFECTIONS.

Small-pox.	Purpura.
Malaria.	Scurvy.
Yellow fever.	Leukemia.
Hemophilia.	Endocarditis.
	Acute fevers.

Detection of Hematuria.—The recognition, microscopically, of red blood-cells in the urinary sediment is positive evidence of the existence of hematuria, and unless the examiner is skilled in laboratory methods, will prove his most reliable test. (See also tests for blood-pigment in feces, p. 515.)

Recognition of Hemoglobinuria.—Whenever the urine is of a bloody color and it is impossible to detect by microscopic examination red blood-cells in the urinary sediment, the condition in question is probably one of hemoglobinuria.

Method.—In order to demonstrate the presence of hemoglobin in the urine powdered tannic acid is added to the filtered urine until a heavy precipitate is produced. This precipitate is collected on a filter-paper, thoroughly washed with distilled water, and allowed to dry in the air. A small granule of this precipitate is then put on a microscopic slide with a granule of sodium chlorid of the same size, and a few drops of glacial acetic acid are added. A cover-glass is then put on the mixture and the slide is warmed over a flame until the acid steams. If the acid evaporates completely, more is added, until there is a brown color in the fluid. After cooling, the specimen is examined with a $\frac{1}{6}$-inch objective, and if hemoglobin was present in the urine, the characteristic crystals of hematin will be discovered.

Caution.—In performing this test the slide must not be heated too hot and the cooling must take place slowly. Good crystals are obtained by al-

lowing the mixture of precipitate, sodium chlorid, and glacial acetic acid to stand overnight at room temperature.

LEUKOCYTURIA.

Remarks.—Normal urine, when studied microscopically, will be found to contain an occasional leukocyte, but white cells are seldom demonstrable unless the urine be centrifugalized and a large amount of sediment be employed for microscopic study.

Whenever leukocytes are found in a urinary sediment in large number, pus is said to be present.

Pathologic leukocyturia is found whenever there is congestion of the kidneys or any irritation of the pelves of the kidneys, ureters, bladder, prostate, or urethra, at which times the number of leukocytes present depends entirely upon the degree of irritation and the size of the area of mucous membrane affected. Leukocytes often enter the urine as the result of an inflammatory process of the prostate or of the seminal vesicles. Leukocyturia is also a feature of endometritis, vulvitis, vaginitis, and sexual excesses. Again, leukocytes may appear in the urine whenever an inflammatory process is present in any of the structures adjacent to the urinary tract.

PYURIA.

Definition.—The presence of pus in the freshly voided urine.

Naked-eye Appearance.—There is a white or milky white sediment that collects if the urine is allowed to stand for a few hours, and upon shaking the bottle this sediment is found to be extremely heavy, and to float up through the clear liquid, assuming a more or less fringed-rope appearance. Pus may be found in either acid or alkaline urines, although it is more common in the latter.

Microscopic Appearance.—Microscopically, this sediment is found to contain many pus-cells (Fig. 256), and at times red blood-cells.

Pus-cells are round objects of fairly uniform size, each of which contains a polymorphous nucleus. In some specimens the nucleus is so obscured by the granules in the cell cytoplasm as not to be easily distinguishable. In such a case a crop of 50 per cent. acetic acid, if allowed to flow under the cover-glass will dissolve the granules and bring the polymorphous nucleus into view. The pus-cells, or leukocytes, are the smallest round granular cells seen in urinary sediments. Renal epithelium, the cells of which have a single nucleus, in contradistinction to the polymorphous nucleus of the pus-cell, is about one and one-half times as large as the pus-cell; while round, pelvic, ureteral, vesical, and prostatic epithelial cells are from two to seven times as large as the pus-cell.

Clinical Significance.—Pus-cells appear in the urine as the result of inflammatory processes along the genito-urinary tract. In pyonephrosis, pyelitis, and the more severe forms of cystitis and urethritis erythrocytes are also present. Pyuria not infrequently results from the escape of pus from other tissues into the urinary tract, and it may depend upon the admixture of leukorrheal discharge.

It is of great importance to determine the origin of the pus in a given specimen of urine, and by examining the genito-urinary tract, it is usually possible to determine this point. It has been stated that pus-cells coming from the kidney and from the pelvis of the kidney are equally disseminated throughout the urine, but in our experience we have found many exceptions

to this rule. The deposit of pus as a thick, ropy sediment depends, in great measure, upon the amount of mucus present in the specimen.

ALBUMINURIA.

Remarks.—Albumin may appear in the urine as the result of a number of varied pathologic conditions, and some writers believe that the urine may contain albumin under normal conditions—the so-called physiologic albuminuria. The question that concerns the clinician most is, whether or not, in a given case, the albumin is renal in origin; and this point it is frequently difficult to determine. We have found many cases of albuminuria in which it

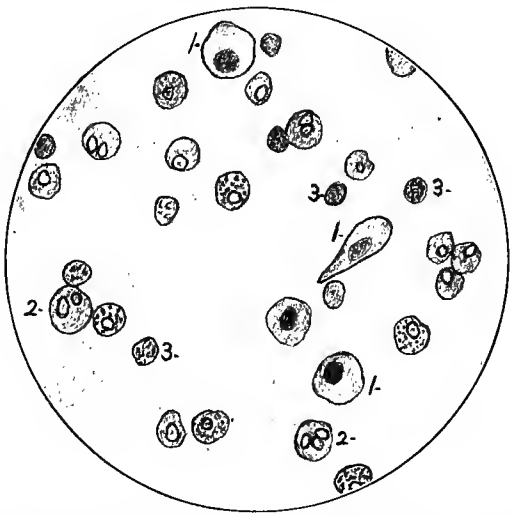


FIG. 256.—URINARY SEDIMENT FROM CASE OF PYELITIS (Boston).
1, Epithelial cells, probably from pelvis of kidney; 2, large pus-cells; 3, small-pus cells (obj. Spencer one-sixth).

was quite impossible to determine whether or not we were dealing with a true renal albuminuria. For convenience of study we have considered albuminuria under the following subheadings:

Renal Albuminuria.—A symptom resulting from temporary irritation of the renal tissue, or inflammatory or degenerative disease of the kidney, and depending upon the escape of albumin into the uriniferous tubules. If there should be disease of the pelvis of the kidney, albumin might enter the urinary tract at this point, and the condition should be considered as one of renal albuminuria. The changes capable of exciting the escape of albumin into the uriniferous tubules are of two types—(*a*) Inflammatory, in which there is congestion or inflammation of the kidney substance; and (*b*) degenerative changes, in which case evidences of acute inflammation are wanting. The former variety of albuminuria is to be seen occasionally in acute nephritis; the latter type is best exemplified in the chronic nephritides. Without doubt we have, at times, to deal with albuminuria resulting from the combined action of these two pathologic processes.

During the early stages of acute nephritis the amount of albumin that

escapes with the urine is extremely high, and may equal one or one and one-half per cent. by the Esbach method.

Caution.—The urine of a patient with nephritis which is voided after eating and exercise will contain more albumin than that collected after rest and sleep.

Toxic Albuminuria.—Renal albuminuria may be toxic in origin, and is to be seen after the administration of certain renal irritants, as, *e. g.*, copaiba, turpentine, phenol, following ether anesthesia, etc.; acting probably in a similar manner we find the toxins of certain acute diseases (diphtheria, scarlet fever, typhoid fever, pneumonia) capable of exciting the escape of a large amount of albumin with the urine. It is quite impossible to separate febrile from toxic albuminuria, since they frequently occur together.

Intermittent and Remittent Albuminuria.—Renal albuminuria may be intermittent, remittent, or continuous in character, and, in fact, it is not unusual to find all three of these types of albuminuria in chronic diseases of the kidney, as, *e. g.*, in interstitial nephritis, mild parenchymatous nephritis, and early during amyloid disease. In these chronic conditions the quantity of albumin passed may be comparatively small, and, in fact, it is necessary to concentrate the urine before one is able to detect this body. Clinically speaking, we do not consider albuminuria a pathologic condition unless the albumin is capable of detection by the methods ordinarily employed for this purpose. Orthostatic, postural, alimentary, and cyclic albuminuria are special varieties of the intermittent type.

During the course of an acute exacerbation of a chronic type of nephritis the amount of albumin passed during the twenty-four hours is high, and may even exceed that excreted in primary acute nephritis. In general, however, the larger the amount of urine excreted, the lower the percentage of albumin present.

(*a*) **Traumatic albuminuria** may follow injury to the kidney, abdominal massage, traumatism to the head, and severe injury to the extremities.

(*b*) **Alimentary Albuminuria.**—Following the ingestion of foods rich in albumin, the urine not infrequently contains albumin in pathologic amounts. We have repeatedly seen this form of albuminuria in patients taking from six to twelve eggs a day. It seems that when uncooked eggs are taken, albuminuria is most likely to follow, yet our series of experiments is not sufficiently large to enable us to state positively that this is the rule, although it was found by us in nearly 60 cases.

(*c*) During the course of certain chronic and malignant maladies there may be a decided impoverishment of the blood, in consequence of which albumin escapes from the kidney into the uriniferous tubules. Thus we find albuminuria in both primary anemias (leukemia) and secondary anemias. The albuminuria that accompanies chronic lead-poisoning does not belong strictly to this second class, and there is room for question as to whether or not the albuminuria of anemia is not toxic in origin.

Extrarenal Causes of Albuminuria.—General Consideration.—Inflammatory processes involving the pelvis of the kidney, ureter, bladder, prostate, or urethra are capable of exciting albuminuria, and in this connection it may be well to mention especially such maladies as stone and tuberculosis of the pelvis of the kidney, stone in the ureter, and torsion of the ureter due to movable kidney; vesical polypi, ulcerative cystitis, vesical calculus, and tuberculosis and carcinoma of the urinary tract may in turn produce albuminuria.

Pus generated along the genito-urinary tract or escaping from other tissues with the urine may be responsible for albuminuria, and blood and

blood-serum (filariasis; Bilharzia disease), when added to the urine, cause this condition.

Prostatic Albuminuria.—Experiment has shown that in massage of the prostate the seminal fluid passes into the bladder without any escape of semen from the meatus; again, the urine may be free from albumin prior to prostatic massage, whereas following this operation the urine may contain albumin. In view of the foregoing facts this variety of albuminuria has been termed prostatic, and we regard the albuminuria or prostatic, ovarian, and uterine congestion as extrarenal in origin.

Albuminuria of prostatic origin is most likely to develop after exercise, hot and cold baths, etc.; this renders it difficult to distinguish between it and the so-called intermittent, cyclic, orthostatic, and transitory albuminuria, which is common after inflammatory processes of the urethra, prostate, or in conditions known to irritate these tissues. In prostatic albuminuria the urine is likely to contain spermatozoa.

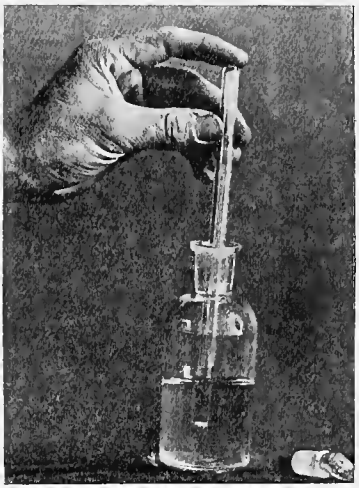


Fig. 257.—Boston's Method of Filling Lower Portion of Pipet, that Contains Urine to be Tested, with Nitric Acid.

Parasitic Albuminuria.—Infection with the Schistosomum hæmatobium causes hemorrhage into the bladder, and consequently albuminuria. There may be infection of the bladder with round worms—ascaris, oxyuris, anguillula—or with rhabiditiform embryos, all of which excite albuminuria. The animal parasites known to infect the kidney in man are: Eustrongylus gigas, which usually inhabits the pelvis of the kidney, and is quite common in the lower animals, and the Tænia echinococcus, which most often invades the substance of the kidney. Infection of the kidney with these parasites may be accompanied by both hematuria and albuminuria, and we have studied cases representing each of these types of infection.

Recognition of Albumin.—Boston's Pipet Method.—*Reagents.*—(1) Concentrated nitric acid; or (2) nitric acid, 1 part, and saturated solution of magnesium sulphate, 9 parts.

"Albumin causes a white cloud to appear in the form of a ring at the zone of contact of the two liquids (reagents and urine) (Figs. 257, 258), and this test, when carefully applied, must be regarded as one of great value."

1. A pipet is filled for a distance of from one inch to one and one-half inches with the urine to be tested. The urine is then removed from the surface of the pipet by washing or by wiping.

2. The pipet, with its contained urine, is then placed near the bottom of a bottle containing nitric acid, when the pressure of the index-finger is lessened and the acid allowed to flow gradually up into the pipet.

3. When the pipet is seen to contain about equal amounts of acid and urine, the finger is again pressed firmly upon the top of the pipet, which is then removed from the bottle and held toward the light on a level with the eye. If albumin is present, a distinct white ring of coagulated albumin appears at the junction of the urine and the reagent. "The ring is often in-

tensified by placing the pipet in different lights or against a dark background. The hand, when placed back of the pipet and carried slowly above and then below the level of the ring, serves this purpose."

The Heat and Nitric Acid Method.—Fill a test-tube three-quarters full of filtered urine, and boil the top layer. In the presence of phosphates or albumin a precipitate will be produced. The addition of one drop of nitric acid will dissolve this precipitate if it be due to phosphates. If it be due to albumin, the precipitate will not be dissolved, and the drop of acid, as it falls through the unboiled portion of the urine, will produce a second precipitate by its coagulating action on the albumin.

Fig. 258. — Boston's Method. Pipet Containing an Upper Stratum of Urine, a Lower Stratum of Nitro-magnesium Solution, and Showing a White Line (Albumin Ring) at Zone of Contact.

Heller's Test.—About three cubic centimeters of nitric acid are placed in a test-tube, and filtered urine is allowed to flow down the side of the tube so that it lies in a layer above the acid. In the presence of albumin a white line of coagulated albumin will be seen at the junction of the two layers of fluid. A brownish ring due to acid urates is formed a short distance above the line of junction of the two fluids, and is to be differentiated from the ring of albumin. After the administration of copaiba and other balsamic drugs the urine contains substances that produce a brownish ring below the junction of the two fluids.

The Acetic Acid and Potassium Ferrocyanid Test.—A small quantity of filtered urine is acidulated with five drops of acetic acid, and a 10 per cent. solution of potassium ferrocyanid is added to the mixture one drop at a time. In the presence of albumin a white precipitate will be produced.

Significance.—The heat and nitric acid test is the most reliable method for the detection of serum-albumin in the urine. The other tests will give positive reactions for other albuminuous substances than serum-albumin. It occasionally happens that the amount of serum-albumin in the urine is so small, or that fermentative changes have taken place in the specimen to such an extent, that the heat and nitric acid test is difficult of interpretation. Under such circumstances the other reactions suggested may be used as confirmatory tests. The acetic-acid-potassium-ferrocyanid test will give a precipitate with mucin and other albuminoid bodies, and is not to be used in the routine examination of urine for albumin.

Frequency.—Certain foreign authors claim to find albumin in about 7 per cent., and others in 20 per cent., of cases studied; in patients under our observation we have not found albuminuria so common. In fact, we incline to the belief that those who found so high a percentage of urines to contain albumin must have been considering all the coagulable substances, and that they have included reactions caused by the presence of the albumoses (peptones), mucin, phosphates, and the like.

Esbach's Method.—The Esbach method for the quantitative determination of albumin is the best method available for clinical work. The instru-

ment has been tested against estimations made by precipitating out and weighing the albumin and has been found to be quite accurate. The following solution (Esbach's solution) is used: Picric acid, 10 gm.; citric acid, 20 gm.; distilled water, 1000 c.c.

Process.—Fill an Esbach tube with filtered acid urine to the mark U, and add the reagent until it reaches the mark R. Then place a cork in the mouth of the tube, and invert it several times to insure perfect mingling of the urine with the reagent. The tube should now be placed in a special receptacle (Fig. 259) and allowed to stand for twenty-four hours, when the sediment that collects at the bottom of the tube will consist of serum-albumin, serum-globulin, albumoses, uric acid, and kreatinin. The amount of sediment is read directly from the scale, and indicates the amount of albumin in grams in 1000 cubic centimeters of urine.

Caution.—When the specific gravity is above 1.008, the specimen should be diluted with water to reach this density. The temperature of the room should be 15° C. (59° F.). The urine must be acid in reaction.

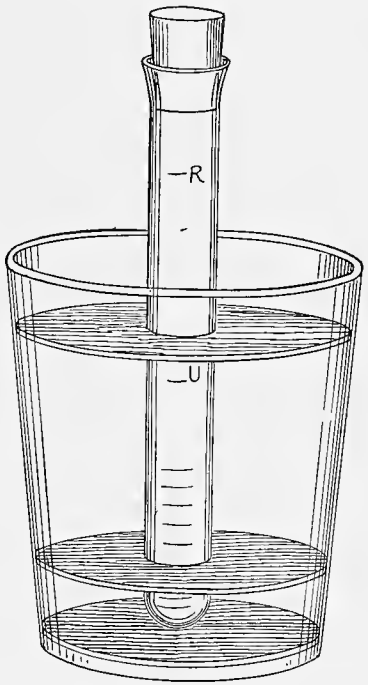


Fig. 259.—Receptacle for Esbach's Albuminometer, Devised by Dr. W. G. Mudie.

In the bottom of an ordinary tumbler place a piece of heavy cardboard and cut central openings in two other cardboards which are glued in position.

SPECIFIC GRAVITY.

Consideration.—The specific gravity of the urine for healthy American is given at 1.020 and for Chinese 1.004 to 1.012. It usually varies in inverse ratio to the quantity excreted daily. If a large quantity of urine is excreted daily, and such urine is of high specific gravity,—1.030 to 1.040,—glycosuria is to be suspected. Urines rich in solids display a high specific gravity, and when the quantity of urine voided during the twenty-four hours is far below the normal, a high specific gravity is usual. A diet rich in albumins also tends to increase the specific gravity of the urine. As a rule, the specific gravity is high in acute nephritis, during the fastigium of acute fevers, in chronic parenchymatous nephritis, and also in specimens recovered from persons who live luxurious lives and take insufficient exercise.

A *low* specific gravity occurs after ether anesthesia, after *hysteric seizures*, in diabetes insipidus, chronic interstitial nephritis, amyloid disease of the kidneys, and after imbibing too freely of liquids. During convalescence from acute nephritis and from acute fevers the specific gravity is comparatively low.

REACTION OF THE URINE.

Normal urine is acid in reaction, such acidity depending upon the presence of acid sodium phosphate, (NaH_2PO_4). The total acidity of the twenty-

four-hours' product is estimated to be equivalent to 14 grams of sodium carbonate.

The acidity of the urine is **increased** by violent muscular exercise, a diet rich in meats, mineral acids, during certain fevers, in scurvy, diabetes mellitus, diabetes insipidus, leukemia, gout, rheumatism (chronic and acute), and in the acute infections. Increased acidity is also present in persons who eat too heartily, and who take insufficient exercise. It is characteristic of the uric-acid diathesis and of oxaluria, and is commonly observed when the quantity of urine passed during the twenty-four hours is below the normal. When an oxalic or a uric-acid stone of either the bladder or the kidney exists, the urine is often highly acid.

Decreased acidity may follow the ingestion of a light meal, a vegetable diet, and the ingestion of alkaline carbonates. Decreased alkalinity is also seen following profuse sweating, paroxysmal vomiting, and more rarely under other circumstances. The acidity may be lowered during certain hours of the day. Standing lessens the acidity of the urine.

Neutral and Amphoteric Urines.—Rarely, one encounters a urine which causes no change in either red or blue litmus; such a specimen is neutral. Occasionally, the urine will give both an acid and alkaline reaction with litmus-paper, when it is styled amphoteric. This phenomenal reaction is dependent on the presence of both acid and neutral sodium phosphates, which substances are held in equal suspension. This reaction is of no clinical significance.

CHLORIDS.

Remarks.—Sodium in combination with chlorin forms the chief alkaline constituent in normal urine, from 10 to 15 grams being excreted during the twenty-four hours. It should be stated that chlorin, in combination with calcium, potassium, ammonium, and magnesium, is also present in normal urines.

Decrease.—During health the amount of sodium chlorid excreted is in direct proportion to the quantity and quality of the food taken. The chlorids are decreased in such febrile conditions as scarlet fever, smallpox, typhoid fever, typhus fever, acute hepatic atrophy, and in disease in which starvation occurs. It is asserted that the chlorids are diminished or absent in croupous pneumonia. The chlorids are diminished slightly in certain diseases of the insane in proportion to the degree of involvement of the kidney. In maladies attended with an excessive drain upon the system, such as diarrhea, and in disease in which the body tissues are imperfectly nourished (*e. g.*, carcinoma), the chlorids are much below the normal limit.

Increase.—A marked increase is seen to follow the administration of potassium salts, whereas a less pronounced increase results from the absorption of the serous exudates, during diabetes insipidus, convalescence from fever, the afebrile stage of intermittent fever, and after epileptic seizures.

Recognition of Chlorids.—Reagents required: (1) Nitric acid; (2) solution of silver nitrate (1 dram of the crystalline salt to the ounce).

The foregoing solution of silver, when added to albumin-free urine, is capable of precipitating the chlorids when the urine is first acidulated by adding a few drops of nitric acid. This precipitate appears as an opaque, milk-white silver chlorid. Normal urine contains from $\frac{1}{2}$ to 1 per cent. of chlorids.

Application.—1. From 10 to 15 c.c. of albumin-free urine are placed in a

test-tube—and to it are added a few drops of nitric acid, and the mixture shaken gently.

2. The silver solution is added drop by drop, careful note being made of any changes that may occur.

Each drop of the solution causes a curdy white clump to fall to the bottom of the tube; in normal urine this mass does not become disseminated upon shaking the tube, nor does the entire liquid tend to become milky.

Should the chlorids be reduced to 0.1 per cent., the drop of solution merely causes an opalescence, whereas in the absence of chlorids, no change is observed, a feature seen in lobar pneumonia. A copious precipitate is indicative of an increase in the amount of chlorids present. The precipitate due to chlorids is soluble in ammonia, but *insoluble in nitric acid.*

Although the quantitative estimation of chlorids is desirable in certain instances, it is not within the scope of this volume to give the details of such a test.

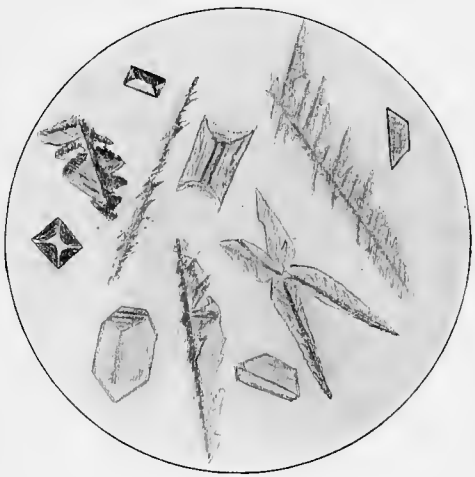


Fig. 260.—Crystals of Phosphates (Boston).

PHOSPHATES.

Consideration. — The twenty-four hours' urine should contain from 2.05 to 3.05 grams of phosphoric acid. Most of the phosphoric acid enters into combination with sodium, whereas the remainder of it is found to be united with calcium and magnesium. The earthy phosphates (calcium and magnesium) are found in a proportion of 33 to 67, and constitute one and one-half grams of the twenty-four-hours' product.

Alkaline phosphates (sodium and potassium) constitute the greater portion of the daily excretion, sodium phosphate being by far the more abundant. Approximately, four grams of alkaline phosphates are excreted daily.

Increase.—The urinary phosphates are found to be decidedly increased during convalescence from the acute fevers, in diabetes insipidus, leukemia, diabetes mellitus, and phosphatic diabetes.

Drugs.—An increase in the excretion of phosphates may be due to the administration of drugs, as, *e. g.*, alcohol, chloral, chloroform, vegetable acids, and the bromids. Violent exercise, mental strain, anxiety, and hot baths are likewise followed by a moderate increase.

Phosphatic Diabetes.—This term is descriptive of a condition in which the symptoms of diabetes accompany phosphaturia. Here the most decided increase in the urinary phosphates is seen, and the twenty-four-hours' product may exceed four grams.

Decrease.—The urinary phosphates are diminished in conditions in which the vitality is greatly lowered, in most forms of anemia, rheumatism, chronic plumbism, and atrophic hepatic cirrhosis. The phosphates have been found below the normal in melancholia, but observations upon the insane are highly unsatisfactory.

In estimating the amount of phosphates, it is important to ascertain that the patient has not been taking cocain, strychnin, alcohol, valerian, quinin, and phosphoric acid, since these drugs cause a temporary diminution in the excretion of phosphates.

Estimation of Phosphates.—For a description of the methods of estimating the amount of phosphates in the urine the reader is referred to special works upon clinical diagnosis.

SULPHATES.

Remarks.—During health, from two to three grams of sulphates are secreted during the twenty-four hours, the amount being influenced largely by the quantity of proteid food ingested, and by the tissue destruction that is taking place.

Increase.—The daily excretion of sulphates will exceed three grams when the patient is fed upon a diet rich in animal proteids. During acute fevers and in inflammation of the meninges and of the serous sacs there is a rise in the output of urinary phosphates. A decided increase is occasionally seen in diabetes mellitus, eczema, pseudohypertrophic paralysis, muscular atrophy, and myeloid leukemia.

Drugs.—It must be remembered that sodium salicylate, antifebrin, morphin, and the bromids cause a somewhat marked increase in the urinary sulphates.

A feature of great clinical importance is that whenever the percentage of hydrochloric acid is lessened, the ethereal sulphates are increased; consequently an increase is present in intestinal fermentation.

Decrease.—The total sulphates are diminished following a diet rich in vegetables. After diarrhea, depleting conditions, and when the gastric juice is found to contain lactic and butyric acids in excess, the ethereal sulphates are also diminished.

Recognition of Sulphates.—*Reagents.*—1. Solution of barium chlorid (1 : 8).

2. Acetic acid (specific gravity, 1.04).

Application.—1. Place 10 c.c. of urine in a test-tube.

2. Acidify with acetic acid.

3. Add about 3 c.c. (one-third volume) of barium chlorid solution, 1 c.c. at a time, shaking gently after each addition.

Reaction.—A white, milky precipitate indicates the presence of sulphates in normal amounts; but should the liquid assume the consistence of cream, sulphates are present in excess. If the liquid becomes opalescent, sulphates are diminished.

For the quantitative estimation of sulphates see special works upon Laboratory Methods, since the clinical employment of such knowledge scarcely warrants further description in this volume.

SULPHUR.

Loosely combined sulphur, when present in the urine, is a characteristic feature of disease of the bones (myelomata), and this unusual condition is, as a rule, associated with albumosuria. See Myelomata and Bence-Jones' Albumosuria (p. 1004). Urine containing sulphur when heated with a solution of lead acetate causes a brown or blackish precipitate.

UREA.

Physiologic Quantity.—From 20 to 40 grams (300 to 600 grains) of urea constitute the normal quantity for twenty-four hours.

Increase.—The amount of urea excreted is influenced by the rate of tissue destruction; consequently after prolonged exertion the urea-content is in excess of 40 grams a day. An increase is also a feature of scurvy, leukemia, pernicious anemia, paralysis, diabetes mellitus, cyanosis, epilepsy, intestinal fermentation, chorea, and pregnancy.

Drugs.—The prolonged use of such drugs as caffein, the chlorids, morphin and its derivatives is followed by an increase in the urea output, as is also the drinking of lithia waters. Owing to direct effect upon the tissues, the proportion of urea increases after the application of electricity and in poisoning by phosphorus.

Decrease.—The quantity of urea is said to be decreased in acute yellow atrophy of the liver, and following such decrease leucin and tyrosin, the result of destructive changes in the liver, appear in the urine. A lessened excretion of urea is expected in such chronic conditions as hepatic cirrhosis, jaundice, lead-poisoning, melancholia, paresis, nephritis, and hysteria; this is also commonly seen in Addison's disease and in certain nervous affections. It is generally believed that gastro-intestinal derangements materially influence the excretion of urea, but when the amount of urea is studied in conjunction with the various conditions that may influence the output of this secretion, it becomes difficult for the clinician to draw any definite deductions from the amount of urea excreted during the twenty-four hours.

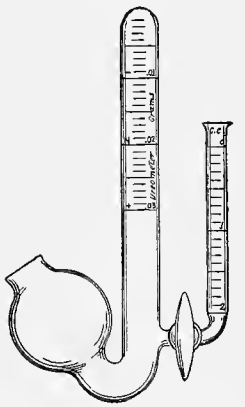


Fig. 261.—Hinds' Modification of the Doremus Ureometer.

Recognition of Urea. — Hypobromite Method.—The procedures of physiologic chemistry give the only accurate methods for the estimation of urea, the chief nitrogenous excrementitious product in the urine. The total nitrogen content should be determined by the Kjeldahl method, and this may be expressed as urea. Such a procedure, however, is too complicated for clinical work, and we are obliged to use a less accurate method for such a determination. It must be remembered, however, that the determination of urea by any method is of no value unless a portion of a twenty-four-hour specimen of urine is used.

The Hypobromite Method.—The hypobromite method is the most convenient for clinical work; but it has been shown by Ryan and Marshall that an average of 92.56 per cent. of the nitrogen of urea is liberated as free nitrogen gas in the tube.

The Doremus-Hinds ureometer is the instrument most used in this method. The vertical tube and bulb are filled with a solution of sodium hypobromite made by dissolving 100 grams of sodium hydroxid in 250 cubic centimeters of water and adding 25 cubic centimeters of bromid. The side arm of the tube is then filled with urine to the one cubic centimeter mark, after opening the stopcock sufficiently to allow the reagent to fill its lumen. The stopcock is now opened wide and the one cubic centimeter of urine is allowed to mix with the reagent. The reaction liberates nitrogen gas, which collects

in the vertical tube, and after the bubbles have subsided the volume is read off. The graduations represent grams of urea per cubic centimeter of urine. This figure multiplied by the number of cubic centimeters in the twenty-four-hour specimen will give the amount of urea eliminated during the day.

URIC ACID.

Consideration.—The daily elimination of uric acid for normal man is given as 0.2 to 0.5 grams. After a time, uric acid usually collects at the bottom of the specimen in the form of a brick-red, crystalline sediment. Microscopically, these crystals are seen to take the form shown in the accompanying illustration (Plate XIII). Crystals of uric acid precipitate from urines of high and low specific gravity, but, as a rule, this sediment is most copious in urines in which the specific gravity is above 1.025.

Increase.—The amount of uric acid excreted is influenced largely by the foods ingested, *e. g.*, such animal foods as liver, thymus gland, brain, and kidney cause an increase in the amount excreted daily. Violent exercise results in cell destruction, affecting particularly the leukocytes, which is followed by a similar increase. In from four to six hours after a meal rich in meats, during the paroxysmal stage of gout, during high temperature, and in acute articular rheumatism an increased elimination of uric acid occurs. The urine of leukemia contains a high percentage of uric acid.

Decrease.—A vegetable diet, lead-poisoning, nephritis, muscular atrophy, and chlorosis show a decrease in the amount of uric acid excreted. A decrease is also usually associated with the primary and the secondary anemias. The great difficulty encountered in estimating the uric acid of the urine renders a knowledge of this excretion of comparatively little clinical value. Personally, we have been unable to draw any deductions from our own observations, and even a review of the literature has not provided us with sufficient facts from which to draw valuable conclusions.

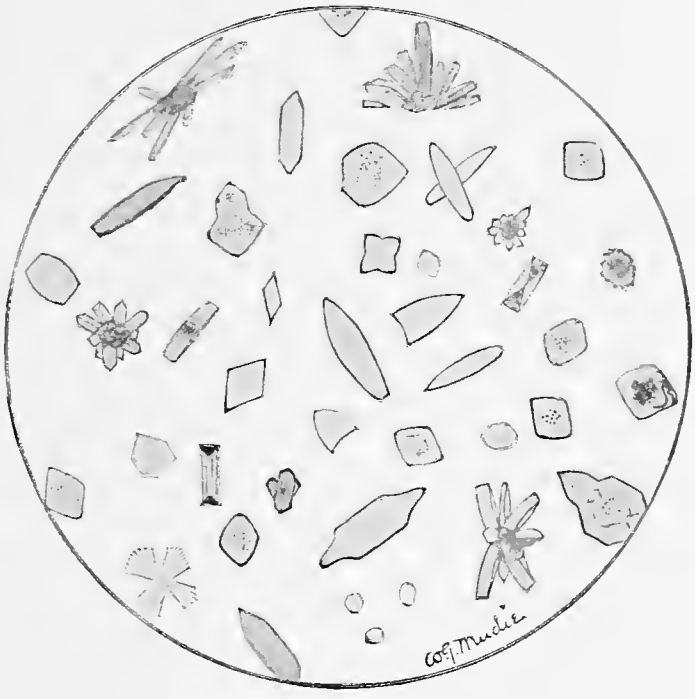
CARBOHYDRATES.

Clinical Consideration.—Clinically, we are concerned with but two sugars that occur in the urine: glucose and lactose. Occasionally, levulose is to be detected in urines in which glucose is also present, and less commonly maltose, saccharose, and pentose are found in the urine.

Glucose.—We shall first discuss the clinical significance of glucose (dextrose or grape-sugar) when it appears in the urine. The presence of glucose in the urine is usually considered under the heading of glycosuria. It may be stated here that the mere presence of glucose in the urine does not denote that diabetes mellitus exists, unless the other essential symptoms of the disease—progressive weakness with emaciation, intense thirst, excessive appetite, and polyuria—are also present. If any of these symptoms are absent, we are possibly dealing with alimentary glycosuria.

Clinical Significance.—Under normal conditions slight traces of glucose are found in the blood (*glycemia*), but it is doubtful whether it is present in sufficient amounts in the urine to be detectable by the clinical methods ordinarily employed for its recognition, "except after the ingestion of an excess of food rich in saccharin or starchy substances" (Anders). Consequently whenever this substance is present in the urine in sufficient amounts to induce a reaction with Fehling's solution, glycosuria exists. (See Diabetes Mellitus. p. 992, for clinical varieties of glycosuria.)

PLATE XIII



Crystals of Uric Acid from Permanently Mounted Specimen Slides (obj. B. and L. one sixth; eye-piece 2). (Boston.)

Alimentary glycosuria follows the free ingestion of carbohydrates, and is not infrequently seen in those who eat heavily and take but little exercise. Again, alimentary glycosuria may be seen in certain diseases in which malnutrition is prominent.

Toxic glycosuria follows the administration of lethal doses of hydrochloric acid, sulphuric acid, mercury, strychnin, glycerin, alcohol, nitrobenzol, lead, arsenic, phosphorus, potassium iodid, caffein, thyroid extract, and tuberculin.

Caution.—After the administration of any of the aforementioned drugs, and when any of the coal-tar products have been taken, the fermentation test should always be employed before deciding that glucose is present, since these drugs may cause a reduction of the copper in Fehling's solution.

Pathologic toxic glycosuria is the term applied to a condition in which glycosuria is found to develop during the course of acute and chronic infections as, *e. g.*, cerebrospinal meningitis, cholera, relapsing fever, typhoid fever, diphtheria, phthisis, some of the exanthemata (scarlet fever), hepatic cirrhosis, rachitis, gastritis, malarial paroxysms, scarlatinal nephritis; chronic interstitial nephritis, cholelithiasis, syphilis, asthma, and whooping-cough.

Glycosuria may occur during the course of cerebral hemorrhage, brain tumor, brain abscess, epilepsy, neuralgia, sclerosis of the spinal cord, and the various forms of insanity. Maladies in which there is general loss of *circulatory equilibrium*, as, *e. g.*, exophthalmic goiter, myxedema, etc., are not infrequently accompanied by glycosuria.

Nervous glycosuria is that form of the disease in which the nervous manifestations are most prominent, *e. g.*, neuroses, psychoses, traumatic neuritis, brain injury, and permanent lesions of the nervous system. Certain *emotions*, anxiety, mental strain, financial embarrassment, etc., are also capable of causing a temporary glycosuria, and if such contributing causes persist for an indefinite period, a permanent glycosuria may follow.

Traumatic Glycosuria.—Traumatism to the head, trunk, and extremities is occasionally followed by glycosuria.

Puerperal glycosuria is somewhat unusual, whereas lactosuria is of common occurrence during the latter months of gestation and during the puerperal period.

Intermittent Glycosuria of Arthritis.—Glycosuria may develop during the course of certain chronic affections of the joints, and is most likely to make its appearance while one or more joints are acutely involved. This form has been called the "glycosuria of gout and obesity."

Test for Glycosuria.—Experience with the various tests for the recognition of sugar in the urine has caused us to employ Fehling's test for routine work. Although we have found other recognized tests—*e. g.*, Nylander's (bismuth) test and the phenylhydrazin tests of value, though less practical than the Fehling's test as now employed.

Fehling's Test.—*Reagents*—*Solution A.*—Powder, 34.64 gm. of pure crystallized sulphate of copper, and dissolve in 200 c.c. of warm distilled water; cool, and add distilled water to make 500 c.c. *Solution B.*—Crystallize Rochelle salts, 180 gm., and dissolve in 300 c.c. of distilled hot water; filter, and add 70 gm. of pure costic soda; cool, and add distilled water sufficient to make 500 c.c. This solution should be kept in a colored-glass stoppered bottle.

Collection of Urine for Examination.—It is our practice to secure 2 to 4 oz. of the first urine that is passed upon rising in the morning, and an

equal quantity passed during the evening, preferably three hours after the heaviest meal of the day. In specimens thus collected the morning urine usually shows the smallest, while that voided after exercise and a full meal is liable to contain the largest amount of sugar that is present during the twenty-four hours.

All specimens thus collected should be filtered before tested, and it is further to be remembered that ammoniacal urine may not give a satisfactory reaction with Fehling's solution.

(1) Place in a test-tube an equal quantity of Solutions A and B, which, when mixed thoroughly, results in an "alkaline solution of potassic cupric tartrate" (1 c.c. of this solution is reduced by 5 mgm. of glucose). When employing the test for qualitative analysis, add to the above mixture approximately three times its volume of water, which will result in a deep amethyst blue solution.

(2) Fill a test-tube two-thirds with the diluted Fehling's reagent and heat the upper portion of the reagent to the boiling-point. Then should it remain clear add drop by drop from a pipet the urine to be tested, boiling after each additional drop.

(3) In the presence of glucose in pathologic amount the upper and heated portion of the solution becomes first slightly turbid, and changes to a reddish or yellowish color. (See Plate.) The color produced by glucose varies, greatly depending upon the quantity of this substance present. The addition of from five (5) to twenty (20) drops of filtered urine free from albumin is usually sufficient to give a characteristic reaction should glucose be present. Whenever the reaction is uncertain, allow the tube containing it to stand for a time, then if the substance in question be sugar it will fall to the bottom of the liquid as a granular precipitate.

Caution.—Never employ a urine for analysis that has not first been filtered and proved to be free from albumin. The upper portion of the liquid in the test-tube should not be boiled briskly, and the lower part should not receive any heat from the Bunsen burner.

Where the amount of glucose present is but slight, a characteristic precipitate may not appear until the mixture (diluted reagent and urine) is allowed to cool.

Substances Reducing Copper.—While many of these are given in our own experience, but two of such substances have actually confused the reaction for glucose, these being glycuronic acid and lactose. Among the several precipitates seen when adding urine to diluted Fehling's solution as previously outlined none of these substances are seen to fall through the clear unheated reagent as is characteristic of glucose.

Quantitative Estimation of Sugar.—This may be readily accomplished when it is remembered that 1 c.c. of a solution containing equal parts of reagent A and reagent B is reduced by five (5) mgm. of pure glucose.

Fermentation Test.—This method is probably quite as reliable as are certain of the more complicated laboratory methods, and is much more readily employed by the general practitioner.

Method.—After it has been shown by Fehling's or some other reliable test that sugar is present in a given urine render a portion of such urine acid by the addition of tartaric acid, after which boil for several minutes. Add to the urine a portion of a cake of yeast ($\frac{1}{8}$ to $\frac{1}{4}$ inch square), shaking until the mixture is free from lumps, and then place this prepared urine in an Einhorn Saccharimeter, nearly filling the expanded portion, then place the thumb over the mouth of the apparatus and incline the tube so as to

compel the yeast mixture to occupy the graduated portion. Place the filled saccharimeter at a temperature of from 77° to 95° F.; when at the expiration of twelve hours should sugar be present, the carbon dioxid formed will have collected at the top of the tube and have displaced the urine, as shown by the graduation on the perpendicular limb of the saccharimeter. The reading obtained from the saccharimeter should be multiplied by the degrees of dilution (2 to 10) of the urine employed.

Caution.—Since yeast may give rise to the formation of a small volume of gas, in the absence of sugar it is advisable to conduct at the same time a control test with normal urine. Maltose, lactose, and levulose, when present in the urine, may give a similar reaction to that obtained with glucose, although these substances are usually excluded through the cautious employment of Fehling's solution.

Lactosuria.—Whenever there is a reduction of Fehling's solution or a reaction by the bismuth test and negative results are obtained with the phenyl-hydrazin and fermentation test, the presence of lactosuria should be suspected.

Clinical Significance.—Lactosuria frequently occurs during the period of lactation, and is seen to develop when there is some interference with the flow of milk. The amount of lactose in the urine is no direct guide to the degree of interference with the mammary function. Lactosuria is likely to develop on the second or third day after delivery, and disappears in from five to seven days (physiologic lactosuria). It should be remembered that milk-sugar may be placed in the urine by hysteric individuals.

Pentosuria.—Clinical Features.—Traces of pentose are present in normal urine, and pathologic pentosuria may develop during the course of glycosuria. Cases have been reported which show that pentosuria, like glycosuria, may develop in several members of the same family. A fact to be borne in mind is that pentose is capable of reducing Fehling's solution, and that it gives the phenyl-hydrazin test for glucose. The clinical significance of pentosuria remains doubtful.

Test for Pentose.—To five cubic centimeters of filtered urine add an equal quantity of hydrochloric acid (sp. gr. 1.19) and 30 milligrams of phloroglucin. Mix the ingredients and warm. In the presence of pentose a red color appears in the solution, and, on examining it with the spectroscope, an absorption band will be seen in the green. Lactose and galactose give the same color reaction as do the pentoses; but, of course, a different spectroscope absorption band.

CHOLURIA.

General Consideration.—Both bile-pigments and the bile acids may enter the urine as the result of disease, the rule being to find these two substances in the same specimen, the pigment occurring in profusion while the acids are but scanty. The yellow color of the urine, the presence of a heavy yellow froth, and the detection, microscopically, of bile-stained epithelial cells serves as positive evidences of choluria. There are many clinical tests by which bile in the urine may be detected, but they are also reactions for other organic substances, a fact that further emphasizes the importance of the color of the froth and of the staining of microscopic organic cells as valuable diagnostic points.

Clinical Significance.—Choluria results when there is any interference with the hepatic circulation or with the flow of the bile through the

hepatic ducts or through the common bile-duct, or when the hepatic cells are diseased. Choluria develops during the course of cholelithiasis, cholecystitis, parasitic disease of the liver (echinococcus cyst, ascaris infection, liver flukes), hepatic abscess, carcinoma of the common bile-duct, gall-bladder, or head of the pancreas, and duodenal catarrh. Pressure of new-growths upon the liver or upon its ducts may cause choluria.

Test.—In the presence of bile the urine froth is yellow. (See tests for bile in gastric fluid, p. 473, also 464.)

ACETONE.

In the progress of diabetes mellitus acetone, diacetic acid, and β-oxybutyric acid are found in the urine. Acetone is the first of these bodies to appear; when from 0.4 to 0.5 gram is present in the twenty-four-hours' urine, diacetic acid may also be found; but β-oxybutyric acid does not usually occur until the amount of acetone exceeds one gram. After the β-oxybutyric acid appears it is the substance to which the increase in these acid substances is chiefly due. Sometimes the β-oxybutyric acid excretion reaches as high as 180 grams in twenty-four hours, while acetone and diacetic acid together rarely exceed 7 or 8 grams.

Acetone.—250 c.c. of urine and 5 c.c. of strong sulphuric acid are placed in a distilling flask and 5 c.c. or 10 c.c. of distillate are collected in a test-tube (a condenser is not necessary). The distillate is rendered alkaline with 10 per cent. sodium hydroxid solution and a few drops of Lugol's solution are added (iodin, 1.0; potassium iodid, 2.0; water, 300). In the presence of acetone yellow crystals of iodoform are produced which have a characteristic odor.

Another but less satisfactory test is the sodium nitroprussid test. A quarter of a test-tube of filtered urine is mixed with an equal quantity of freshly prepared dilute solution of sodium nitroprussid and five drops of acetic acid are added. A 10 per cent. solution of sodium hydroxid is then added, one drop at a time, and, in the presence of acetone, a strong purple color is produced.

Significance.—Acetone may appear in the urine when the patient is suffering from any disease accompanied by high temperature, such as typhoid fever, scarlet fever, pneumonia, measles, and smallpox. The condition is known under such circumstances as *febrile acetonuria*. *Diabetic acetonuria* is the most common and most grave form. Acetonuria occurs sometimes in cases of carcinoma independent of inanition.

It occurs in persons who are not sufficiently nourished, consequently in cases of gastric ulcer in which sufficient nutriment is not being absorbed. It is seen in certain of the psychoses, in digestive derangements, as an expression of autointoxication, and in chloroform narcosis.

Diacetic Acid.—A small quantity of filtered urine is treated with a 10 per cent. solution of ferric chlorid. If a white precipitate of phosphates forms, it should be filtered out and more ferric chlorid solution added. In the presence of diacetic acid a Bordeaux red color is produced. Salicyluric acid gives the same color reaction with solution of ferric chlorid; but salicyluric acid is not volatile, while diacetic acid is volatile. Consequently, when this test is positive upon first application, a fresh portion of the urine should be boiled briskly, and, after cooling, the ferric chlorid solution should be added. If in this specimen the Bordeaux red color fails to appear, the original reaction was due to diacetic acid. If the

Bordeaux red color develops in this boiled specimen, salicyluric acid is present.

β-Oxybutyric Acid.—The tests for β-oxybutyric acid are too complex and require too expensive instruments for use in the laboratory of the general practitioner, and consequently are not described in this work. In a case of diabetes mellitus, however, the presence of acetone and diacetic acid in the urine in increasing amounts, as shown by the intensity of the reactions, may be taken as an indication of the existence of β-oxybutyric acid and of the imminence of coma.

OXALURIA.

Consideration.—The oxalic acid in normal urine is probably derived from two sources: as the result of a vegetable diet and from tissue destruction. Oxalic acid is also produced by oxidation of uric acid and from the imperfect oxidation of carbohydrates. Under normal conditions from 10 to 20 mgm. (0.31 grain) of oxalic acid are excreted daily. Oxalic acid, when present in pathologic amounts, may be detected by the microscope, the envelop crystals being found. Should no crystals collect in the sediment, cautiously neutralize the urine by adding a few drops of ammonia, and stand the specimen aside for a few hours, when a copious sediment, rich in crystalline calcium oxalate (Fig. 262), results if oxalates are present in the urine in excess.

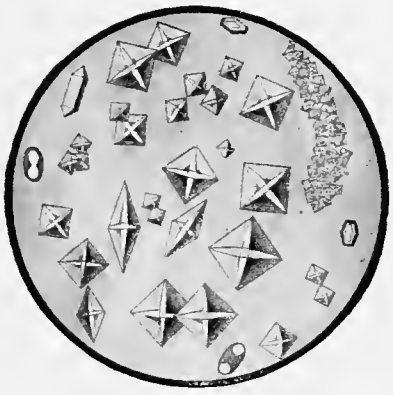


Fig. 262.—Calcium Oxalate Crystals (Jakob).

Increase.—A pathologic increase in the oxalic acid excreted with the urine is observed after the ingestion of large amounts of certain vegetables, among which should be mentioned spinach, carrots, tomatoes, string-beans, celery, onions, rhubarb, and asparagus. Apples and grapes cause a similar increase. Oxaluria often accompanies gastro-intestinal derangements, and this in all probability depends upon the imperfect oxidation of carbohydrates.

Calcium oxalate is often present in the urine during the course of chronic diseases of the skin. Localized erythematous areas affecting the backs of the fingers, the nose, the eyes, the lips, and, rarely, portions of the chest and abdomen, disappear when oxaluria subsides as the result of treatment. Decided itching of the skin, particularly at the junction of the skin with the mucous membranes, appears to be occasioned by the excretion of oxalic acid. At times these cutaneous manifestations of oxalic acid intoxication are most pronounced when the amount of oxalic acid excreted daily is comparatively low.

Calcium oxalate is a common constituent of the urine during the course of gastro-intestinal derangements, and particularly during the course of chronic gastritis. Oxaluria is also influenced by obstinate constipation. It must be remembered that oxaluria is most common in early adult and middle life, although we have repeatedly found it present in children under ten years of age and in those over sixty. In our experience oxaluria is unusually

common in those cases in which there is oxalate calculus in the bladder or in the pelvis of the kidney; a few such instances have come under our observation.

The urine of hemophiliacs is often heavily charged with calcium oxalate, and when these patients are unable to take sufficient exercise, the oxaluria can be relieved only with difficulty. In fact, the oxaluria increases with the development of a ravenous appetite.

Course.—In practically all cases the condition is relieved by treatment. The real cause of oxaluria is commonly found in the gastro-intestinal tract or in the faulty hygiene of the patient, particularly as regards his exercise and diet. Oxaluria, when permitted to exist over a long period, is likely to irritate the kidney sufficiently to cause mucous cylindroids to appear in the urine. In fact, we have found cylindruria to accompany oxaluria more often than any other pathologic condition of the urine in which there is no true nephritis.

LEUCINURIA AND TYROSINURIA.

Consideration.—Leucin and tyrosin appear in the urine as a result of the decomposition of albumins. These substances are to be found in the urine of persons suffering from acute yellow atrophy of the liver, acute phosphorus-poisoning, and during the course of the severer forms of small-pox, yellow fever, typhoid fever, and other maladies in which the decomposition of proteids is rapid. The detection of leucin and tyrosin crystals in the urine of pernicious anemia, leukemia, and septicemia is an occasional finding. Leucin and tyrosin are, as a rule, found in the same urine, and may be precipitated out as characteristic crystals (Fig. 263).

Characteristics of Crystals.—Crystals of tyrosin are soluble in both acids and alkalis.

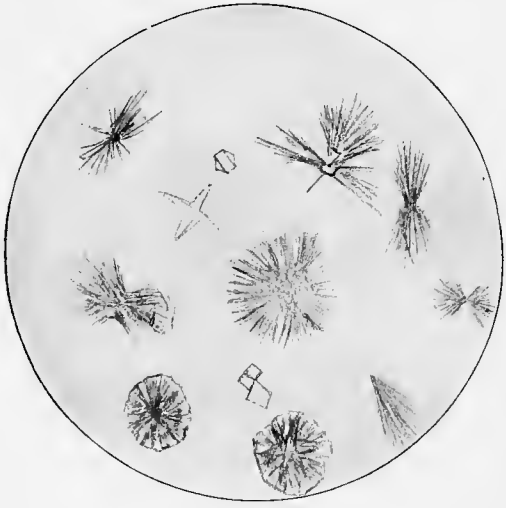


Fig. 263.—Leucin Discs and Tyrosin Crystals (Boston).

Differentiation.—Crystals of tyrosin must be distinguished from the crystals of acid and neutral phosphates. Crystalline tyrosin resembles fat crystals to some extent, from which it is distinguished by the fact that tyrosin is not dissolved in ether, whereas fat is.

CYSTINURIA.

Consideration and Significance.—The presence of large numbers of cystin crystals in the urine is an extremely rare finding, but these crystals, when present, cause a milky-white sediment to appear. Cystinuria has been known to occur in several members of the same family.

Recognition.—Place a drop of the sediment on a slide, and examine under a $\frac{1}{6}$-inch objective, when, if cystin is present, the characteristic crystals (Fig. 264) will appear. These crystals are soluble in ammonia and are reprecipitated by acetic acid, but remain unchanged upon treatment with ether, water, and alcohol.

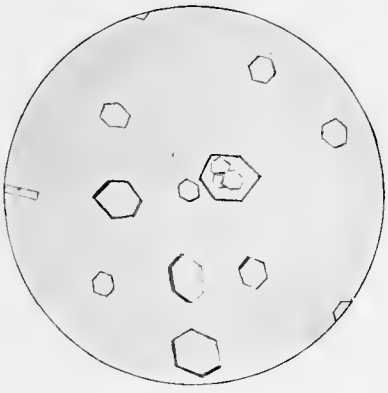


Fig. 264.—Crystals of Cystin (Boston).

CHOLESTERINURIA.

Cholesterin is seldom found in the urine unless a mixture of chyle or of fluid from hydatid, ovarian, or other cysts has been added. Rarely, cholesterin is found during the course of chronic cystitis and in acute nephritis. Cholesterin is commonly found in the fluid from cysts, abscesses of the liver, and fluid from the serous sacs.

Recognition.—Cholesterin is recognized by the appearance of the

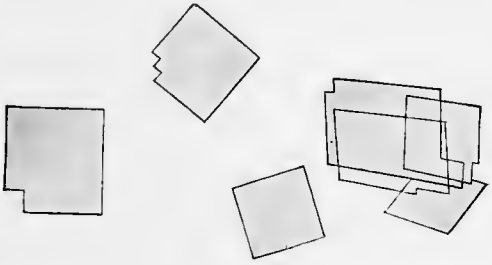


Fig. 265.—Cholesterin Crystals (Ogden).

urine; *e. g.*, upon shaking the bottle containing such urine small, snowflake-like bits are seen floating through the fluid. Microscopically, we find the specimen rich in characteristic crystals (Fig. 265).

INDICANURIA.

General Consideration.—Indican appears in the urine in pathologic amounts in the form of indoxyl-potassium sulphate. It is said to be formed during the decomposition of an excess of albuminous material.

Although the amount of indican eliminated with the urine daily is to a certain degree controlled by the character of the diet taken, Gaffé has fixed the normal excretion at 6.6 mgm. for each 1000 c.c. of urine. It will be found that Gaffé's figures are entirely too low for healthy individuals who live upon a diet rich in animal foods.

Significance.—Indoxyl-potassium sulphate and indoxyl-sodium sulphate are the particular forms of indican present in pathologic urines.

(*a*) Indicanuria is a feature of chronic intestinal obstruction, and while in this instance it is probably due to stagnation of the contents of the intestines, some observers believe it to result from the action of the colon bacillus and other bacteria. Indicanuria is a feature of chronic constipation, acute peritonitis, wasting diseases, such as, *e. g.*, dysentery, cholera, Addison's disease, carcinoma, and other affections. It is absent in diseases of the pancreas.

Indican is found in pathologic amounts in practically all diseases in which a high degree of intestinal putrefaction exists. Clinical observation points strongly to the fact that the amount of indican bears a more or less close relation to the acidity of the gastric juice, and an increase in the amount of urinary indican is often associated with hypochlorhydria. We would suggest that in gastro-intestinal conditions the indican and the hydrochloric acid be studied correlatively. The special diseases in which this variety of indicanuria is seen are gastric carcinoma and ileus.

Exceptions to the foregoing rule are occasionally encountered, *e. g.*, an excessive amount of indican is found in acute and subacute gastritis, and is not infrequently seen in gastric ulcer, a condition in which hyperchlorhydria is present. The maladies in which intestinal putrefaction takes place are so numerous that it appears unwise to mention each condition in which indicanuria may occur. We repeat that the degree of indicanuria is, with few exceptions, an index to the digestive power of the stomach.

(*b*) Diminished peristalsis contributes toward the elimination of an increased amount of urinary indican, in consequence of which indicanuria is the rule in acute and chronic peritonitis and in ileus. It should be borne in mind that the increased production of indican often depends upon some abnormality of the small intestine, and that pathologic indicanuria is not a feature of uncomplicated constipation. Gastroptosis and enteroptosis may be present when indicanuria is a prominent symptom, but either of these conditions may influence both the secretion of hydrochloric acid and the peristalsis of the small intestine, which makes it difficult to determine the exciting cause of indicanuria in these peculiar misplacements of the viscera.

(*c*) In accord with the view formerly held, that albuminous putrefaction results in indicanuria, we find it a feature of pulmonary gangrene, gangrene of the extremities, emphysema, puerperal sepsis, and in certain of the acute infectious diseases. For reasons which are not readily explained indican is often present when the leading primary feature is oxaluria.

Test.—To 10 c.c. of filtered urine add one drop of a 1 per cent. potassium chlorate solution, then 5 c.c. of chloroform, and, lastly, 10 c.c. of pure fuming

hydrochloric acid (specific gravity, 1.19). It is necessary that the reagents should be added in the order given.

Mix thoroughly by pouring repeatedly from one test-tube to another. By this method the indican (indoxyl-potassium sulphate) is oxidized to indigo, which dissolves in the chloroform and imparts a blue color to it. In about ten minutes the maximum coloration has been reached, and the whole should again be thoroughly mixed. The chloroform will be colored more or less blue according to the amount of indigo set free.

If the urine contains iodids, the chloroform will be colored violet. This color may be removed by adding three drops of a 5 per cent. aqueous solution of sodium thiosulphate, whereupon the blue coloration will appear.

Indigo appears in the urine in the form of amorphous débris, in which there are fine, needle-like crystals that possess a variable degree of blueness. The reaction of the fresh urine has little, if any, effect upon the formation of these crystals, although they are commonly present in decomposing urines.

Diazo-reaction.—A reaction commonly present during the course of high fever and dependent upon the presence of a chromogen in the urine.

Reagents.—(1) A solution of sulphanilic acid (1 gm. to every 100 c.c.) in 5 per cent. hydrochloric acid.

(2) Solution of sodium nitrate, ½ per cent. It is necessary that both solutions be fresh.

Method.—Place 10 to 20 c.c. of urine in a test-tube and to it add an equal volume of solution No. 1, shaking gently to effect a perfect mixture; then add from 3 to 6 drops of solution No. 2, and shake until a heavy froth collects. Render alkaline with ammonia. The diazo-reaction consists in the liquid becoming a port-wine color; the froth is also red.

Clinical Significance.—The diazo-reaction is a fairly constant symptom of typhoid fever after the end of the first week of the disease. We have found it present in cases of measles, tuberculosis (with cavity), meningitis, croupous pneumonia, in a number of obscure conditions with high fever; and less often in scarlet fever, acute miliary tuberculosis, erysipelas, pyemia, diphtheria, puerperal sepsis, and tonsillitis. The value of this reaction in diagnosis is limited.

DISEASES OF THE KIDNEY.

ACUTE NEPHRITIS

(ACUTE PARENCHYMATOUS NEPHRITIS; ACUTE GLOMERULONEPHRITIS).

Pathologic Definition.—An acute, diffuse inflammation of the kidney, which may vary greatly as to severity, duration, and extent of destruction of renal tissue. Special varieties of acute nephritis, as, for example, acute degenerative, exudative, and productive, have been described. For clinical purposes we shall consider them all as acute nephritis. Reference may be made to the types of acute inflammation attacking the kidney substance, such as acute tubular, acute glomerular, and acute diffuse nephritis, although it is impossible to distinguish clinically between these pathologic subvarieties. The microscopic appearance of the kidney will be found to vary greatly, depending upon the severity and extent of the infection, but in the average case, however, the organ is slightly enlarged, swollen, and appreciably softened. Before the kidney is sectioned it dis-

plays a somewhat reddened or bluish appearance, and there is a distinct mottling of its surface. Occasionally there are minute hemorrhages beneath the capsule (acute hemorrhagic nephritis). The kidney cuts with ease, and the cut surface of the parenchyma shows decided mottling, while the pyramids are intensely reddened. The capsule strips with ease.

Microscopically, there is infiltration with intrusion upon the tubules, and the Malpighian tufts also show inflammatory changes. Later, cellular necrosis, and also fatty degeneration, may be present.

Predisposing and Exciting Factors.—Age.—This disease occurs at all ages, but is more common in children and during early adult and middle life. Owing to the fact that exposure and contagious diseases are most frequent during the first forty years of life, acute nephritis is likewise more common then.

Sex.—Acute Bright's disease is encountered oftener in males than in females, owing to the fact that males are more exposed to cold and wet.

Heredity is often an important predisposing factor in nephritis; we have in mind a family in which the father died of acute nephritis at the age of thirty-seven; two sons and a daughter developed acute Bright's disease between the ages of twenty-five and thirty, and died before reaching the age of thirty-three; a third son suffered his first attack of acute nephritis at the age of twenty-nine.

Among the *exciting causes* are: (*a*) Both acute and chronic cutaneous diseases, as, *e. g.*, conditions in which the skin is inactive either from disease or from exposure to cold and following extensive burns.

(*b*) *Chemic Causes.*—Either excessive doses or the prolonged use of any one or more of the following drugs may result in the production of acute nephritis: Ether, as in prolonged ether anesthesia, phenol, salicylic acid, iodin, the iodids, turpentine, phosphorus, lead, arsenic, mercury, and potassium chlorid. The kidneys may also be irritated as the result of the ingestion of certain adulterated foods.

(*c*) *Biologic Causes.*—Under this head we must consider the poisons resulting from the development of bacteria within the human economy, as is well exemplified in nephritis complicating scarlet fever (second or third week of convalescence), typhoid fever, relapsing fever, cholera, dysentery, pneumonia, diphtheria, rheumatism, and allied conditions. Septicemia, septicopyemia, and severe pyogenic infection, such as is seen during the course of pulmonary tuberculosis with cavity formation, are also capable of exciting an acute inflammation of the kidneys. Rarely, indeed, nephritis follows measles, chicken-pox, syphilis, and a single attack of malaria, but it must be remembered that repeated infection with the malarial parasite is productive of true nephritis and of hematuria and hemoglobinuria.

(*d*) *Traumatism to the kidney,* and at times traumatism to the trunk and the extremities, is followed by acute nephritis.

(*e*) *Pregnancy.*—Nephritis may develop at any time during gestation, but it is far more common in primiparæ after the seventh month.

(*f*) Latent and insidious chronic nephritis may be the cause of the onset of acute nephritis (Anders).

Principal Complaint.—The patient's description of his illness will vary greatly, depending upon the grade of nephritis present, but, as a rule, he will be found to complain of chilliness or of a series of chilly sensations, slight pains in the loins, with nausea, decreased appetite, and at times vomiting. Within the next twenty-four hours there will be a variable degree of headache, edema, and mental apathy. In children, the onset not infre-

quently begins with a convulsion, the child having been apparently well up to this time. The patient early observes the characteristic swelling of the ankles and puffiness beneath the eyes, and in severe cases the edema of the skin becomes quite general. Epistaxis and conjunctival hemorrhage are among the less common symptoms.

Thermic Features.—The temperature begins to rise after the prodromal symptoms, gradually reaching about 100° to 101° F.; it is of an irregular type, declining to the normal whenever the acuteness of the renal inflammation subsides. Fever, while usually present, is by no means a constant feature of acute nephritis, although in severe cases the temperature may occasionally reach 102° to 104° F.

In mild cases the nephritis is not detected until an examination of the urine is made; we have seen many cases in which malaise was the only other symptom present.

Physical Signs.—Inspection.—In mild cases of acute nephritis inspection is negative, but later slight edema beneath the eyes and at the ankles may be detected. In moderate and in more severe types of the disease the skin is pale, and there is swelling of the feet, ankles, and fingers, with edema of the eyelids and face, all of which vary greatly with the severity of the type of disease present. General anasarca may follow, at which time there is edema of the scrotum, prepuce, and labia. The face may be so distorted as to make recognition impossible.

Palpation.—There is pitting about the ankles, and over all edematous tissues the skin is dry, and at times rough to the touch. The cardiac impulse is forcible, and the apex-beat is usually diffuse. The pulse is accelerated, except when uremic toxemia has developed, when it will be found to be slow and of high tension. When repeated attacks of acute nephritis have occurred, the heart is hypertrophied, and the apex-beat is found below the fifth interspace and to the left.

Percussion.—There may be evidence of the presence of fluid in the pleural sacs; this is manifested by bilateral flatness, which extends only to the top of the fluid, and above about this point there is compensatory hyperresonance. It is ofttimes possible to detect fluid in the peritoneum, and hydropericardium may be present, which gives an increased area of cardiac dullness. This area is conoid in outline, the apex being directed downward. Not infrequently the area of cardiac dullness is increased, owing to hypertrophy of the left ventricle, but this area differs from that caused by pericardial effusion in that the apex of this triangle is directed downward and to the left. In extreme cases the triangular area of dullness is directed downward.

Auscultation.—Early during the course of acute nephritis the heart-sounds are clear and forcible, and their frequency is slightly increased, but after uremia has developed they are slow, clear, and the second aortic sound is decidedly accentuated. During the course of unfavorable and fatal cases the heart-sounds become very rapid, feeble, and intermittent, but it must be remembered that slow cardiac action is characteristic of uremia before cardiac dilatation has developed.

The respiratory murmurs are at first apparently normal; later the respirations may become slightly accelerated, but until circulatory embarrassment and edema of the lungs develop, the breath-sounds are clear. The respirations become slow and shallow at first, but as the disease advances, or after cardiac dilatation supervenes, they become feeble, rapid, and are accom-

panied by numerous bubbling râles. Cheyne-Stokes breathing occurs late.

Nervous Manifestations.—Headache, mental dullness, and twitching of the muscles are commonly seen. Paroxysmal vomiting, difficulty in speaking, marked by thickness of the voice, the floating of specks before the eyes, vertigo, and even convulsions may develop during the course of acute nephritis. When the condition is that of an acute exacerbation of a chronic nephritis, localized paralyses, involving most often the muscles of the arms, face, or eyes, are to be seen. These paralyses are shifting in character, and may disappear promptly upon the administration of the proper remedies, only to reappear and involve some other portion of the body—the so-called shifting paralyses of Bright's disease. Maniacal delirium is occasionally present, and is at times the initial symptom of an acute exacerbation of chronic nephritis. Intense headache and backache may precede the onset of uremia. Uremic coma is quite characteristic, and is to be distinguished from coma due to other causes. (See Differential Table.)

Special Symptoms.—The productive type of acute nephritis most often develops during the course of some other infectious malady, in which case the symptoms of acute nephritis are added to the symptoms of the initial disease; *e. g.*, in typhoid fever the development of a high temperature, maniacal delirium, and the urinary phenomena of acute nephritis are suggestive of this complication, and there may be added dyspnea, diarrhea, vomiting, and coma.

Laboratory Diagnosis.—The amount of perspiration excreted is decidedly lessened during the course of acute nephritis, and the more active the inflammatory process in the kidneys the more reduced is the activity of the sweat-glands.

The Urine.—In mild cases the quantity of urine excreted is moderately diminished, but with the advance of the disease it gradually lessens until, in severe types of nephritis, but a few ounces of fluid may be voided during the twenty-four hours, and, in fact, there may be anuria.

Naked-eye Study.—The urine is of high color, cloudy, acid in reaction, of high specific gravity, and deposits a heavy precipitate upon standing. In hemorrhagic nephritis it is bloody, and a dark-red sediment collects upon standing. Should the hemoglobin escape with the blood-serum into the urine, the urine is of a bloody hue.

Microscopic Study.—In mild cases of nephritis the urinary sediment will be found to contain granular and hyaline casts, a few red blood-cells, and many leukocytes. (Plate XIV.) In severe cases, in addition to the findings just mentioned, there are present renal epithelium, blood-casts, many red blood-cells, and blood-pigment.

Chemistry.—The urine will be found to contain large amounts of albumin, the quantity of which fluctuates with the degree of irritation present in the kidney and with the amount of urine voided during the twenty-four hours. Such urines may give a reaction for hemoglobin.

The inorganic constituents of the urine are lessened in acute nephritis, but during convalescence from such attacks these substances reappear in the urine in abnormally large amounts, the increase, however, being of but short duration.

In mild cases of acute nephritis the *blood-findings* are of little if any clinical value, whereas in nephritis complicating other diseases the blood-findings of the primary disease are present. In severe nephritis in which uremia is well established the blood flows sluggishly from the site of puncture in the

PLATE XIV

Various Forms of Urinary Casts (Boston): 1. Hyaline casts from case of puerperal eclampsia (original) (obj. B. and L. one-sixth).

2. B. J., age twenty-two, female, suffering from puerperal eclampsia. Urine showing large, finely granular casts (original) (obj. Queen one-sixth; eye-piece 2).

3. S. A., age fifty-eight, male. Urine showing granular and fatty casts; post-mortem showed chronic parenchymatous nephritis (original) (obj. Queen one-sixth; eye-piece 4).

4. J. D., age fifty-four, male, suffering from cancer of the common duct and head of the pancreas. Urine showed bile-stained casts (original) (obj. Queen one-sixth; eye-piece 4).

5. A. G., age fifteen, male, suffering from acute nephritis. Urine showed granular casts (original) (obj. Queen one-sixth; eye-piece 2).

6. C. A., age nine, male. Scarlatinal nephritis, third week of convalescence. Urine showed granular casts (original) (obj. Queen one-sixth; eye-piece 2).

skin and is dark in color. The percentage of hemoglobin is often above the normal, as is also the number of red cells in a cubic millimeter, but this increase is dependent upon the presence of general cyanosis. When nephritis accompanies certain acute infectious maladies, such as typhoid fever, microörganisms may be found in the blood in great numbers, and are likewise present in the urine. Judging from the cases reported, no standard as to the degree of importance to be attached to the demonstration of bacteremia and bacteriuria can be established.

Duration of the Disease.—When acute nephritis does not occur during the course of some other disease, it is of but short duration, running its course in from a few days to a few weeks; but if the patient's resistance is undermined by some infection, the nephritis may last longer and may tend toward chronicity. In repeated attacks of acute nephritis each successive attack lasts longer than did the preceding one.

Illustrative Case of Acute Nephritis Followed by Chronic Parenchymatous Nephritis.—B. A., male, aged twenty-seven years; height, 5 feet 8½ inches; weight, 140 pounds, for the past five years.

Family History.—Parents born in Ireland. Father living at fifty-six; mother in apparent health at fifty. No definite record could be obtained with reference to nephritis in either paternal or maternal ancestors. An older brother, however, developed nephritis at the age of twenty-nine and died at thirty-three; another brother, two years older, developed nephritis at twenty-seven and died at the age of thirty years. One sister living and is apparently in good health at the age of twenty-two years.

Previous History.—The patient had the diseases of childhood. He suffered from diphtheria at the age of twelve, from which he made a complete recovery. He had malaria at the age of twenty-two, but at this time there were no evidences of renal disease. On account of the peculiar tendency his brothers have shown to develop nephritis he has had his urine examined at periods of a month for the past five years, and it has never contained either albumin or casts, although it has always been of low specific gravity—1.010 to 1.015.

Social History.—Single, student of medicine, senior class. Habits good; has never been addicted to the excessive use of alcohol or tobacco, and for the past five years has avoided undue exposure to cold and wet. He has been somewhat nervous, and has shown considerable mental anxiety since his second brother contracted renal disease.

Present Illness.—About one month ago he contracted a cold resembling grip, and was confined to the house for a period of one week, but at no time was he compelled to remain in bed during the entire day. As he was thoroughly familiar with the subject of urinalysis, he examined his urine the third day of the cold, and found that it gave a feeble reaction for albumin. Extreme prostration, unusual thirst, and constipation were also present.

Headache was annoying during the onset of the attack, and disappeared by the end of the first week, since which time it has occasionally occurred after a night's sleep.

Cough was at no time severe, although it continued for one week from the beginning of the attack. As the disease advanced it became more annoying, and within the course of one year he suffered from headache a portion of each day. At times he complained of vague pains over the loins and muscles of the limbs.

With the onset of the disease the temperature rose during the first twenty-four hours to 101° F., and ran an irregular course, fluctuating between the normal and 100° F., until the fifth day of the disease, when there was a moderate decline, and by the middle of the second week the temperature was normal, and remained near this point at the morning hours throughout the illness.

An *ophthalmoscopic examination* made eight months following the initial attack revealed the existence of albuminuric retinitis and retinal hemorrhage, distinct impairment of vision developing about six months later.

Physical Examination.—*General.*—When first seen, the general appearance of the patient was quite identical with that usually displayed by those suffering from an attack of influenza. (See Illustrative Case of Bronchopneumonia, p. 110.) Following convalescence from the cold the patient did not gain flesh nor strength, but, on the contrary, there appeared to be progressive weakness, with slight loss in weight. The patient was readily exhausted after slight exercise, and there were restlessness, inability

to sleep during the night, and mental hebetude during the day. The skin and mucous membranes were pale, the cheeks presented a peculiar baggy appearance, and there was puffiness beneath the eyes.

Palpation.—The skin was dry, somewhat roughened, and there was distinct pitting (edema) in the malleolar regions. The pulse was full, strong, and of high tension, the beats numbering 80 a minute. Repeated estimations of the blood-pressure showed it to be decidedly above the normal. The heart impulse (apex) was forcible, and best felt about two inches below and just outside the left nipple. Systolic blood-pressure was 154.

Percussion.—The area of cardiac dullness was somewhat increased downward and to the left.

Auscultation.—The first sound of the heart was loud and booming in quality.

Laboratory Findings.—During the first three days of the acute, grip-like cold the urine contained only a trace of albumin. By the sixth day there was a decided reaction for albumin, and the microscope revealed the presence of numerous pale, hyaline casts following the cold, and for a period of six weeks casts became more numerous; at this time some of them were slightly granular in appearance. Many leukocytes and a few erythrocytes were also present. The specific gravity of the urine fluctuated between 1.010 and 1.016 for a period of about six months, when the specific gravity was found to be increased, ranging between 1.020 and 1.025. At first the total quantity of urine excreted approximated fifty to sixty ounces for the twenty-four hours, but as the disease advanced there was a progressive lessening in the quantity of urine, until the renal condition had merged into the chronic parenchymatous type, when the urine excreted did not exceed twenty ounces a day. With the course of the disease the urinary findings underwent a change, and at length were typical of those described for chronic parenchymatous nephritis. (See p. 671.) The amount of urea excreted during the twenty-four hours was slightly below that of normal at the first examination, and there was a noticeable progressive decrease in the amount of urea excreted throughout the course.

The *hemic* changes were those of a chloranemia, and a blood examination made at the sixth week of the illness showed: hemoglobin, 72 per cent.; red cells, 3,600,000; white cells, 7200. After the disease had advanced to the stage of chronic parenchymatous nephritis, the degree of anemia became more pronounced.

Diagnosis by Induction from Clinical Data.—Here the clinical history was of unusual importance, and after obtaining this, we immediately proceeded to make an analysis of the urine, which gave findings characteristic of nephritis.

Course of Illustrative Case.—During the first three weeks of the illness the clinical course resembled somewhat that of obscure influenza, but convalescence was protracted and albuminuria persisted. The patient did not regain strength, as is ordinarily the case following an acute cold or an attack of influenza, and he continued to lose weight for from three to six months, when the characteristic symptoms of chronic parenchymatous nephritis developed. Treatment materially modified the quantity and character of the urine after parenchymatous nephritis had developed, but during the first six weeks of the disease it had but limited effect upon this secretion. Including the stage of chronic parenchymatous nephritis, the patient's condition fluctuated between periods of improvement and exacerbations, ending in uremic coma two and one-quarter years after albuminuria was detected.

Summary of Diagnosis.—The recognition of acute nephritis depends almost entirely upon the detection of albumin and of casts in the urine, since these two findings afford positive evidence of the existence of the disease. Dryness of the skin, thirst, parched lips and tongue, and constipation are early manifestations of the disease. Later, the quantity of urine is decreased, and headache, mental dullness, and even stupor may supervene. In children nausea, vomiting, chilly sensations, and headache appear to be among the cardinal complaints, whereas in the adult there are usually stiffness and soreness of the muscles, slight pain in the back, and general malaise.

Course and Gravity of Disease.—The prognosis is dependent, to a great extent, upon the primary disease or causal factor of the nephritis, as well as upon the degree and character of the renal inflammation. When nephritis is the result of exposure to cold and wet, a permanent recovery is likely to ensue. Postscarlatinal nephritis is far less likely to be followed by

permanent restoration of the function of the kidney than is the previously described variety. In the acute infections (typhoid fever, diphtheria, etc.) and in pregnancy the acute parenchymatous degenerative type of renal infection is present, and recovery is the rule. Occasionally one encounters a virulent type of renal infection during the course of some other infectious disease, and this grade of nephritis is not unusual in acute yellow atrophy of the liver, cholera, and following poisonous doses of mercury, phosphorus, etc. In this last class of cases the manifestations of renal insufficiency are grave, and the patient grows rapidly worse until uremic symptoms appear.

The factors that warrant a favorable prognosis are an increase in the quantity of urine excreted, the amount of urea and other solids being also increased, the skin, at the same time, recovering its normal color and moisture. The edematous areas disappear rapidly after the increased flow of urine, as does also the fluid that has accumulated in the serous sacs (pleura, pericardium, peritoneum).

Among the most serious symptoms of acute nephritis are edema, effusion in both pleural sacs, and such nervous manifestations as stupor, partial paralysis, convulsions, and coma. The development of complications such as pneumonia, meningitis, and pericarditis renders the prognosis unfavorable. Throughout the course of acute nephritis the prognosis is either favorable or unfavorable, depending directly upon the quantity of urine excreted during the twenty-four hours (the smaller this quantity, the less favorable the prognosis), upon the presence or absence of complications, and upon the ability of the physician to institute proper hygienic and therapeutic measures. After the renal process has assumed a productive character, the life of the patient is, as a rule, prolonged over months and at times years, but complete recovery seldom follows.

ACUTE INTERSTITIAL NON-SUPPURATIVE NEPHRITIS.

Pathologic Definition.—An acute inflammation of the kidneys, either localized or diffuse, resulting in the production of a non-suppurative exudate in the interstitial tissue, without essential accompanying degeneration of the parenchyma. The kidney is enlarged and its surface mottled. There is a distinct proliferation of the cells in the interlobular tissue, and these changes are in excess of those found in the parenchyma. The proliferative changes are especially conspicuous about the venous and capillary epithelium. Plasma cells, lymphocytes, and polymorphonuclear leukocytes are present in the exudate.

General Consideration.—Thus far the majority of recorded cases of this type of nephritis have developed after such acute infections as scarlet fever and diphtheria, although they have been known to follow typhoid fever, pneumonia, meningitis, and measles. A pathologic study of the kidneys reveals the presence of streptococci in the interstitial tissue. Some authors believe that this type of nephritis is the result of the action of powerful toxins, and that bacteria enter the kidneys secondarily.

Clinical Picture.—The characteristic clinical features of this type of nephritis are: (1) That it develops during the course of some other infection; (2) the patient rapidly enters a moribund state; and (3) edema is slight and often absent.

CHRONIC NEPHRITIS (EXUDATIVE)

(CHRONIC BRIGHT'S DISEASE; CHRONIC PARENCHYMATOUS NEPHRITIS; CHRONIC DIFFUSE NEPHRITIS WITH EXUDATION; CHRONIC TUBAL AND CHRONIC DESQUAMATIVE NEPHRITIS; CHRONIC GLOMERULONEPHRITIS; LARGE WHITE KIDNEY; SECONDARY OR FATTY AND CONTRACTED KIDNEY).

Pathologic Definition.—A diffuse, chronic, inflammatory process, involving both kidneys, and characterized by epithelial degeneration, with the formation of permanent connective tissue and the escape of certain portions of the blood (serum and pigments) into the renal tubules.

Several types of pathologic kidney are present in this disease, but the distinctive differences as to size, etc., are dependent upon the varying causal factors and the stage and duration of the case in question.

(1) The large white kidney (without amyloid degeneration) is either enlarged or of normal size, and of a pale or yellowish color. The surface of the organ is smooth, and its capsule strips with ease. The cut surface displays a yellowish-white color throughout, with certain opaque areas and here and there some mottling with red. Microscopically, the destructive changes are pronounced; the renal epithelium is swollen; and hyaline, granular, and fatty degeneration is conspicuous. The glomeruli are appreciably enlarged, owing to overgrowth of the capsular epithelial cells. The interstitial tissue is seen to be increased.

The small white kidney is generally believed to be but the result of a later stage of the preceding variety, in which, owing to advanced degeneration and overgrowth of connective tissue, contraction has taken place. The organ, in addition to being small, is firm and resistant to the knife, and its capsule is adherent. The cut surface is grayish or yellowish in color, and at times mottled. Distinct foci of fatty degeneration are usually disseminated throughout the cortical portion of the organ.

The large red kidney, in addition to being swollen and congested, or mottled, frequently shows distinct irregularities or humping on its surface. Here the capsule is somewhat adherent, especially at the points of indentation. The cut surface of the organ also shows many irregularities, and at times slight hemorrhages, and its mottling is shown microscopically to depend upon various stages of degeneration. In certain respects the microscopic changes simulate those described for the large white kidney.

Varieties.—This type of nephritis has been divided into several varieties, according to the peculiar grade of pathologic change present in the kidney; in our experience it has been found generally difficult, and sometimes impossible, to ascertain, either by an examination of the urine or by other clinical methods, the exact type of kidney present.

Predisposing and Exciting Factors.—Age.—Chronic parenchymatous nephritis may be found at practically all ages, but it is especially common in children who have suffered from scarlatina, as well as in young adults, and the disease is by no means uncommon during the fourth, fifth, and sixth decades. The age at which both men and women are subjected to hard work and exposed to cold and wet appears to be the most susceptible period.

Persons who eat heavily of rich foods and who take but a moderate amount of exercise are especially prone to this malady, as are also those who imbibe too freely of alcohol, beer, malt, and other intoxicating liquors. Exposure to cold and wet, and employment in which the temperature is extremely high, as in the case of firemen, workers about furnaces, etc., are

predisposing factors—dependent either upon the intense heat or upon the sudden change of temperature in passing from a hot into a cold atmosphere.

The toxins of acute infectious diseases excite in themselves an acute nephritis that becomes subacute, and eventually chronic, in nature. Practically all conditions that favor the development of acute nephritis figure prominently in the etiology of chronic parenchymatous nephritis.

Climate is believed to contribute slightly toward the development of this disease, and it is said that the disease prevails in humid and marshy localities. We have observed it in certain persons living in regions known to be highly malarial. Persons suffering from any malady in which chronic suppuration is a feature are especially likely to develop this type of nephritis; this variety has been considered under the head of amyloid disease of the kidney.

Principal Complaint.—The symptoms present in acute nephritis are nearly all present during the course of chronic parenchymatous nephritis,

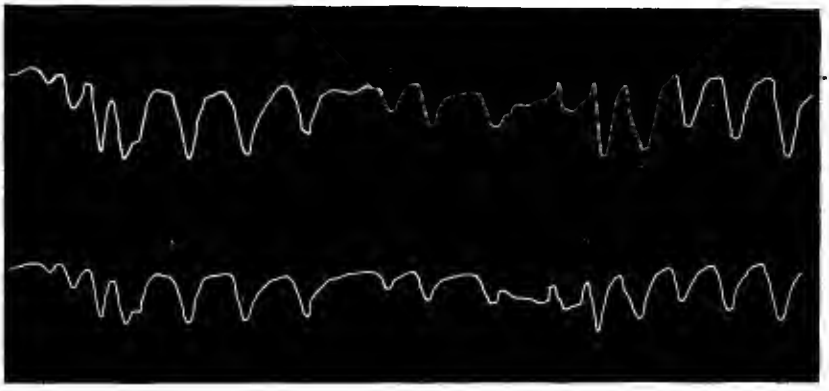


FIG. 266.—BILATERAL PNEUMOGRAM SHOWING RESPIRATORY ARRYTHEMIA, FROM A CASE OF UREMIA STUDIED AT THE PHILADELPHIA GENERAL HOSPITAL.

Note especially the great variations in amplitude of the curve, also variations in time of the respiratory movements (distance between summits). (See Movements of Chest, p. 131.)

although they are less conspicuous, since each individual symptom is not so acute, but more persistent. Headache is a conspicuous symptom in this type of nephritis, and is present every day, in various degrees of severity, affecting either the frontal or the occipital region. There are also dull, wandering pains in the muscles and progressive weakness, which eventually becomes extreme. Nausea is commonly experienced, and the appetite is lessened at the onset of the disease and lost entirely in extreme cases. Paroxysmal attacks of indigestion, accompanied by headache, mental dullness, sleeplessness, and vertigo, occur. Coma may follow a gastric attack. In many cases we have observed an inordinate appetite for eggs and other albuminous foods.

With the progress of the disease the patient observes that he cannot open his eyelids widely when he awakens in the morning, and that there are also present bagginess of the skin of the cheeks and swelling of the ankles, which latter symptom increases rapidly until anasarca appears. The swelling of the face present on rising in the morning lessens during the day, whereas the swelling of the feet is less evident in the early morning hours and becomes more apparent after the day's exertions.

Dyspnea is a common complaint, and becomes more marked as the disease advances. It may be toxic and nervous in character, or it may depend upon mechanic interference with the pulmonary circulation. Cardiac dyspnea, which is dependent upon faulty action of the heart, is always present in the course of chronic parenchymatous nephritis, and is aggravated on assuming the recumbent posture. Dyspnea not due to cardiac insufficiency may be dependent upon vasomotor constriction, and should be regarded as of serious prognostic moment; indeed, we have studied cases of chronic nephritis in which renal asthma was the chief complaint; for this reason an analysis of the urine should be made in all cases of asthma.

Catarrhal bronchitis is frequently associated with this form of Bright's disease. Without doubt it contributes toward the dyspnea and cough, as well as to the free expectoration.

The patient's discomfort is materially increased by the presence of complications, among which should be mentioned retinitis with failing vision, diseases of the skin, pericarditis, endocarditis, pneumonia, and colitis. Certain of these conditions may give rise to fever, which is not present in uncomplicated chronic nephritis. The symptoms common to these complicating maladies are added to those of the primary nephritis.

Physical Signs.—Inspection.—During the first few months inspection is practically negative, but as the disease advances there are decided pallor of the skin, edema of the face, eyelids, and extremities, particularly of the ankles, and prominence of the superficial veins of the face, calves of the legs, abdomen, and chest; small red blotches may also appear on the skin. The hair and that portion of the skin covered by clothing are lusterless, slightly roughened, and covered with fine scales, which are seen to surround the roots of the hair.

After the accumulation of fluid in the pleural, pericardial, and peritoneal sacs has taken place, there is prominence of the abdomen. The respirations are hurried, and the attitude of the patient is altered in accordance with the degree of serous exudation, in order to enable him to breathe more easily. Before pericardial effusion occurs the cardiac apex-beat is displaced downward and to the left, whereas after the accumulation of fluid in the pericardium it is seen at the third or fourth interspace and in the midclavicular line. In ascites the breathing is thoracic, and the superficial veins of the abdomen and chest are seen to be greatly enlarged. There is generally decided edema of the lower extremities and of the genitalia, as well as edema of the hemorrhoidal veins and hemorrhoids.

Palpation.—The skin is often dry and harsh, but when it is edematous, it pits upon pressure. Palpation serves to confirm inspection as to the position of the apex-beat of the heart; it is further found to be forcible early during the course of this malady, but late in the disease it is weak and irregular, and in both pericardial effusion and acute cardiac dilatation it may be scarcely perceptible. The expansion of the chest is greatly lessened when either pleural or pericardial fluid has accumulated.

In order to demonstrate the presence of free fluid in the peritoneal cavity an assistant makes pressure with the ulnar border of one of his hands in the median line of the abdomen, while the examiner, with the palm of one hand pressed against the skin of one flank, taps gently against the skin of the opposite flank with the tip of one of the fingers of his other hand. If fluid is present, a transmitted wave will be apparent to the palm of the palpating hand. Polyhydramnios, large ovarian cyst, hydronephrosis, chylous cyst, and a distended bladder may give such a wave.

Percussion.—The area of cardiac dullness is increased downward and to the left early, and it is decidedly increased in the transverse diameter, owing to hypertrophy of the left ventricle. In pericardial effusion the area of cardiac dullness is likewise enlarged, but forms a triangular area, the base of the triangle being directed downward, whereas its apex rises to the second left interspace. The three conditions that cause the area of cardiac dullness to be increased are: (1) Hypertrophy of the left heart; (2) pericardial effusion; (3) acute cardiac dilatation. These are readily differentiated by the aid of physical signs other than percussion, and it is important to remember that they may all be present at different stages during the course of chronic parenchymatous nephritis. (See Differential Table.)

Hydrothorax is readily detected by the fact that it gives bilateral dullness at the base of the chest, which dullness changes with the position of the patient.

The presence of free fluid in the abdominal cavity in cases of nephritis with associated cardiac failure is indicated by dullness on percussion in the flanks and above the pubes, with tympany above the dullness. If both ascites and pleural effusion are present, a continuous area of dullness may be detected throughout the lateral region of the trunk from the axilla to the brim of the pelvis.

Auscultation.—The breath-sounds are unaltered at first, but later, and especially after the accumulation of fluid in the serous sacs, numerous small crackling and large bubbling râles are audible over both lungs. The respiratory murmurs become more and more rapid, depending upon the amount of fluid in the serous sacs and the degree of cardiac embarrassment. The heart-sounds are first accentuated, most marked over the aortic cartilage, and this bounding element in the sounds persists while the hypertrophy continues, but when the hypertrophy has reached its limit and cardiac dilatation begins, the sounds become weak, rapid, and fetal in character. If there is pericardial effusion, the cardiac tones are extremely weak, distant, and muffled.

Laboratory Diagnosis.—The quantity of urine voided during the twenty-four hours may be normal or moderately increased, at times falling to between ten and thirty ounces. The color is high, and the specific gravity ranges between 1.016 and 1.030. (The larger the quantity of urine, the lower the specific gravity.) Chemically, this urine contains quite a large amount of albumin. It is generally conceded that the greatest amount of urinary albumin is lost in this type of Bright's disease. The amount of solids voided during the twenty-four hours is, as a rule, decreased, and this decrease becomes more and more pronounced with the advance of the disease.

Microscopically, the urinary sediment is found to contain many long and short granular casts and a few epithelial and leukocytic casts. Leukocytes, which are present in normal urines, appear in pathologic numbers during the entire course of parenchymatous nephritis. Red blood-cells are seldom seen except during an acute exacerbation of this condition, but when found, they are of grave clinical significance.

Hematologic Study.—Throughout the entire course of parenchymatous nephritis the percentage of hemoglobin will be found to be below the normal limit; with the progress of the malady it shows a decided decline—to even below 50 per cent. in the latter stages of the disease. The erythrocytes likewise show a proportionate decrease in number, falling to below 2,000,000 during the height of the disease. The leukocytes are normal in number early, but may be found moderately increased in advanced cases. The skin is less moist than normal, and the moisture of the breath is above

85 per cent. Widal, Weil, and Laudat* have discovered that lipemia is rather common in those cases which show retinal hemorrhage. Chauffard found that cholesterinemia is also present and that it runs parallel with the degree of lipemia. Likewise cholesterinemia may be present in other conditions where there is a so-called simple albuminuria, and where there is retention of chlorids or nitrogen. The naked-eye appearance of the blood-serum simulates that of milk.

Summary of Diagnosis.—Progressive loss of strength and of weight, anemia, dyspnea upon exertion, diminished excretion of urine, which is rich in albumin, and numerous granular casts serve as the cardinal symptoms of chronic parenchymatous nephritis. When, associated with these findings, there are cardiac hypertrophy, dropsy, headache, and gastro-intestinal disturbances, the diagnosis becomes quite clear, irrespective of the urinary findings, which are in themselves characteristic of the disease.

Differential Diagnosis.—Chronic parenchymatous nephritis is differentiated from chronic interstitial nephritis with difficulty, since both processes may be progressing in the same patient. In pure types, however, the urine in the case of chronic parenchymatous nephritis is usually less than normal in quantity; of rather high specific gravity; and contains a moderate amount of albumin—0.1 to 0.5 per cent. by the Esbach method, and pale and dark granular and hyaline casts. The urine in the typical case of chronic interstitial nephritis, on the other hand, is increased in quantity; is of low specific gravity; and contains little or no albumin and a few hyaline casts. The associated arteriosclerosis and the more marked nervous manifestations are important characteristics of chronic interstitial nephritis. A history of prolonged suppuration or of syphilis is highly suggestive of amyloid kidney. The table below shows the chief characteristics of chronic interstitial nephritis.

TABLE SHOWING THE DIFFERENTIAL FEATURES BETWEEN CHRONIC PARENCHYMATOUS NEPHRITIS, AMYLOID KIDNEY (WAXY), AND CHRONIC INTERSTITIAL NEPHRITIS.

Clinical Features.

Chronic Parenchymatous Nephritis.	Amyloid Kidney (Waxy).	Chronic Interstitial Nephritis.
1. History of one or more attacks of acute Bright's disease.	1. History of prolonged suppuration or of syphilis involving the bones.	1. Commonly follows high living and excessive indulgence in alcoholic liquors.
2. Commonest during early adult and middle life, but may occur at any age.	2. May appear during childhood or early adolescence.	2. Usually noticeable after the age of forty.
3. Duration, two to seven years.	3. May persist over a long period, and apparent recovery or improvement follows removal of the cause or the institution of proper treatment.	3. Duration, ten to thirty years. Death usually results from some intercurrent infection, as, *e. g.*, pneumonia, influenza, etc.
4. Dropsy of the face and ankles, with general anasarca, common.	4. Not pronounced, although moderate edema of the face and ankles may be seen.	4. Dropsy absent, unless it be the result of cardiac insufficiency or during an acute exacerbation of the renal process.

*Semaine Medicale, Nov. 6, 1912.

Clinical Features.—(Continued.)

Chronic Parenchymatous Nephritis.	Amyloid Kidney (Waxy).	Chronic Interstitial Nephritis.
5. Anemia with extreme pallor develops early and increases steadily with the progress of the disease. The hemoglobin and red cells are relatively reduced.	5. Hemoglobin and red cells show decided reduction.	5. Less marked anemia in proportion to the duration of the disease.
6. Leukocytes uncommon.	6. Leukocytes common, but not constant.	6. Leukocytes may be present during the latter stages and during complications.
7. Nervous symptoms do not appear until the other clinical evidences of the disease are prominent.	7. Not a characteristic feature.	7. Nervous manifestations appear early, as, *e. g.*, shifting paralyses, headache, neuralgia, asthma, and coma.
8. Pallor of the conjunctiva and of the retina. Retinal hemorrhages rather common until disease is well advanced.	8. Extreme pallor of conjunctiva and retina. Amyloid deposits in retina	8. Retinal hemorrhages common, and choking of the disc is occasionally seen.
9. Liver and spleen of normal size.	9. Enlargement of spleen and liver common.	9. Liver usually small.
	Laboratory Findings.	
10. Quantity of urine voided during the twenty-four hours normal or subnormal.	10. Normal or increased.	10. Decidedly increased—70 to 150 ounces a day.
11. Specific gravity, as a rule, above 1.018.	11. Specific gravity normal or often extremely low—1.015 to 1.005.	11. Specific gravity low—1.015 to 1.005.
12. A high percentage of albumin is present.	12. A low percentage of albumin.	12. Merely a trace of albumin, but when this is studied in comparison with the large quantity of urine voided, the amount of albumin lost during the twenty-four hours is quite large.
13. Numerous short, thick, granular casts, with few hyaline casts present.	13. Most of the casts are of the hyaline variety, and at times there are to be seen the so-called amyloid casts (Fig. 268). Wide hyaline casts with hyaline-like epithelial cells upon their free surface common.	13. Hyaline casts predominate, and they are, as a rule, long and narrow, at times appearing as mere shadows. Wider casts are seen during the early course of the disease. Renal epithelial cells are often present.

Course and Duration.—During the first six months there are slight edema of the face and ankles, some pallor, and pronounced dyspnea, after which the disease advances, as a rule, rapidly from bad to worse.

Judicious treatment may prolong life for a period of several years. We have seen cases showing slight pallor, somewhat scanty urine of high specific gravity, with albumin, but with no other complaint for years, develop severe attacks, with dropsy and dyspnea, lasting for several months, thus showing that the disease may run an intermittent course. We have also seen severe cases that terminated fatally within a few months, during which time the patients manifested uremia, dropsy, acute cardiac dilatation, and intercurrent complications, such as bronchopneumonia. When parenchymatous nephritis has existed for more than a year, recovery is not likely to occur. Rarely, indeed, an apparently permanent recovery follows this type of nephritis in the young, and we have had under our care several such cases in which there was no recurrence for a period of several years.

CHRONIC INTERSTITIAL NEPHRITIS

(CHRONIC NEPHRITIS (NON-EXUDATIVE); CHRONIC BRIGHT'S DISEASE; PRIMARY OR GENUINE CONTRACTED KIDNEY; CIRRHOTIC KIDNEY; RED GRANULAR KIDNEY; CHRONIC PRODUCTIVE (DIFFUSE) NEPHRITIS WITHOUT EXUDATION (DELAFIELD); GOUTY KIDNEY).

Pathologic Definition.—A chronic disease of the kidneys, characterized by the presence of inflammatory changes, with the extensive production of fibrous tissue and a consequent lessening in the size of the organ, obstruction to some of the uriniferous tubules, and a tendency toward the formation of cysts in the parenchyma of the kidney.

The size of the kidneys is greatly diminished, being reduced to about one-third or one-half that of the normal. The capsule is appreciably thickened, often opaque, and decidedly adherent. The surface of the organ is usually reddened, somewhat granular, and may display distinct nodules and cysts. The kidney cuts with difficulty, and the cut surface shows the cortical portion to be greatly thinned (the result of atrophic changes), and mottling is effected by dark-red bands that course through paler areas of the organ. Cysts, varying in size from that of a millet-seed to that of a walnut, may be seen in any part of the cortical portion.

The essential microscopic change is an increase in connective tissue, and such increase is most conspicuous in the cortical portion of the organ, and is always accompanied by a variable degree of atrophy and degeneration of the renal parenchyma.

(*b*) Heredity stands as a prominent predisposing factor in interstitial nephritis.

(*c*) Adult and advanced middle life are the periods at which this type of nephritis is most likely to develop.

(*d*) Sex figures prominently, males being far more commonly affected than females.

(*e*) The prolonged use of such toxic substances and chemic irritants as alcohol, chronic lead-poisoning, the so-called uric-acid diathesis, gout, chronic gastritis, etc., are believed to favor sclerotic change in the kidney substance. Again, such biologic irritants as the toxins of malaria and syphilis serve as potent factors in the production of chronic interstitial nephritis.

(*f*) Persons who indulge too freely in rich foods and in alcoholic drinks are especially likely to suffer from this form of Bright's disease, and it may here be stated that the prolonged daily use of small quantities of alcoholic stimulants is also followed by sclerotic change in the kidney.

(*g*) Those who are inactive or whose occupations necessitate confinement indoors during the day are frequently attacked by this affection.

(*h*) Nervous strain, the result of bereavements, financial embarrassment, and anxiety, is believed to exercise a decided influence upon the production of this type of renal affection.

(*i*) Sclerotic changes in the kidney may follow irritation of these organs from hydronephrosis, pyelitis, retention of stone, either in the bladder or in the renal pelvis, and any interference with the flow of the urine through the ureters.

Principal Complaint.—The disease comes on insidiously, and may exist for years without causing the patient much discomfort, although his friends may see that his health is gradually failing. Ofttimes the first symptoms observed by the patient do not appear until late in life, at a time when the kidneys may be in an advanced stage of degeneration. Indeed, the physician often detects nephritis while making a routine examination of the urine or of the cardiovascular system. Attacks of uremia are occasionally one of the early symptoms, and among the phenomena that accompany these should be mentioned headache, stupor, nausea, vomiting, dyspnea, especially upon exertion, and later convulsions. At times the patient hears a constant roaring, and states that he hears his heart beat when lying at rest. He further describes attacks of palpitation, and may be annoyed by the forcible beats of his heart. Epistaxis may be an early and distressing symptom.

Curiously enough, many of the most distressing of these symptoms may disappear for an indefinite period, to return with increased violence. Even during the intervals of comparatively good health the patient complains of drowsiness or insomnia, headache, dyspnea, and indigestion, but all these symptoms are of a mild form. Early during the course of chronic interstitial nephritis there is frequent micturition, and following an interval of subsidence of the symptoms a severe uremic attack may occur which may terminate fatally. If, however, the patient recovers, there will be progressive loss of weight and strength and failing vision, which is due to retinal hemorrhages. At times the patient is annoyed by specks floating before his eyes, and these may be described even before retinal hemorrhage has taken place.

Uremic Asthma.—Spasmodic dyspnea may be an early symptom of chronic interstitial nephritis, and a correct diagnosis is reached only by making a chemic analysis of the urine, and the additional fact that the dyspnea is promptly relieved after free diaphoresis has been effected.

Paralyses.—Attacks of monoplegia and of paraplegia may be experienced at any time during the course of chronic interstitial nephritis, but these paralyses, like the spasmodic dyspnea, may disappear promptly upon the institution of treatment. Indeed, the paralyses may disappear from one side or from one portion of the body, to reappear in a few days or weeks on the opposite side.

Physical Signs.—Inspection.—Early during the course of this disease there is a variable degree of pallor, which becomes more marked as the disease advances. The skin of the face and ears is also roughened, of a slight lemon tint, and there is an absence of luster. The nails are clubbed and brittle. The temporal arteries stand out prominently, and in advanced cases they are often tortuous and may show slight pulsation. The superficial fat appears to be fairly well preserved, although the skin hangs in folds and wrinkles. The apex-beat is usually displaced down and to the left, and if there is an organic lesion of the heart, pulsation of the vessels of the neck and

over the main arteries occurs. During the later stages of chronic interstitial nephritis there may be cardiac dilatation, at which time there will be noticed pulsation in the epigastrium, at the third interspace on the right, and pronounced throbbing of the vessels of the neck. The tongue is likely to be dry and coated, and the patient describes a sticky condition of the mouth.

Palpation.—The skin is dry and rough, and gives a somewhat graty feel to the palpating hand. The hair is also dry. Throughout the entire course of chronic interstitial nephritis the arterial tension is increased, the pulse is small and wiry, and the arteries display an unusual hardness; in advanced cases the radials are wiry, and the temporal arteries are readily outlined by the finger. A fact to be borne in mind is that persons suffering from chronic interstitial nephritis are especially prone to have, in conjunction with the general arteriosclerosis, which is so characteristic of this disease, disease of the valves of the heart, and consequently they display the symptoms of organic heart disease. As a rule, therefore, a patient who has suffered from chronic interstitial nephritis for a long time will show the pulse more or less imperfectly characteristic of either mitral or aortic disease, and it is for this reason that we seldom find a pulse that can be said to be characteristic of chronic interstitial nephritis that has advanced for several years. With loss of compensation the pulse becomes weak, rapid, dicrotic, and irregular. The apex-beat is felt to be displaced downward and to the left, and is decidedly forcible during the early stage of the disease, but after dilatation has resulted, there is diffuse feeble pulsation over the precordium, and, depending upon the variety of organic lesion of the heart, impressions may be conveyed to the hand. Edema of the skin is seldom present except in the later stages of the disease or after cardiac dilatation has taken place. During an acute exacerbation of a chronic nephritis there may be edema of the skin of the extremities and of the face, and, in fact, ascites may result from the same cause, in which case a wave is transmitted over the abdominal fluid.

Percussion reveals nothing of special importance, and simply confirms previous findings—cardiac hypertrophy or dilatation and, rarely, the presence of fluid in the peritoneum, pericardium, or pleural sacs. The area of cardiac dullness is shown to extend downward and to the left, ofttimes reaching the midaxillary line at the lower border of the seventh rib transversely. This degree of cardiac hypertrophy will be seen to increase gradually from year to year, so long as compensation remains complete. Cardiac dilatation is likely to follow after the rupture of compensation, and it is in these cases that we find the greatest area of cardiac dullness. We have seen cases in both hospital and private practice in which the transverse diameter of the area of cardiac dullness during acute dilatation extended for ten inches, and we have in mind a recent case in which the transverse diameter of the heart extended from the angle of the vertical diameter during the stage of dilatation.

Auscultation.—There is decided accentuation of both the aortic and pulmonic second sounds, and the first sound of the heart is also forcible early during chronic interstitial nephritis. After myocarditis and consequent cardiac dilatation have developed the heart-sounds are weak, feeble, and even fetal in character.

Laboratory Diagnosis.—Urinary Findings.—The quantity of urine voided during the twenty-four hours is always above that of the normal, and, in fact, often exceeds 100 ounces. This urine is pale, clear, of a specific

gravity of 1.005 to 1.016, acid in reaction, and does not show an abundant sediment upon standing. Chemically, the urine is found to contain a small percentage of albumin, except in those cases in which there is an acute exacerbation of the renal condition, and also after cardiac compensation has been ruptured, when the urine contains a comparatively large amount of albumin. It may be absent altogether, especially from the urine voided in the morning. The apparent trace of albumin present in the urine of chronic interstitial nephritis, when studied in relation to the large quantity of urine voided during the day, discloses the fact that the patient is constantly losing a large amount of this substance—a feature of vital clinical importance, and one that deserves most careful consideration.

The solids (urates, phosphates, sulphates) and the percentage of urea are lessened in the urine of chronic interstitial nephritis, and the clinical evidence that solids are not eliminated from the body is strongly suggestive that other substances (toxic in character) that should be eliminated with the urine are likewise retained in the body tissue. It is supposed to be the retention of excrementitious products of metabolism that explains the frequent occurrence of profound nervous symptoms in chronic interstitial nephritis.

Microscopically, the urine is found to be deficient in crystalline substances, and to contain a few small, narrow hyaline casts, with an occasional granular cast. Leukocytes and epithelial cells are present, and the latter may be seen clinging to the surface of the hyaline casts.

Blood Findings.—A study of the *blood* reveals the presence of secondary anemia. Rarely, the blood is decidedly concentrated in chronic interstitial nephritis, consequently the number of red cells in a cubic millimeter may be but moderately reduced, or, rarely, it may be above that of the normal, whereas the total number of cells in the body is far below the normal. After cardiac insufficiency and cardiac dilatation have developed, the number of red cells in a cubic millimeter may be far above the normal average (5,000,000 in a cubic millimeter), this peculiarity depending upon the presence of cyanosis, and it may be well to mention that, late in the course of kidney disease, cyanosis is the commonest cause of error in making an estimation of the number of red blood-cells.

The *hemoglobin* usually falls to between 75 and 50 per cent., except late in the disease, and shortly prior to a fatal termination, when the hemoglobin in the circulating blood may register near the normal (80 to 95 per cent.); this apparent increase in the percentage of hemoglobin, like the pseudo-increase in the number of red cells, is dependent upon the existence of cyanosis, and is of somewhat unfavorable prognostic significance.

The moisture of the skin is below that of the normal, and may fall to 40 or even to 25 per cent. The moisture of the breath is increased, registering above 80.

Illustrative Case of Chronic Interstitial Nephritis.—Henry M., male, aged fifty-four years; apparent age, sixty-five years; height, 5 feet 10¼ inches; weight, 140 pounds; at the age of thirty-five the weight was 160 pounds.

Family History.—Father died of heart disease at the age of fifty-four; mother died at the age of fifty-six, the cause being unknown. A paternal uncle now living has been a victim of gout since the age of fifty, and another uncle died of hepatic cirrhosis at the age of forty-seven. No record of malignancy in ancestors.

Previous History.—Does not recall having had any of the diseases of childhood except measles and scarlet fever at the age of ten years. Was subject to frequent attacks of sore throat between the ages of ten and twenty-five, but since then has suffered but slight inconvenience. Had typhoid fever at thirty, and influenza at thirty-three.

Social History.—Engaged in mercantile business, and bears the responsibilities

of a concern that employs at least fifty clerks. Married at the age of twenty-five, and has three children living, all in apparent health. He has used alcohol since the age of twenty, taking from two to six drinks of what he regards as the best of gin or whisky each day. Uses tobacco freely, smoking from six to ten cigars daily. His work does not necessitate exposure to cold and wet, and, indeed, he has been unusually careful in this respect since his attack of influenza.

Present Illness.—At the age of thirty-eight he observed that he tired easily, and that he felt a throbbing in the head following violent exertion (climbing stairs, etc.). The latter condition increased in severity for a period of about one year, when he consulted his physician for an attack of vertigo that yielded promptly to treatment. Since that time he has occasionally been annoyed by a similar throbbing sensation in the head, and he states that he cannot rest upon the left side when in bed on account of hearing the sounds of his heart. Weakness has been progressive, although he is still able to follow his usual occupations, but he complains greatly of exhaustion during the afternoon hours of each day. The appetite is disturbed, and he is unable to eat at the morning meal because of nausea, although he seldom vomits. He does not appear to be able to endure cold, and is uncomfortable unless dressed in heavy clothing. States that his hands and feet are continually cold, even during the summer months. Constipation is obstinate, sleep is not restful, and he is continually tossing from side to side while in bed and awakens repeatedly during the night.

Pain.—At present there is more or less aching of the muscles of the lower limbs and of the back, and this is most pronounced after a day's work. Headache has been more or less persistent for the past three years, and the patient states that at present it is unusual for him to be free from headache upon arising in the morning.

His temperament has altered materially during this illness, and he is extremely irritable. He has observed that at certain times he is more nervous than at others, and he has found that a hot bath at night before retiring usually insures him a restful sleep, and that following such bath and sleep he is less likely to be annoyed by headache.

Physical Examination.—*General.*—The face is somewhat drawn, the skin and mucous membranes are pale, and the surface of the skin is unusually dry. Upon rising in the morning there is slight puffiness beneath the eyes, and by firm pressure it is possible to detect slight pitting in the malleolar regions. While sitting, the patient is more or less nervous, shifting about in his chair.

Local Examination.—Inspection.—The hair appears to be poorly nourished, dry, and sparingly distributed over the scalp. The finger-nails are somewhat brittle, and the patient has difficulty in keeping the surface of the nails smooth. The tongue is slightly coated, and the mucous surface of the lips and mouth is pale. The apex impulse of the heart is forcible, and is seen 1½ inches below and an inch outside the left nipple-line. There is slight pulsation of the vessels of the neck, and epigastric pulsation is also visible.

Palpation confirms what has already been stated under general examination and inspection, the apex-beat being strong. The pulse is slow, the beats numbering 65 to 77 a minute, of high tension, and there is distinct hardening of the arteries. The temporal arteries stand out prominently and are readily palpable, and even the brachial artery can be distinctly felt for a short distance of its course.

Percussion.—The area of cardiac dullness is increased downward and to the left. The area of liver dullness is distinctly decreased, its superior border beginning at the sixth rib; it does not extend to the costal margin; indeed, in the midclavicular line it is found to be from one to one and one-quarter inches above the costal border. The area of liver dullness is also confirmed by auscultatory percussion, there being an appreciable decrease in the size of the organ.

Auscultation.—The heart-sounds are strong, booming in quality, and a distinct diastolic murmur is heard at the aortic cartilage, which is transmitted downward along the left border of the sternum. A murmur of moderate intensity is also heard at the apex of the heart, and is well transmitted for 2½ inches toward the left axilla. No murmur is audible at the ensiform cartilage, although the venous pulsation in the right carotid region strongly suggests the existence of tricuspid regurgitation.

Laboratory Findings.—The first urinalysis was made about six and one-half years ago, and at that time the attending physician detected a feeble reaction for albumin. Three years ago a more complete analysis was made, and at this time the quantity of urine voided during the twenty-four hours varied between 50 and 70 ounces. The specific gravity was 1.010; the urine was pale in color, and a precipitate did not collect when it was permitted to stand for several hours. Microscopically, many hyaline casts, a number of leukocytes, and a few epithelial cells were detected. At present the urinary findings are practically the same as those obtained four years ago, except that casts are more numerous, and that occasionally a granular cast is present. Some

of the hyaline casts are unusually long and narrow, and occasionally they assume a spiral outline.

The hemic changes are those characteristic of secondary anemia, the hemoglobin ranging between 68 and 75 per cent., and the red cells fluctuating between 3,500,000 and 4,000,000 in a cubic millimeter.

Diagnosis by Induction from Clinical Data.—The history of the vitality having been below that of normal for several years and the additional evidence that he had lost twenty pounds during this period give some clue to the diagnosis. Progressive weakness, mental irritability, and gastro-intestinal disturbances are also to be considered important symptoms. The diagnosis was confirmed only by making an analysis of the urine.

Course of Illustrative Case.—It is clearly evident that the patient's general condition, including the loss of weight and progressive weakness, has for several years steadily become worse, and although he is still able to attend to his duties, their performance has become a hardship, and he has observed that he is unable to cope with business propositions with the same astuteness that he did in former years.

Summary of Diagnosis.—Progressive anemia, pallor, and polyuria in persons in whom the skin is dry and the breath emits an odor of urine serve as points on which to base a diagnosis of chronic interstitial Bright's disease. The presence of relatively small amounts of urinary albumin, together with the finding of small, narrow hyaline casts and a few leukocytes and renal epithelial cells, are almost conclusive evidence of the existence of this form of nephritis. The marked tendency toward nervous symptoms, the progressive weakness, the frequency of headache, and the duration of the malady are all common to chronic contracted kidney.

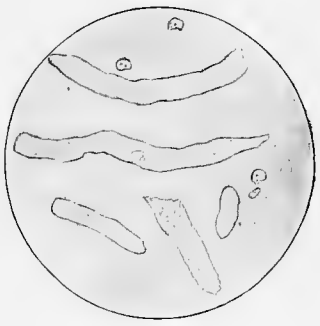


Fig. 267.—Hyaline Casts (Narrow) from Case of Contracted Kidney.

Observed at Philadelphia Hospital (obj. B. and L. one-sixth).

Differential Diagnosis.—Contracted kidney is seen after middle life, and there is often a history of overeating, alcoholism, gout, chronic rheumatism, and sedentary habits. The symptoms of uremia, when manifested, are practically the same in all forms of nephritis. Shifting paralyses and retinal hemorrhages are far more common in chronic interstitial than in other forms of nephritis. The low specific gravity of the urine and the large quantity voided during the twenty-four hours serve to differentiate this disease from chronic parenchymatous nephritis. (See Differential Table, p. 672.)

The absence of edema, anasarca, and ascites in chronic interstitial nephritis is valuable. The foregoing symptoms aid when this malady is to be differentiated from chronic parenchymatous nephritis, in which these symptoms are common. During the course of an acute exacerbation of chronic interstitial nephritis it is impossible to differentiate chronic interstitial from chronic parenchymatous nephritis, either by the urinary findings or by the general clinical picture.

Clinical Course of Disease.—Chronic interstitial nephritis extends over a period of from ten to thirty years, although the duration depends largely upon the presence or absence of certain other acute infectious conditions (*e. g.*, pneumonia). In the absence of these intercurrent infections and of accidents, such as cerebral apoplexy, the course of the disease may be lengthy.

The prognosis as to life is good, but as to permanent recovery, it is unfavorable. Persons with chronic interstitial nephritis seldom, if ever, enjoy

perfect health, although, by the institution of judicious treatment, many of them are enabled to attend to their duties and derive a limited amount of pleasure out of life, going on from year to year, and showing no decided change in their physical condition.

AMYLOID DISEASE.

Pathologic Definition.—Amyloid degeneration of the kidney, while at the present time generally conceded to be a rare disease, is encountered during the course of certain chronic conditions (suppuration, syphilis). It differs widely from other forms of nephritic degeneration. The kidney is usually enlarged, pale in color, displays some mottling, and does not offer decided resistance to the knife. The cut surface of the organ shows the cortical portion to be thickened and waxy in appearance. Microscopically, there is amyloid degeneration of the epithelium.

Predisposing and Exciting Factors.—Amyloid kidney may occur at any age, although it is more common during early adult and middle life. The conditions that predispose to amyloid kidney are: (1) General amyloid disease, which is usually marked by amyloid degeneration and enlargement of the liver and spleen; (2) prolonged suppuration, *e. g.*, pulmonary tuberculosis with cavity, and syphilis with lesions of the bones.

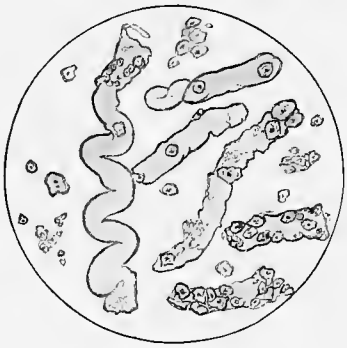


FIG. 265.—EPITHELIAL AND AMYLOID CASTS (Boston).

Patient a female, aged forty-two years, suffering from septicopyemia with amyloid kidney.

Principal Complaint.—In a few instances the patient appears to enjoy fairly good health, and when symptoms arise, they are dependent on the marked secondary anemia; consequently the patient complains of general weakness, dyspnea, palpitation, tinnitus aurium, anorexia, chronic dyspepsia, and attacks of apparent acute gastritis. The abdominal protrusion may be distressing, and is due to enlargement of the liver and of the spleen, although in some cases ascites has been seen. Rarely, a moderate amount of edema of the ankles is present. Headache is the rule, but severe nervous symptoms are unusual. The patient complains early of specks floating before the eyes, but albuminuric retinitis is uncommon until late in the disease. Chronic dysentery may be distressing. The general complaint differs slightly from that of chronic parenchymatous nephritis, with which it may be confounded, and the differentiation will depend chiefly upon the clinical history, the evidences of general amyloid disease, and the late appearance of edema.

Laboratory Diagnosis.—The quantity of urine voided during the twenty-four hours is, as a rule, increased, although it has been seen to be normal, and in two instances the quantity was slightly subnormal.

A feature of great importance in connection with amyloid disease of the kidney is that the quantity of urine excreted varies at different stages of the disease (the more advanced the condition, the larger the quantity of urine) until late, when it may display a great diminution. The quantity of urine voided is also influenced by the presence of complications, being lessened when such conditions as dysentery, diarrhea, and paroxysmal vomiting coexist. The specific gravity of the urine fluctuates with the quantity

voided during the twenty-four hours, but is seldom above 1.015; as a rule, it is between 1.005 and 1.010. A well-marked trace of albumin is always present in the urine of amyloid kidney, and a high percentage is not unusual, the amount of albumin lost during the twenty-four hours being quite large. The urinary solids are diminished.

Microscopically, the urine contains many hyaline casts (Fig. 267) some of which are wide, plump, and tapeworm-like in outline—the so-called amyloid casts (Fig. 268).

Summary of Diagnosis.—The diagnosis of amyloid disease is dependent largely upon a clinical history of suppuration or of syphilis and upon the urinary findings, neither of which, however, is characteristic of this affection. We have sometimes found cases of amyloid disease of the kidney at autopsy in which there were no positive evidences of this condition during life.

Course.—Usually, when the disease complicates pulmonary tuberculosis with cavity formation, the course of the disease is protracted; but should it develop during chronic bone suppuration, surgical interference may effect an apparently permanent cure. In syphilis, specific treatment often gives relief, and life may be prolonged for a number of years.

HYDRONEPHROSIS.

Pathologic Definition.—An accumulation of urine within the pelvis and calices of one of the kidneys. The fluid, by exerting pressure, may produce pyelitis, dilatation of the renal pelvis, atrophy, and cystic degeneration of the parenchymatous tissue; an abdominal tumor may also be present, which may suddenly disappear after the passage of a large quantity of urine.

Predisposing and Exciting Causes.—Hydronephrosis is generally a secondary condition, although it may be classified as either congenital or acquired. It is always dependent on occlusion of the lumen of the ureter. Among the causes of hydronephrosis are:

1. Congenital malformation in the urinary passages, and in this class of cases it may be bilateral. Instances are recorded in which congenital hydronephrosis in the fetus was known to complicate labor.
2. Hydronephrosis among adults is far more common in the female than in the male, and this is especially true of women who have borne many children. Rarely, indeed, the disease is bilateral in the adult, and when such is the case, the obstruction is in either the bladder or the urethra.
3. Impaction of renal calculi in the pelvis of the kidney.
4. Inflammatory disease of the ureter, which results in narrowing of the lumen of this membranomuscular tube.
5. Floating or movable kidney, with torsion of the ureter.
6. Adhesions following pelvic and abdominal operations.
7. Pressure upon the ureter from abdominal tumors, among which should be considered new-growths, ovarian cysts, fibroid uterus, prolapsed spleen, ectopic gestation, and normal pregnancy.
8. Impaction of a calculus at the junction of the ureter with the bladder. There are but few recorded instances of this condition being the predisposing cause.
9. Tumors and sclerotic changes of the bladder, with the production of new tissue and the consequent closure of the orifice of the ureter.

10. Urethral obstruction, dependent on either an enlarged prostate or upon urethral stricture.

11. Vesical paralysis.

Principal Complaint.—In the presence of an abdominal tumor in the infant, hydronephrosis should be suspected. The mother often describes increasing prominence of the child's abdomen, and this abdominal distention is less likely to disappear in children than it is in the adult. It is possible for a congenital hydronephrosis to exist without evincing any marked symptoms, and the patient may continue in comparatively good health, the condition being discovered later in life, and possibly not until uremic symptoms develop.

In bilateral hydronephrosis the patient shows symptoms of uremia early. In adults the intermittent form of hydronephrosis may be marked by the presence of a periodic or a constant tumor in the abdomen. The tumor mass decreases in size or disappears with the passing of an increased quantity of urine, and, on the other hand, while the mass gradually increases in size, there is a diminished flow of urine. The patient may complain of a tumor-mass in the abdomen, which is decidedly prominent, and which does not show any apparent change in size—a variety of hydronephrosis usually seen after the pelvis of the kidney has been dilated for months or even years. There is some loss in flesh, but secondary anemia is not common, unless uremic intoxication is also present.

Pain occurs in practically all cases, and in intermittent hydronephrosis it disappears with the subsidence of the tumor, but returns with the reappearance of the abdominal distention. In the majority of instances the patient believes that straining or violent exercise induces the accumulation of fluid in the pelvis of the kidney, and not infrequently there is a history of pains having followed some violence of this kind. The pain, which is often excruciating, may last for from one to twelve hours, after which the patient observes a gradual swelling of the abdomen.

Sufferers from hydronephrosis may continue in fairly good health for weeks, months, and even years during the intervals, or while the pelvis of the kidney is not distended with urine.

Acute symptoms are by no means uncommon in hydronephrosis, and following the initial pain there may be a chill, succeeded by an elevation of temperature to 102° to 104° F., after which there is a drenching sweat. These three stages simulate somewhat closely the malarial paroxysm. Nausea, obstinate vomiting, and increased respiration and pulse-rate are often present. Constipation is frequently an annoying symptom, yet it is by no means constant in hydronephrosis.

Nervous Symptoms.—The majority of women suffering from hydronephrosis are of the neurasthenic type, and suffer from headache, neuralgia, and the like. Paraplegia has been known to complicate hydronephrosis, and there may be extremely acute pains, which are described as shooting in character, and radiating down the thighs to the calf muscles and the ankles; cramp of the lower limbs is often distressing.

Thermic Features.—When, as previously stated, the obstruction is due either to torsion of the ureter or to plugging of the ureter with a calculus, the temperature may rise suddenly. If the severe pain continues over a period of several hours, the temperature will first rise suddenly to say 100° to 103° F., and may then fall to the normal, or in severe cases to a subnormal, level, and remain at this point for an indefinite period—the so-called renal

intermittent fever. During the stage of hypopyrexia the skin is blanched, cold, and clammy, and the general condition is that of shock.

Physical Signs.—**Inspection** reveals the presence of asymmetric abdominal distention.

Palpation.—It may be possible to palpate the kidney and to find it freely movable in the abdominal cavity when the pelvis is not filled with urine. When, however, the pelvis of the kidney is distended, a distinct mass is readily palpable, and a wave may be transmitted through the fluid. This mass is usually soft and doughy, and commonly occupies the brim of the pelvis, although a hydronephritic tumor may be found on either side of the abdomen, quite irrespective of the particular kidney involved.

Percussion is of but little value in making a diagnosis of hydronephrosis unless the intestines are comparatively free from gas; in our experience we have found this condition to be generally associated with a variable degree of tympanites.

Peritoneal adhesions are also likely to anchor portions of the bowel over the tumor-mass, which interferes materially with the value of the percussion-note.

Laboratory Diagnosis.—The quantity of urine passed during the twenty-four hours is usually diminished prior to and during the development of the tumor, and in intermittent hydronephrosis an increased flow of the urine is observed during the disappearance of the tumor. The urine is usually pale, of low specific gravity, and at times contains a trace of albumin. We have seen two cases in which bloody urine followed hydronephritic tumor.

Microscopically, the urine contains many leukocytes and few solids, and red blood-cells are occasionally seen.

Summary of Diagnosis.—The diagnosis is based largely upon the somewhat rapid development of a tumor in the abdomen, accompanied by colicky pains, and the passing, from time to time, of a large quantity of urine, irrespective of whether or not there has been a subsequent decrease in the flow of urine. The disappearance of the tumor coincident with the discharge of an abnormally large quantity of urine is almost positive evidence of the presence of hydronephrosis. In abdominal tumors of the new-born and during infancy hydronephrosis should be considered a prominent cause.

Differential Diagnosis.—This is reached by excluding such abdominal masses as *ovarian cysts, a distended urinary bladder*, and *chylous cysts*. The table on p. 684 shows the distinctive features of five of these abdominal conditions.

Rarely, if ever, does the tumor of hydronephrosis attain sufficient size to warrant its being mistaken for ascites.

Percussion is of little value in diagnosticating hydronephrosis, since in most abdominal growths the colon is anterior to the tumor-mass. Tapping of these cystic or fluid tumors by abdominal puncture cannot be recommended as a safe diagnostic measure, although a study of the aspirated fluid aids materially in distinguishing between such tumors of the abdomen. Urea is found in small amounts in both the fluid from the pelvis of the kidney and in that from an ovarian cyst, whereas red and white blood-cells may be present in practically any fluid tumor of the abdomen. Catheterization of the ureters serves as a means of diagnosis when unilateral hydronephrosis is present.

Clinical Course.—It is common for hydronephrosis to run a somewhat chronic course, although this is in a measure modified by the etiologic factors in each case. Surgical intervention is necessary to correct the condition.

TABLE SHOWING THE DISTINCTIVE DIFFERENCES BETWEEN HYDRONEPHROSIS, DISTENDED GALL-BLADDER, FLOATING SPLEEN, OVARIAN CYST, AND DISTENDED BLADDER.

Hydronephrosis.	Distended Gall-bladder.	Floating Spleen.	Ovarian Cyst.	Distended Bladder.
1. A history of renal colic or of intense pain in the region of the kidney.	1. History of pain in the region of the liver. Pain radiates to right shoulder.	1. Pain less common.	1. There is seldom, if ever, pain.	1. Distress in the region of the bladder, which may increase to a dull pain.
2. Commonest in hysteric women, after traumatism, and following repeated pregnancies.	2. Occurs after middle life and after several attacks of gall-stone colic.	2. Common in those displaying a hysteric temperament.	2. Usually occurs after the age of thirty.	2. Commonest in the male as the result of urethral stricture and of enlarged prostate.
3. Pain cramp-like, and may radiate to the pubic region. Dull, constant pain at times.	3. Pain may persist for several hours, and then disappear suddenly. Radiates to right shoulder.	3. Pain epigastric or in left hypochondrium.	3. Pain absent.	3. Pain confined to pubic region, may be at meatus urinarius.
4. Abdominal tumor develops quite abruptly and disappears suddenly.	4. Tumor may disappear suddenly, following which there is usually one or more copious bilious discharges from the bowel.	4. Tumor may develop suddenly, but there is no change in its size.	4. Develops slowly, but with constant increase in size.	4. Develops within twenty-four to forty-eight hours, during which time the quantity of urine passed is small.
5. Tumor is not affected by passing catheter into bladder.	5. Tumor not changed by catheterization.	5. Tumor not changed by catheterization.	5. Tumor not changed by catheterization.	5. Tumor disappears upon passing catheter into bladder.
6. During the disappearance of the tumor the patient passes a large quantity of pale urine of low specific gravity.	6. Urine bile-stained.	6. Urine not affected.	6. Urine not affected.	6. Urine not affected.
7. The appearance of the tumor is decidedly intermittent.	7. Tumor intermittent where it follows hepatic colic, always in right hypochondrium.	7. Tumor unchanged in size, and on left side of abdomen.	7. Tumor unchanged in size or in position.	7. It is unusual to have repeated attacks.
8. There may or may not be disease of the prostate.	8. Prostate normal.	8. Prostate normal.	8.	8. Urethral stricture and prostatic disease common in male subjects.
9. Jaundice rare.	9. Jaundice persistent.	9. Absent.	9. Absent.	9. Absent.

PYELITIS (PYELONEPHRITIS; PYONEPHROSIS).

Pathologic Definition.—A purulent inflammation involving the pelvis of one or of both kidneys. The suppurative process may also extend from the renal pelvis to the kidney substance, and, rarely, both the pelvis and the parenchyma of the organ become distended by pus (pyonephrosis). The condition may be found to depend upon infection with pus-producing bacteria, and in many instances tuberculosis of the pelvic mucosa has preceded the condition. Stone in the pelvis of the kidney is at times the exciting factor. An important feature in this connection is that infection of the pelvis is commonly secondary to a similar suppurative process involving the bladder.

Varieties and Pathology.—(1) **Catarrhal pyelitis** is a condition in which the pathologic changes in the pelvis of the kidney are mild, and consist of reddening and swelling, with loss of luster of the mucous membrane. The greater part of the mucous membrane is covered with a mucopurulent exudate in which many desquamated epithelial cells are present.

(2) The **moderately severe forms,** in which, in addition to what is found in catarrhal pyelitis, there are numerous ecchymoses into the mucous membrane. This type is often the result of renal calculi or of virulent infection with pyogenic bacteria. All urine contained in the pelvis of the kidney is purulent from the admixture of pus.

(3) **Calculous Pyelitis.**—Calculi not infrequently set up a catarrhal inflammation of the pelvis of the kidney, which in turn becomes infected with pyogenic bacteria.

(4) **Extraneous Pyelitis.**—Abscess of the kidney occasionally empties into the renal pelvis and excites a purulent inflammation of the lining of this mucous surface.

(5) **Pseudomembranous Pyelitis.**—The formation of a false membrane in the renal pelvis has been known to develop during the course of certain infectious fevers.

(6) **Tuberculous Pyelitis.**—In tuberculosis of the pelvis of the kidney it is customary to find small irregular ulcers in the mucous membrane. The surface of these ulcers is covered with mucopurulent exudate. Their edges are thin and slightly irregular. Tuberculosis of this structure may be chronic in its course, in which case caseous masses may be formed within the walls of the pelvis of the kidney.

(7) **Suppurative Pyelitis.**—When the pelvis of the kidney becomes infected from the bladder (*e. g.*, following a severe cystitis), it is usually referred to as ascending pyelitis, and when the suppurative process extends to the renal parenchyma, it is called surgical kidney.

(8) **Obstructive pyelitis,** which is dependent on obstruction to the flow of urine from the kidney, is described under Predisposing and Exciting Factors.

Predisposing and Exciting Factors.—Pyelitis is usually secondary to some preëxisting pathologic condition, and among the numerous maladies and conditions it complicates or in which it appears as a sequel should be mentioned.:

(*a*) Renal calculus. (*b*) Torsion of the ureter and hydronephrosis. (*c*) Direct extension of an inflammatory process from the ureter (ureteritis), bladder (cystitis), urethra (urethritis), or enlarged prostate (prostatitis). (*d*) Dilated pelvis of the kidney, in which the urine may become decomposed. (*e*) Acute nephritis. (*f*) Renal tuberculosis. (*g*) Renal carcinoma.

(*h*) Foreign bodies in the bladder, such as vesical calculus. (*i*) Such chemic irritants as turpentine, cantharides, cubebs, and copaiba contribute toward the development of pyelitis. (*j*) It has been known to follow infectious fevers, as, *e. g.*, typhoid fever, scarlet fever, smallpox, diphtheria, typhus fever, etc. (*k*) Obstructive pyelitis may follow plugging of the ureters with a small calculus, and the constant irritation produced by the passing of large amounts of uric acid is also capable of exciting true pyelitis. Portions of large calculi occupying the pelvis of the kidney would in turn irritate its mucous membrane. (*l*) Severe traumatism to the kidney is also followed by the development of obstructive pyelitis. (*m*) Pyelitis occurs during the course of such nervous conditions as hemiplegia and paraplegia, but here it is probably secondary to cystitis.

Parasitic pyelitis may result from infection of either the kidney or its pelvis with the Tænia echinococcus, Eustrongylus gigas, and filaria.

Age.—Pyelitis may develop at practically any age, but it is somewhat more common after the thirtieth year.

Principal Complaint.—This varies greatly with different stages of the disease, and also with the virulence of the type of infection. When pyelitis occurs during the course of the infectious fevers, it does not cause any inconvenience to the patient unless the suppurative process is a severe one. Generally speaking, however, even in mild types of infection of the renal pelvis there are backache, tenderness upon deep pressure in the loins and over the kidneys. As the disease progresses all these symptoms become exaggerated until the distress in the loins is painful, and the patient may describe pains that are boring or tearing in character.

At the onset of the disease there is usually a chill or a series of chilly sensations, which may last for from a few minutes to several hours, and, as a rule, is followed by moderate fever. The effect of the suppurative process upon the general health becomes apparent early, and the patient complains of weakness, emaciation, anorexia, palpitation, headache, dyspnea, and lassitude. Night-sweats are occasionally a troublesome symptom late in the disease.

We have seen cases in which, after several weeks, general infection of the kidney developed, which was followed by septic foci in different parts of the body. One of our cases terminated in abscess of the brain, and another in ulcerative endocarditis. Septicemia has been known to follow pyelitis, and in such instances the symptoms are those of septicemia plus the symptoms resulting from infection of the special organ showing most marked involvement (brain, lung, heart, etc.).

At times pyelitis becomes chronic, and this is especially true when the infection is tuberculous in nature.

Thermic Features.—Pyelitis, when it develops during the course of any other infectious condition, causes an exaggeration of the symptoms of the initial disease present, causing a moderately increased elevation in the temperature. If the fever has been regular, it may be converted into either the remittent or the hectic type. In mild pyelitis the temperature is usually elevated from one-half to two degrees, but in the severer type it is common to see an elevation of from two to four degrees in the afternoon. When pyelitis alone exists, the temperature is that of general sepsis displaying evening exacerbations with morning remissions, and at times intermissions—the so-called septic temperature. A fact ever to be borne in mind is that suppuration of a mucous surface may exist without any coincident marked febrile changes.

Septicemia not infrequently follows, when the temperature assumes a continuous type, and the patient suffers from the so-called typhoid state. Should meningitis, endocarditis, multiple abscesses of the kidney, or abscesses of the perinephritic tissue develop during the course of pyelitis, a high temperature of the continuous type is usually observed.

Physical Signs.—Inspection is negative except in cases in which the pelvis of the kidney is greatly distended with pus, or in which perinephritic suppuration is associated, when the physical signs are practically those of perinephritic abscess. (See p. 697.) Early during the course of pyelitis there may be but slight, if any, evidence of anemia, but as the disease advances extreme pallor and a hectic flush appear, and at times a muddy or dusky complexion is presented.

Palpation discloses slight rigidity of the muscles of the affected side of the abdomen and of the loin. Upon deep pressure with the finger-tip it is possible to outline a tumor-mass when the nephritic or perinephritic tissue is involved.

Catheterization of the Ureters.—This means of diagnosis is of great aid, and is especially applicable in pyelitis in the female, but even in male subjects it is possible to catheterize the ureter of the affected side and thereby determine whether or not the urine from that kidney contains pus.

Cystoscopic examination is often invaluable, revealing, as it does, the presence of suppuration of the bladder; it may also disclose the vesical condition to which pyelitis is secondary. (See Contributing and Exciting Causes, p. 685.)

Laboratory Diagnosis.—The healthy kidney may become hypertrophied, in order to compensate for its fellow when pyelitis has incapacitated one kidney, and it is for this reason that a knowledge of the quantity of urine voided during the twenty-four hours is of but little, if any, diagnostic value. The characteristic feature of the urine is that it contains pus-cells. This secretion also shows the presence of shreds of mucus, red blood-cells, leukocytes, and bacteria. Whenever the calices and larger renal tubules become involved, large hyaline and granular casts are commonly present. Cylindric shreds of mucus that are covered with bacteria are probably the most characteristic microscopic findings in pyelitis.

Chemically, mucin is one of the earliest urinary findings in pyelitis, and we have observed cases in which mucinuria persisted for months without giving any other symptoms referable to pyelitis. The reaction of the urine may be either acid or alkaline, being controlled largely by the variety of pyelitis present, and by whether or not it exists as a complication of some infectious fever. *Albuminuria* is an early symptom, and continues throughout the course of the disease, the amount of albumin fluctuating with the type of infection present. Indicanuria and hematuria are occasional findings.

A bacteriologic study of the urine is of great service in diagnosticating tuberculosis of the renal pelvis, since tubercle bacilli (Plate IA, p. 83) in the urine are quite common in this condition. Other bacteria are nearly always present, and it is the rule to find colon bacilli and many pyogenic cocci and bacilli in the urine in cases of chronic pyelitis.

The Blood.—Pyelitis may exist for prolonged periods without showing any decided effect upon the blood, a feature characteristic of suppurative processes of the mucous surfaces in general. When there are suppurative foci in the renal tissue or in the perinephritic structures, leukocytosis develops, and this increase in the number of white cells affects for the most part the polymorphonuclear elements. The red cells and hemoglobin are relatively

decreased in advanced cases of pyelitis, and the blood displays the other findings characteristic of secondary anemia. (See p. 356.)

A bacteriologic study of the blood may show the presence of bacteriemia when there is general sepsis or such complications as ulcerative endocarditis, meningitis, hepatic abscess, and the like.

Summary of Diagnosis.—Pain and tenderness over the region of the kidney, with a possible history of traumatism or of tuberculosis, together with pyuria, are essential factors in formulating a diagnosis of pyelitis. A history of repeated attacks of renal calculi or of inflammatory processes of the bladder contributes liberally toward the formation of a diagnosis, and catheterization of the ureters, with the recovery of urine containing pus from the affected side, is conclusive evidence of the existence of pyelitis.

Calculous pyelitis may be revealed by an *x*-ray examination, and the detection of tubercle bacilli in the urine likewise indicates the existence of tuberculous disease of the renal pelvis. Lumbar pains and tenderness on one side of the back and abdomen are likewise suggestive of pyelitis. It is to be remembered that in certain cases the pain of pyelitis is vesical rather than lumbar, and that vesical tenesmus may be present.

Differential Diagnosis.—Pyelitis is to be distinguished from perinephritic abscess, cystic kidney, tuberculosis of the suprarenal body, and impaction of the colon. The accompanying table may serve, in a measure, to differentiate these conditions, which are distinguished only with extreme difficulty.

TABLE SHOWING THE DISTINCTIVE FEATURES BETWEEN PYELITIS, NEW-GROWTHS OF THE SUPRARENAL CAPSULE, TYPHLITIS, WITH IMPACTION OF THE COLON AND PERINEPHRITIC ABSCESS.

Pyelitis.	New-growths of the Suprarenal Body.	Typhlitis with Impaction of the Colon.	Perinephritic Abscess.
1. History of renal calculi common.	1. Tuberculous diathesis.	1. History of obstinate constipation which has existed for a long time.	1. History of renal colic the rule. Traumatism to the region of the kidney.
2. Pain and tenderness in the region of the kidney. In extreme cases the pain becomes tearing or boring in character. Pain continuous, and may rarely be localized in the bladder.	2. Pain boring in character, and located at the tenth rib on the right and at the eleventh on the left.	2. Some pain and tenderness along the ascending colon.	2. Pain quite general, and limited to one or other loin.
3. Temperature 100° to 103° F., and is usually of the hectic type.	3. Temperature normal.	3. Temperature normal or but slightly elevated.	3. Temperature of the hectic type with morning remissions and evening exacerbations. Fluctuates between normal or subnormal, and 102° to 104° F.

Pyelitis.	New-growths of the Suprarenal Body.	Typhlitis with Impaction of the Colon.	Perinephritic Abscess.
4. Contour of the loin not distorted.	4. Often slight fullness posterior and below the twelfth rib.	4. Contour of the loin distorted, and there may be bulging of the abdomen over the impacted colon.	4. Distortion of the normal contour of the loin. (See Figs. 269–271.)
5. In uncomplicated pyelitis the kidney is not palpable.	5. Hard tumor palpable above kidney.	5. A soft, dough-like tumor which is readily outlined.	5. It is usually possible to palpate a mass in the region of the kidney.
6. Uiine contains pus, albumin, mucus, and at times blood. Large casts covered with bacteria are occasional findings.	6. Urine normal.	6. Urine contains a large amount of indican.	6. It is usually possible to palpate a mass in the region of the kidney.
7. Urine contains tubercle bacilli or the bacteria of suppuration.	7. Bacteriologic study negative.	7. Bacteriologic study negative.	7. Bacteriologic study negative unless complicated by pyelitis.
8. Blood may show evidences of secondary anemia, with a moderate increase in the number of leukocytes.	8. Secondary anemia, leukocytes normal.	8. No leukocytosis.	8. A decided leukocytosis develops early, and continues throughout the disease.
9. *x*-Ray may reveal the presence of calculi in the renal pelvis.	9. *x*-Ray negative.	9. *x*-Ray negative.	9. *x*-Ray negative.
10. Catheterization of the ureter of the affected side recovers urine containing pus.	10. No pus in urine.	10. No pus in urine.	10. No pus unless kidney tissue is involved.

Course and Duration.—Mild cases of pyelitis are of but short duration, and, in fact, in many instances would not be detected unless an examination of the urine were made. Usually, the milder types, which are seen during the course of infectious fevers, go on to recovery in from a few days to one or two weeks.

Calculous pyelitis tends to assume a chronic condition unless the stone is removed from the pelvis of the kidney.

Tuberculous pyelitis, so far as we are aware, is not curable, yet we have known patients to suffer from this type of the disease for a number of years. In one instance, a private patient, the diseased kidney was removed, and after three years the patient was in good health.

Severe and chronic types of pyelitis may last for from a few months to several years, during which time the patient suffers extreme pain and displays the general symptoms of secondary anemia.

NEPHROLITHIASIS.

Pathologic Definition.—A condition characterized by the formation of hard, stone-like masses in either the substance of the kidney or the renal pelvis; these concretions may be either large or small, smooth or rough, and regular or irregular in contour. When located in the renal pelvis, the mucous surface first becomes congested, and later a catarrhal inflammation develops, which has a tendency to become infected by pathogenic bacteria.

Varieties and Pathology.—Nephrolithiasis may be divided primarily into renal nephrolithiasis and pelvic nephrolithiasis. The condition is again classified in accordance with the size and character of the concretions present: (*a*) Renal sand, in which the inorganic substance is composed of small particles that appear to have been finely pulverized. (*b*) Renal gravel, composed of concretions varying from the size of a millet-seed to that of a pea. (*c*) Renal calculi, that vary from the size of a hazel-nut to a mass that fills the entire renal pelvis; these large stones, as a rule, present rough surfaces. (*d*) Dendritic or coral calculi, which, as their name implies, are irregular in contour, may fill a large portion of the renal pelvis, and present irregular indentations and projections that extend into the kidney.

Renal calculi may be classified according to their chemic composition. The uric-acid calculus is the most common variety; although it is possible to find a renal calculus that displays, upon section, a more or less laminated appearance, the different strata being ofttimes composed of various inorganic substances. Other varieties of calculi may be composed, for the most part, of calcium oxalate, calcium phosphate, or ammonium urate. Occasionally we encounter renal calculi composed largely of xanthin or of cystin (light yellow color), displaying an amyloid luster. Calcium carbonate fibrin, indigo, and mucous débris may form renal concretions, but these are uncommon.

The pathologic changes induced by the presence of renal calculi vary in direct proportion to the intensity of the irritation produced by them, and to the length of time this irritation has existed. The changes caused by a dendritic calculus are more pronounced than are those of calculi with smooth surfaces. "In one of my own patients the left kidney was, apparently, nearly twice the normal size, owing to the presence of a large coral calculus (uric acid and urates), connected by an isthmus with a rounded stone in the inferior portion, quite as large as a large walnut. The pelvis of the right kidney also contained a dendritic calculus" (Anders).

Stones may be lodged in the kidney pelvis for years without causing decided pathologic change, although pelvic calculi in nearly all instances excite a pyelitis. (See Pathology of Pyelitis, p. 685.) By pressure large renal concretions may cause necrosis of the pelvis of the kidney, and the projecting spines of dendritic calculi pressing upon the kidney tissue may cause renal atrophy. Small calculi may become lodged in the ureters. (See Hydronephrosis, p. 681.)

Predisposing and Exciting Factors.—Age.—Practically all ages are subject to nephrolithiasis. We have seen this condition present in early childhood, in young adults, and in the aged, although our experience has been that renal calculi are far more common before the age of fifteen and after the age of fifty.

Males appear to be affected oftener than females. The uric-acid or

lithemic diathesis and gout serve as contributing factors. Persons of sedentary habits and those who indulge too freely in rich foods and alcoholic intoxicants are especially subject to this disease. Nephrolithiasis is frequently seen in several members of the same family, which fact would suggest that heredity plays a rôle in the production of this disease.

Catarrhal pyelitis, through the production of mucus in the renal pelvis, contributes liberally toward the condition, since these particles of mucus act as foreign substances around which the urinary solids collect, and the prolonged use of renal irritants in a similar way favors nephrolithiasis. Chemic changes that enhance the precipitation of the normal solids of the urine certainly play an important part in the production of renal calculi. When the urine contains abnormal quantities of uric acid, calcium oxalate, cystin, phosphates, or chlorates, nephrolithiasis is especially likely to supervene.

Again, pus and blood, when of renal origin, may become lodged in the pelvis of the kidney and become covered with renal sand. Parasites in the pelvis of the kidney may excite pyelitis. In the vast majority of instances the crystalline substances of the urine collect upon particles of mucin, epithelium, bacteria, blood, renal casts, or the ova of parasites, and it is highly probable that these figure prominently in the production of renal calculi.

Principal Complaint.—Symptomatology.—There is often gradual failing in the health, and the patient becomes weak and anemic, and has a sensation as of weight or soreness, which is localized to one or the other side of his back. In fact, it is possible for a calculus to remain in the pelvis of the kidney for a number of years without giving rise to any acute symptoms, and patients often pass large amounts of uric acid for a period of many years without any attending discomfort. Tenderness over the kidney, slight backache, and occasional attacks of hematuria are complained of by nearly every subject of nephrolithiasis.

Thermic Features.—When a renal calculus becomes lodged in the pelvis of the kidney, and in turn excites a variable degree of inflammation of the mucous surface of the renal pelvis and of the kidney itself, moderate fever, ranging between 99° and 102° F., is likely to be present. The so-called renal intermittent fever is due to the lodging of a calculus in the ureter, and is ushered in by renal colic, following which the temperature rises suddenly to from 101° to 104° F., and falls by crisis to normal or subnormal as soon as the calculus has been dislodged or expelled into the urinary bladder. In a few instances the calculus remains in the ureter for a long time or the ureter itself may rupture—in either case the temperature falls to normal or subnormal. There may be a subsequent rise of temperature the result of traumatism received by the ureter. Renal intermittent fever closely resembles Charcot's intermittent fever, which is associated with cholelithiasis.

Uremic Manifestations.—There may, at times, be a destruction of the renal tissues, and such patients may develop symptoms of uremia, among which are headache, vertigo, numbness of the hands and feet, renal asthma, dimness of vision, and floating of specks before the eyes. Nervous twitchings may occur, and, in fact, coma may follow in this class of cases.

Frequency of micturition and pain are also among the symptoms of renal calculus, and may result from—(*a*) Inflammatory processes in the kidney, ureter, or bladder; (*b*) pain from an impacted stone in the ureter; (*c*) pain from pressure of large calculi lodged in the renal pelvis; (*d*) pain from the passage of concentrated urine, as is not only found in nephrolithiasis,

but also in certain acute fevers, gout, and after the administration of renal irritants; (*e*) it is often a reflex symptom, and is more or less constant in inflammatory disease of the genito-urinary tract. As a rule, the patient does not complain of frequency of micturition, and it is only when the physician makes inquiry as to the number of times the patient is compelled to rise during the night that this symptom is detected.

Renal Colic.—In several cases we have obtained a history of violent exercise having been indulged in just prior to the development of renal colic, and it has been asserted that the impacted calculus is often dislodged by violent exertion.

The onset is sudden, and there may be a slight chill or chilly sensations, followed by intense pain in one or the other loin. The patient describes this pain as descending lower and lower, following the course of the ureter from the kidney to the bladder. The pain may radiate to the back, but more often it passes to the inguinal and pubic regions. Retraction of the testicle is common, and the pain may at times extend down to the thigh, in the region of the femoral ring, when it is likely to be excruciating in character.

The pain of renal colic is described as tearing in character, and continues until the stone has been dislodged from the ureter. In a few instances we have seen cases of renal colic in which the pain began in the region of the kidney, and was then reflected over the entire abdomen, but in these the characteristic area of distribution of the pain developed a few hours later. Vesical tenesmus may be present, and is due to spasm of the vesical sphincter.

Collapse.—If the pain is severe or is prolonged for several hours, the patient may enter into a state of collapse, in which case there are nausea, vomiting, a weak, thready pulse, decided tremor, restlessness, and syncope. After an attack of renal colic the patient usually falls into a deep sleep, from which he awakens drenched in perspiration. Between attacks of renal colic the patient usually enjoys fairly good health, although he may experience occasional attacks of slight pain in the region of the kidney, together with vesical tenesmus and hematuria.

Physical Signs.—Inspection of the loins is negative, unless nephrolithiasis is complicated by perinephritic abscess, in which case there is a fullness over the area of the affected kidney. (See Figs. 269, 270, 271.) The patient's attitude is quite characteristic: If sitting, he is bent forward and reclines toward the affected side; if seen in bed, he is usually somewhat restless, and the thigh of the affected side is, as a rule, flexed upon the abdomen. When a stone has been lodged in the ureter for some time, the patient's expression is anxious, the face is beaded with perspiration, there is extreme pallor, and the general appearance is that of collapse.

Palpation.—It is possible to isolate the area of tenderness in the region of the kidney, and while the stone is passing through the ureter, its exact position can often be ascertained by making deep pressure over the course of that structure (Fig. 252). If the ureter remains obstructed for an indefinite period, hydronephrosis follows, and it is then possible to palpate a tumor-mass in the renal region. The abdominal muscles may be somewhat rigid during the attack of colic, and in certain cases this rigidity is most pronounced upon the affected side. The testicle on the affected side is retracted and tender.

Percussion is of but little value in making the diagnosis unless nephrolithiasis is complicated by hydronephrosis or by perinephritic abscess.

Roentgen Diagnosis.—Stones in the kidney and in the ureter may be recognized by means of the *x*-ray, and this is a most reliable diagnostic measure.

Lenard* states: "The most striking proof of the accuracy of the Roentgen method is that it has shown that ureteral colic and ureteral calculi are more frequent than renal colic, and that it has led to a differentiation between the symptoms of these two conditions." Again, in speaking of calculi of the ureter, he remarks: "There is no greater evidence of its accuracy (Roentgen diagnosis) than the fact that it has shown ureteral calculi to be much more frequent than was previously supposed, and, in fact, they have been shown to be more frequent than renal stones in a series of 330 cases examined. Their ratio has been 33 to 66, including cases not operated upon, yet confirmed by the passage of the calculus, and 29 to 40 in cases confirmed by operation or the passage of the urethral calculi."

Laboratory Diagnosis.—When the gravel passed is small, it is customary to find uric acid crystals (Plate XIII), crystals of the phosphates (Fig. 260), cystin (Fig. 264), and amorphous urates in the urinary sediment. A marked feature of the urine of nephrolithiasis is that, upon standing, it precipitates a heavy sediment rich in one of these inorganic substances. When the calculus is large, the urinary sediment may contain crystals of the substance of which the stone is composed. It is customary for a calculus lodged in the renal pelvis to be composed of the same substance that is being passed in crystalline form with the urine. When the calculus produces sufficient irritation in the pelvis of the kidney or in the kidney proper, the urine contains mucus, large flakes of epithelial cells, pus, blood, and, sometimes, casts of the larger renal tubules.

Stone in the kidney substance does not give rise to urinary symptoms suggestive of its existence. Urine obtained by catheterization of the ureter of the diseased side may contain blood, mucus, and other substances suggestive of renal inflammation, whereas the urine from the opposite side is generally clear and approximately normal.

Summary of Diagnosis. — Among prominent diagnostic features should be mentioned catarrh of the renal pelvis and highly acid urine, which has been present over a prolonged period. The existence of mucinuria and the presence of an excess of uric acid or of phosphates are valuable features in arriving at a diagnosis of renal calculus, and are present even before renal colic has developed. Renal colic is recognized by the cramp-like pain, which radiates to the penis or pubis, and which is felt down the side along the course of the ureter. Frequent micturition, with the passing of bloody urine, is valuable evidence in diagnosing renal calculus. In children, the examination of the urine is a practical method of arriving at a diagnosis, since a description of the characteristic pain cannot be obtained.

Repeated attacks of colic, intermittent fever, and sweats are always suggestive of renal calculus. The *x*-ray provides a positive means of recognizing renal and ureteral calculi.

Differential Diagnosis.—Renal calculus is to be distinguished from hepatic calculus, cystic calculus, and intestinal colic. The accompanying table serves to point out the distinctive differences between these four conditions:

* Lancet, xvii, 1905, p. 1635.

TABLE SHOWING THE DISTINCTIVE FEATURES BETWEEN RENAL CALCULUS, HEPATIC CALCULUS, VESICAL CALCULUS, AND INTESTINAL COLIC.

Renal Calculus.	Hepatic Calculus.	Vesical Calculus.	Intestinal Colic.
1. History of pain and soreness over the region of the kidney.	1. Soreness in the right hypochondrium, and possibly history of previous attacks of jaundice.	1. History of an irritable condition of the bladder.	1. History of a too liberal ingestion of rich, uncooked or unripe foods.
2. Patient complains of pain upon receiving a sudden jar, as in alighting from a carriage or in descending a stair.	2. Jar does not cause pain.	2. Jar excites pain at pubes.	2. Jarring does not cause localized pain.
3. Pain in the region of the kidney, described as boring or tearing in character, and radiating along the ureter to the pubes, femoral ring, and thigh.	3. Pain in the region of the liver and radiating to the angle of the right scapula.	3. Pain in the pubic and perineal regions, over the bladder, and at the glans penis.	3. Pain in the epigastrium and umbilicus, reflected to a variable degree over the entire abdomen.
4. Deep palpation elicits tenderness over the kidney and along the course of the ureter.	4. Tenderness in the epigastrium and over the gall-bladder.	4. Tenderness over the urinary bladder.	4. Tenderness seldom localized.
5. Nausea and vomiting may follow when the pain is intense. The vomitus is usually composed of mucus and particles of food.	5. Vomiting common.	5. Vomiting unusual.	5. Vomiting of undigested food. Vomitus oft-times bile-stained.
6. Temperature rises abruptly to 102°, 103°, or 104° F., and falls by crisis to normal or subnormal with the cessation of the pain.	6. Temperature resembles that of renal colic, but is usually higher.	6. Temperature is usually normal unless there is acute cystitis, when a temperature of 100° to 102° F., of the continuous type, may be seen.	6. Temperature 100° to 103° F. at onset, but may become normal within a few hours. No decided rise and fall, as is seen in renal and hepatic colic. A subnormal temperature is seen when purging is severe.
7. Frequent painful micturition, with the passage of bloody urine.	7. Frequency of micturition not affected.	7. Frequent micturition, and the urinary flow may end abruptly, to return in a few minutes. Pain is reflected to head of penis.	7. Frequency of micturition not affected.

Renal Calculus.	Hepatic Calculus.	Vesical Calculus.	Intestinal Colic.
8. No discoloration of the skin and conjunctivæ.	8. Jaundice one or two days after the attack.	8. Jaundice absent.	8. Catarrhal jaundice may follow.
9. Urine contains blood and mucus during the attack, but is usually clear twenty-four hours later.	9. Urine clear during the attack, bile-stained twenty-four to forty-eight hours later.	9. Urine may contain blood, pus, mucus, and albumin.	9. Urine of high color, high specific gravity, and rich in indican
10. Bowels not affected.	10. Stools clay colored, and show an absence of bile during the attack, with an excess of bile following the dislodgment of the gall-stone. Calculus may be found in the feces. Feces rich in fats during attack.	10. Feces normal.	10. Diarrhea and purging the rule, and indol and skatol conspicuous.
11. Examination by Roentgen-rays discloses the presence of a stone in the pelvis of the kidney or in the ureter.	11. *x*-Rays may show the existence of stone in the gall-bladder, although this is less definite than in renal calculus.	Vesical calculus is easily recognized.	11. *x*-Ray examination negative.

Clinical Course.—The urine may contain renal sand for a period of several years, and yet the patient suffer no inconvenience. When evidences of a catarrhal pyelitis are present, the malady becomes more serious, and the likelihood of renal colic is materially increased. It is not unusual to find persons who have had many attacks of renal colic, and who apparently enjoyed perfect health during the intervals between such attacks. When renal calculus gives rise to acute symptoms, surgical intervention may become necessary. Such complications as acute nephritis, nephritic abscess, pyelitis, and perinephritic abscess add materially to the seriousness of the disease. A fatal termination is rare in uncomplicated cases.

PERINEPHRITIC ABSCESS.

Pathologic Definition.—A condition characterized by an accumulation of pus in the retroperitoneal tissues surrounding the kidney.

Anatomic Consideration.—The perinephritic tissue becomes the seat of suppuration, usually posterior to the kidney. It may be limited to a single abscess of varying size, or, in severe cases, the pus may be found to have infiltrated nearly all of the retroperitoneal tissue. The pus shows a tendency to burrow into the pelvis, and may follow the course of the psoas muscles, commonly pointing in the vicinity of Poupart's ligament. When the course of the pus is deflected backward, the abscess may open on the skin surface. Rarely, the pus is reflected upward and forced through an

opening in the external arcuate ligament of the diaphragm, when the signs of empyema will appear coincidentally with a lessening in the size of the lumbar tumor. Occasionally, the lung may be adherent to the parietal pleura, when the pus will escape directly into the bronchi, whereupon the symptoms of purulent bronchitis with copious expectoration follow.

There are numerous records of perinephritic abscess having perforated the urinary bladder, the gall-bladder, the general peritoneal sac, the colon, and the vagina. The relative frequency of points of rupture in places other than the loin is best shown by Küster's table of 230 cases of perinephritic abscess, of which 34, or 14.78 per cent., belonged to this class:

Pleura and bronchi	18
Intestine	11
Peritoneal cavity	2
Bladder and vagina	2
Bladder alone	1

The surrounding peritoneum is thickened, and shows evidence of prolonged inflammation.

Predisposing and Exciting Factors.—Traumatism serves as a potent etiologic factor, although in the vast majority of cases perinephritic abscess is secondary to a similar suppurative process elsewhere, *e. g.*, in the pelvis of the kidney, in the ureter, appendix, liver, Fallopian tubes, and bladder; it may also be secondary to abscess of the pelvis and caries of the spine.

Renal colic, hepatic colic, and stone in the urinary bladder each contribute toward the development of perinephritic abscess. Indeed, perinephritic abscess may be directly traceable to a suppurative ureteritis, following which infection of the upper portion of the urinary tract has occurred.

Küster, in his recent complete report of 230 cases, found that 59, or one-fourth of the total number of cases, followed abscess of the kidney, and that of these, 31 were due to renal calculus. Guiteras, in a recent paper, holds that in nearly all cases perinephritic abscess is secondary to renal diseases. His analysis of 15 cases gives:

Calculi	4
Tuberculosis	4
Pyonephrosis	3
Ascending pyelonephritis	2
Rupture of kidney	1
Empyema	1

In a few recorded cases perinephritic abscess has followed echinococcus cyst and tuberculosis of the suprarenal body and adjacent structures. Stab wounds and gunshot wounds are not infrequently followed by the formation of perinephritic abscess.

Age and Sex.—In Guiteras's series of 15 cases the age of the oldest was fifty; the youngest was twenty, and the average age was thirty-three years. Perinephritic abscess is found at practically all ages: it has been observed at the fifth week of life, but is more common during early adult life or between the ages of twenty and forty-five.

Sex figures prominently in this connection, two-thirds of all cases being found in males.

Perinephritic abscess is occasionally seen as a complication of typhoid fever, smallpox, and typhus fever.

Varieties.—**Secondary abscess,** resulting from a similar process elsewhere, is by far the commonest variety.

The perinephritic tissue may follow traumatism and tuberculosis of the kidney or suprarenal body. In some instances the pus of a large empyema may burrow its way through the diaphragm, posterior to the peritoneum, accumulating in the perinephritic tissue. Bilateral perinephritic abscess is rare, Guiteras having found it but twice in an analysis of 197 reported cases.

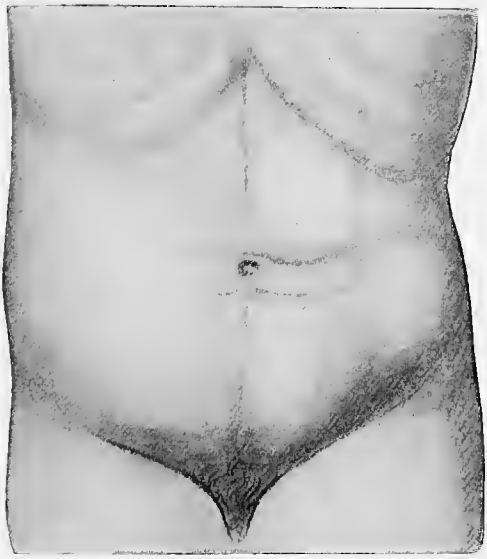


Fig. 269.—Showing the Obliteration of the Curve under the Ribs in a Case of Perinephritic Abscess on the Right Side (Guiteras).

Principal Complaint.—One of the most pronounced symptoms is a dull, throbbing *pain* immediately over the affected area, the pain being aggravated by motion and by deep pressure. Occasionally the pain is described as radiating to the front of the pelvis and down the thigh, being reflected along the course of the nerve-trunks. From pressure of the abscess tingling and numbness of the hips and lower extremities of the affected side at times occur. There is progressive loss of flesh and strength, together with a more or less pronounced secondary anemia. Headache, vertigo, palpitation, insomnia, and anorexia are common symptoms in this affection.

Fig. 270.—Fullness of Left Loin in Perinephritic Abscess, Illustrative of Private Case.

When the pus of perinephritic abscess burrows posteriorly and upward it may, as previously stated, pass through the arcuate ligament of the diaphragm and escape into the pleural sac; here it may accumulate rapidly, and give rise to cough, dyspnea, and cyanosis, and increased pulse and respiration rate may develop coincidentally with a lessening or disappearance of the swelling in the loin (Fig. 270). Occasionally the pus gains access to the bronchi, and when this occurs, a copious expectoration of blood-streaked pus follows.

If the pus ruptures into the peritoneal cavity, general peritonitis results. In a few reported instances

the abscess ruptured directly into the intestine, and the discharge of a large quantity of purulent material from the rectum, together with a lessening in the size of the tumor in the loin, followed. Guiteras and others have cited instances in which the abscess ruptured into the ureter or bladder and the pus appeared in the urine. Statistics show that 85 per cent. of these abscesses point externally, in which case the general symptoms of abscess of the superficial tissues are present.

Thermic Features.—The temperature early becomes hectic in char-

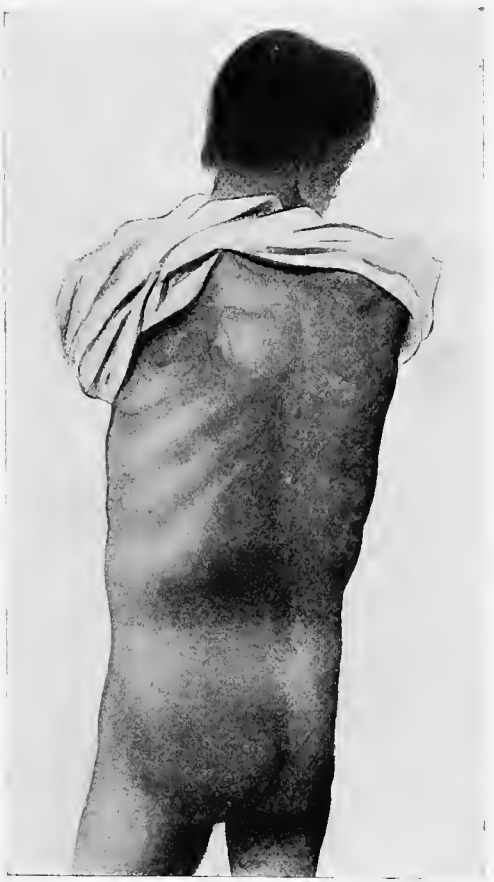


Fig. 271.—Perinephritic Abscess of Left Side. Notice Obliteration of Natural Curve of Loin (Guiteras).

acter, showing decided evening exacerbations with morning remissions; at times it may be of the intermittent type, particularly if pyemia supervenes. These exacerbations of temperature are usually followed by profuse sweats, after which the patient is profoundly prostrated. In severe cases the temperature may continue high with but slight remissions for a period of days or even weeks—the typical continued type of pyrexia. The temperature is likewise influenced by the nature of the infecting organisms and by

the presence of a fistulous communication with the kidney, the intestine, or the skin.

Physical Signs.—Inspection.—The sufferer always inclines toward the affected side, both when standing or sitting; and when resting in bed, he flexes the thigh of the affected side well upon the abdomen and the leg upon the thigh—all of which positions are assumed to relieve the general tension. When the patient is standing with the arms folded across the chest or with the hands elevated on a level with the top of the head, a decided bulging of the affected side (Fig. 271) is to be seen; bulging of the loin is equally conspicuous on an anterior or a posterior view of the patient (Figs. 269 to 272). The natural curve of the loin is obliterated in perinephritic abscess, and, indeed, a decided bulging of this region is frequently observed (Fig. 269). In walking the patient inclines toward the affected side, and takes short steps with the corresponding foot, lifting it but slightly above the floor.

The skin and mucous membranes are pale, and ofttimes present a muddy tinge. The superficial fat seems to have disappeared, and the bones are quite prominent.

Palpation.—The skin covering the abscess is tense and pits upon pressure, and deep pressure increases the pain. The edema of the skin may extend for some distance around the abscess. Fluctuation is commonly present, and is usually more conspicuous at some one point of the distended area. The rigidity of the abdominal muscles is decidedly increased on the affected side.

The pulse is rapid, and after the abscess has existed for some time, it is likely to be weak, thready, and irregular.

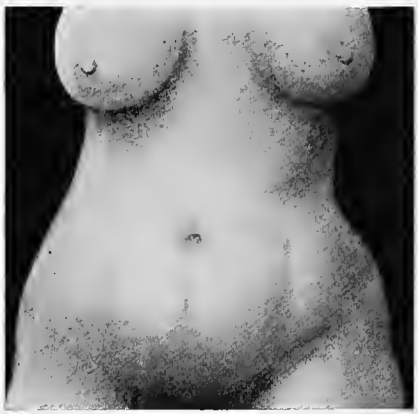


Fig. 272.—Illustrative of Case of Right Perinephritic Abscess, Showing Prominence of Right Flank.

Percussion is of but limited value in the majority of instances in which the pus is confined to the abdominal cavity, but it is invaluable in cases in which the pus has escaped into the pleura.

Auscultation is negative over the affected area. The heart action is rapid, and its sounds are accentuated, a soft, systolic murmur may be heard over the base of the heart in cases in which anemia is prominent. When the pus drains through the lungs, numerous mucous and bubbling râles are heard over the affected side.

Laboratory Diagnosis.—The urine is practically normal in quantity, and contains albumin in varying amounts. If the abscess does not communicate with the urinary tract, but little albumin may be present in the urine, but when there is a direct communication between the abscess and the pelvis of the kidney, the urine is highly albuminous. A microscopic study shows the presence of pus-cells, shreds of mucus, and red blood-corpuscles. A bacteriologic study of the urinary sediment will reveal the presence of streptococci, staphylococci, and, in certain cases, tubercle bacilli.

The blood is deficient in hemoglobin, and the red cells are correspondingly

reduced, whereas a leukocytosis of from 10,000 to 25,000 is usual, and concerns, for the most part, the polymorphonuclear elements, which may equal 85 to 90 per cent. of the total number of leukocytes.

Stained specimens show degeneration of the erythrocytes.

Summary of Diagnosis.—The diagnosis of perinephritic abscess is based largely upon the character of the pain, the presence of edema and tenderness over the loin, bulging of the affected side (Figs. 269 to 271), and the presence of pus and of pus-producing bacteria in the urine.

Differential Diagnosis.—Perinephritic abscess is to be distinguished from a **large dendritic calculus** and from **pyelitis,** both of which conditions it simulates closely; it is, in fact, often secondary to these affections. The continuous boring pain and the hectic temperature, together with the bulging of the affected side, serve as the salient points that favor the existence of perinephritic abscess. Among other conditions for which perinephritic abscess may be mistaken are **lumbago, nephralgia, Pott's disease, psoas abscess, lumbar hernia in Petit's triangle, hematoma** following renal injury, **new-growths** of the kidney, and **hydronephrosis,** but in the vast majority of instances a careful consideration of the clinical history, together with a physical examination, will enable one to distinguish between them.

Clinical Course.—Whenever a diagnosis is attained the surgeon should be consulted. The course of the abscess varies greatly: (*a*) It may become walled off by connective tissue, and eventually be absorbed; (*b*) it may rupture into the peritoneal cavity and terminate in general peritonitis; (*c*) it may rupture into any of the abdominal viscera and be evacuated through the urinary or intestinal tracts; (*d*) it may discharge its fluid through the diaphragm into the pleura or the lung, and the pus be expectorated; (*e*) it may rupture into the gall-bladder; (*f*) a far more common termination is for the abscess to rupture externally.

The duration varies greatly in different cases, but, as a rule, perinephritic abscess lasts from a few weeks to several months.

CARCINOMA OF THE KIDNEYS.

Consideration.—Renal carcinoma may be either a primary or a secondary condition, and, as a rule, it develops late in life, although it is occasionally observed in children. The abdominal growth is usually associated with decided pigmentation of the skin and an abnormal growth of hair in the pubic and axillary regions. Renal hemorrhage is a most prominent symptom, and when there are repeated profuse hemorrhages, the condition is most likely to be malignant. Pain may or may not be present until late in the disease, when, as a rule, a dull pain occurs in the loin and lumbar region. Varicocele may develop as the result of pressure upon the spermatic veins.

Urinalysis gives negative results except in those cases in which renal hemorrhages have occurred. *Microscopically,* the urine may contain red blood-cells and pus, and certain observers have found fragments of carcinomatous tissue in the urinary sediment.

SARCOMA OF THE KIDNEY.

Consideration.—This malignant growth may be either primary or secondary in nature, and may occur at any decade of life. The statistics of

Roberts, collected from the literature, show that of 75 cases, 67 occurred in children under ten years of age. Sarcoma of the kidney, therefore, holds a prominent place among the abdominal tumors of children, and the possibility of its existence should always be considered in examining a child with a unilateral abdominal enlargement.

Principal Complaint.—In many cases the only complaint is of slight discomfort in one or the other loin, which eventually becomes painful. The pain is dull, is confined to the lumbar region, and seldom radiates to the pelvis and thigh. A history of repeated attacks of hematuria is often obtained.

Physical Signs.—Inspection.—As a rule, the skin retains its normal luster, and the mucous surfaces are not extremely pale. The normal curve of the loin is lost (see Figs. 269 to 272), and there is bulging of the abdomen anteriorly, which sometimes extends beyond the median line; we have, however, seen a child three and one-half years old whose entire abdomen was greatly distended, the autopsy revealing a sarcomatous kidney weighing 9¼ pounds. In another case, that of a child of four and one-half years, the kidney removed at autopsy weighed 5¼ pounds. There may be pigmentation of the chest, abdomen, face, and extremities. In girls, there is a precocious growth of hair in the pubic and axillary regions.

Palpation.—Palpation will outline a smooth, elliptic tumor, located well posteriorly in the abdomen. The tumor is covered anteriorly by the colon, which gives a more or less doughy sensation to the palpating finger. Mensuration is of great importance in the diagnosis of sarcoma of the kidney, since these tumor-masses grow with great rapidity. In sarcoma of the kidney the tumor is always firm, whereas in rapidly growing carcinoma of the kidney the tumor is soft and less symmetric in contour. Upon deep respiration the tumor-mass is seen to rise and fall with the movements of the diaphragm. Few observers have noted pulsation over a sarcomatous tumor of the kidney.

The pulse is slightly accelerated early, and continues to increase in rapidity with the general interference with the circulation produced by the development of the tumor.

Percussion confirms the findings of both inspection and palpation. A tympanitic note is generally obtained anteriorly, due to the colon overlying the tumor, whereas at the side and posteriorly the note is dull. In a few instances the growth may be of such extraordinary size as to flatten the portion of the colon which is anterior to it, when a variable degree of dullness will be obtained by percussion over the anterior surface of the tumor.

Laboratory Diagnosis.—Early during the course of sarcoma of the kidney the blood is practically normal, but when there is a decided evidence of malnutrition, secondary anemia is the rule. Hematuria occurring during early life, especially when the attacks are frequent and the quantity of blood lost is large, is always suggestive of sarcoma, and this is especially true if it occurs in young females.

Summary of Diagnosis.—A rapidly developing unilateral abdominal distention in a child is always suggestive of sarcoma of the kidney. The smoothness of the tumor, the presence of the colon anteriorly to the mass, and the limited motion of the tumor on deep respiration favor a diagnosis of renal sarcoma. The absence of secondary anemia early during this malady points toward sarcoma, whereas in carcinoma secondary anemia develops early, and, while the tumor is yet small, constitutes a prominent feature.

Duration.—Sarcoma of the kidney terminates fatally in from a few months to one and one-half years.

ADDISON'S DISEASE.

Pathologic Definition.—A chronic malady characterized by degeneration of the suprarenal capsules, the semilunar ganglia, a bronze pigmentation of the skin, and, at times, fatty degeneration of the heart, liver, and kidneys. The pathologic findings at postmortem vary considerably in different cases.

Predisposing and Exciting Factors.—A history of tuberculosis may be obtained, as may also a history of traumatism to the trunk. Males are affected in approximately 70 per cent. of cases. Young adults are most often diseased, although this condition may be observed at the extremes of life.

General Complaint.—Before definite cutaneous manifestations are detectable there are experienced a progressive asthenia, pronounced lassitude, and loss of physical and mental energy. Later fatigue, dyspnea, headache, tinnitus aurium, and vertigo develop. The appetite is poor and nausea with paroxysmal attacks of vomiting are common. Pain in the epigastrium and lumbar region is frequent, and this, at times, assumes the neuralgic type. Diarrhea may be a late and annoying symptom. The mind generally remains clear, although mental depression and delirium are occasional features.

Physical Examination.—**General.**—The patient's general appearance is that of an old person. He moves slowly, his general attitude is one of exhaustion. The skin displays a variable degree of pigmentation, which varies through the successive shade of a dusky yellow, bronze, olive, greenish brown, or black. Pigmentation is seldom uniform over the entire body. It is deepest, however, where normally pigmentation is present, *e. g.*, face, neck, back of hands, abdomen, groin, nipples, and genitalia. Pigmentation spots are often seen on the various mucous membranes. A brown line is occasionally apparent at the junction of the mucous membrane of the lips with the skin. Those portions of the body that receive pressure from the clothing also show an unusual degree of pigmentation. Decided irregularity in the pigmentation of both the skin and the mucous surfaces is a rather prominent feature of Addison's disease. Moderate enlargement of the superficial glands is at times present.

Local.—The mucous membrane of the buccal cavity often shows white patches, while certain portions of the skin may present similar areas of leukoderma. The apex-beat is feeble and there may be seen pulsation over the great vessels. The hair is at times rough, and apparently poorly nourished.

Palpation.—The *apex* is felt to be weak, the muscles flabby, the skin rather harsh, and the extremities cold. The pulse is lacking in both volume and force. Tenderness in the epigastrium and abdominal region is rather common.

Auscultation.—The heart-sounds are weak, and there is an appreciable lessening in the booming quality of the first sound.

Laboratory Diagnosis.—The quantity of urine voided during the twenty-four hours may exceed that of normal, and is often rich in indican.

The quantity of urea excreted during the twenty-four hours is subnormal. Tubercle bacilli have been found in the urine of those suffering from Addison's disease. There is a moderate reduction in both the red and the white blood-cells, and in the hemoglobin, although complicated cases may show leukocytosis. Where Addison's disease develops during the course of extensive tuberculous glandular involvement of the abdomen, the feces may contain an abnormal amount of fat, and rarely an absence of bile, due to involvement of both the liver and the pancreas. The feces rarely shows tubercle bacilli.

Differential Diagnosis.—Among the maladies with which Addison's disease may be confounded, especial mention should be made of (*a*) sarcoma and carcinoma of the peritoneum, diabetes (hepatic); (*b*) hepatic cirrhosis, and chronic venous, hepatic congestion (liver spots); (*c*) pregnancy during its latter stage, and diseases of the uterus and its appendages may at times be accompanied by extensive pigmentation of the skin (chloasmata); (*d*) vagabondism, the irritation from pediculi and dirt together with exposure is often followed by cutaneous pigmentation; (*e*) exophthalmic goiter; (*f*) the posteruptive stage of syphilis; (*g*) the prolonged use of nitrate of silver and the mixture of race-bloods may one and all, in exceptional cases, produce conditions that must in turn be distinguished from Addison's disease.

THE BLADDER.

DISEASES OF THE BLADDER.

VEGETABLE PARASITES IN THE URINE.

Under normal conditions the freshly voided urine is practically free from bacteria and fungi, but on standing exposed to the air, normal urine soon becomes loaded with organisms that are, as a rule, saprophytic in nature.

Fungi are common in diabetic urine. When there is general infection with the *actinomyces*, the urine may contain this fungus, and in local actinomycotic infection of the genito-urinary tract the actinomyces is present in the freshly voided urine. In general aspergillosis the Aspergillus fumigatus may invade the kidneys and appear in the urine. Both the aspergillus and the actinomyces have been detected in the urine of persons suffering from *acute cystitis*.

BACTERIURIA.

It must be remembered that the urine may contain both pathogenic and non-pathogenic bacteria; when, however, they occur in large numbers, their presence is to be regarded as a serious symptom, regardless of the variety of bacterium present. Bacteriuria is an essential symptom of the

acute infectious maladies in which bacteriemia exists; and the best example of this type of bacteriuria is seen in typhod fever, in which it is found in more than 30 per cent. of cases at some time during the course of the disease (Fig. 273). Bacteriuria occurs, however, during the course of ulcerative endocarditis, pyemia, gonorrhea, septicemia, streptococcemia, glanders, syphilis, and relapsing fever.

Bacteriuria is one of the symptoms of acute nephritis, and both cocci and bacilli are commonly found in the urine of children suffering from nephritis. In the acute nephritis complicating pneumonia, diplococci are occasionally present, but in sixteen cases in which cultured studies were made by us, pneumococci were not recovered from the urine. In one of our cases, that of a youth of fourteen years, the urine contained large numbers of streptococci for a period covering several months, following an attack of tonsillitis. Bacteriuria is encountered in cystitis, pyelitis, obstinate constipation, diphtheria, and scarlet fever. This finding has but limited clinical significance, except in cases of cystitis and when tubercle bacilli are found. The following diagram is designed to classify the various bacteria that are found in the urine and the types of infection present when bacteriuria is a symptom:

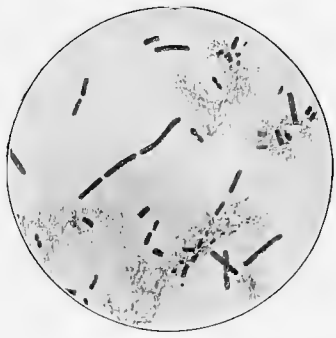


Fig. 273.—Typhoid Bacilli in Urine. Third week of disease. Stained with carbolfuchsin (Boston)

General Infections Excited by:

Streptococcus pyogenes. Pneumococcus. Micrococcus melitensis Gonococcus Staphylococcus pyogenes aureus. B. Typhosus. B. coli communis. B. Paratyphosus.	Nephritis need not necessarily be present.

Local Infections of the Genito-urinary Tract:

Nephritis. Pyelonephritis Ureteritis. Cystitis. Prostatitis.	B. coli. B. tuberculosis. B. pyocyaneus. B. pneumonia (Friedländer's bacillus). Streptococcus pyogenes. Pneumococcus. Gonococcus. Staphylococcus pyogenes. Streptococcus pyogenes.
Urethritis	Gonococcus. Staphylococcus pyogenes aureus and albus. Pneumococcus. Streptococcus pyogenes. Micrococcus catarrhalis.

Acute Cystitis.—Probably 50 per cent. of all urines in cases of acute cystitis show the presence of the Bacillus coli communis (commonly in pure

cultures). A high degree of acidity is seen in colon bacillus infection, whereas in a staphylococcus infection the acidity is seldom marked. The staphylococcus pyogenes albus occurs in from 15 to 25 per cent. of cases, and the Staphylococcus aureus is far less frequently found. The Bacillus pyocyaneus reveals its presence by the greenish color it lends to the urine; this green color is increased if the urine is kept at body-temperature. Brown* reports a case of infection with the Bacillus pyocyaneus, and we have found it present in the urine of two cases of acute cystitis—once in pure culture. The Bacillus proteus vulgaris has been found by Brown in alkaline (ammoniacal) urine containing both blood and pus.

Chronic Cystitis.—In a study of 24 cases Brown† recovered the Bacillus coli communis from the urine eleven times and the bacillus tuberculosis once; Staphylococcus pyogenes aureus was present three times, Staphylococcus pyogenes albus twice, and in two cases pyuria existed with sterile urine.

Acute and Chronic Pyelitis.—In nearly 50 per cent. of cases in which the urine is obtained by catheterization of the ureters the colon bacillus will be found, and the reaction of the urine will be acid. The Bacillus proteus vulgaris figures in from 20 to 30 per cent. of cases (urine alkaline). When the process is tuberculous, the bacillus tuberculosis is found by staining and by inoculation of animals with the suspected urine; cultivation of this organism from the urine is, however, impracticable.

Tubercle Bacilli.—In cases of tuberculosis of the bladder and of the kidney, tubercle bacilli may be found in the urine. It is always necessary to differentiate between bacillus tuberculosis and bacillus smegmæ, which is a normal inhabitant of the external genitalia in both sexes. To accomplish this differentiation care must be taken in the collection of the specimen, and an acid-alcoholic solution must be used in the decolorization process, since the latter organism is not alcohol-fast, although it is acid-fast. The acid-alcohol solution to be used in decolorizing is composed of sulphuric acid, 2.0; 95 per cent. alcohol, 25.0; water, 75.0.

Method.—Withdraw the urine by a sterile catheter into a sterile conical urine glass. The urine so withdrawn is allowed to stand until the sediment collects. The supernatant fluid is poured off and the sediment is centrifuged. The supernatant fluid is poured out of the centrifuge tube, the tube filled with distilled water that is free from tubercle bacilli, shaken so as to wash out the urinary salts, which interfere with the staining, and centrifuged again. This process is repeated once or twice. The sediment remaining after the third centrifugation is smeared on clean glass slides and allowed to dry in the air. The smears are fixed by passing three times through the flame, and then stained for five minutes with warm carbolfuchsin, washed in water, submitted to the action of the acid-alcohol solution for one minute, washed in water, counterstained with Löffler's alkaline methylene-blue for thirty seconds, dried, and examined under a $\frac{1}{12}$ inch oil immersion objective. If acid-fast bacilli are found by this method, a second smear should be decolorized in the acid solution overnight, washed in water, and counterstained in the Löffler's alkaline methylene-blue in order to be sure that they are not smegma bacilli.

Urine obtained by the catheterization of the ureters may be treated in the same way.

* Johns Hopkins Hosp. Rep., vol. x, No. 1, 2.
† Ibid., vol. x, No. 1,.2.

ACUTE CYSTITIS.

Pathologic Definition.—A disease characterized by an acute inflammation of the mucous membrane of the bladder, with or without punctate hemorrhages or ulcerations of the mucous surface. The surface of the bladder may be greatly congested, somewhat mottled, and covered with a catarrhal exudate, which may be mucous during the early stage of the disease, but later is frequently purulent.

Anatomic Features.—By means of a cystoscopic examination congestion, redness, edema, and at times minute hemorrhages and ulcers of the vesical mucosa may be detected antemortem. The point of the mucous surface showing the most redness is in the vicinity of the trigonum. The entire surface of the vesical mucous membrane is covered with a thick, tenacious mucus that is at times purulent in character. Areas over which the epithelium has sloughed from the mucous surface are not usually seen. When the exciting cause is sufficiently virulent, the entire mucous lining of the bladder may be so swollen as to make the distinction of any of its blood-vessels impossible.

Phlegmonous cystitis is a condition in which small, punched-out ulcers and necrotic areas are scattered over the vesical mucous surface. Post-mortem findings are confirmatory of those obtained by the aid of the cystoscope.

Exciting and Predisposing Factors.—1. Among the exciting factors should be mentioned disturbance of the vesical circulatory system, which results most often from exposure to cold and wet, and here cystitis is possibly preceded by an inactivity of the skin.

2. Retention of urine may be the only apparent cause for the production of an acute attack of cystitis, and tumors pressing on the bladder and new-growths of the bladder-wall are potent exciting factors. Obstruction to the flow of urine from whatever cause is capable of exciting cystitis. Failure of the bladder completely to empty itself during urination,—a condition commonly seen in paralysis of the bladder or of the lower extremities,—as well as cystocele and enlarged prostate, are among the causes. Exfoliating cystitis, in which there is an extensive destruction of the lining epithelium of the vesical mucosa, may follow overdistention of the bladder, but, as a rule, there has also been exposure to cold and wet.

3. A high degree of acidity of the urine and a urine too rich in solids, particularly in uric acid and in calcium oxalate, is capable of exciting cystitis.

4. Bacterial infection of the bladder by unclean instruments that have been introduced into this organ, and bacteria that have gained access to the bladder from other sources,—the circulation,—are responsible for what is known as septic cystitis.

5. Cystitis sometimes results from the direct extension of gonorrhea from the urethra.

6. Again, septic cystitis may develop during the course of scarlet fever, diphtheria, acute articular rheumatism, and tuberculosis, and it is probable that all such cases are bacterial in origin.

7. Certain drugs, when introduced into the system in both medicinal and lethal doses, are capable of exciting an acute inflammation of the vesical mucous membrane; among these are copaiba, capsicum, cubebs, turpentine, cantharides, phenol, mercury, and certain of the coal-tar products.

8. Traumatism to the bladder and the pressure induced by labor are frequent causes of cystitis. Impaction of feces and foreign bodies, such as

cystic calculus, may in turn inflict true traumatism on the bladder. Operation upon the bladder is likely to cause local, and at times diffuse, cystitis.

9. Direct extension of inflammation from adjacent structures—vaginitis, urethritis, pyelitis, ureteritis—and malignant disease of any of the pelvic viscera are common causes of acute cystitis. In several of our cases cystitis has followed salpingitis and pelvic abscess.

10. Parasitic cystitis results from infection of the bladder or of its wall with Schistosomum hæmatobium. Rarely the vinegar eel and the oxyuris are found in the bladder, and cause acute cystitis.

Principal Complaint.—Acute cystitis is usually preceded by such prodromal symptoms as malaise, anorexia, muscular soreness, lumbar distress, and constipation. Pain is the chief source of complaint in acute cystitis, and this is first described as a mere, dull ache in the lumbar region; later it becomes severe, and is accompanied by frequent micturition. After the disease has existed for several hours vesical tenesmus becomes the most prominent symptom, and is usually accompanied by more or less rectal tenesmus. The quantity of urine voided at each act is small, and may not exceed a few drops, and it is in this last described condition that vesical and rectal tenesmus is most marked.

Pain.—In severe cases the pain is most marked over the suprapubic region, radiating from this point to the sacral area, perineum, along the upper portion of the thighs, and to the glans penis. It is most severe preceding micturition and for some time after emptying the bladder, but when the bladder is empty, the patient is comparatively free from pain. The pain of acute cystitis is relieved by assuming the recumbent posture, whereas firm suprapubic pressure aggravates it. In cases of abscess of the bladder-wall pain and tenderness are also present, there is an almost constant desire to urinate, and the vesical and rectal tenesmus are most agonizing. In severe acute cystitis accompanied by an exfoliation of the epithelial lining of the bladder the patient rapidly enters into the so-called "typhoid state." The lips are parched and fissured, and may display large blackish areas of sloughing epithelium. The tongue is brown and furrowed and the teeth are covered with sordes. The patient's general condition progresses from bad to worse until decided nervous symptoms are added.

Nervous Symptoms.—In mild cases of cystitis there are restlessness, slight headache, and insomnia; but in the severer types, and particularly in those suffering from malignant cystitis, there are generally intense headache, which may be frontal or occipital, delirium, subsultus tendinum, persistent vomiting, stupor, and coma.

Thermic Features.—An attack of acute cystitis is occasionally preceded by a distinct rigor or chill, but more often the patient experiences a slight chill or a series of chilly sensations. There is usually a moderate elevation in temperature, which ranges between 100° and 102° F., the fever continuing until the acuteness of the vesical inflammation subsides.

In the malignant cases the temperature rises rapidly to from 103° to 105° F., and may remain high for several days, displaying either slight or decided morning remissions, which are controlled by the type of the infection. In pseudomembranous cystitis a continued type of temperature is common, although this is by no means a constant feature.

Physical Signs.—Firm palpation over the suprapubic region elicits deep-seated vesical pain that radiates to the sacral region and to the peri-

neum and is reflected down the anterior surface of the thighs. Pressure over the bladder also produces a desire to urinate, and an intense, cramp-like pain that is reflected to the glans penis.

Laboratory Diagnosis.—The quantity of urine voided during the twenty-four hours is approximately normal. The urine is usually acid in reaction; it is cloudy, and macroscopically numerous small, flake-like particles are seen. Upon standing, such urine deposits a heavy sediment, that is divided into three layers: an inferior layer, which is quite dark, a middle layer, which is lighter, and a superior pale zone. Microscopically, the sediment contains desquamated vesical epithelial cells, shreds of mucus, pus-cells, and, in cases in which there is virulent infection, red blood-cells are also seen.

Chemically, the urine of acute cystitis may contain albumin in small quantities, although this is by no means a constant finding.

Illustrative Case of Acute Cystitis.—Jane A., female, aged seventeen years; weight, 115 pounds.

Family History.—Father and mother living at the age of forty-two and thirty-five years respectively; an older brother and two younger sisters living and in apparent health. No history of tuberculosis, nephritis, diabetes, malignancy, or other inheritable maladies in the family.

Previous History.—The patient had the usual diseases of childhood, and during the past six years has suffered from two attacks of tonsillitis. She had a fracture of the left arm at the age of twelve years. Reached puberty at the age of thirteen, since which date she has not consulted a physician.

Social History.—She is attending school, and does not recall having missed a day during the past three years. The mother states that the child's record as a student is unusually good, that she does not find it difficult to perform the general work assigned her, and that upon returning from school each day she takes an hour or more of outdoor exercise.

Present Illness.—Two days ago she joined a coasting party, and, owing to the low temperature, she was exposed to undue cold; upon her return she discovered that her clothing was wet. The following morning she was awakened by severe distress in the lower portion of the abdomen and in the loins, and when seen by her physician, she complained of frequent urination. The quantity of urine voided at each act was not large, and its discharge was accompanied by a variable degree of tenesmus. There were headache, chilly sensations, anorexia, and general malaise.

At first she complained of mere distress and of a sense of weight in the back and loins, but later this was described as a dull pain. At the end of the first twenty-four hours following the onset, whenever a small quantity of urine accumulated in the bladder, pain became intense and continued until shortly after the bladder was emptied. Headache, which was present at the onset, disappeared during the first twenty-four hours. As the result of treatment pain was relieved, but there was no abatement until such treatment was instituted.

When seen a few hours after the symptoms had developed, the temperature was 101.4° F., and continued an irregular course for three days, when it declined to the normal by lysis.

Physical Examination.—*General.*—The patient was well nourished, the skin and mucous surfaces were of normal color, and she was somewhat restless, both when sitting and when lying in bed.

Local Examination.—*Palpation.*—Firm pressure immediately above the pubes caused a sense of discomfort, but did not excite true pain. The pulse was of good volume, the beats numbering 90 to 100 a minute.

Laboratory Diagnosis.—The quantity of urine voided during the twenty-four hours approximates that of normal. The specific gravity is 1.022, and upon standing, a heavy precipitate collects. Microscopically many amorphous urates are seen. Leukocytes are numerous, a few red blood-cells are present, and great numbers of both small and large epithelial cells are present.

Diagnosis by Induction from Clinical Data.—History of exposure to cold followed by frequent micturition which was accompanied by tenesmus, the fact that pain in the lower abdomen became well marked when the bladder became filled

with urine, and that such pain continued until the urine was emptied from the bladder, were considered in themselves fairly characteristic symptoms of the disease.

Course of the Disease.—The patient was compelled to void urine every half to one and one-half hours, until treatment was instituted, when this annoying symptom subsided. Local applications of heat over the bladder and over the loins also appeared to give relief. By the end of the third day the patient suffered little or no inconvenience, although she was directed to remain in bed until the fifth day, from which time on convalescence was uninterrupted.

Summary of Diagnosis.—Frequent micturition and the voiding of a small quantity of urine at each act, together with the presence of suprapubic pain, which radiates to the back and thighs, and vesical tenesmus, are the cardinal symptoms. Both the macroscopic and the microscopic study are of inestimable service in formulating a diagnosis.

Differential Diagnosis.—Acute cystitis may be confused with the frequent urination that occurs during the early months of **gestation** and with **chronic cystitis.**

Duration and Prognosis.—The duration of acute cystitis varies in accordance with the type of infection of the bladder mucosa and with the form of treatment instituted. In the majority of cases the acute symptoms subside in from twelve to forty-eight hours after treatment is begun. In a few instances, however, the condition progresses rapidly until a false membrane is formed, in which case a fatal issue commonly ensues. Cystitis developing during the course of one of the acute fevers may last for from a few days to two weeks.

CHRONIC CYSTITIS.

Pathologic Definition.—A disease of the urinary bladder, characterized by a chronic inflammation of its lining mucous membrane, a variable degree of hypertrophy of the mucosa and bladder-wall, and the formation of indentations or pockets in the mucous membrane.

Early during the course of chronic cystitis the mucous surface of the bladder becomes swollen and lusterless, but with the progress of the disease the mucosa gradually thickens and becomes pale. In advanced cases the entire bladder-wall is found to be thickened, and there are numerous ridges and deep furrows upon the mucous surface, which form small indentations and sacculations. It is these sacculations that prevent the bladder from completely emptying itself at each act of urination.

The prostate gland is hypertrophied, and the mucous lining in the region of the vesical trigone in such cases displays marked change. Vesical calculus more or less completely encysted in the bladder-wall and new-growths of the bladder are frequently found in chronic cystitis. The adjacent structures of the pelvis are likely to become involved by direct extension of the pathologic process of the bladder; thus a similar inflammation of the ureters and the pelvis of the kidney may follow chronic cystitis.

Predisposing and Exciting Factors.—Acute cystitis may terminate in a chronic form of the disease. Enlarged prostate, atony of the bladder, and urethral stricture are among the commonest causes of chronic cystitis. Vesical calculus, vesical polypi, and infection with animal parasites are frequent causes, and occasionally there is extension of inflammation from the pelvic viscera. Carcinoma and tuberculosis of the urinary bladder always give rise to chronic cystitis. Chronic cystitis frequently develops during the course of locomotor ataxia, monoplegia, hemiplegia, and arthritis

deformans; and, generally speaking, it may be said to complicate practically all diseases of the spinal cord. We have found chronic cystitis quite common among hospital patients suffering from paretic dementia and other mental diseases. In women it frequently follows injury to the urethra and pelvic inflammatory conditions.

Principal Complaint.—Frequent micturition, with vesical and lumbar pain that may radiate to the pubic and perineal regions and thighs, forms the leading symptom of chronic cystitis. When chronic cystitis complicates different forms of insanity and diseases of the spinal cord, there may be an absence of pain. Frequent desire to void urine is less decided in chronic than in acute cystitis, and, in fact, the bladder may hold the normal amount of urine without causing any great discomfort. When chronic cystitis is caused by vesical calculus, there is intermittent pain, which is caused by jarring the body, as in alighting from a carriage or in walking downstairs. Headache, malaise, and anorexia are common.

Thermic Features.—There may be no elevation in temperature during the entire course of chronic cystitis, even if the urine contains a large amount of pus. A feature ever to be borne in mind is that, unless ulceration of the bladder mucosa exists, fever (in chronic cystitis) is suggestive of inflammatory or suppurative conditions elsewhere.

Physical Signs.—Deep palpation over the suprapubic and perineal regions may elicit tenderness, although it seldom causes pain.

Laboratory Diagnosis.—The quantity of urine voided during the twenty-four hours is practically normal. The urine may be either acid or alkaline in reaction, and upon standing precipitates a heavy white or grayish-white sediment, which, when agitated, floats up through the supernatant liquid, giving it the so-called "ropy" appearance. Upon attempting to lift this sediment it will be found to be highly tenacious.

Microscopically, the urinary sediment contains many pus-cells, epithelial cells, and shreds of mucus. Red blood-cells are found when there is vesical calculus or ulceration of the bladder mucosa. Crystals of calcium oxalate, ammoniomagnesium phosphate, and ammonium urate are frequently found, and in two instances we have seen crystals of cystin in the urine of chronic cystitis.

Clinical Course.—The course of the disease varies in direct accordance with the etiologic factors concerned. In cases in which there is atony of the bladder or paralysis the course is protracted, but when cystitis is dependent upon an irritating urine, vesical calculus, or enlarged prostate, the disease is amenable to treatment.

NEUROSES OF THE BLADDER.

Pathologic Definition.—A condition characterized anatomically by mild congestion of the vesical mucous membrane immediately surrounding the orifice of the urethra, and clinically by frequent urination, which is increased by nervous excitement.

Anatomic Features.—Owing to the fact that these cases seldom, if ever, terminate fatally, its pathology remains obscure. Cystoscopic examination reveals the presence of a moderate congestion of the vesical mucous membrane, particularly in the region of the trigone, and, on the other hand, it has been asserted, upon good authority, that in certain cases no change in the mucous membrane is apparent, although there is a marked hypersensitiveness of the mucous membrane immediately surrounding the urethra.

This congestion of the vesical mucosa may extend for some distance from the opening of the urethra and ureters.

Predisposing and Exciting Factors.—The majority of sufferers are of the neurotic type; consequently this condition occurs, as a rule, in hypochondriasis, hysteria, and melancholia. Severe grief, financial difficulties, menstrual irregularities, dysmenorrhea, ovaritis, endometritis, and masturbation are often precursors of vesical irritability. Vesical neurosis may be but one manifestation of a general trend of symptoms known to follow such gastro-intestinal derangements as dyspepia and hyperchlorhydria. Among other predisposing factors should be mentioned improper hygienic surroundings, malnutrition, and weakness.

Lithemia no doubt contributes liberally toward the development of vesical neurosis, and in such instances the neurosis may result from local irritation caused by a highly acid urine. Neurosis of the bladder not infrequently accompanies disease of adjacent structures, as, *e. g.*, the ureter, urethra, rectum, and uterus, in which case it should be termed sympathetic neurosis. It may accompany cephalalgia and facial and lumbar neuralgia. Attacks of vesical neurosis may also occur with pronounced periodicity, and are often seen to follow the acute infectious fevers.

Principal Complaint.—Irritability of the bladder is manifested by the following symptoms: frequent micturition, together with vesical and rectal tenesmus. Emptying of the bladder does not always relieve the pain, and occasionally it may be equally marked or more pronounced after urination than it was before the beginning of the act. When there is spasm of the muscle of the bladder, there is likely to be extreme pain for some seconds, and at times for several minutes, after the emptying of the bladder. In a few of our cases the pain was sufficiently severe to necessitate the use of morphin. In the severer types of vesical neurosis the urine may be voided spasmodically, or there may be vesical spasm at some time during the act of urination

Laboratory Diagnosis.—The quantity of urine voided during the twenty-four hours approximates the normal, except in hysteric individuals, in whom periodic attacks of polyuria occur.

Summary of Diagnosis.—Frequent painful micturition developing in persons of neurotic temperament, the urine showing none of the urinary findings of cystitis, is highly suggestive of vesical neurosis.

TABLE SHOWING THE DISTINCTIVE DIFFERENCES BETWEEN NEUROSIS OF THE BLADDER, PREGNANCY, CYSTITIS, AND IRRITABILITY FOLLOWING DISEASE OR TRAUMA OF THE URETHRA.

Neurosis of the Bladder.	Cystitis.	Neurosis of Pregnancy.	Irritability Following Disease or Trauma of the Urinary Tract.
1. History of previous attacks accompanying a general breaking down of the nervous system. No history of organic disease of the bladder or of the urethra.	1. There may or may not be a history of previous attacks, but it is not associated with general nervous debility.	1. History of similar complaint during the early months of gestation only.	1. History of gonorrhea, urethral stricture, or surgical operation upon the urethra or bladder.

Neurosis of the Bladder.—(*Continued.*)	Cystitis.—(*Continued.*)	Neurosis of Pregnancy.—(*Continued.*)	Irritability Following Disease or Trauma of the Urinary Tract.—(*Continued.*)
2. The patient is of a neurotic temperament.	2. May develop in any individual, irrespective of temperament. Frequently follows exposure to cold and wet, catheterization or washing of the bladder.	2. Usually develops about one month after conception, and continues until the third month of pregnancy, when it is lessened in severity or disappears to reappear after the eighth month of gestation.	2. Urethral stricture of long standing and enlargement of the prostate.
3. Pain severe, and may be worse after micturition.	3. Pain marked before micturition, and continues during the act, but is relieved after the bladder is emptied.	3. Pain is uncommon, but a decided desire to empty the bladder is present.	3. There is a more or less constant desire to void urine, and pain may be slight, absent, or at times severe.
4. The constitutional symptoms are those of nervous depression.	4. Symptoms vary with the severity of the type of infection.	4. Symptoms absent.	4. There is seldom evidence of constitutional symptoms unless the condition has existed over a long period.
5. The duration is from weeks to months, and is controlled by the general nervous state of the patient.	5. Of short duration, except in chronic cases, in which it may last for months or even years.	5. Present during first to third and the latter month of gestation.	5. Usually of long duration.
6. The urine is that of hysteria, being at times pale, of low specific gravity, acid in reaction, and excessive in quantity, while at intervals the urine may be concentrated and highly acid.	6. The quantity of urine is approximately normal; microscopically it contains desquamated epithelium, pus, shreds of mucus, albumin, and in severe cases blood.	6. Approximates normal, although some writers claim to have found an increase in the phosphates.	6. Does not reveal anything of diagnostic value.
7. Cystoscopic examination is, as a rule, negative. Rarely there is hypersensitiveness of the mucosa of the trigone and about the vesical orifice of the urethra.	7. Cystoscopic examination reveals a pathologic condition of the vesical mucosa. May detect vesical calculus or tumor.	7. Cystoscopic examination negative.	7. Cystoscopic examination negative.

Neurosis of the Bladder.	Cystitis.	Neurosis of Pregnancy.	Irritability Following Disease or Trauma of the Urinary Tract.
8. The duration is always protracted.	8. In acute cystitis it varies from three to fifteen days, but in the chronic form it may continue for months or even years.	8. Duration short.	8. Usually continues for a long period and until the cause is successfully treated.

Course.—A fatal termination is practically unknown, yet in the majority of instances this malady continues for several months or even for years. Recovery may follow judicious treatment, but there is a great tendency for a repetition of the attacks whenever the general nervous vitality of the patient becomes reduced.

INCONTINENCE OF URINE.

Definition.—Inability to retain the urine owing to hyperesthesia of some portion of the urinary tract or to disease of the spinal cord.

Predisposing and Exciting Factors.—Incontinence of urine is sometimes due to a lesion of the spinal cord that involves the sphincteric center of the bladder; it is also a symptom of such nervous maladies as locomotor ataxia, tumor of the spinal cord, and disease of the central nervous system, a condition commonly referred to as paralytic incontinence. Prostration with general bodily weakness, as seen after the acute infectious diseases, abnormal conditions of the bladder, such as malformation, diminution in size, etc., urethral stricture, and traumatism to either the bladder or the urethra from whatever cause may result in partial or complete paralysis of the sphincter muscles. Overdistention of the bladder with partial paralysis of the muscles is ofttimes responsible for incontinence, and is usually referred to as "*the incontinence of retention.*" Vesical calculi, vesical polypi, anteflexion of the uterus, vesical parasites, and cystitis may each in turn be contributing factors.

Irritability of the compressor muscles of the bladder (*spasmodic incontinence*) is, as a rule, the result of the passage of a highly irritating urine, although Bierhoff believes that the essential or ultimate condition is a hypersensitiveness of the deep urethra. Local irritation, however, may depend, in the male, upon contraction of the urethral meatus, masturbation, an elongated and adherent prepuce, or neglect of the proper toilet of the child. In girls there may be adhesions of the labia minores, with a binding down of the clitoris.

Nocturnal enuresis is a term employed to describe a condition common to neurotic children, and believed to depend upon a neurosis of either the bladder or the urethral mucous membrane. In this condition a pricking sensation is felt in the urethra. At times the affection appears to be psychic, the child giving a history of having had a peculiar dream, or of being frightened during sleep.

Principal Complaint.—**Nocturnal enuresis** as seen in children is a condition in which the child is unable to control the sphincter muscles of the bladder. As a result, during the hours of sleep, usually in the early part of the night, the bladder is partially or completely emptied, the child being generally unconscious of having voided urine until he awakens some hours later.

Paralytic enuresis is characterized by a more or less constant dribbling, which alternates with spurts, of urine, whenever the voluntary or involuntary muscles are brought into action, as in coughing, sneezing, laughing, or during violent exercise. Paralytic incontinence may be the result of a general weakness, and is likewise seen after prostration. We have repeatedly seen this form of incontinence during the later stages of pulmonary tuberculosis, typhoid fever, and other maladies associated with marked prostration.

When incontinence is the result of overdistention of the bladder, pain is unusual. When the incontinence depends upon the presence of vesical calculus, polypus, vesical ulceration, or similar conditions that are accompanied by cystitis, pain is present. Enuresis the result of a hypersensitive condition of the bladder or urethral mucous membrane seldom gives rise to intense pain.

Physical Signs. — **Palpation.** — When incontinence results from lesion of the spinal cord, abnormal sensory changes may take place over the

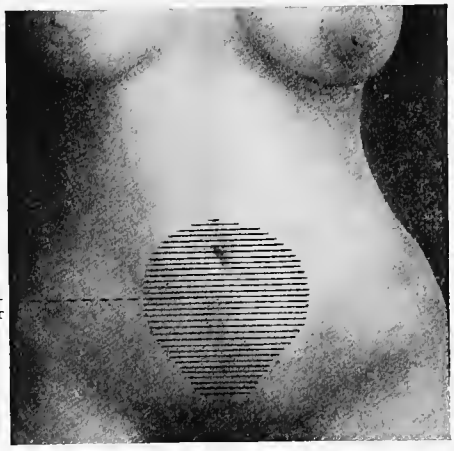


FIG. 274.—AREA OF FLATNESS DUE TO EXTREME DISTENTION OF THE BLADDER BY URINE.

limbs and abdomen; among these are anesthesia, paresthesia, and hyperesthesia. Deep pressure over the bladder discloses the presence of tenderness in certain cases in which vesical calculus, tumors, or cystitis may be present. If the incontinence is dependent upon overdistention of the bladder a tumor-mass may be readily outlined in the region of the bladder (Fig. 274). In females a hypersensitiveness of the clitoris and adhesions of the labia may be present, and the vulva may be swollen and inflamed.

Percussion is of diagnostic value only when the bladder is distended with urine, in which case there is dullness over the lower portion of the abdomen.

Clinical Course. — Enuresis of children disappears when the exciting cause is removed, although in many cases we have known it to continue until the child was ten to twelve years of age. The condition is, as a rule, amenable to treatment, but in the adult, medication has but limited effect. Incontinence depending upon lesion of the cord or upon other grave nervous and mental maladies is seldom curable.

When the enuresis is dependent upon general prostration or upon delirium, it disappears as soon as the general vitality and the mental condition of the patient become normal.

ACUTE INFECTIOUS DISEASES.

FEVER.

Definition.—Fever in its unrestricted sense is a term used to designate a rise in body temperature. Elevation of temperature, however, does not *per se* constitute fever. In our modern conception of fever we recognize an abnormal condition, occupying a certain interval of time characterized by an elevation of body temperature, general malaise, cerebral phenomena, weakness, loss of appetite, thirst, quickened pulse and respiration, slight albuminuria, and derangement of metabolism.

Determination of Body Temperature.—The only accurate method for the determination of body temperature is by the clinical thermometer. The places usually selected for the taking of the temperature are (*a*) mouth, (*b*) axilla or armpit, (*c*) the rectum, and (*d*) the vagina.

The temperature of the mouth is about one degree higher than that in the axilla and lower than of the rectum. The mouth is a convenient and accurate place to take the temperature, the thermometer being placed under the tongue. On account of possibilities of the thermometer being a carrier of disease-germs the axilla has been found a more suitable place.

The temperature as recorded in the axilla is lower than that in the mouth and rectum. All undue moisture should be wiped away before the instrument is placed and care should be taken that it does not project beyond the posterior folds and that it is not caught in a fold of the clothing.

The rectal or vaginal temperature is more nearly that of the body. It is the place of selection in case of children and those who are in a state of delirium or coma and in those cases where the accurate determination of the temperature is of great importance.

Normal temperature is said to be between 98° and 99.5° F. (36.7° and 37.5° C.). It is subject to physiologic variations, among which may be mentioned: (*a*) time of day, (*b*) exercise, and (*c*) age. The temperature rises from seven to eight in the morning and reaches the maximum between seven and eight in the evening. It then begins to gradually fall, and may even be subnormal between 12 P. M. and 4 A. M. Exercise tends to raise the temperature. After violent and prolonged gymnastic exercise it has been known to rise to 104° F. In infants and young children the temperature is somewhat higher and is subject to greater variations. In old age it is usually lower, and may even be subnormal.

Causes of Fever.—The temperature of the body is kept within normal limits by means of the heat regulatory mechanism. It is accomplished by (*a*) the regulation of heat production—thermogenesis; and (*b*) by controlling heat dissipation—thermolysis. Normally, these two functions so counterbalance each other that the temperature under various conditions is kept within tolerably narrow limits. It is evident, therefore, that the causes of fever act through increasing thermogenesis or decreasing thermolysis, or both. It is due to certain substances circulating in the blood,

which may be: (1) toxins, the result of (*a*) infection by microörganisms, or (*b*) intoxication, on account of faulty metabolism or introduced from without; and (2) the infection of the blood by products of putrefaction—sapremia. It is probable that the rise of temperature sometimes attributed to fright, emotion, violent pain, etc., is due to derangement of physiologic processes whereby toxins are produced.

Among the clinical phenomena that may accompany fever the following deserve special mention:

1. **Pyrexia,** or increased temperature.
2. **General malaise,** or subjective symptoms of illness.
3. **Increased pulse-rate.** In many cases of febrile disease the pulse is accelerated, and this acceleration usually corresponds to the intensity of the fever. It is estimated that there is an increase of eight beats to every 1.8° F. increase above the normal. The ratio between the pulse and temperature is of diagnostic and prognostic value. Thus, in tuberculosis with moderate or no fever the pulse-rate is usually increased. On the other hand, in basilar meningitis and yellow fever the pulse is slow, although the temperature may be high. In typhoid fever acceleration of the pulse is moderate in comparison to the temperature; this fact being of aid in the differential diagnosis between it and acute miliary tuberculosis or septicemia, in which conditions the pulse-rate is high. Furthermore, in diphtheria and peritonitis the pulse-rate is greatly increased, although there may be but moderate fever.
4. **Increased respiration.** The respiration is increased in almost all cases of fever. It is due partly to the toxins which produce the fever and partly to the stimulating effect of the heated blood. It is of clinical importance to note that greatly increased respiration in the absence of disease complicating the respiratory apparatus is of very unfavorable prognostic significance.
5. **Nervous symptoms,** such as prostration, headache, pain in the back, convulsions (especially in children), somnolence, stupor, delirium, and coma. The prostration may be pronounced and is often present in the early stages of febrile disease and bears no relationship to the wasting of the tissues which later takes place. If the fever persist, a low asthenic state may supervene. The eyes and expression become dull and heavy, the face congested, the pupils contracted. The sensibilities are blunted and the mental processes become sluggish. Prostration is profound and may be attended with a low, muttering delirium. When venous stasis is present, the pulse becomes small, feeble, and dicrotic, the breathing shorter and labored. This complete or profound adynamia is commonly referred to as the typhoid state. An opposite condition may, however, exist. Instead of prostration and depression great strength with active, violent, or maniacal delirium is exhibited. Intense headache may be present. The pulse is strong; the face flushed and bright red in color, while the eyes are active, bright, and injected. This condition, the direct opposite of the typhoid state, has been called ataxia or the ataxic state.

Headache and pain are of diagnostic value. In tonsillitis and smallpox the pain in the back may be severe. In cerebrospinal meningitis and enteric fever the headache is very marked and is of a protracted, throbbing variety in the latter disease.

The delirium which may be present may be mild and wandering or active and maniacal, and appears to bear no relationship to the intensity of the fever. Thus, in relapsing fever with a temperature of 106° F. the mind remains clear,

while in certain cases of typhoid fever marked delirium may attend a temperature of 103° F.

6. **Derangement of normal secretions** is quite prominent and becomes manifest on the part of the gastro-intestinal tract by a coated tongue, thirst, loss of appetite, impaired digestion, and constipation or diarrhea. The urine is scanty, dark-colored, and of high specific gravity. The skin may be unusually dry, while in some cases there may be sweating. The latter is apt to occur at the time of defervescence, although it may take place throughout the course of the disease or at certain stages. In deep-seated suppuration, diseases of bones, and tuberculosis it is cold and clammy.

7. **Emaciation.** Wasting is a very pronounced symptom even in febrile disease of moderate duration. Changes occur in the solid structures as well as in the blood. The changes which occur in the blood are a decrease in the number of erythrocytes and the progressive loss of the albumins of the plasma.

Classification of Fever.—Fever has been divided according to (1) the degree of pyrexia and (2) type.

According to the height of the temperature fever is spoken of as (*a*) subfebrile (apyrexia); (*b*) moderately febrile; (*c*) highly febrile, and (*d*) intensely febrile, or hyperpyrexia. The following table shows the temperature to which the foregoing terms apply:

Fahrenheit.	*Centigrade.*	
99.5°—100.5°	37.5°—38.1°	sub-febrile.
100.5°—103.1°	38.1°—39.5°	moderately febrile.
103.1°—105.8°	39.5°—41°	highly febrile.
Above 105.8°	Above 41°	hyperpyrexia.

Type of Fever.—Fever is clinically divided according to its range into three distinct types: (*a*) continued, (*b*) remittent, and (*c*) intermittent. Of less importance, the inverse and the atypical type may be mentioned. The recognition of the different types in the various febrile diseases is of importance and forms a positive aid to diagnosis.

Continued fever is that type of fever in which the diurnal range (fluctuation) does not exceed 1.8° F. It is seen in typhus fever, pneumonia, scarlet fever, etc.

The remittent type of fever is characterized by diurnal fluctuations of more than 2° F., but which, however, does not reach the normal.

Intermittent fever is that type of fever in which there are a succession of rises and of remissions, to or below the normal. This type of fever is seen in intermittent malarial fever, relapsing fever, hepatic colic, suppuration, tuberculosis with cavity, etc.

Inverse Type.—In fevers of the continued and in some of the remittent type diurnal oscillations occur which, while corresponding to the normal in time, yet are of greater range. In some cases this condition is reversed, the remission taking place in the evening and the exacerbation in the morning. It is then called the inverse type of fever and is occasionally met with in tuberculosis and more rarely in typhoid fever.

Atypical fever is encountered in some of the febrile diseases. It is of diagnostic importance, its presence militating against the existence of any febrile disease known to have a characteristic type of temperature.

Course of Fever.—Fever has been divided into four stages: (*a*) The stage of prodromes; (*b*) the onset or invasion; (*c*) the fastigium, and (*d*)

the defervescence. In the various febrile diseases the different stages are quite characteristic and of definite duration. Their recognition is of great clinical significance from a diagnostic as well as prognostic standpoint.

The onset or stage of invasion may be very short (a few hours) or it may extend over a period of several days. The temperature accordingly rises either abruptly or gradually until the maximum is reached. When the rise of temperature is abrupt, it is usually accompanied by a chill which frequently corresponds in intensity with the abruptness of the invasion. Its intensity may vary from a subjective sensation of cold with pallor and cyanosis of the lips and fingers to a severe and prolonged chill, with chattering of the teeth, cyanosis, and a pallid and pinched face. The temperature of the surface of the body is cool, although the internal temperature as recorded in the mouth or rectum may be very high. In children convulsions not infrequently take the place of a chill.

A sudden invasion with rapid rise of temperature is seen in erysipelas, lobar pneumonia, influenza, scarlet fever, tonsillitis, middle-ear disease or mastoid inflammation, osteomyelitis, intermittent fever, gastro-intestinal disorders of children, etc.

A gradual invasion is typically seen in typhoid fever and attends quite a number of febrile diseases.

The Fastigium or Acme.—In this stage the temperature has attained its maximum. It may last only for a very short time or it may be prolonged for three or more weeks. During this time diurnal variations usually occur which while corresponding in time to the normal are yet of greater latitude. The temperature of the surface of the body is usually the same as the internal, although it may at times and in certain diseases vary. When the maximum temperature is transient the term acme is usually employed to designate this stage.

The Stage of Defervescence.—In this stage the temperature falls to the normal. This fall may be abrupt—a fall by crisis or critical defervescence—or it may be gradual—lysis. Febrile diseases characterized by a sudden invasion usually terminate by crisis, while those in which the invasion is more gradual terminate by lysis.

When the fever terminates by crisis, the temperature drops to or below the normal in a very short time (twelve to twenty-four hours). It is usually attended by copious diaphoresis—the critical sweat—the passage of large quantities of urine or large, loose stools. The pulse and respiration also improve with the fall in temperature. A knowledge of the condition of the pulse and respiration at this time is of great clinical significance, inasmuch as a rapid fall in temperature without improvement in the pulse and respiration may indicate collapse or impending death. In typhoid fever it indicates perforation if followed by a rise of the temperature, abdominal pains, or bloody stools.

Erysipelas, measles, lobar pneumonia, relapsing fever, and typhus fever usually end by crisis.

Typhoid fever, acute rheumatism, bronchopneumonia, and pleurisy usually terminate by lysis.

ACUTE TONSILLITIS.

Pathologic Definition.—An acute, self-limited, inflammatory process, involving first the mucous membrane and the tonsillar crypts, then the tonsillar parenchyma, and terminating in resolution, suppuration, or chronic tonsillitis.

Clinical Types.—(*a*) Acute catarrhal tonsillitis; (*b*) acute parenchymatous tonsillitis; (*c*) phlegmonous tonsillitis; (*d*) peritonsillar abscess or quinsy; (*e*) necrotic tonsillitis.

Predisposing Factors.—**Age** is the chief predisposing factor, the disease being common during childhood and early adolescence, but rare during infancy. In children with a rheumatic inheritance there is often a special susceptibility to the development of tonsillitis. Rheumatism figures prominently as an etiologic factor in tonsillitis in persons between the ages of fifteen and thirty years.

Season also has its influence, the greatest number of cases developing during the spring months. Exposure to cold and wet and to extreme climatic changes is followed by an increase in the number of cases of acute tonsillitis.

Sex.—The disease is far more common in boys and young men than in females. One attack predisposes to others, which are likely to develop once or twice annually. Those suffering from a permanent enlargement of the tonsils are exceedingly prone to attacks of acute tonsillitis. Acute infectious diseases of the throat also favor the development of tonsillitis.

Exciting Causes.—Inflammation of the tonsils and of the peritonsillar tissues may be caused by a number of microörganisms: staphylococcus aureus, streptococcus pyogenes, bacillus pyocyaneus, sarcinæ, and others. These organisms may be cultivated from the exudate covering the mucous membrane and from the pus contained in superficial or deep collections.

Principal Complaint.—The *general symptoms* are often severe and appear abruptly, as a rule, preceding the local symptoms. The disease is sometimes ushered in with a distinct chill or a series of chills. In children there may be vomiting and, rarely, diarrhea. Marked soreness and intense pain in the back, loins, and muscles of the extremities, together with severe headache, are among the initial symptoms of acute tonsillitis.

Within a few hours after the chill there are certain definite *local symptoms*, the first being soreness and lancinating pains upon swallowing, and stabbing pains in the ear. There may be difficulty in expelling the excess of saliva from the mouth, and even movements of the head or of the jaw excite intense pain.

Profuse acid sweats are a great annoyance, and are most likely to occur during sleep. The following train of symptoms, known to practically every disease that is ushered in with a chill, are likewise present in tonsillitis: (1) Fever; (2) sweat; (3) increased respiration; (4) increased frequency of the pulse; (5) constipation; (6) a diminution in the quantity of urine excreted, the fluid being of high specific gravity, high in color, and rich in solids.

Thermic Features.—Immediately following the chill the temperature reaches 102° to 105° F., and remains high for from one to several days. The height of the fever is influenced materially by treatment and by the type of tonsillitis present.

Special Varieties.—(*a*) **Acute catarrhal tonsillitis** is a part of a general catarrh of the mucous membrane of the throat. The local manifestations are soreness and difficulty in swallowing, with pains radiating to the angles of the jaw or to the ear. Swelling of the tonsils and of contiguous tissues gives a peculiar nasal tone to the voice. In some of these cases there is an excess of saliva, whereas in others the mouth is comparatively dry, pain being more pronounced when the secretion is diminished.

The *exudate* that collects in the throat is thick and highly tenacious, and may excite paroxysmal coughing, which is followed by the expectoration of a

large quantity of this mucopurulent material. Occasionally *stomatitis* is seen to accompany catarrhal tonsillitis, and in such cases salivation is the rule.

Physical Signs.—Inspection.—The mucous membrane of the tonsils, of the pillars of the fauces and pharynx is intensely congested. In the early stages it presents a glazed appearance, but later it becomes covered with a mucopurulent exudate which is easily detached and leaves a dry, reddened surface.

Palpation.—The tenderness is localized to the surface of the tonsils, yet it is not infrequent to find extreme tenderness in the region of the pillars and the uvula. On making external palpation at the angle of the jaw, a variable degree of tenderness is elicited.

Clinical Course and Duration.—The general systemic evidence of disease is not well marked in acute catarrhal tonsillitis, and the duration of the disease is from two to four days, when recovery follows.

Among the *complications* of tonsillitis should be mentioned otitis media, pharyngitis, and peritonsillar abscess.

(*b*) **Follicular Tonsillitis.**—This is a severe type of tonsillitis, common in children and young adults, involving the mucous lining of the tonsillar crypts and the mucosa covering the tonsils. The local symptoms of this type of the disease are practically the same as those described under Acute Catarrhal Tonsillitis, except that they are more severe.

Physical Signs.—Inspection.—The surface of the tonsils is covered with small, slightly elevated, yellowish areas, over which creamy white exudate is spread. The number of these areas seen upon one tonsil may vary from three to fifty. Pressure upon the tonsil may force small, yellowish-white, plug-like masses out of the crypts.

The *constitutional symptoms* of follicular tonsillitis are marked, the chill, headache, muscle pains, and fever being far more severe than in catarrhal tonsillitis.

Follicular tonsillitis may develop during the course of endocarditis, pleurisy, and other febrile conditions. In certain cases the material that collects in the crypts of the tonsil remains there for an indefinite period and becomes calcareous in structure—the so-called "chalk-plugs" of chronic tonsillitis. (See differential table below.)

TABLE SHOWING THE DIFFERENTIAL DIAGNOSIS BETWEEN DIPHTHERIA AND FOLLICULAR TONSILLITIS.—(*Modified from Anders.*)

Diphtheria.	Follicular Tonsillitis.
1. History of an epidemic or of exposure.	1. History of previous attacks.
2. Symptoms develop somewhat slowly, except in malignant cases. There may be headache, chilliness and occasionally a distinct chill.	2. Symptoms develop abruptly, with a chill and profuse acid sweats.
3. A tough, ashy-gray, continuous, and uniform pseudomembranous deposit in the tonsils.	3. A soft, pultaceous, yellowish-white deposit occurs in spots or patches situated over the follicles, with intervening areas of redness.
4. The exudate is very adherent, and can be torn off only in strips, leaving a bleeding surface.	4. The exudate is easily removed, leaving a smooth but reddened surface.
5. The pillars of the fauces and uvula are involved, and the membrane may extend to the nasal mucosa.	5. The deposit is limited to the tonsils.
6. Removal of the membrane is followed by reformation within twenty-four hours.	6. If the creamy deposits unite to form a continuous layer, removal is not immediately followed by reformation.

Diphtheria.	Follicular Tonsillitis.
7. Bacillus diphtheriæ is present in all cases. Other bacteria (cocci and bacilli) may also occur.	7. Cultures from the tonsillar mucous membrane fail to show bacillus diphtheriæ.
8. Temperature rises slowly to from 99½° to 102° or 103° F., but the fever continues longer than in tonsillitis.	8. Following the chill the temperature rises to from 102° to 104° or 105° F., and remains high for one or two days, when a rapid decline is witnessed.
9. In the majority of cases albuminuria develops by the end of the first week, and may be present throughout the disease.	9. Not present in uncomplicated cases.
10. There is usually enlargement and hardening of the cervical lymph-nodes.	10. Glandular enlargement rare. Tonsillar abscess an occasional complication.

(*c*) **Acute Parenchymatous Tonsillitis.**—This type of tonsillitis differs from those forms previously described in that it occurs more commonly in adults than in children. The symptoms are severe, and the substance of the tonsil, in addition to being decidedly congested, shows a special tendency to go on to suppuration.

Principal Complaint.—The general complaint does not differ markedly from that described under Acute Follicular Tonsillitis until the stage of suppuration is reached, when the pain is excruciating and radiates to the ears. The temperature rises to 104° to 105° F., and the pulse reaches 120 a minute, and is full and bounding. Delirium is uncommon.

The secretion of the throat and mouth is scanty at first, but later a viscid mucus is produced, but salivation is seldom, if ever, present. With the accumulation of pus in the tonsils dyspnea develops, which is caused by edema of the cellular structures of the throat.

Physical Signs.—Inspection.—The patient opens his mouth only with great difficulty, and thus a thorough inspection of the throat is not possible. The tonsils are markedly enlarged, and may meet in the median line; in cases in which only one tonsil is involved, this may occupy nearly the entire throat. The voice is husky, and articulation is indistinct. The entire mucous membrane of the affected side, and often of both sides, is intensely congested, as are also the faucial arches and the soft palate. The uvula will be pushed to one or the other side of the throat, or be pushed forward as the result of the tonsils occupying nearly the entire cavity of the throat.

Palpation.—The tonsils and edematous tissues of the throat and palate are hard at first, but later, with the accumulation of pus, they become soft and spongy.

By palpation of the tonsils it is often possible to locate the pus, and it not infrequently happens that the abscess ruptures and discharges its contents into the mouth. Glandular enlargement is present at the angle of the jaw.

Laboratory Diagnosis.—Pus from an abscess of the tonsil is likely to contain streptococci, the Staphylococcus pyogenes aureus, and the Staphylococcus pyogenes albus.

The *urine* is highly colored, of high specific gravity, and displays a red froth when agitated, and a heavy sediment upon standing. The urinary solids are decidedly increased, whereas the quantity of urine voided during the twenty-four hours is diminished; consequently the total amount of solids excreted during the day is approximately normal. Albuminuria and glycosuria are unknown in uncomplicated cases of tonsillitis. The diazo-reaction is commonly present.

Clinical Course.—After the pus has been released, either by incising the tonsil or by spontaneous rupture, the vast majority of cases terminate favorably. Irrespective of surgical interference, the average duration of tonsillar abscess is from eight to twelve days.

(*d*) **Peritonsillar Abscess.**—This is a condition that develops as a complication in acute parenchymatous tonsillitis, and is due to the pus invading the surrounding cellular tissues, and forcing its way between the tonsil and the pterygoid muscles. In peritonsillar abscess the pus may burrow its way as low as the clavicle, or may infiltrate the contiguous structures of the pharynx, mouth, and larynx.

(*e*) **Phlegmonous tonsillitis** occurs much less frequently in children than in adults. It is distinguished from the previously described types of tonsillitis by the fact that it is seldom, if ever, bilateral. Sore throat, stiffness of the muscles of the neck, pain, and salivation are more pronounced than in acute parenchymatous tonsillitis.

Clinical Course.—Phlegmonous tonsillitis terminates in recovery within a period of three or four days after the pus has been released. A few cases are on record in which the pus from peritonsillar abscess has penetrated the veins and arteries of the neck.

(*f*) **Necrotic Tonsillitis.**—An acute localized infection of the tonsils, characterized by the formation of a false membrane, which is followed by extensive ulceration of the tonsillar mucous membrane. Generally speaking, necrotic tonsillitis is regarded as being due to infection with the Klebs-Löffler bacillus, but we have made cultural studies of a number of cases in which there has been extensive sloughing of the tonsil, and in which only the bacteria common to the mouth and throat were recovered.

Principal Complaint.—According to the symptomatology of necrotic tonsillitis, there appear to be at least two types of this affection; one in which the necrosis may not extend deeper than the mucous membrane, although the accompanying constitutional symptoms are comparatively severe, resembling those of acute follicular tonsillitis.

The second type of necrotic tonsillitis is seen in young adults, and is not accompanied by any marked constitutional disturbances. There is but slight soreness of the throat, and practically no pain is felt upon opening the mouth or upon moving the head. The temperature ranges between 99° and 99.6° F. The pulse is but slightly, if at all, accelerated, and the appetite is undisturbed.

Physical Signs.—Inspection.—This last described variety of necrotic tonsillitis is unilateral. The initial lesion is oftenest seen on the anterior surface of the tonsil, and in appearance closely resembles that seen in diphtheria. Within from twenty-four to seventy-two hours extensive ulceration and necrosis of both the mucous membrane and the deeper tissues of the tonsil may take place. In ordinary cases it is possible to pass a probe for a distance of from $\frac{1}{8}$ to $\frac{1}{2}$ inch into the substance of the gland. These areas of excavation are bounded by ragged margins that are covered with a thick, yellowish, tenacious mucus. The tonsil is not painful to the touch, nor is there marked edema of the surrounding tissues. Diphtheria bacilli are absent.

Clinical Course and Duration.—Recovery usually takes place in from two to three weeks. After the ulcer has healed, the affected tonsil is much smaller than its fellow, and there is often a decided depression at the original site of the necrotic process.

CHRONIC TONSILLITIS.

Pathologic Definition.—General enlargement of the tonsils following repeated attacks of acute tonsillitis. It is unusual for enlargement of the tonsils to cause any decided inconvenience to the patient unless adenoid vegetations in the pharynx are associated with it. Enlargement of the tonsils may cause obstruction of the Eustachian tube and consequent impairment of hearing, and it is also responsible for heavy breathing and snoring during sleep.

Fetid breath may be an annoying symptom of chronic enlargement of the tonsils, and the patient may be able, by pressing the tonsils, to cause the escape of peculiar yellowish plugs from the tonsillar crypts. Permanent enlargement of the tonsils is a condition for the specialist.

TUBERCULOUS ENTERITIS (TUBERCULOSIS OF THE INTESTINE; TUBERCULOUS DYSENTERY; CONSUMPTION OF THE BOWEL).

Pathologic Definition.—Either a primary or a secondary infection of the mucosa, and of the deeper coats of the bowel, by tubercle bacilli, with the formation of ulcers that are arranged transversely to the long axis of the colon, and characterized clinically by frequent watery stools, tubercle bacilli in the feces, progressive emaciation, profound weakness, and the blood changes and other symptoms of secondary anemia.

General Remarks and Varieties.—The lesions present in the colon may be—(*a*) primary, but, as a rule, they are (*b*) secondary to tuberculosis of the lung, throat, or other structures.

Primary tuberculosis of the intestine is oftenest seen before the tenth year, a fact that is explained in part by the large amount of milk that is ingested by children. It is difficult to separate tuberculosis of the intestine from tuberculosis of the peritoneum, since the two conditions often develop conjointly. While the patient complains of the general symptoms produced by the tuberculous enteritis, he is likely to suffer from local or general tuberculosis of the peritoneum. In many autopsies performed on the bodies of children dead of tuberculosis of the intestine it has been difficult to ascertain whether the tuberculous infection originated in the bowel or in the peritoneum; and, indeed, in many instances the pathologic changes suggested that the two conditions developed hand in hand. In certain cases the infection was probably first manifested in the retroperitoneal lymph-nodes.

"The *secondary variety* occurs in more than one-half of the cases of pulmonary tuberculosis" (Anders). Any portion of the colon may be involved, yet the lower one-third of the ileum serves as the common site for the initial intestinal lesions. The clinical picture is but slightly different, whether tuberculosis of the intestine be primary or secondary.

Symptomatology.—In children, the mother usually states that the child has suffered from a somewhat chronic intestinal catarrh for weeks, or possibly for months. Moderate colicky pains and diarrhea are present and the dejecta may be blood-stained. The child has not gained in weight since the intestinal symptoms developed, and, indeed, in most instances there has been a gradual loss in flesh throughout the course of the illness.

Tuberculosis of the intestine in the adult gives rise to symptoms quite similar to those described for children. In adults, as previously stated, it is extremely common to have the symptoms of gastro-intestinal catarrh, with probably two, three, or even six copious, watery stools daily. Consti-

pation may persist for one or more weeks, during which time the patient complains of intestinal discomfort.

Thermic Features.—In primary tuberculosis of the mucous coat of the intestine there may be slight fever, or the temperature may be subnormal during the morning hours. When the deeper coats of the intestine become involved, the temperature will range between 99° and 101° F. When tuberculosis of the intestine is secondary to tuberculosis of the lungs with cavity formation, the temperature will be found at or near the normal during the morning hours, with a rise to between 102° and 104° F. during the afternoon and evening hours. The hectic temperature is probably dependent, for the most part, upon the pulmonary condition, and not upon the intestinal lesion.

Physical Signs.—Inspection.—There is pallor of the skin and mucous membranes, and the tongue is, as a rule, heavily furred, although where there is a great amount of intestinal irritation, it may be red and glazed. The respirations become hurried, and cyanosis and edema of the ankles are present late during the disease. Early during the course of intestinal tuberculosis the abdomen is scaphoid in shape, and later, as the result of associated peritonitis and tympany, it becomes distended. Should the peritoneum become involved, ascites develops. (See Ascites, p. 567.)

Palpation gives negative results until the disease is well advanced, when there is some tenderness over the course of the colon, and particularly in the region of the appendix. As the disease advances tenderness becomes more marked, and finally the features characteristic of general peritonitis are present. In some cases a tumor-like ridge lying transversely just above the level of the umbilicus may be felt.

Percussion.—After the disease is well advanced an increased tympanitic note over the entire abdomen is obtained.

Laboratory Diagnosis.—When there are ulcers in the mucous surface of the colon, the feces contains tubercle bacilli, shreds of necrotic tissue, pus, and blood. Large numbers of intestinal epithelial cells are also present, which give the liquid stool a granular appearance.

The *hematologic changes* present are those of secondary anemia. In probably 50 per cent. of all cases of tuberculous enteritis the sputum will be seen to contain tubercle bacilli, and in several cases we have found tubercle bacilli in the urine of persons suffering from this affection. In nine cases bacteriologic study of the venous blood gave negative results.

Illustrative Case.—J. P., male, aged nine months; fed upon modified cow's milk. He was apparently in perfect health until two months ago, when the mother noticed that the child did not gain in flesh, and that he refused to take his usual amount of nourishment at each feeding. Since his illness he has lost four and one-half pounds in weight, and is now very fretful; he frequently awakens and cries during the night, and has paroxysms of crying during the day. Together with the loss of weight pallor and wrinkling of the skin have developed. There are from six to ten copious, watery bowel movements daily. The features are pinched, the expression old, and the child cries upon the slightest movement. The tongue is covered with a white fur. The abdomen is tender and somewhat tympanitic.

The *temperature* ranges between 99° and 102° F., the higher temperature being seen during the afternoon and evening hours. A *microscopic examination* of the feces revealed the presence of tubercle bacilli, streptococci, and other bacteria.

Summary of Diagnosis.—The existence of tuberculous enteritis is to be suspected whenever a history of progressive loss in weight, pallor, and weakness, together with diarrhea, is given. The detection of tubercle bacilli in the feces confirms the diagnosis in all cases, and serves to distinguish tuberculosis of the intestine from other forms of *intestinal catarrh.*

Clinical Course.—The prognosis is unfavorable, the majority of cases terminating in death in from a few weeks to six or more months. We have found tubercle bacilli present in the feces of adults over a period of one year, but it is seldom that the disease is so prolonged in children. In one child, a patient seen in the Philadelphia Hospital, tubercle bacilli were present in the feces during a period of seventeen weeks.

PHLEGMONOUS ENTERITIS.

Pathologic Definition.—A local or diffuse purulent inflammation of the submucous coat of the large intestine.

Predisposing Factors.—Septicemia, pyemia, and abscess of any portion of the body frequently antedate phlegmonous enteritis.

Duration.—The majority of cases terminate fatally in from twenty-four to seventy-two hours.

CATARRHAL ENTERITIS (SUMMER DIARRHEA; ACUTE GASTRO-INTESTINAL CATARRH; DIARRHEA OF CHILDREN; CHOLERA INFANTUM; MYCOTIC DIARRHEA).

General Remarks.—Summer diarrhea is a term applied to a series of gastro-intestinal conditions that are most likely to develop during hot weather. Diarrhea frequently occurs in epidemics, although endemic diarrhea is seen in both tropical and temperate climates. The malady is announced by a sudden onset, with pronounced irritation of the stomach and bowel. High fever, extreme prostration, and nervous symptoms soon follow.

Varieties.—(1) **Diarrhea Resulting from Dietetic Errors.**—This includes those cases caused by the ingestion of improper or indigestible food, which virtually acts as a foreign body. The irritation caused by such foods may, in the milder cases, produce only increased secretion and peristalsis, but in the severer forms inflammation of the gastro-intestinal mucosa may ensue. The stools are first seen to contain the somewhat hardened contents of the intestine; later they are semiliquid, and if the irritation is great, the stools become watery.

A *bacteriologic study* of the feces shows only those bacteria that are present during health.

(2) **Eliminative Diarrhea.**—The cases considered under this head are all those in which, clinically, the diarrhea appears to be nature's means of expelling from the body certain toxic substances that are known to circulate in the blood. An example of this type is the diarrhea that develops late during the course of renal disease—the so-called "uremic diarrhea." It is also probable that diarrhea developing during the course of certain other acute or chronic maladies may in reality belong to this subclass.

Eliminative or "toxic diarrhea" is to be carefully distinguished from other types of the affection, since to arrest such a diarrhea would be disastrous.

(3) **Acute Intestinal Indigestion.**—The ingestion of food that is not readily digested, and consequently remains unaltered in the intestines, is the exciting cause of this type of diarrhea. The undigested food causes a mechanic irritation, but it is also likely to undergo putrefactive changes in both the stomach and the intestine. Putrefaction may result in the generation of certain toxic substances (gases, ptomains) which, in turn, irritate the gastro-intestinal mucous membrane and aid in exciting diarrhea.

The symptoms (*e. g.*, vomiting, diarrhea, convulsions, and pain) may vary greatly with the degree of gastro-intestinal irritation present. This type

of the disease is usually severe in those under two years of age, and proportionately lessens in virulence up to the tenth year, after which the condition is uncommon and seldom dangerous to life.

If intestinal irritation continues for more than a few hours, acute inflammation of the mucous coat results. The submucous coat becomes involved later. The severity of both the local and the constitutional symptoms serves as an index to the degree of intestinal inflammation present.

A *bacteriologic study* of the feces may reveal the presence of pathogenic organisms (streptococci, staphylococci, and bacilli) in severe cases. Blood may be found either by the microscope or by chemic methods.

(4) **Nervous Diarrhea.**—Both mild and prolonged nervous impressions are capable of producing diarrhea in neurasthenic individuals. Among such conditions should be mentioned atmospheric changes, mental excitement, public speaking, rapid chilling of the surface of the body, profound exhaustion, shock from fright, and dentition. Again, the introduction of either liquid or solid substances into the stomach may be followed almost immediately by a bowel movement. *Increased peristalsis and malnutrition* are cardinal symptoms in this type of diarrhea.

A *bacteriologic study* of the feces is negative as to pathogenic organisms. The feces contain particles of food that have passed through the alimentary canal without being digested.

(5) **Diarrhea Due to Drugs.**—A protracted diarrhea may result from the use of cathartics, even though they be administered in moderate doses. The prolonged use of arsenic is, as a rule, followed by diarrhea, and, indeed, this type of the condition is closely allied to that subclass previously described as "eliminative diarrhea."

Predisposing and Exciting Factors.—(1) **Bacteriology.**—In the mild forms of diarrhea (simple diarrhea) only those bacteria present during health are to be found in the dejecta. When diarrhea continues for several days, other microörganisms appear in the feces, *e. g.*, bacilli and cocci. The bacillus of dysentery and streptococci are not infrequently present when diarrhea has lasted for one week. The colon bacillus, while normally present in the feces, may at times assume a pathologic rôle in this condition.

(2) **Age.**—Children display an unusual susceptibility to diarrhea, and this susceptibility is amply borne out by the statistics of Crandall, whose analysis of 3000 cases gave the following: Under six months, 14 per cent.; between six and twelve months, 29 per cent.; between twelve and eighteen months, 24 per cent.; only 16 per cent. developed after the children were two years of age. The disease is decidedly less common after the tenth year of life, and becomes less and less frequent until the debility of old age asserts itself.

(3) **Season.**—In a temperate climate the majority of cases of diarrhea is seen during the months of June, July, August, and September, July furnishing the greatest number of cases. Hot weather, together with the unhygienic conditions that are likely to prevail during the summer, predisposes to all varieties of diarrhea. Owing to the intense heat large quantities of water are taken, milk is likely to contain a great many bacteria in a cubic centimeter, or become decomposed before use, and these factors, together with the ingestion of unripe fruit, play an important rôle in the production of summer diarrhea.

(4) **Environment.**—Individual surroundings and circumstances hold prominent places as etiologic factors in diarrhea, as is shown by the fact that this condition is far more common in cities than in rural districts. Poverty

predisposes materially to diarrhea, but the children of well-to-do parents do not escape.

(5) **Uncleanliness** is considered by many as contributing toward diarrhea. Statistics show that artificially fed children are far more likely to develop diarrhea than are those fed on breast-milk; therefore, the food with which the child is nourished figures prominently as a causative factor. The contamination of food-supplies by flies and street-dust is an important etiologic factor.

(6) **Dentition.**—It is often difficult to show the exact relation between dentition and diarrhea; the fact remains that the two conditions appear simultaneously. The general belief that diarrhea is dependent upon dentition when it develops during the summer is an erroneous one in many instances.

(7) **Malnutrition.**—Any condition or conditions that tend to lower the general nutrition and vitality of the patient increase the tendency to develop diarrheal disease. Even when such children are placed amid the most hygienic conditions possible, the mortality rate is extremely high.

Symptomatology.—There is always an increase in the number of stools during the twenty-four hours. The *temperature* may be normal, or there may be a slight febrile reaction. The child is somewhat restless, particularly at night. The previously mentioned symptoms continue for two, three, or more days, during which time there is a progressive increase in the number of stools. In some cases the onset is insidious, with anorexia, nausea, vomiting, and acute intestinal pain; in others it is sudden, with fever that rises to 104° or 105° F. In those cases with acute onset convulsions occasionally occur, the abdomen is tender, and the child rests upon its back, with the thighs flexed upon the abdomen.

Laboratory Diagnosis.—The stools contain undigested food, and in children fed upon milk they display large "curd-like" masses. By the end of the second or third day the stools emit a decidedly offensive odor. If symptoms develop acutely, the stool is streaked with a yellow or greenish-yellow substance; in other cases the naked-eye appearance of the stool is identical with that described under the milder type (p. 548) of diarrhea.

Microscopically a great many classes of bacteria are found, but practically all are common to the intestinal tract. After acute congestion of the intestinal mucous membrane has developed, pathogenic bacteria (streptococci, staphylococci, bacilli) appear in the feces.

Clinical Course and Duration.—Mild uncomplicated cases go on to recovery in from seven to fourteen days, and the duration of such an attack is materially shortened by the application of judicious treatment. Relapses are common, and it is after one or more relapses that the child is likely to develop true enterocolitis. When acute indigestion with diarrhea is accompanied by extreme prostration or pronounced nervous symptoms, a fatal issue may follow.

DYSENTERY.

Pathologic Definition.—A condition in which the mucous membrane of the large intestine presents a general redness, with swelling and the exudation of blood-stained mucus, and there may be many ulcers of varying size and depth. The disease may be due to a specific bacterium (bacillus dysenteriæ), when it is called bacillary dysentery; or to a protozoan parasite (entamœba histolytica), when it is called amebic dysentery or intestinal amebiasis.

General Remarks.—Generally speaking, acute dysentery should be regarded as an epidemic disease, although it occurs endemically in tropical and subtropical climates.

Varieties.—Clinically, acute dysentery is classified as—acute bacillary dysentery, acute catarrhal dysentery, and acute sporadic dysentery.

Exciting and Predisposing Factors.—The exciting factors of acute dysentery will be discussed under each special type of the disease described.

Season.—Among the predisposing factors of those types of dysentery supposedly of bacterial origin *season* heads the list, the disease being more common during the summer and autumn months.

Age is a prominent favoring cause in all forms of acute dysentery, and although no age is immune, by far the greatest number of cases are seen during the first ten years, and also between the tenth and twentieth years.

Temperature is not without influence, since many cases develop after sudden climatic changes, as, *e. g.*, the extremes of temperature, humidity, etc.

Climate is a prominent factor, and dysentery is more common in subtropical and tropical districts, although epidemic outbreaks may be seen in the far north during the summer months.

Unhygienic surroundings, as is shown by the epidemic outbreaks seen in prisons, asylums, and armies, doubtless constitute a potent factor in the production of local epidemics.

The **existence of previous maladies** that have lowered the patient's general vitality lends a predisposition to all forms of acute dysentery; consequently dysentery is common when malaria prevails, and persons suffering from gastro-intestinal catarrh are likewise especially subject to infection.

Diet.—The condition is frequent among those who have committed dietary errors.

Acute Bacillary Dysentery.

Remarks.—This condition is the epidemic variety of acute dysentery excited by the Bacillus dysenteriæ (Shiga). Sporadic cases of bacillary dysentery, however, are by no means unusual in certain portions of the United States and Europe, as well as in tropical countries.

Pathologic Definition.—The bacillus of Shiga gives rise to an acute inflammation of the large intestine, which is soon followed by the occurrence of numerous minute ulcers on the intestinal mucosa. The mucous surface is covered with a somewhat tenacious serosanguineous exudate. The exudate and the severity of the ulcerative process vary greatly in different cases.

Varieties.—For convenience of study, bacillary dysentery is divided into two subclasses: (*a*) Catarrhal dysentery; (*b*) pseudomembranous dysentery.

Etiology.—According to the researches of Shiga, Kitasato, Flexner, Vedder and Duval, and many other observers, the weight of opinion is that all types of acute catarrhal dysentery are due to the bacillus of Shiga (Bacillus dysenteriæ). Most of the observers just mentioned, however, are in doubt as to whether or not the bacillus of Shiga is always an essential factor in the production of epidemic dysentery, but this point cannot readily be determined, since many other bacteria that may at times assume a pathologic rôle are also present in the intestine.

Acute Catarrhal Dysentery.

Pathologic Definition.—The large intestine is attacked, and there is hyperplasia, followed later by necrosis of the solitary follicles, with the ultimate formation of small ulcers. There may be an extensive purulent inflammation of the mucous surface of the colon, and the involved area displays numerous superficial ulcers. In exceptional cases the ulcerative process may extend from the large intestine to the ileum.

General Complaint.—The patient feels indisposed for one or more days, during which time he suffers from a somewhat indefinite type of gastro-intestinal catarrh, characterized by a lack of desire for food, slight intestinal pain, and mild diarrhea. Following these prodromes more characteristic symptoms develop, *e. g.*, intestinal colic, which is followed by frequent evacuations of the bowel. At first the stools number from two to ten daily, but they gradually become more and more frequent, and by the third or fourth day the number has increased to from 20 to 100 a day. The movements are accompanied by tenesmus. With the progress of the disease an almost constant desire to empty the bowel develops, and there is a continuous burning sensation in the rectum.

The patient complains of extreme weakness and of a feeling of faintness and vertigo upon slight exertion. Sufferers from acute dysentery refrain from unnecessary talking, and the voice is harsh and rasping.

Thermic Features.—Early in the disease the temperature may range between 99° and 100° F., and may rarely reach 104° F. by the fifth or the sixth day. Generally speaking, the temperature in dysentery is irregular, but in no way characteristic.

Physical Signs.—Inspection.—The expression is anxious, the cheeks are sunken, the lips are pale and fissured, and the tongue is dry and heavily coated. The extremities present a "waxy appearance"; the abdomen is scaphoid in shape, and the patient, as a rule, rests in the recumbent posture, so as to avoid all possible exertion.

Palpation is generally negative, although in some cases slight tenderness may be elicited over the course of the colon.

The pulse becomes weak, thready, and irregular, the beats numbering 110 to 140 a minute. The skin is cold and clammy, and in those cases in which prostration is extreme, the skin is beaded with drops of perspiration.

Auscultation.—The heart-sounds are weak and rapid. There is a decided gurgling over the abdomen, and particularly along the course of the colon.

Laboratory Diagnosis.—At first the number of bowel movements varies from four to ten a day, and the dejecta contain many scybalous masses. Later the stools become mucoid in character, and finally they are mucopurulent, seropurulent, or bloody. The quantity passed at each movement of the bowels may be extremely small—not exceeding from one-half to four drams.

Microscopically, the stool will be found to contain mucus, pus, and blood. If a severe type of infection is present, the watery stool contains many fat-globules and epithelial cells. Cultures from the dejecta will show the presence of the bacillus dysenteriæ in addition to many other bacteria.

The *urine* is scanty, high in color, and of high specific gravity; it is rich in indican, and may display a trace of albumin.

Blood.—Owing to the extreme depletion of liquids from the body the blood becomes concentrated, and during the height of the attack the number

of red cells in a cubic millimeter is likely to be above the normal, but during convalescence the usual evidences of secondary anemia are present, and both the red cells and the hemoglobin are decreased. The Bacillus dysenteriæ (Shiga strain) has been recovered from the peripheral circulation.

Serum Diagnosis.—In dilutions of 1 : 100 to 1 : 40 the serum of persons suffering from acute bacillary dysentery is capable of agglutinating the bacillus dysenteriæ.

Illustrative Case of Acute Catarrhal Dysentery.—C. C., female, twelve years of age.

Family History.—Parents and two brothers living and in perfect health.

Previous History.—The first record of illness was that of whooping-cough at the age of three years; scarlet fever developed at seven years of age, and measles before the tenth year, but in none of these conditions did any complications arise.

Social History.—Female, attending school during the fall and winter months, during which terms she was not compelled to be absent because of illness.

Present Illness.—The patient had been in perfect health until August 15th, when, after having eaten unripe fruit, she developed a mild form of constipation, which was later followed by anorexia, abdominal pain, and diarrhea. The number of stools a day varied between 10 and 40; at first they were scybalous, later small in quantity, and consisted chiefly of mucus, blood, and serum. Extreme prostration and intense thirst were present. Pain was usually localized to the region of the umbilicus, but with each evacuation of the bowel rectal tenesmus was distressing. The temperature fluctuated between 99° and 102° F., and was of an irregular type.

Physical Examination.—*General.*—The child usually rested for prolonged periods in one position, with the thighs flexed upon the abdomen, and showed no inclination to move about in bed. The mucous surfaces and skin were pale. She was rather stupid and the voice was husky.

Local Examination.—Inspection.—Her expression was anxious, the cheeks were sunken, and the features were pinched. The respiratory movements were feeble, and the abdomen was scaphoid in shape.

Palpation.—There was tenderness upon deep pressure along the course of the colon; the heart impulse was feeble, and the radial pulse was weak, small, and increased in frequency.

Laboratory Findings.—The urine was diminished in quantity, of high specific gravity, rich in indican, and contained a trace of albumin. The mucous stools were found to contain red blood-cells, leukocytes, desquamated epithelial cells, and granular débris. A bacteriologic study of the feces showed them to be rich in streptococci, staphylococci, and bacilli. Cultural studies showed the presence of the Streptococcus pyogenes and the colon bacillus.

Course and the Effect of Treatment.—During the first twenty-four hours abdominal pain, rectal tenesmus, and the general symptoms progressed from bad to worse, but after the application of judicious treatment the pain was relieved and the number of mucous stools fell from fifty to six a day. Within the course of ten days the child had recovered sufficient strength to be able to sit in bed, and developed an appetite for certain light foods. By the end of the second week she was able to leave her bed, and an uninterrupted recovery followed.

Complications.—Most serious among the complications are to be mentioned severe nervous symptoms, *e. g.*, the various types of delirium, followed by coma; in which class of cases a fatal termination is imminent. Cardiac failure is an occasional complication, as are also general peritonitis, bronchopneumonia, and meningitis.

Differential Diagnosis.—The maladies that simulate acute catarrhal dysentery are extremely few, similar symptoms, however, being occasionally seen in persons suffering from strangulated hemorrhoids, syphilitic disease of the rectum, and rectal epithelioma. In all the previously named conditions there is no history pointing toward an epidemic of dysentery, and the onset is slow, and the course continues over an indefinite period. Physical examination of the rectum will always serve to differentiate organic rectal disease from true catarrhal dysentery. Cultivation of the Bacillus dysenteriæ from the stool makes the diagnosis positive.

Duration and Clinical Course.—Mild cases continue for from eight to ten days, whereas in the more severe forms the dysentery may last for three or more weeks. In the majority of cases the disease goes on to recovery in less than four weeks, and probably one-half of all cases are able to be about the room in from ten to fifteen days. If the type of infection during an epidemic is unusually virulent, a high percentage of deaths follows.

Sequelæ.—Relapses are extremely common, and each attack increases the patient's susceptibility to another. Constipation and other gastro-intestinal disturbances are frequently seen to follow bacillary dysentery. Mitchell has seen paraplegia follow as a sequel to bacillary dysentery, and in a few instances stricture of the bowel has been reported.

Pseudomembranous Dysentery.

Pathologic Definition.—An acute inflammation of the colon with ulceration, caused by the Bacillus dysenteriæ, and characterized by the accumulation of a grayish-yellow exudate upon the mucous surface of the colon, with necrosis of the epithelial cells. In mild cases this process may be limited to the upper surface of the folds of the colon. In virulent types of the disease the deeper layers of the bowel may be involved, and in this class of cases the mucous membrane is yellowish-brown in color. The entire colon may be implicated. As a rule, the disease attacks the flexures of the colon and the rectum. Extensive sloughing, with the formation of large ulcers, may take place.

General Remarks.—This particular type of bacillary dysentery is seen more often in the tropics than in temperate climates, although epidemics and sporadic cases occur in practically all parts of the civilized world.

Exciting and Predisposing Factors.—The *exciting* cause is the bacillus dysenteriæ.

Among the *predisposing factors* should be mentioned **age,** young adults being most susceptible to the disease; it may, however, be found at any time of life.

Temperature also predisposes to this type of dysentery, and a warm climate probably facilitates infection, either from the drinking-water or from the ingestion of improper foods. Persons residing in barracks, asylums, and homes are not infrequently attacked, and those dwelling in cities are more often affected than are those residing in rural districts.

Season.—*Summer* and *autumn* appear to furnish the greatest number of cases in subtropical districts.

Principal Complaint.—There may be a history of some gastro-intestinal or obscure febrile condition antedating the attack of dysentery, but the rule is for acute pseudomembranous dysentery to begin abruptly, with the early development of severe local and general symptoms. The patient often gives a history of a mild chill, or, in rare instances, of a distinct rigor. Following the chill the patient observes that his face is flushed. The temperature will be found to register 100° to 103° F. within the first few hours. There is great weakness early, and in children delirium develops within the first forty-eight hours, whereas in adults nervous manifestations appear later. There is diarrhea, the stools numbering ten to fifty or more a day. Cramp-like abdominal pain is present, followed later by rectal tenesmus.

Physical Signs.—Inspection.—The face is flushed at first, but later there is a decided pallor of both the skin and the mucous surfaces. The tongue at first is red and glazed, but later may become brown and deeply

fissured. The cheeks are sunken and the expression is anxious. The patient hesitates to move, and his respirations are hurried and shallow.

Palpation.—During the first twenty-four hours the skin is hot and dry, but later, in severe types of infection, the skin may be cold and clammy and beaded with perspiration. There may or may not be tenderness, and, as the result of tympany, the abdominal wall is tense at times. The pulse soon becomes weak, rapid, small, dicrotic, and compressible.

Percussion.—In selected cases a tympanitic note may be elicited over the course both of the colon and of the small intestine.

Auscultation.—The heart-sounds are accelerated at first, but soon become feeble, rapid, and irregular. Decided gurgling is heard over the course of the colon and near the umbilicus.

Laboratory Diagnosis.—The discharges from the bowel are numerous, and with the unaided eye they are seen to contain shreds of sloughing tissue, and even portions of casts of the lower bowel. These shreds of tissue are usually milky-white in color, but if there has been hemorrhage from the intestinal mucous membrane, they are dark or brownish, and emit a fetid odor. Blood, pus, and mucus are also present in the stool.

Microscopically, the dejecta are found to contain shreds of mucous membrane, many epithelial cells, red blood-cells, blood-crystals, large numbers of leukocytes, and pus. Stained specimens of the mucus from the dejecta will display a profusion of bacteria. Slender bacilli, resembling bacillus typhosus in morphology (Bacillus dysenteriæ), and colon bacilli are present in great numbers. Cultures from the mucous or bloody exudate will develop the Bacillus dysenteriæ, which organism, when properly grown (see Widal reaction), will be found to agglutinate with the serum of persons suffering from this type of dysentery.

Illustrative Case.—A. L. B., male, aged twenty-three years; previously healthy, had been indisposed during the day, and developed a distinct chill, followed by a temperature of 102° F. Within the course of a few hours there were present quite severe abdominal pains, with frequent stools, which at first were scybalous, but later consisted of mucus and blood and contained a few shreds of membrane. By the end of the first thirty-six hours great intestinal pain and rectal tenesmus were present. Mild delirium appeared on the third day, and later the patient developed carphologia, subsultus tendinum, maniacal delirium at night, and finally became comatose.

Both the naked eye and the microscopic examination of the feces revealed the characteristics of pseudomembranous dysentery. (See Laboratory Diagnosis.) The pulse soon became weak, and later it was rapid and thready, and there was a tendency toward circulatory collapse. The urine contained a trace of albumin. With the progress of the disease evidences of profound toxemia became more and more marked, the case terminating in death by the end of the fourth day.

Summary of Diagnosis.—The diagnosis is made positive by the recognition of but few symptoms, for example: (*a*) The character of the stools and the presence of large shreds of brownish membrane; (*b*) the early development of nervous symptoms; (*c*) the tendency toward circulatory collapse, and (*d*) a positive serum reaction. All these serve to distinguish pseudomembranous dysentery from other maladies in which diarrhea is a symptom.

Complications.—Among these, special mention should be made of intestinal perforation, with the subsequent development of localized or generalized peritonitis. Acute ulcerative endocarditis and pericarditis have been known to complicate this form of dysentery. Pleurisy, acute parenchymatous nephritis, and bronchopneumonia may also develop. Myocarditis may occur in cases tending toward recovery.

Clinical Course.—The mortality rate is extremely high, many cases terminating fatally as the result of the profound toxemia. Permanent recovery may follow, although in these cases the disease runs a chronic course and restoration to health is protracted.

Secondary Pseudomembranous Dysentery.

Pathologic Definition.—A disease characterized by the formation of a false membrane on the intestinal mucosa.

Predisposing Factors.—Any condition that impoverishes the patient's general nutrition predisposes to the development of secondary pseudomembranous dysentery. The diseases during the course of which it is most likely to develop are: pneumonia, diabetes insipidus, pulmonary tuberculosis, chronic interstitial nephritis, chronic parenchymatous nephritis, gastric carcinoma, chronic suppuration, hepatic cirrhosis, essential anemia, and valvular heart disease. In these conditions the dysentery belongs to the group of terminal infections.

Chronic Dysentery.

Pathologic Definition.—A chronic catarrhal and ulcerative condition of the colon, secondary either to acute bacillary or to amebic dysentery. In the majority of cases there are ulcerative changes in the colon, and in certain cases these ulcerations show a tendency toward healing after an acute attack of dysentery, whereas in other cases there is a tendency for the ulcer to heal and form a constriction of the bowel as the result of the formation of scar tissue. At the site of the ulcers the intestinal mucosa is deeply pigmented, and displays a slate-gray or blackish color. There is generally some hypertrophy of the submucous and muscular coats of the colon, and the lumen of the bowel is frequently narrowed. In atypical cases actual ulceration of the intestine does not occur, although there is extensive formation of fibrous tissue, with some puckering of the mucous membrane.

Exciting and Predisposing Factors.—Chronic dysentery is usually secondary to one or more acute attacks. The disease often follows an unusually mild grade of amebic dysentery.

Among the *predisposing factors* are *age* and *sex*, the disease being more common in adult males than in women or children.

Principal Complaint.—The patient complains of moderate prostration, loss of flesh, mental hebetude, and restlessness; when questioned closely, he will state that he is free from pain and rectal tenesmus. The majority of patients with chronic dysentery suffer from acute exacerbations of the condition every three to twelve months, and at such times there may be intestinal pain and tenesmus. The average number of stools is from three to twelve daily, but this is controlled largely by the character of the food taken. Much undigested food escapes with the stool, and when the patient is upon a diet rich in starches, the stool is white and covered with froth. During an acute exacerbation, there may be blood and pus in the stool. Intervals of constipation are fairly common, and the degree of constipation that exists in a given case depends upon the character and location of the disease of the colon. The patient may complain that he has a more or less constant sense of fullness in the abdomen, but this seldom becomes painful.

The appetite is fairly good, although in some instances it is appreciably impaired. The patient often maintains that the character of food taken in no way influences the dysentery. When chronic dysentery has extended

over a prolonged period, mental hebetude is apparent, and late in the disease the patient may become stupid.

Thermic Features.—The temperature is normal, or possibly slightly below normal in the morning hours, except during an acute exacerbation, when there may be mild but irregular fever.

Physical Signs.—Inspection.—There are evidences of emaciation, and the general appearance is that of asthenia. The skin is pale, and at times yellowish or dusky; the tongue is clear at one time, bright red and glazed (beefy) at another, and probably heavily furred at the next examination.

Palpation.—The surface of the skin is harsh and dry, and always feels cool. With the progress of the disease the pulse gradually weakens and later becomes rapid, irregular, and dicrotic upon slight exertion.

Summary of Diagnosis.—The diagnosis rests first upon the history of a long-standing condition and the fact that there have occasionally been acute exacerbations. The number of stools a day—from four to ten—serves as a positive clinical evidence of chronic dysentery.

Differential Diagnosis.—Chronic dysentery is to be distinguished from *tuberculous ulceration* of the mucous coat of the colon. The distinctive differences between tuberculous enteritis and chronic dysentery are: (1) In the former condition there is commonly a history of tuberculosis of the lung or of other portions of the body; (2) tubercle bacilli are present in the feces. (See Examination of the Feces, p. 518.)

Duration and Clinical Course.—The duration of chronic dysentery varies between two and ten or more years. Dysentery, when it has not existed for a year, is generally considered as subacute. Cases will be encountered in which a dysenteric condition has lasted over a period of thirty or more years, and yet such patients, although never enjoying good health, are able to go about, although they are unable to do any form of labor. The duration of chronic dysentery is also influenced by judicious treatment, a number of cases terminating favorably in from one and one-half to three years.

Complications.—The complications are practically the same as those described for acute dysentery. Death, as a rule, results from the development of some intercurrent condition, *e. g.*, bronchopneumonia, pulmonary tuberculosis, and chronic kidney or liver disease. Chronic gastritis is a frequent complication.

ASIATIC CHOLERA.

Definition.—An acute infectious disease that may occur either sporadically or epidemically, excited by the bacillus choleræ (Vibrio choleræ Asiaticæ), and characterized clinically by copious watery discharges from the bowel, vomiting, intestinal and muscular cramps, suppression of the excretions, and collapse.

Incubation Period.—This varies greatly, and may be from a few hours to four or five days.

Clinical Types.—(1) **Premonitory Diarrhea.**—During the prodromal period the patient is, comparatively speaking, well, although he may exhibit slight local symptoms, *e. g.*, nausea, abdominal discomfort, and occasionally slight pains in the abdomen. The initial symptoms are somewhat severe; languor is experienced at this time, and the patient becomes easily fatigued.

(2) **Mild Type—Cholerine.**—In this type the symptoms are extremely

mild, and in many cases they are less severe than in cholera morbus (see p. 548). Although the general clinical picture of cholerine simulates that of true cholera, none of the symptoms are pronounced. Mild muscular cramps, slight prostration, a trace of albumin in the urine, and a cold, clammy skin, particularly of the hands and feet, are quite characteristic. It is important to bear in mind that in the mild type of cholera the stools are not characteristic, but, on the contrary, are feculent in character. In uncomplicated cases the duration seldom exceeds from seven to ten days.

(3) **Usual Types.**—The general clinical picture of this type will be given as the Principal Complaint.

(4) **Foudroyant or Asphyxic Type.**—In this type the disease develops suddenly, and the symptoms are so severe that the patient dies within a few hours. Vomiting and purging may or may not be present. The virulence of this type of infection is the only explanation offered for this clinical phase of the disease. **Cholera sicca** should also be included under this type.

Exciting and Predisposing Factors.—**Bacteriology.**—An essential factor in the development of cholera is infection with bacillus choleræ, and this bacterium may be isolated from the intestinal contents and from the watery discharges of persons ill with or dead of the disease.

Infection from Without.—The bacillus choleræ is found not only in the dejecta of persons suffering from the disease, but has also been isolated from drinking-water, and in 1892 Fränkel detected it in flowing water during certain epidemic outbreaks.

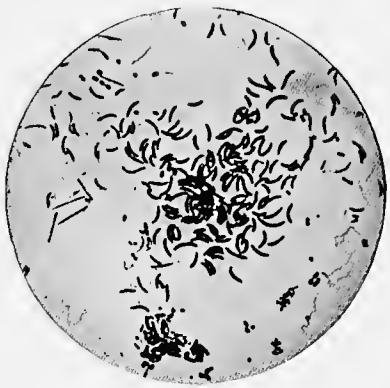


Fig. 275.—The Bacillus of Cholera (from the Mouth); ×1000 (Günther).

Geographic Distribution.—Those residing in the tropics are far more likely to develop the disease than are those in temperate and subtropical districts. Cholera tends to spread along the lines of commerce, consequently persons residing at or near the sea-coast and at prominent ports are especially prone to acquire the disease. Excessive humidity has been said to cause a predisposition to cholera, and a high temperature certainly favors the development of the spirillum.

Season.—Cholera prevails epidemically in subtropical districts during the warm months, although an epidemic may be continued well into autumn. The majority of European and American epidemics developed late during the summer months, and ended with the approach of cold weather.

Individual Susceptibility.—Intestinal catarrh from whatever cause and particularly that following the ingestion of unripe fruits, and the like, materially predisposes the individual to infection with the bacillus choleræ. Rigid sanitation exercises a great influence, and those living amid such environment are less likely to develop the disease than are others less fortunately surrounded.

Age and Sex.—Age and sex appear to exercise but little, if any, influence upon the development of cholera.

Previous Attack.—Persons having suffered from a previous attack of cholera are, as a rule, not immune to the disease.

Clinical Picture.—1. Usual Type, First Stage.—The *stools* are very frequent and painless. In *cholera sicca* the serous diarrhea is absent, death soon taking place. *Gastric symptoms* develop early, and consist of vomiting and intense thirst. The patient has no desire for food; his *tongue* at first is moist and coated, but later, if much liquid has been abstracted from the body, the tongue is dry and parched. He experiences a feeling of pressure or of discomfort in the abdomen, but real pain is unusual. Intestinal cramp and rectal tenesmus are occasionally seen. *Prostration* is extreme.

Nervous Symptoms.—The *mental faculties* may be retained until near death, but, as a rule, the patient is apathetic, or delirium may develop and coma ensue.

The *muscular symptoms* are severe and occur early, cramps affecting the various muscles (calves of the legs and feet) being perhaps one of the most distressing symptoms in a mild attack of cholera, although they are also severe in the more violent types of infection.

Thermic Features.—Ordinarily, the temperature, as taken by the axilla, falls to a subnormal level during the first hour, usually reaching 96° F. At the same time the rectal temperature will be found to vary between 101° and 105° F.

Cardiovascular Features.—These form a prominent part in the clinical picture, and are given at length under Physical Signs.

Physical Signs.—Inspection.—The expression is anxious, the face is pinched, the cheeks are sunken, the lips are pale and fissured, whereas the skin and tongue are dry and wrinkled. The eyes have a peculiar glare, and, owing to the high grade of cyanosis, the complexion is dusky or bluish. Cyanosis of the fingers is conspicuous.

Palpation.—The skin is cold, dry, and rough to the feel. The abdomen is, as a rule, soft, but may be tense. Pressure over the calf muscles excites discomfort, and at times pain. The reflexes are diminished or absent.

Early during the disease the pulse is rapid,—120 to 140 beats a minute,—and when the degree of liquid excreted from the body is large, it becomes smaller and smaller, until at last it is almost imperceptible.

Auscultation.—Owing to concentration of the blood the heart action becomes very rapid at the onset of the disease, and there may be distressing palpitation. Later the heart-sounds again become increased in frequency, the muscular quality being now absent, the sounds growing less and less distinct, until venous stasis occurs. Owing to dryness of the vocal cords and the other organs of speech the voice becomes feeble and husky.

2. Algid Stage (Ordinary Type).—During this stage of cholera the clinical manifestations described under the first stage of the disease are practically all present, but are appreciably intensified. The patient may be regarded as being in a state of asthenia, and the pulse is imperceptible, the cyanosis extreme, the skin and extremities very cold, the respirations shallow and frequent. Coma is likely to develop within a short time.

The copious watery discharges present in the first stage are here absent, although there is often a continuous dribbling of serous material from the rectum. No urine is excreted during this stage.

3. Stage of Reaction.—Reaction may follow the first stage of cholera, in which case there is an amelioration of all the symptoms and the patient goes on to recovery in from ten days to a few weeks. The kidneys again functionate, the cutaneous and rectal temperature approaches normal, the mucous surfaces become moistened, the heart action less rapid, the pulse

stronger, and the voice clearer. It is to be borne in mind that reaction may possibly develop during the second stage.

Cutaneous complications may develop during this stage, and among these are purpuric, roseolar, macular, and erythematous eruptions. The clinician should be ever alert for the development of serious nervous symptoms of a uremic character, since at this stage acute nephritis may be seen.

Laboratory Diagnosis.—All the secretions of the body are diminished, *e. g.*, there are scanty sputum and an absence of saliva, and the urine is diminished or suppressed during the first and second stages of cholera. During the stage of reaction the flow of urine is increased in favorable cases, although such urine is at first albuminous. If true nephritis develops, the urine will be found to contain an abundance of albumin and many casts. Leukocytes and red blood-cells may be present.

Stools.—At the onset the number of stools is great. Within a short time the stool presents a peculiar "rice-water" appearance. *Microscopically*, the small granules floating in the watery dejecta are composed of epithelial cells from the intestine. A *bacteriologic study* of the stool reveals the presence of bacillus choleræ (Fig. 275). The colon bacillus and other bacteria common to the intestinal tract are also present.

Blood.—If the number of watery discharges from the bowel has been large, the blood becomes concentrated, and the number of red cells in a cubic millimeter will be found to range between 8,000,000 and 12,000,000. The hemoglobin percentage of such blood is above the normal. After convalescence has been established the number of red cells falls to the normal, and later a decided anemia occurs.

The *vomitus*, which is also of the "rice water" type, contains the bacillus choleræ.

Illustrative Case.—E. X., male, aged twenty-two ears; formerly a resident of England, and now a private in the English army, has been in India for six months, during which time he has suffered from a mild attack of dysentery. For the past three weeks he has been in comparatively good health, except for indigestion. He was seized suddenly by violent muscular cramps, profound vomiting, and copious watery discharges from the bowel, which were not accompanied by intestinal pain.

During the first hour the patient's features became pinched, the expression anxious, and the pulse weak, the beats numbering 140 a minute. The cutaneous temperature fell below the normal, whereas the rectal temperature was 102.6° F. By the end of the third hour of his illness there was anuria, accompanied by a continuous dribbling of watery material from the bowel. Mental hebetude soon advanced to mild coma, and all the mucous surfaces became extremely dry. The dejecta presented a rice-water appearance, and these rice-like nodules were composed of desquamated epithelial cells. The bacillus choleræ and many other bacteria were also present.

The clinical picture of the patient now indicated a most critical condition, when, suddenly, there was a cessation of the symptoms previously described, the pulse became stronger, the cutaneous temperature rose steadily to the normal, and the tongue and other mucous surfaces became moistened. The kidneys now began to functionate, and convalescence was comparatively soon established.

Summary of Diagnosis.—A history of exposure or of residence in a district where cholera is epidemic, or even endemic, is of great importance. The diagnosis is based largely upon the character of the stools and of the vomitus, together with the existence of muscular cramps, a small, rapid pulse, and the early tendency toward collapse, marked by subnormal temperature, anxious expression, mental dullness, and coma. Recovery of the bacillus choleræ from the dejecta and from the vomitus renders the diagnosis

positive, but the acme of the disease is often reached before such cultural studies can be completed.

DIFFERENTIAL DIAGNOSIS BETWEEN ASIATIC CHOLERA AND CHOLERA MORBUS.

Asiatic Cholera.	Cholera Morbus.
1. History of an epidemic or of exposure in tropical districts.	1. History of dietetic errors, *e.g.*, eating unripe fruit or decomposed foods.
2. Diarrhea and vomiting are not accompanied by severe intestinal pain.	2. Intestinal colic a prominent symptom.
3. First vomitus contains particles of food, but soon resembles rice-water.	3. Vomitus contains food, and mucus may be present.
4. Vomitus contains the bacillus choleræ.	4. Bacillus choleræ absent.
5. Stools, although frequent, are without odor, and resemble rice-water in appearance.	5. Odor very offensive, stools feculent.
6. Rectal tenesmus extremely uncommon.	6. Rectal tenesmus may be prominent.
7. Collapse develops early and coma is common.	7. Collapse develops later and coma is unusual.
8. Axillary temperature becomes subnormal.	8. Temperature seldom below normal.
9. Anuria the rule.	9. Anuria very rare.
10. Complications somewhat common.	10. Complications unusual.

Clinical Course.—Cholerine terminates in recovery in practically all cases. The asphyxic type, which represents the other extreme of the disease, usually terminates in death. The mortality rate is found to vary greatly in different epidemics, ranging between 20 and 80 per cent. During the algid period, and still more often during the period of convalescence, nephritis and lung complications increase the gravity of the disease.

Cholera is extremely fatal in the asthenic, in those suffering from chronic disease, in alcoholics, and in the aged. The mortality rate is greatly diminished in those epidemics in which it is possible to institute treatment early.

Complications.—Complications are, as a rule, due to secondary infection. Septicemia and pyemia may develop, and pseudomembrane formation occasionally involves the mucous surfaces, *e. g.*, colon, throat, and vagina. In those cases in which the nervous symptoms are prominent bronchopneumonia is common, whereas pleurisy and parotitis are occasionally seen.

SPRUE (Psilosis).

Pathologic Definition.—A chronic disease characterized by atrophy of the walls of the bowel.

Exciting and Predisposing Factors.—The exciting cause is unknown, yet facts prove that it is most probably of microbic origin. Residence in the tropics is the chief predisposing factor.

Clinical Features.—Manson groups these as irregular action of the bowels, with characteristic stools, *i. e.*, stools that are copious, pale, drab, frothy looking, and that emit an offensive but sweetish odor.

The patient becomes cachectic and the skin is somewhat bronzed; mental hebetude and loss of strength are common. A characteristic feature of sprue is soreness of the mouth and of the rectum. Sprue is not essentially a fatal disease.

TYPHOID FEVER.

Pathologic Definition.—An infectious disease characterized by congestion, proliferation and ulceration of Peyer's patches and the solitary follicles. There are associated bronchitis, enlargement of the spleen, congestion and moderate enlargement of the liver, and a tendency toward such complicating conditions as bronchopneumonia, nephritis, intestinal perforation, and phlebitis.

Clinical Remarks.—There is bacteriemia excited by infection with the Bacillus typhosus, characterized clinically by an incubation period of from ten to twenty-five days, and by three stages:

(*a*) *Invasion*—a gradual daily rise in temperature, headache, lassitude, muscular pains, weakness, nose-bleed, constipation, followed by slight diarrhea.

(*b*) *Fastigium*—marked by continued fever, characteristic eruption, Widal serum-reaction, diarrhea, dilated pupils, pronounced nervous symptoms, abdominal tenderness, tympanites, and a tendency toward complications (intestinal hemorrhage, intestinal perforation, nephritis, and bronchopneumonia).

(*c*) *Stage of defervescence*—in which there is a gradual decline in the fever and an amelioration of all symptoms, followed by convalescence.

Exciting and Predisposing Factors.—Bacteriology.—The bacillus typhosus is the exciting cause of typhoid fever, although there are certain steps in the "postulates of Koch" that have not yet been completed, *e. g.*, the inoculation of an animal with a given organism, known to have excited the disease in another animal suffering from or dead of the disease, and recovering this organism from the second animal's tissues after inoculation, and at a time when it displays the symptoms of the disease in question.

Loris-Melikoff in studying the anaërobic bacteria of the intestines in typhoid fever found the Bacillus satillitis almost constantly present. This bacillus is agglutinated by serum from typhoid patients (1 : 100).

Animals infected by this organism show lesions similar to those seen in man. The Bacillus satillitis has also been recovered from the stomach of healthy oysters.

Another anaërobic organism commonly found associated with the Bacillus satillitis is the Bacillus perfringens. This organism, like the Bacillus satillitis, is capable of producing indol and phenol, as well as hyperemia and congestion of Peyer's patches.

It has been demonstrated that the Bacillus perfringens produces ulceration of the intestine. These anaërobic bacteria appear clinically to be potent factors in the production of typhoid fever. Confirmatory experiments are needed in order that we accept one or both of these organisms as exciting or contributing factors in the production of the many signs and symptoms that go to make true typhoid fever.

The Bacillus edematis maligni and Bacillus sporogenes have also been found by Loris-Melikoff in the dejecta of typhoid fever subjects.

Distribution of the Organism in the Human Body.—The bacillus is found in the lymph-glands, the contents of the intestines, the spleen, the liver, the blood, the bile, the rose-spots, the urine, the sputum, and the nasal secretions. (See Laboratory Diagnosis, p. 750.)

Distribution Outside of the Body.—The bacillus cannot readily maintain a permanent existence outside of the body. From time to time, however, the conditions indispensable to the growth and development of the bacillus typhosus prevail, and corresponding with such periods, more or less extensive

epidemic outbreaks of the disease may occur. It is known that the typhoid bacillus may retain its virulence for from seven to fourteen days in water. It disappears from water after this time, however, on account of the saprophytic organisms present. Multiplication of the bacilli may take place in water, in milk (very rapidly), and in the soil, where, under favorable conditions, they may live for an indefinite period. Freezing does not kill them, and they may live in ice for several months. According to the experiments of M. P. Ravenel, bacillus typhosus was not killed even by exposure to the temperature of liquid air—240° F. below zero.

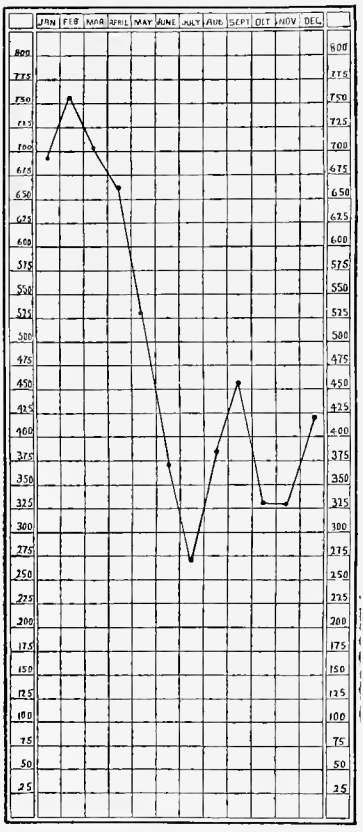


FIG. 276.—AVERAGE NUMBER OF CASES OF TYPHOID FEVER OCCURRING DURING EACH YEAR. STATISTICAL ANALYSIS OF 68,943 CASES (Sallom).

In a paper entitled "History of "Typhoid Fever," one of us (Anders) set forth the evidence offered by the different epidemics that occurred in armies in Europe, Africa, and America, and showed that typhoid fever did not remain in a camp when all the sick were removed, the bedding and linen used by such camp being destroyed by fire or left behind, and the healthy members of the army removed to new quarters. These facts indicate strongly that typhoid bacilli must have the power of existing upon clothing, and that they may be transmitted from such clothing to healthy persons, and in such persons excite the disease.

It has been shown that in armies the number of cases of typhoid was greatly reduced when all the patients suffering from the disease were safely screened from the attacks of flies and other insects. Much evidence has been adduced that goes to support the belief that the housefly acts as a carrier of the organisms concerned in the production of typhoid fever. The habits of the fly, particularly that of alighting upon dejecta, soiled linen, and food-stuffs, would certainly appear to be a possible means of infection, and one of the methods by which this disease is transmitted.

Dejecta known to be rich in typhoid bacilli, when thrown into small streams, may in turn find their way to the water-supply of towns and cities, and this method, although it was formerly believed to be the most common means by which bacillus typhosus gained access to the human body, still has many ardent supporters, yet bacteriologic research contributes but moderately toward strengthening this original view.

So far as we are aware, bacillus typhosus has never been found in the drinking-water used in the city of Philadelphia, yet there is probably no other city in the world that furnished so large a percentage of typhoid fever cases before the introduction of filtered water. Our own studies have shown that bacillus typhosus is unable to live in Philadelphia water for more than

from two to ten days, and that the number of bacilli present is greatly diminished after the first forty-eight hours.

Typhoid Carriers.—Persons who have had typhoid fever and recovered from it may show the exciting organism of the disease for an indefinite period after all symptoms have subsided. Some persons, doubtless, are hosts of the typhoid bacillus, while being themselves immune to the disease. Consequently such persons at times act as carriers of the bacilli and thus become responsible for the spread of the disease. It is believed that the habitat of the organism in the body of such carriers is chiefly the gall-bladder, whence they escaped by way of the bowels. In some cases bacilli are discharged in the urine for many years after convalescence from typhoid.

Geographical Distribution.—Typhoid fever is found to prevail, for the most part, throughout the temperate zone, and in America it is the most important of infections. Epidemics of the disease, however, have been reported as far north as Norway and Iceland. Typhoid fever is unusually common throughout the northern portion of the United States and in Canada, but is less frequently seen along the Gulf of Mexico and in the southwestern

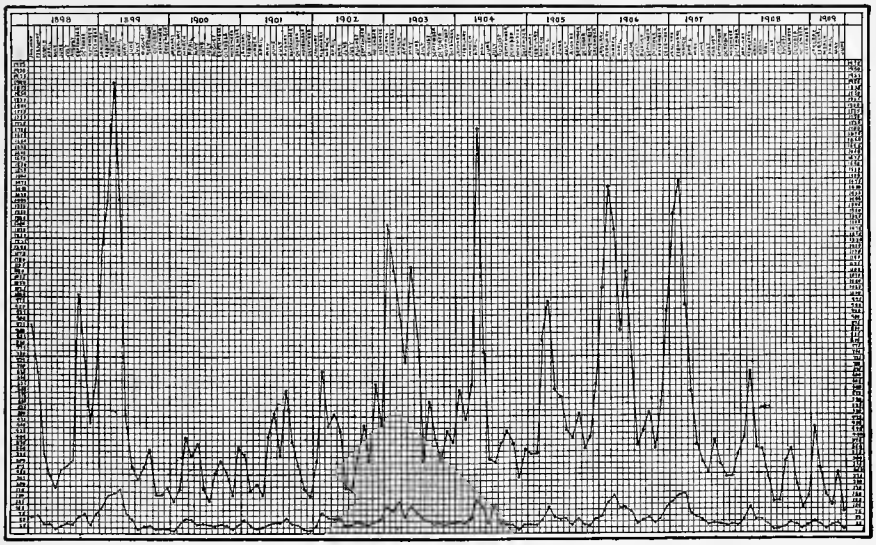


Fig. 277.—Chart Showing the Number of Cases and Mortality of Typhoid Fever by Months. Statistical Analysis of 68,943 Cases (Sallom).

part of this country. The disease is far less common in Europe than in America, and, owing to its infrequency, is greatly dreaded in European countries.

In large cities typhoid fever prevails endemically, with one or two epidemic outbreaks each year. Epidemics in the rural districts are not uncommon, but the disease seldom prevails endemically in such sections.

Season.—Available statistics go to show that the greatest number of cases are reported during the summer and autumn months—August, September, and October. More cases are frequently seen to occur during the late autumn and early winter months than during the spring. Sallom's analysis of 68,943 reported cases of typhoid fever for Philadelphia gives the largest number of cases as reported in February (see Fig. 276). The epidemology of typhoid fever is well shown in the accompanying chart (Fig. 277).

Temperature figures prominently as a predisposing factor, since epi-

demics of typhoid fever are unusually common after a long spell of hot and dry weather. It must be remembered that extensive epidemics of typhoid fever may occur at any time during the year, and that the so-called "house epidemics," developing in hotels, apartment-houses, colleges, asylums, etc., are but slightly influenced by season.

Clinical Varieties.—(1) The **usual form** of typhoid fever will display a *temperature* of from 100° to 103° F., and a pulse of from 100 to 120, which later tends to become dicrotic. The *tongue* is at first moderately coated, but this coating grows more intense with the progress of the disease, and by the seventh to the tenth day the organ is heavily furred. *Constipation* obtains during the first two to four days, when it is relieved by a moderate grade of diarrhea, the stools numbering from two to ten a day.

Some mental dullness, continuous headache, and mild delirium at night may be present. A slight amount of abdominal tenderness and tympany may also be present late during the first week. The *spleen* is enlarged, readily palpable and tender, and in from seven to nine days the characteristic rose-spots appear over the lower portion of the trunk and abdomen.

From the seventh to the fourteenth days mental dullness is more marked; the headache, which is prominent during the first week, gradually subsides, the pulse quickens, the temperature ranges from one to two degrees higher than during the preceding week, and tympanites is present. The mouth is dry and the tongue is dry and parched, the teeth and lips are covered with sordes, and all the symptoms previously described are intensified.

From the fourteenth to the twenty-first days the fever of 100° to 102° shows a moderate decline, although the pulse remains at about the same rate (90 to 110). Weakness is now extreme, and emaciation is noticeable.

Between the twenty-first and the twenty-eighth days the fever usually falls gradually to near the normal, the diarrhea subsides, the quantity of urine is increased, abdominal distention from tympanites disappears, the mouth becomes moist, the tongue clears, the patient becomes rational, and, in addition, develops a ravenous appetite.

This variety of typhoid fever is, as a rule, free from complications, although it may be well to state, in this connection, that no case of typhoid fever is so mild that serious complications may not develop during the third and fourth weeks of the disease.

(2) **Severe Form.**—In this variety all the symptoms described under the mild form of typhoid are intensified from the onset. The temperature soon reaches 104° to 106° F., the tongue is dry early, and by the second week becomes brown and deeply fissured; its surface may be bleeding, and in fact the organ may be coiled upon itself well back in the oral cavity. The fever remains high, and at times does not reach the normal level before the beginning of the sixth or the seventh week.

Nervous symptoms develop early, and carphologia, subsultus tendinum, and low muttering delirium are present. The urine and feces may be expelled involuntarily, and coma in many instances ends the scene.

The *pulse* becomes rapid by the end of the first week, and by the fourteenth day it is often 140 a minute, displaying a tendency toward dicrotism. Diarrhea develops early, although obstinate constipation is an equally serious symptom.

The *heart-sounds* are weak and distant, and suggest myocardial degeneration. There is a distinct *bronchitis*, and numerous fine râles are audible over the greater portion of the lungs posteriorly. The *urine* is scanty, of high color, of high specific gravity, and contains albumin.

The mortality rate in this variety of typhoid fever is extremely high, and in those cases that tend toward recovery convalescence is always protracted and complications are extremely common.

(3) **Mild Form.**—In this variety the disease seldom continues for more than two weeks, and by the twenty-first day at the longest the patient is able to leave his room. The onset is insidious and all the symptoms are mild.

The *temperature* seldom, if ever, exceeds 103° F., and in many cases ranges between 99° and 101.1° F. The *eruption*, a characteristic symptom, is, as a rule, scanty, and there may be but a single crop of rose-spots. Splenic enlargement is moderate, and slight tenderness is elicited only upon firm pressure. There is but slight, if any, tenderness over the cecum, a more constant sign being that of distinct gurgling in the region of McBurney's point.

The *Widal* serum reaction is present in the majority of these cases, but may not appear until the third or the fifth week of the disease, or even after convalescence is well established. (See Laboratory Diagnosis, p. 750.)

(4) **Latent (Walking) Typhoid.**—The symptoms described during the first week of the usual form of typhoid are so mild in this type that they do not even arouse suspicion as to the nature of the disease until the second or third week, when certain of the symptoms of typhoid appear. Many of these cases go undetected until some complication arises, when a careful study reveals the true nature of the condition in question. The *eruption* is said to appear in the majority of cases of walking typhoid, and this sign, together with the *Widal serum-reaction*, may be the only feature to indicate the nature of the malady in question.

When a patient suffering from walking typhoid has been permitted to go about his duties until the tenth or fifteenth day of the disease, he may then develop characteristic symptoms of the usual form of typhoid, and, indeed, it not infrequently happens that these patients at this time present many of the symptoms known to the severer form of the disease.

Complications frequently develop in cases of latent typhoid when the disease was not detected until the second or third week, but when the true nature of the condition is recognized early, cases of latent typhoid usually make an early and uninterrupted recovery.

(5) **Typhoid Fever of the Aged.**—When typhoid fever develops in persons after the fifth decade, it is characterized by mild fever, marked cardiac weakness, and a decided tendency to develop complications. The symptomatology of this form of typhoid is misleading, since the eruption and splenic tenderness are often absent. The Widal serum-reaction, however, is fairly constant.

(6) **Afebrile Typhoid.**—This type is certainly rare, although there are authentic records of such cases. Afebrile typhoid is said to present mild headache, slight weakness, some impairment of the appetite, questionable splenic enlargement, and a slow pulse. A typical eruption has been observed. Strictly speaking, this should not be considered as afebrile, but as a mild form of typhoid, the rule being for the patient to develop a temperature of from 99° to 101° F. during the afternoon and evening hours at some time during the disease.

(7) **Nephrotyphoid.**—In a small percentage of patients suffering from typhoid infection the most alarming lesions appear to be in the kidneys, taking the form of an acute parenchymatous nephritis. Symptoms develop abruptly; the urine becomes scanty, highly colored, rich in albumin, and contains hyaline, granular, and blood casts. Both red and white blood-cells

are present, and, indeed, hematuria may be one of the most prominent features of the disease. The bacillus typhosus may be cultivated from the urine.

Nervous symptoms appear early and are of a serious nature, the patient eventually developing uremia. The *temperature* varies between 100° and 104° F. The *pulse* does not become weak and dicrotic early, as is the case in the severe type of typhoid, showing the same nervous symptoms, but at first resembles the pulse of uremia. In nephrotyphoid the prognosis is decidedly unfavorable.

(8) **Cerebral Typhoid.**—Here the disease is ushered in by intense headache, facial neuralgia, nausea, vomiting, photophobia, twitching of the muscles, rigidity of the cervical muscles, with some retraction of the head and, rarely, convulsions. Extreme stupor and coma may end the scene.

This form of the disease is to be distinguished from epidemic meningitis, the distinction being made positive by lumbar puncture or by the Widal serum-reaction; spinal puncture is negative in typhoid fever, whereas the Widal serum-reaction is negative in epidemic meningitis and positive in typhoid fever.

(9) **Laryngeal Typhoid.**—This type of typhoid infection pursues a mild course, the invasion being unusually gradual; abdominal symptoms, *e. g.*, tympanites, tenderness, and diarrhea, may be slight or absent. The fever is not high, and the degree of prostration is only moderate.

Between the second and fourth weeks involvement of the larynx generally occurs, characterized by harshness of the voice, a hard, rasping cough, some soreness of the throat, and at times dyspnea. *Ulceration* may be present on the mucous surface of the larynx, epiglottis, or adjacent structures. (See Tonsillotyphoid, p. 744.) *Cultures* made directly from the ulcerating mucous surface, as a rule, show the presence of typhoid bacilli.

There are authentic records of aphthous patches having appeared on the mucous membrane of the mouth and nose, the result of typhoid infection. Perforation of the nasal septum may result from typhoid ulceration.

Typhoid of the upper respiratory tract, as a rule, tends toward recovery, the most imminent danger being infiltration of the larynx and interference with respiration.

(10) **Tonsillotyphoid.**—During the course of typhoid infection the only evidence of such lesions may be an infiltration of the mucous membrane of the tonsils. Small kidney-shaped, superficial ulcers may occur on the buccal mucous membrane from the seventh to the tenth day of the disease, and may be present in any clinical variety of typhoid fever. These ulcerations are usually bilateral, located on the fauces just above and to the outer side of the tonsil (so-called "Bouveret" ulcer). The concavity of the ulcer is usually directed toward the median line, the ulcer varying greatly in size from $\frac{1}{4}$ to $\frac{3}{4}$ inch in length, and from $\frac{1}{3}$ to $\frac{1}{2}$ inch in width. Ordinarily these ulcerated surfaces are covered by a grayish slough. Their duration varies from that of a few days to one week. Clinically they are of diagnostic importance, since they are present before the appearance of the Widal reaction in about 5 per cent. of cases. Bouveret ulcers have been reported by Johanns and Devic, who observe them as a precursor of typic typhoid relapses. Tonsillotyphoid is, as a rule, mild in nature, and commonly runs a short course, with a slight tendency to develop complications.

In all these special types of typhoid infection of the throat and upper respiratory tract the Widal serum-reaction and the recovery of the bacillus typhosus from the ulcerated surface are the most distinctive clinical features.

(11) **Pneumotyphoid.**—One occasionally encounters typhoid fever in which the leading symptoms are referable to the lungs, and there is a somewhat severe acute bronchitis that develops early during the attack, which is likely to be followed later by bronchopneumonia, lobar pneumonia, or acute pleurisy with effusion. Pneumotyphoid, as a rule, displays somewhat marked nervous and circulatory symptoms, and the prognosis in this type of the disease is but guardedly favorable.

(12) **Typhoid Septicemia.**—Here the general clinical picture is that of profound sepsis, and the symptoms of this condition usually progress from bad to worse until a fatal termination is reached. The prognosis in the so-called septic type of typhoid is unfavorable, but a small percentage of the cases going on to recovery. All the symptoms described in the usual type of the disease are greatly intensified in this septicemic form.

(13) **Typhoid of Children.**—The typhoid of childhood is a fever characterized more often by nervous than by intestinal symptoms. The *onset* is sometimes sudden, with well-marked symptoms, *e. g.*, fever, prostration, emaciation, and vomiting are not uncommon. The disease is also seen to begin with lassitude, headache, coated tongue, anorexia, and a gradual rise in temperature. In cases developing abruptly it often appears as though acute indigestion had been the means of precipitating the attack. Vomiting is, as a rule, the initial symptom. Epistaxis may occur, but is less common in children than in adults. The course is mild, as a rule, and the mortality low.

Diarrhea.—There is no constant relation between the severity of the intestinal lesions and the condition of the bowels. Diarrhea is present in about 50 per cent. of cases, the average number of stools being from two to four a day. There is nothing about the stool that may be said to be characteristic. Constipation is a feature in many cases in which typhoid develops before the tenth year, and according to Morse's statistics, over 60 per cent. of children manifest constipation during typhoid infection. Constipation is the general rule at the onset of the fever, a condition equally true of typhoid in the adult.

Abdominal distention, due to tympanites, is less constant in children than in adults, and tympany, when present, is usually a feature of those cases showing diarrhea due to colonic ulceration and marked catarrhal enteritis. Other abdominal features of typhoid, *e. g.*, gurgling and tenderness in the iliac fossa, are not constant.

Eruption.—An eruption appears in approximately 60 per cent. of all cases, but children, as a rule, develop but slight eruption, and there may be only a single crop of rose-spots. In the relapsing typhoid of children an eruption occurs with each relapse.

Thermic Features.—The temperature will be found to vary from that described under the typhoid of adults in the following features: In children under three years of age the fever lasts for from eight to fourteen days. Wolberg's analysis of 277 cases shows that the fever was of less than fourteen days' duration in 70 per cent. of cases, and in 2.8 per cent. of these, it did not continue for more than eight days. A subnormal temperature is the rule during the first week in those cases in which typhoid develops insidiously. Marked elevation of temperature during the second week of the disease is suggestive of complications.

The *nervous symptoms* are in direct relation with the degree of fever, and the severe forms of delirium so common in the adult are extremely rare in children.

Principal Complaint.—Stage of Incubation.—The average length of this period, until the first definite symptoms of typhoid appear, is not definitely known, but is probably, in the majority of cases, between seven and ten days, although in rare instances it may be three or more weeks. The patient at first appears to be in good health, but as the disease progresses, definite symptoms develop, and he complains of prodromes, *e. g.*, languor, anorexia, headache, nose-bleed, muscular pains, nausea, and constipation, which continue for from three to seven days, when a mild diarrhea begins.

Clinical Stages.—For convenience of study, typhoid fever is divided into three distinct stages; thus, in the moderately severe cases the first week represents the stage of development; the second and third weeks, the fastigium; and the fourth week, the stage of decline.

(1) *Stage of Development.*—The period of invasion is, as a rule, gradual, the symptoms being chilliness and slight fever, with an increase in the severity of the prodromal symptoms. At or about this time epistaxis may reveal the nature of the disease. Headache is continuous. The symptoms described are quickly followed by prostration marked enough to compel most patients to take to their beds.

It is best to regard the time of occurrence of the previously mentioned symptoms (elevation of temperature, with its attendant discomforts) as the stage of onset, since many patients continue at their accustomed vocations for days after the first symptoms appear. The onset may be marked by symptoms resembling influenza, and muscular pains and pharyngeal irritations are by no means uncommon at the onset.

With the progress of the initial period the symptoms usually increase in severity daily; the temperature (Fig. 278) is higher each day, until the fourth or sixth day, when the fastigium is reached. The appetite is lost, there is intense thirst, the face is flushed, the skin is hot and parched, and there may be profuse sweating in the axilla and groins. Distinct flushings, alternating with chilly sensations may occur. The symptoms and signs of a mild bronchitis are present in more than 50 per cent. of all cases.

The *pulse* has gradually increased in frequency with the progress of the disease, until it is full and strong, the beats numbering from 90 to 110 a minute.

Upon palpation tenderness is often detected in the right iliac region, and distinct intestinal movements can be felt at this point. Moderate splenic enlargement is the rule, and the organ is often quite tender.

(2) *The Fastigium.*—In typical cases this begins on the fifth to seventh day of the disease, and lasts about two weeks. During the first week of the fastigium (the second of the disease) all the general symptoms become intensified. The evening temperature ranges between 103° and 105° F., and approaches the continued type. The pulse is accelerated, but full and of fair strength. Headache, a prominent symptom during the first stage, gradually disappears, and in its stead there are seen mental hebetude, slowness of speech, and a moderate degree of deafness. There may be delirium, which is most likely to occur at night.

Cough with the physical signs of bronchitis are present, and there is, at times, slight expectoration. Diarrhea—from two to eight stools daily—is present during this stage.

Inspection.—The cheeks are somewhat sunken, the lips are dry and fissured, and the tongue is heavily coated, and often brown and bleeding. Sordes is seen on the lips and the teeth. The abdomen is distended, and

there may be profuse sweating and urticaria. About the eighth day of the disease a number of rose colored spots appear on the trunk. The rose spots of typhoid fever disappear when the skin is stretched or when pressure is made upon them, but they reappear when pressure is relieved.

Palpation.—The abdomen is moderately tender, peristaltic movements of the bowel are prominent in the right iliac fossa, and the spleen is readily

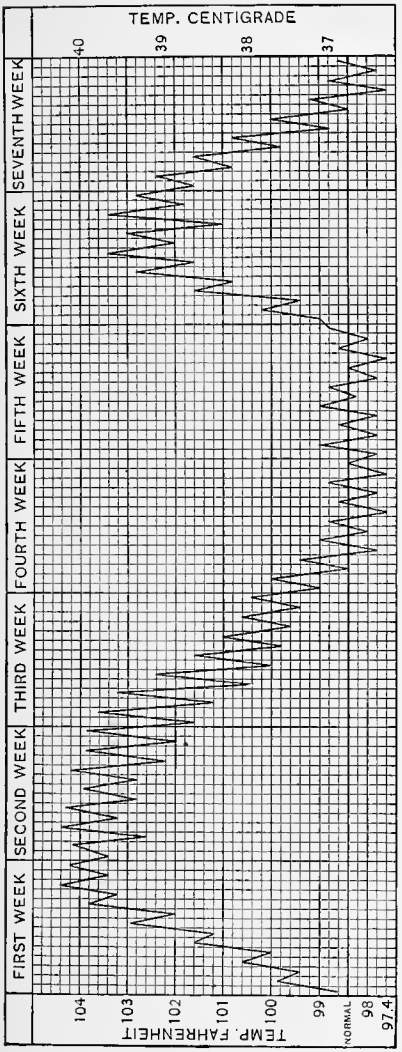


FIG. 278.—TYPICAL TYPHOID TEMPERATURE CURVE WITH RELAPSE BEGINNING THE SIXTH WEEK OF DISEASE.

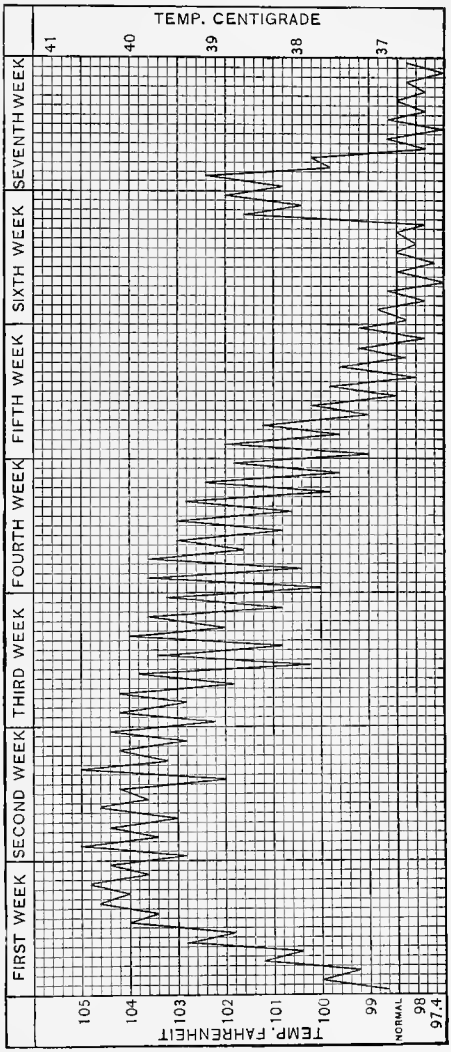


FIG. 279.—TEMPERATURE CURVE IN SEVERE TYPHOID, SHOWING RECRUDESCENCE LATE DURING THE SIXTH WEEK.

palpable and may extend some distance—two to four inches—below the costal margin. At the close of the second week complications are most likely to occur. (See Complications, p. 755.)

The third week of the disease, and the second week of the fastigium, finds the symptoms previously detailed at their height, and other more serious symptoms and even complications may be added.

(3) *Defervescence.*—At the end of the second stage, and about the twenty-third day of the disease, the fever begins to decline in favorable cases, and with this change the other general and local symptoms become gradually ameliorated, which improvement continues for one week and is followed by an establishment of convalescence.

In protracted and unfavorable cases the fourth week of the disease may present the same clinical indications as did the third, and, in fact, when the type of infection is unusually virulent, the symptoms outlined during the third week may be greatly intensified.

In unfavorable cases the pulse may range between 120 and 140 beats a minute, and become weak, dicrotic, compressible, and irregular. Nervous symptoms are also likely to be pronounced, all types of delirium and coma being occasionally seen. The abdomen is greatly distended, and the involuntary discharge of urine and feces is occasionally observed.

The fever may remain high during the fourth week, and we have seen cases in which the fever continued above 102° F. during the fifth, sixth, and seventh week of the disease. Instances are recorded in which the fever has continued for a period of fifty and even seventy days.

In those cases in which convalescence is established late, relapses, as well as recrudescences, are extremely common. (See Figs. 278 and 279.)

Clinical Picture.—Thermic Features.—In *typical* cases the temperature rises gradually during the first week, reaching 103° to 104° F., with morning remissions of one to one and one-half degrees. (See Fig. 278.) During the third and fourth weeks the morning temperature becomes normal, but there is a persistence of fever during the evening hours, which in uncomplicated cases is from one to two degrees above the normal. By the end of the fourth week this evening rise of temperature has entirely subsided, and the morning record is subnormal, whereas the evening registers at or below the normal line.

Atypical Temperature.—(1) *The Inverse Type.*—A low evening and a high morning temperature are occasionally displayed. (2) The fever may terminate at the end of the second week, the temperature declining somewhat rapidly, and reaching the normal in from forty-eight to seventy-two hours. (3) An abrupt development of fever is seen in children and in those cases in which the disease is ushered in by a chill or a series of chilly sensations. (4) *Intestinal perforation and hemorrhage* are marked by a sudden fall in the temperature when the thermometer registers below normal. (5) A rapid rise in temperature during convalescence is the result of dietetic error, constipation, overexertion, or mental excitement, and is, as a rule, of but short duration. Such thermic exacerbations are generally regarded as a "recrudescence"—fever without the other symptoms of typhoid. (6) In those cases in which there is questionable involvement of the bones, joints, pleura, gall-bladder, or other portions of the body, an evening temperature of 99° to 100° F. may continue indefinitely. (7) During relapses the fever is seldom as high as it was during the initial seizure, unless the primary attack has been abnormally light, is of shorter duration, and, like all the other symptoms, is of a mild nature.

Gastro-intestinal Symptoms.—Anorexia is present early during the course of the fever; nausea and vomiting are occasionally present as the disease progresses, but are by no means constant. Vomiting indicates either a severe grade of infection or the development of serious complications. *Diarrhea* is a fairly constant symptom, although in the majority of cases it appears during the second week of the disease. The *stools* are thin, semi-

liquid, or pea-soup-like in consistence, and vary in the average cases, from two to six daily. The stools may be more frequent, and in those patients suffering from either severe gastro-intestinal catarrh or extensive ulceration in the colon there may be ten or even twenty movements a day.

Stools containing blood are seen to occur during the third week, and even during convalescence. When the hemorrhage into the bowel is large, the stool may contain clotted blood, which is of a bright red color, but, as a general rule, the blood is retained in the bowel for some hours, when the dejecta have a tar-like color and consistency. Chemically, the stools contain minute quantities of blood in the majority of all cases of typhoid fever.

Constipation is present during the first week, as a rule, and may continue throughout the course of the disease. (See Typhoid of Children, p. 745.)

Circulatory Symptoms.—The *pulse* is, as a rule, but moderately accelerated, and the rapidity of the heart-beats is not in proportion to the degree of fever present—*e. g.*, a pulse of 90 beats a minute may accompany a temperature of 100° to 104° F. The pulse may occasionally become weak early during the course of the fever. In severe types of infection there is a tendency for the pulse to become thready, dicrotic, and compressible. The heart-sounds also bear a close relation to the character and frequency of the pulse. (See Physical Signs, p. 749.)

Respiratory Symptoms.—The respirations are slightly increased in frequency, and a harsh, non-productive cough, that may, though rarely, become quite annoying, is present. (See Complications, p. 755.)

Epistaxis is one of the early symptoms, and is common, particularly in the typhoid of young adults. Bleeding from the nose may be profuse, but in the average case there are three or four attacks, the patient losing but a small quantity of blood. Epistaxis may occur during the fastigium, and may then prove serious.

Nervous Symptoms.—Persistent headache is prominent during the progress of the disease, but by the end of the second week it gradually disappears, and at this time a moderate amount of mental apathy and stupor is apparent. The patient's hearing is somewhat dulled, his speech is thick, and his words are hesitating.

As the acme of the disease is reached the various types of delirium are likely to develop—*e. g.*, low, muttering delirium, which is present only at night, carphologia (picking of the bed-clothing), tremor (both fine and coarse), subsultus tendinum, and maniacal seizures may develop. Following the previously described types of delirium, the patient may fall into a semi-comatose state, or coma-vigil and profound coma may follow.

Convulsions are uncommon, except in those cases in which there is involvement of the kidneys or of the meninges. In typhoid of the meningeal type (see Varieties), strabismus, photophobia, ptosis, and hyperesthesia are to be seen.

Physical Signs.—Inspection.—Throughout the entire course of the fever the face is flushed, unless the patient has had severe intestinal hemorrhage. The expression is somewhat dull and the pupils are moderately dilated. At first the tongue is slightly furred over its superior surface, but later it becomes heavily coated, and in severe cases it may be bright red and deeply fissured, and portions of the surface may be brown from minute hemorrhages. It is not uncommon, during the height of the disease, to find the tongue somewhat rolled upon itself, the patient being unable to

protrude it beyond the teeth. The lips become fissured when the temperature continues high and the teeth are covered with sordes. Swelling and congestion of the tonsils and of the pharynx are occasionally seen. (See Varieties, p. 742.)

Between the seventh and ninth days a crop of small, circular or lenticular rose-spots is seen upon the abdomen; these disappear upon pressure and on stretching the skin, but immediately reappear after such pressure is removed. The eruption of typhoid is likely to occur every two or three days in successive crops. We have studied several cases in which the eruption was profuse and covered almost the entire body. In seven such cases studied at the Philadelphia Hospital, intestinal hemorrhage was a complication in six, and the remaining case showed severe nervous symptoms. From this small collection of cases, however, it cannot be positively asserted that a profuse eruption is always followed by severe symptoms.

The abdomen is distended, the patient usually resting upon his back, and showing a disinclination to turn from side to side. In all cases of typhoid the respiratory movements are somewhat quickened, and the impulse of the apex-beat is, as a rule, diffuse. Late during the course of the disease pulsation of the vessels of the neck, at the epigastrium, and in the second intercostal space may be detected.

Palpation.—The skin of the face and abdomen is usually dry and hot early. As the patient becomes decidedly prostrated, the axillary and inguinal regions are bathed in perspiration.

By the beginning of the second week of the fever the spleen is felt below the costal border, and in severe types of infection it may extend to or even below the umbilicus. The spleen is, as a rule, somewhat tender, although this peculiarity may be absent. There is also moderate hepatic enlargement.

There may or may not be distinct localized tenderness in the right inguinal region and along the course of the ascending colon. On placing the hand gently over the right iliac region, a distinct gurgling movement of the bowel is often felt, but its clinical significance is limited.

Percussion.—Tympanites is the rule, and when present, an increased area of liver and splenic dullness may not be detected. In the absence of gaseous distention the area of splenic dullness is perceptibly and often greatly increased, and there may be a moderate increase in the area of liver dullness.

Percussion over the region of the bladder is an important measure when delirium is present, since by this means retention of the urine, which is an occasional complicating feature, may be detected.

Auscultation.—Borborygmus is heard over the entire abdomen, but is more pronounced in the right iliac fossa and along the course of the ascending colon.

The heart-sounds are at first increased in frequency, but the muscular quality is normal. With the progress of the disease, and in protracted cases, the first sound of the heart becomes greatly weakened, and, indeed, the first and second sounds may be very similar during the third and fourth weeks of the disease.

A variable degree of bronchitis is always present, so that both moist and dry râles are audible over the chest.

Laboratory Diagnosis.—The *Widal* serum-reaction is present at some time during the course of an attack of typhoid fever in from 95 to 98 per cent. of all cases. As a rule, this reaction may be obtained about the

end of the first week of the disease. Rarely, indeed, it is absent until the second or third weeks, and we have seen cases in which it did not appear until convalescence was well established. (See Serum-reaction, p. 344.) Leukopenia is the rule in typhoid fever, the number of *leukocytes* in a cubic millimeter of blood being, in a typical case, between 5000 and 6000. When inflammatory complications are present, such as bronchitis, pleuritis, and bronchopneumonia, a slight leukocytosis may be found. The *differential count* of the leukocytes shows a relative increase in the number of lymphocytes and a diminution in the number of polymorphonuclear neutrophile cells. The leukopenia is often most marked during convalescence. During the first week the red cells are but slightly altered in number, but as the disease progresses, and during convalescence, the number is diminished. In those cases in which there are complications that embarrass the circulation and produce cyanosis the number of red cells in a cubic millimeter may be greater than normal.

The alkalinity falls to a point below that indicated by the color-index prior to the appearance of the Widal reaction.

The hemoglobin is diminished and the color-index is low.

Cultures from the blood will be found to develop typhoid bacilli in from 20 to 30 per cent. of all cases.

*Prendergast's Vaccine Reaction.**—Take 1 drop of ordinary typhoid vaccine and to it add 20 drops of sterile saline solution. Mix thoroughly, and then introduce into the skin, by means of an ordinary hypodermic, a few drops of this mixture. Direct the needle so that the solution when injected will be located near the surface of the skin, and endeavor to produce with the solution a rather conspicuous blister. Within twenty-four hours following the introduction of the diluted vaccine into the skin patients not suffering from typhoid will display an area of redness surrounding the site of the injection, while typhoid patients show no evidence of such reaction. Practically, all evidences of the typical reaction disappear within forty-eight hours.

Generally speaking, the *urine* is that ordinarily regarded as febrile in character. The quantity voided during the twenty-four hours is at first slightly increased, but with the increasing fever it is lessened, of high color, and of high specific gravity. A trace of albumin is ordinarily present, and in severe types of infection casts, leukocytes, and red blood-cells are found. Indican is present throughout the greater part of the febrile period; the diazo-reaction may be obtained during the first week of fever, commonly continues throughout the febrile period, and may even be seen after convalescence has been established.

A *bacteriologic study* of the urine will show the presence of the Bacillus typhosus in about 20 per cent. of all cases.

Bile.—Certain clinicians attach importance to an early bacteriologic study of the bile in the recognition of typhoid fever. Kiralifi has called attention to the fluid of the duodenum being practically free from bacteria in health, and he further claims that bacteria of the colon group are present in great numbers during the early stage of typhoid.

Test.—Give the patient 200 c.c. of sweet oil (olive oil) (in event of the pylorus being relaxed duodenal fluid is regurgitated into the stomach). Recover the oil-test-meal, by means of the stomach-tube, and examine it bacteriologically. Brinton has found that by administering into the stom-

* Medical Record, Dec. 30, 1911.

ach 400 to 600 c.c. of sterilized water practically the same results are obtained as are given under the oil-test breakfast. Both Kiralifi and Brinton's tests are valueless unless there be relaxation of the pylorus at the time of their application.

Feces.—During the prodromal stage of typhoid constipation is the rule, but as the disease progresses from four to six semiliquid stools a day, like pea soup, are the rule in mild types of infection. In severe forms of typhoid the number of movements from the bowel may be greatly increased, and during the second and third weeks intestinal hemorrhage may occur. Hemorrhages may be frequent and small in quantity; but in unfavorable cases hemorrhage is often profuse. Occult blood is present.

Cultures from the feces during any stage of typhoid will show the presence of typhoid bacilli.

Illustrative Case of Typhoid Fever.—D. D., male, aged twenty-three years; weight, 146 pounds; height, 5 feet 8½ inches.

Family History.—Parents, three sisters, and a brother living and in apparent health.

Previous History.—The patient has had the diseases of childhood, but has no recollection of having had either scarlet fever or diphtheria. He has not been compelled to consult a physician in the past five years, but prior to that period he had occasional attacks of tonsillitis.

Social History.—The patient is single; he has lived in the country until six weeks ago, when he moved to the city. A clerk by occupation, his work requiring that he be confined indoors during the greater part of the day. His habits in regard to eating and drinking are good.

Present Illness.—This began October first, when he observed that his appetite was failing and that he felt greatly exhausted after his day's work. For a period of one week before consulting his physician he suffered from malaise and aching of the muscles of the legs and of the back, a variable amount of headache, and two mild attacks of epistaxis. During this time he also suffered from constipation and from ill-defined sensations over the abdomen. At the beginning of the second week of the disease the patient experienced an acute lancinating pain in the region of the spleen, and although this severe pain lasted for six hours, it was not accompanied by any decided fall in the temperature or slowing of the pulse.

Upon his admission to the hospital on the eighth day of his illness, he had a slight cough, which was unaccompanied by expectoration. Headache, which became worse upon exertion, was present.

Mild delirium and restlessness were present during the night from the eighth to the fourteenth day after admission, whereas from the fourteenth to the twentieth days delirium continued during the greater portion of the twenty-four hours, but at no time was it maniacal.

The temperature upon admission was 101° F., but within the course of two or three days it had reached 103° to 104° F., during the evening hours, with slight remissions during the early morning hours, but in general it was more or less of the continued type, until the twenty-first day, when it fell gradually, and reached the normal by the end of the fourth week. When the temperature remained above 102.4° F., for a period of one or two hours the patient was given a cold pack or sponged with alcohol and water, and with either treatment the temperature fell from one to two degrees.

Physical Examination.—*General.*—Upon admission the patient was pale, his gait was feeble, and he rested his head upon his hand while sitting. When placed in bed, he assumed the supine position, with the thighs moderately flexed upon the abdomen. On extending the hands, there was distinct trembling. Both skin and tendon reflexes were approximately normal.

Local Examination.—Inspection.—Upon admission the abdomen presented nothing abnormal, but two days later there was a distinct typhoid eruption. From the fifteenth to the seventeenth days of the disease the abdomen became markedly distended as the result of tympanites. The skin was commonly bathed in perspiration, and those portions of the body upon which the patient rested most of the time became reddened. The tongue was coated early and later became brown and fissured. The conjunctivæ were congested.

Palpation.—Throughout the greater part of his illness the abdominal wall was more or less tense. The spleen could be felt to extend for a distance of two finger-breadths

below the left costal margin, and upon making firm pressure over this area tenderness and even pain resulted. The *pulse* was of good volume at first, the beats numbering 90 to 100 a minute, but during the third week of the disease, and at that time when delirium was present, the number of pulse-beats varied between 110 and 120 a minute. The force of the pulse was greatly lessened at this time, and there was a tendency toward intermittence and dicrotism. On palpating the abdomen in the right iliac fossa distinct gurgling could be detected from the beginning of the second until the middle of the third week of the disease.

Percussion.—During the early stage of his illness the percussion-note was practically normal over the abdomen, but when tympany existed, it was greatly altered. The area of cardiac dullness and that of pulmonary resonance were not altered.

Auscultation.—On placing the stethoscope over the colon it was possible to detect an unusual amount of gurgling during the first and second weeks after his arrival at the hospital. During the third week of the disease the first sound of the heart appeared to have lost its booming quality, yet there was no evidence of endocardial disease. Fine sibilant râles were heard over both lungs posteriorly.

Laboratory Findings.—The blood count showed that the number of leukocytes in a cubic millimeter was below normal; the red cells numbered 4,100,000 in a cubic millimeter and the hemoglobin content was 80 per cent. The Widal reaction was positive during the first week of his stay in the hospital.

The number of bowel movements a day varied between three and thirteen during the second and third weeks of the disease, and a small quantity of blood twice escaped with the dejecta. The urine was high in color, and contained a trace of albumin during the febrile period.

Diagnosis by Induction from Clinical Data.—The age of the patient and the existence of prodromal symptoms, such as pains in the muscles, headache, and epistaxis, were considered as favoring the possible existence of typhoid. Later in the course of the disease the temperature became characteristic, and at this time the positive Widal reaction was considered sufficient to warrant a diagnosis.

Differential Diagnosis.—During the height of the disease the nervous symptoms, the pulse, and temperature suggested the possible existence of acute miliary tuberculosis, from which malady the condition was distinguished by the following clinical facts: (*a*) The blood gave a positive Widal reaction; (*b*) the temperature had risen gradually, and showed a conspicuous decline after the eighteenth day; (*c*) diarrhea became a prominent feature during the second week of the disease; (*d*) epistaxis appeared during the prodromal stage; and (*e*) tympanites was present.

Course of the Disease.—By the beginning of the fifth week of the disease the patient was able to sit up in bed, and continued to make an uninterrupted recovery.

Summary of Diagnosis.—The diagnosis of typhoid is made positive by—(*a*) The history of the patient having never before suffered from typhoid; (*b*) the characteristic temperature-curve; (*c*) the splenic tumor; (*d*) the characteristic eruption; and (*e*) a positive Widal reaction. Many other symptoms, previously mentioned, go far to support a diagnosis of typhoid fever, but are not essential to the recognition of the disease. Season, the presence of an epidemic, and the age of the patient, although they are often of great value in strengthening a doubtful diagnosis, are equally likely to mislead the clinician.

Differential Diagnosis.—**Typhus fever** is to be differentiated from typhoid fever by the presence of an epidemic, by its sudden onset, the presence of stupor, the dulled expression of the features, conjunctivitis, and the pin-point pupils, all of which conditions are but rarely, if at all, seen in typhoid fever. Typhus is characterized by a macular eruption that appears upon the fourth day and may become petechial. The fever, as a rule, runs a shorter course in typhus than in typhoid, and the Widal serum-reaction is negative.

Acute miliary tuberculosis is frequently mistaken for typhoid fever, and the distinctive features between these two conditions are shown in the accompanying differential table:

TABLE SHOWING THE DIFFERENTIAL POINTS BETWEEN TYPHOID FEVER AND ACUTE MILIARY TUBERCULOSIS.

Typhoid Fever.	Acute Miliary Tuberculosis.
1. History of an epidemic or of change of residence from the country to the city.	1. May be history of cough or of a tuberculous ulcer, abscess, fistula, etc.
2. Fever rises gradually, in step-like form, reaching its height in from seven to eight days, and declines during the third or fourth week.	2. Fever rises more abruptly, and remains high (continued type) until death.
3. Respirations are moderately increased, with few râles over the lungs.	3. Respirations from 40 to 60 a minute, with numerous crackling râles and at times increased respiratory murmurs over both lungs.
4. Cough may develop early, but is mild, and usually abates during the second week of the fever.	4. Cough increases gradually throughout the disease, and there may be blood-streaked sputum.
5. Cyanosis is observed only late in the disease, when there is cardiac failure or pulmonary complication.	5. Cyanosis early.
6. Tenderness in the right iliac fossa. Tympanites and from two to six semi-liquid stools daily.	6. Constipation the rule.
7. Widal serum-reaction positive after the first week.	7. Serum-reaction negative.
8. Characteristic eruption appears over the abdomen about the end of the first week of the fever.	8. Eruption absent.
9. Cultures from the blood show the presence of typhoid bacilli in from 20 to 30 per cent. of all cases.	9. Culture may develop tubercle bacilli if proper culture-medium is employed.
10. Pupils are moderately dilated early during the course of the disease.	10. Pupils less likely to be dilated and tuberculous retinitis common.
11. Feces contain the bacillus of Eberth.	11. Tubercle bacilli often present in the feces.
12. Intestinal hemorrhage and intestinal perforation may complicate typhoid fever.	12. Intestinal hemorrhage and perforation absent.

Malarial Fever.—Typhoid fever is distinguished from malarial infection by an examination of the blood alone. The protozoa of malaria are always present in both the fresh and the stained blood, and their detection indicates the type of infection present. The Widal serum-reaction is also absent in malaria. One seldom finds a case of malaria in which abdominal symptoms and signs resembling those of typhoid fever are also present. The temperature of malaria seldom assumes the form characteristic of typhoid, although in estivo-autumnal types of malarial infection a continued type of fever is common.

Meningitis exhibits hyperesthesia, intolerance of light and of sound, altered reflexes, and rigidity of the neck muscles. Vomiting and constipation are common in both epidemic and tuberculous meningitis. The temperature remains at a lower level, on the average, and is more irregular. The eruption is not continued in character or in time of appearance, and never resembles that of typhoid. The nervous symptoms assume greater prominence early during the course of meningitis, which is in striking contrast to the nervous manifestations of typhoid.

In cases suffering from tuberculous meningitis tubercles may be found upon the choroid.

In either acute or subacute meningitis lumbar puncture will recover fluid containing some bacterium.

The Widal serum-reaction is also negative in both epidemic and tuberculous meningitis. In epidemic meningitis a leukocytosis of from 12,000 to 25,000 is to be expected, whereas in uncomplicated typhoid fever leukopenia (6000 to 2500) obtains. In tuberculous meningitis a differential leukocyte count is likely to show an increase in the percentage of lymphocytes present.

In the meningeal type of typhoid fever lumbar puncture, estimation of the number of leukocytes in a cubic millimeter, and the Widal serum-reaction are essential to the formation of a correct diagnosis.

Dysentery.—During the Spanish-American War typhoid fever was frequently mistaken for dysentery—a fact amply proved by the history of the many soldiers treated in the hospitals of Philadelphia. In dysentery the temperature is, as a rule, moderate and more irregular than is that of typhoid, the stools are more frequent, and the degree of prostration becomes marked early. In typhoid prostration is not profound until the second week of the disease. The Widal reaction for typhoid fever is of great importance in distinguishing between these two conditions, and a serum-reaction, with the bacillus of Shiga, while less commonly employed, is also of service in determining the existence of bacillary dysentery. Absence of the characteristic eruption strongly favors dysentery.

Prognosis and Duration.—These are dependent upon three distinctive conditions: first, the severity of the type of infection; second, whether or not treatment, including nursing, can be carried out systematically in certain individual circumstances; and third, the presence or absence of complications.

A severe type of infection is usually marked by high fever. A temperature of 105° F., if prolonged for more than three days, renders recovery unlikely. A fever of 106° F. generally proves fatal.

Marked nervous symptoms, delirium, etc., are also expressions of a severe type of infection, and make the prognosis guardedly favorable. Relapses tend somewhat to lessen the favorable outlook, but a recrudescence is seldom of serious moment.

When typhoid fever is recognized early and appropriate treatment is established and continued throughout the disease, the mortality-rate is greatly lessened.

Complications of whatever nature render the prognosis less favorable. Bronchopneumonia, lobar pneumonia, nephritis, intestinal hemorrhage, and intestinal perforation, the latter in particular, are of serious prognostic moment; other less serious complications likewise proportionately lessen the probability of recovery. Uncomplicated cases of the ordinary type of the disease go on to recovery in from four to eight weeks.

Complications.—Complications may consist of an exaggeration of a symptom or of symptoms other than those known to the severe type of typhoid fever.

Exaggeration of an Ordinary Symptom as a Complication.—Excessive tympanites, when it embarrasses the action of the heart and respiration, forms a complication of serious moment. By stretching the intestine, tympanites favors intestinal perforation and intestinal hemorrhage—two serious complications. Severe diarrhea greatly depletes the patient, and favors the development of other serious complications.

Gastro-intestinal and Abdominal Complications.—The ulcers of typhoid are rarely found in the esophagus, pharynx, and on the tonsils. Suppuration of the parotid gland, although extremely unusual, is a grave complication.

Cholecystitis is marked by the development of jaundice and other symp-

toms, and this complication may rarely be followed by the formation of gall-stones or by hepatic abscess, both of which conditions are of serious moment.

Rarely, indeed, the greatly enlarged *spleen* may rupture, and shock follow.

Intestinal hemorrhage occurs during the third or fourth week in from 3 to 5 per cent. of all cases, and is fatal in probably 30 per cent. of such victims. The indications of hemorrhage are a sudden fall in temperature, with signs of collapse and the passing of bloody or tarry stools. In profuse hemorrhage death may occur before the blood escapes from the rectum.

Intestinal perforation may occur at any time after the middle of the second week, and has been known to take place after convalescence was apparently well established. In more than 70 per cent. of cases with perforation this accident is accompanied by sudden acute abdominal pain. In many instances collapse is the first symptom of perforation, and in practically all such cases there is an abrupt fall in temperature, which, however, tends to rise again some hours later. Several hours after the acute pain due to perforation there are abdominal rigidity and tenderness, the result of the associated peritonitis. Leukocytosis speedily develops. In those cases in which tympanites and delirium are prominent features, it is quite difficult to detect perforation until peritonitis has developed. The former exceedingly high death-rate from this accident has been somewhat reduced by timely surgical intervention.

Respiratory Complications.—The so-called "laryngeal typhoid" seldom occurs until after the third week of the disease. When associated with marked edema of the glottis and of the adjacent structures, the prognosis is decidedly unfavorable, and tracheotomy or intubation may be necessary.

Lobar pneumonia occurs quite commonly during the second or third week. Dullness, weakened voice-sounds, and moderately fine moist râles at the bases during inspiration, are found not infrequently in the later stages, and the respirations are quickened. Bronchitis, which is also present, may extend to the smaller vesicles of the lung, and result in bronchopneumonia. Occasionally, portions of the lung become atelectatic. Pulmonary abscess, gangrene, and pulmonary hemorrhage are among the rarest of complications. *Pleurisy* with effusion is occasionally seen. Ulcers of the nose occur, and perforation of the nasal septum may follow.

Nervous Complications.—Paralyses, while extremely uncommon, may develop during convalescence, and appear in the form of paraplegia, four such cases having come under our notice. Hemiplegia and monoplegia are less common. There may be only temporary loss of control of the sphincters. Neuritis may be either local or multiple, and is characterized by pain, tenderness, foot-drop, and wrist-drop. Myelitis is unusually rare. Any or all of the aforementioned nervous complications renders the prognosis decidedly unfavorable.

The so-called *typhoid spine* develops during convalescence, and is ushered in by extreme tenderness and pain upon movement of the limbs. There is no additional fever, and the actual pathology is doubtful.

Meningeal symptoms of typhoid may be prominent in the so-called "nervous type" of the disease, but true *meningitis* may develop as a complication, at which time practically all the characteristic symptoms of meningitis (p. 838) are displayed—*e. g.*, rigidity of the muscles of the neck, photophobia, hyperesthesia, strabismus, and an irregular temperature. Meningitis, when present, may be due to typhoid infection of the meninges or to a mixed infection. The fluid obtained by lumbar puncture will be found to contain typhoid bacilli and also the pneumococcus and the meningococcus. Meningitis complicating typhoid fever is an extremely fatal condition.

Circulatory Complications.—A variable degree of *myocarditis* is present during the later stage of every severe case of typhoid. *Acute endocarditis* occasionally develops, and manifests itself by an infrequency in the heart action and the presence of a distinct endocardial murmur. *Pericarditis*, although less common than endocarditis, may also cause unusual irritability of the heart.

Thrombosis of one of the femoral veins gives rise to swelling and tenderness of the corresponding foot as the first symptom. The left vein is oftenest involved, but both femorals, and even the axillary veins, may be the seat of such complications. Thrombosis of the spermatic vein and orchitis, although extremely rare, deserves mention. Embolism of the femoral artery is still less common than are complications of the veins. Embolus of the brain and localized paralyses have been reported.

Renal Complications.—Acute nephritis may develop early during the course of the fever (*nephrotyphoid*), but it is more likely to complicate typhoid at the height of the disease or during convalescence. Moderate albuminuria, however, is *not* to be regarded as a complication. Hemoglobinuria and hematuria occur in cases displaying profound nervous symptoms. The presence in the urine of a large amount of albumin and of numerous granular and bloody casts renders the prognosis unfavorable. Pyelitis and cystitis may occasionally complicate typhoid fever.

Ear Complications.—Otitis media has been seen to develop at any time during the course of the fever, but is most likely to appear at the fastigium. Otitis media is frequently overlooked by the clinician until there is a discharge from the ear. The acute pain of otitis media is of no value during the development of this complication, since the general stupor prevents the patient from appreciating this symptom. The temperature serves as the best guide to acute inflammation of the middle ear, and an increase of from one to two degrees takes place with the development of the auditory complication, and continues until rupture of the membrana tympani occurs.

Eye Complications.—Conjunctivitis, iritis, and corneal ulcer are among the rarest of complications. Oculomotor paralysis has been reported.

Cutaneous Complications.—Multiple abscesses of the skin form an occasional complication in those cases displaying the so-called "septicemia of typhoid." Bed-sores are frequently responsible for a rise of temperature of one or two degrees for days or even weeks after other symptoms have subsided. They affect by preference the back, buttocks, and heels.

Complications Affecting the Bony Structures.—Necrosis of the tibia has been reported, and is more likely to occur in the typhoid of children. Acute arthritis may complicate typhoid at any stage of the fever, but is oftenest detected during the third or fourth weeks. As in bed-sores, so in bone and joint complications, the convalescence is protracted. Periostitis with abscess formation is fairly common.

PARATYPHOID FEVER.

Definition.—This term is applied to an infection with an organism of the colon-typhoid group, which resembles bacillus typhosus, but is not identical with it.

Predisposing and Exciting Factors.—The conditions that predispose to paratyphoid are practically the same as those given for typhoid fever. The **exciting cause** is a bacillus belonging to the colon group (Bacillus paratyphosus enteritidis). Melkish has studied this bacillus in connection with a disease known to rats.

Symptomatology.—Generally speaking, the period of incubation is

short, and the onset is more abrupt than is that of typhoid fever. At the beginning the headache is more intense than in typhoid.

Nervous symptoms develop early; thus, during the first week of fever it is not uncommon to find the different types of delirium pronounced. (See Nervous Manifestations of Typhoid, p. 749.)

Gastro-intestinal Features.—Constipation usually obtains throughout the febrile period, although diarrhea is not unknown.

Thermic Features.—By the third or fourth day the temperature is likely to rise to 103° or 104° F. Such rise does not display the peculiar, step-like ascent characteristic of true typhoid. The duration of the temperature, as also its mode of decline, is uncertain, although in the majority of instances the temperature falls by rapid lysis. Convalescence is seldom interrupted, and therefore the temperature, after it once reaches the normal, is likely to remain at or near that level.

Physical Signs.—Inspection.—The face is flushed, the expression is dull, the tongue is heavily coated, and the lips are dry and fissured. A rose-colored eruption resembling that of typhoid fever may be seen upon the abdomen and trunk. In certain cases the eruption very closely simulates that of typhoid, both in appearance and in its mode of recurring in successive crops. The rule, however, is to have an atypical typhoid eruption.

Palpation.—There is but slight abdominal distention, and the abdominal walls are not tense. Splenic enlargement is a constant feature, and the degree of such enlargement varies widely in different cases.

Clinical Course and Duration.—In the majority of cases the temperature reaches the normal earlier than it does in true typhoid, and recovery is less likely to be protracted in paratyphoid. There are exceptional cases, however, in which convalescence is delayed for a prolonged period, such delay being due to some complicating condition (bronchopneumonia, melena).

Laboratory Diagnosis.—The differentiation of paratyphoid fever from typhoid fever can be made only by laboratory methods. A case presenting the symptoms of typhoid fever with a negative agglutination test with bacillus typhosus in all probability belongs to the group of paratyphoid fever. Agglutination tests with paratyphoid or paracolon organisms will sometimes give positive results. In other cases cultural methods must be resorted to, inoculations being made of the blood, the feces, or the urine, the last being the most convenient, and the characteristics of the organism recovered being carefully determined.

TYPHUS FEVER

(SHIP FEVER; CAMP FEVER; JAIL FEVER).

Predisposing and Exciting Factors.—Age.—Typhus is a disease of childhood and early adult life, although it may appear at any age.

Sex is without influence where both males and females are alike exposed.

Season appears to figure prominently, epidemics being more common during the winter months. Unsanitary surroundings usually prevail where there is an outbreak of typhus, consequently filth, poverty, famine, and overcrowding serve as potent predisposing factors. Typhus is also commonly encountered among the occupants of institutions, jails, and prisons, and it likewise affects the crews of ships, especially when they have been at sea for a prolonged period.

Typhus does not occur spontaneously in a community where the disease has never before been known, but is always transported to such locality, yet

the exact mode of conveyance of the infection is not definitely understood. In certain selected epidemics the disease appears to have been transported by means of clothing, whereas in other cases mere association in the room with those known to be suffering from the disease serves as the only possible means of dissemination. The medical profession has not as yet accepted any known specific etiologic factor in the production of typhus.

Period of Incubation.—This varies from nine to twelve days, during which period there may be mild prodromal symptoms, *e. g.*, anorexia and malaise, but in the average case prodromes are not distinct.

Principal Complaint.—**Preëruptive Stage.**—The initial symptom

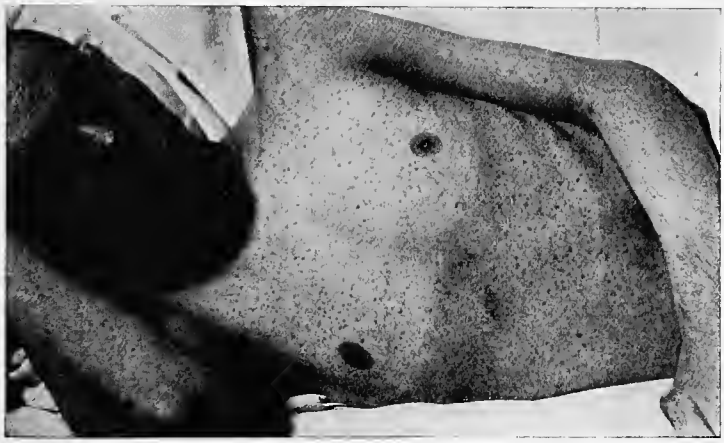


Fig. 280.—Petechial Eruption of Typhus Fever; Patient Recovered (Welch and Schamberg).

may be a *series of chills* or one severe *rigor*, following which the patient complains of *headache, muscular pains, vertigo, tinnitus aurium*, and *profound weakness*. An annoying bronchial *cough* with slight expectoration may be present. *Anorexia* develops early, and there is an inordinate thirst. *Vomiting* is present, and may be an annoying symptom.

Nervous Symptoms.—These appear early, and may even be present with the subsidence of the chill. Delirium is, as a rule, at first mild, but later active, and may terminate in stupor and eventually in coma.

Eruptive Stage.—From the third to the fifth day quite a *characteristic eruption* develops, and with its appearance there is an appreciable decline in the fever. The rash appears first upon the trunk, and then extends over the entire body. It is seldom seen on the face. Two or three days later the crimson red maculæ become petechial, and the skin presents a spotted appearance on account of the coalescence of the isolated patches. Some of the maculæ do not change their hue, but remain as rose-red spots for several days—a feature commonly seen in mild forms of typhus. Again, in the milder form of the disease certain of the rose-spots may disappear upon pressure or on stretching of the skin. The true petechial patches, on the other hand, are unaltered by such manipulation. A variable degree of hyperemia may involve those portions of the skin between the petechiæ.

Nervous Symptoms.—As in the former stage, the nervous symptoms are intense during the stage of eruption, and maniacal delirium is the rule in well-marked cases, and is frequently followed by the various types of coma,

among which coma vigil is common. Even in the milder forms tremor, subsultus tendinum, and carphologia are prone to occur. Owing to the patient's delirious state he persists in resting upon his back. Unusually mild cases of typhus are seen in which both the eruption and the nervous symptoms are not well marked, and the typical clinical picture may not be present at any time during the disease. Multiple neuritis with paralysis of the extensor muscles is an occasional complication, and meningitis is rarely encountered.

Thermic Features.—Quickly following the onset the temperature will be

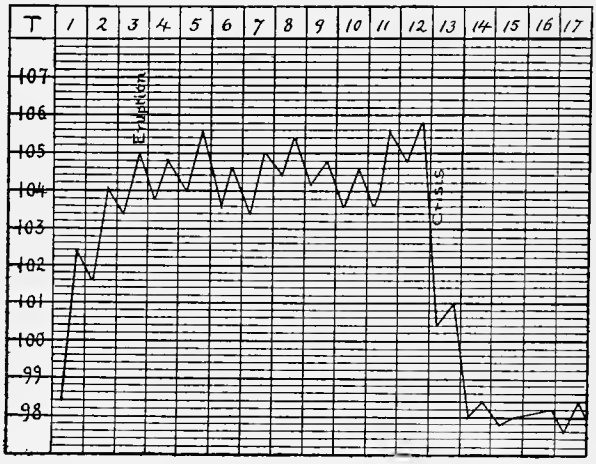


Fig. 281.—Typhus Fever Curve (Register).

found to rise to from 102° to 105° F., reaching the latter height by the second or third day; during the preëruptive stage the fever is of the continued type.

During the eruptive stage, which begins between the third and fifth days, there is not likely to be an appreciable decline in the fever, but between the fourteenth and seventeenth days of the disease, in favorable cases, the fever terminates by crisis. (See chart, Fig. 281.) Immediately before the crisis occurs there is likely to be a sudden rise in the temperature, and sometimes the crisis is interrupted by slight exacerbations. In favorable cases there is a decided amelioration of the symptoms following the crisis.

Physical Signs.—Inspection.—The face is flushed at first, and when coma is well developed, the expression may be dull, anxious, or staring. The cheeks are flushed, the conjunctivæ are congested, the face is expressionless, and the pupils are markedly contracted. From the third to the fifth days the characteristic eruption will appear. (See Eruptive Stage.) The tongue is thickly coated with a yellowish-white fur.

Palpation.—In the majority of cases the spleen is appreciably enlarged, and may even be felt below the costal margin. From the onset the *pulse* shows decided acceleration, and during the eruptive stage the beats will be found to number between 120 and 140 a minute. In those cases in which the nervous manifestations are prominent the pulse may be even more rapid, and is always weak, irregular, and shows a tendency toward dicrotism. There is tenderness over the shin bones and over various localized areas of the subcutaneous tissue. Embolic abscesses may also be present. Tenderness

along the course of the nerve-trunks is a rare finding, and indicates the existence of neuritis. Gangrenous processes of the extremities may also follow extensive neuritis.

Percussion may show the area of splenic and hepatic dullness to be moderately increased.

Auscultation.—The heart-sounds are rapid, and in severe cases the first sound becomes greatly enfeebled with the progress of the disease. Both fine and coarse râles are heard over both lungs, and hypostatic congestion at the bases may be responsible for increased bronchial sounds over this area. Since bronchopneumonia is so commonly seen during the course of typhus, it is important that a physical examination of the lungs be made daily during the entire febrile period.

Laboratory Diagnosis.—The *urine* is scanty, of high specific gravity, high color, and may contain a moderate amount of albumin. In severe types of infection nephritis develops as a complication, and red blood-cells, leukocytes, casts, and albumin are present in the much-concentrated urine.

The vomiting of blood is an occasional symptom, and the spitting of blood that accumulates about the teeth as the result of ulcers of the buccal mucous membrane is an occasional feature.

Illustrative Case.—B. B., male; sailor by occupation, and native of Norway, landed at Philadelphia in 1897 with a history of having been sick for three days. He was removed to the Philadelphia Hospital, where he came under our care for thirty-six hours, during which time—the fourth day of his illness—he developed a typical petechial eruption. Slight cough and the general evidences of bronchitis were present. The temperature ranged between 102° and 103° F. There was complete anorexia. Owing to the patient's inability to speak English, no satisfactory history of a chill was obtained. The following day he was removed to the Municipal Hospital, and from this time nervous symptoms, beginning with maniacal delirium, were well marked. The expression soon became greatly dulled, and the once flushed cheeks assumed a dusky hue. The condition terminated in coma, and continued until death, which occurred on the tenth day of the illness. *Autopsy* showed nothing of special pathologic importance beyond hyperplasia of the abdominal lymph-nodes and dilatation of the heart.

Summary of Diagnosis.—The presence of an epidemic or a clear history that the patient has recently dwelt in a district in which typhus fever prevailed is of great clinical significance. A further knowledge of residence amid unhygienic surroundings (see Predisposing and Exciting Factors, p. 757) is also to be carefully weighed in all diseases in which there is a petechial eruption. Prior to the development of the eruption the diagnosis is, as a rule, doubtful, but the sudden onset with chill, the rapid rise in temperature, which remains of the continued type, together with fairly well-marked nervous symptoms and the presence of an eruption by the fifth day, makes the diagnosis positive.

Differential Diagnosis.—It may become necessary to distinguish between typhus and **epidemic cerebrospinal meningitis.** The latter condition differs from the former in that the headache is usually more intense, there is rigidity of the muscles of the neck, with some retraction of the head, hyperesthesia, intolerance of light and of sound, strabismus, and a tendency to convulsions. Lumbar puncture and a bacteriologic study of the cerebrospinal fluid also give positive results in cerebrospinal meningitis. (See Meningitis, p. 841.)

In those cases of typhus fever in which nephritis is present as a complication it may be necessary to distinguish between complicated typhus and the pure **nephritic condition** with *uremia.* A diagnosis of uremia may be

made largely from the clinical history, headache having lasted over a prolonged period. Uremia rarely develops in those who have been in perfect health, whereas typhus may attack practically any one. In uremia the temperature is seldom, if ever, as high as it is in typhus, and the fever does not fall by crisis. In uremia an analysis of the urine always reveals positive findings. The eruption also serves as a decisive point between typhus with nephritic complications and nephritis.

Malignant measles bears a somewhat close resemblance to typhus fever. In the former the eruption appears first upon the face and then spreads to the trunk and extremities, whereas in typhus the eruption is first seen upon the trunk and may even spread to the extremities, the face being but sparingly affected. Koplik's spots are a precursor of measles and are unknown to typhus.

Typhus fever is distinguished from **typhoid fever** by the abrupt onset and the high temperature by the end of the second day; the termination of the fever by crisis, together with the presence of a petechial eruption, is usually sufficient to rule out the existence of typhoid (see p. 753) and to establish that of typhus fever.

Clinical Course.—This will be found to vary greatly in individual cases with the severity of the type of infection present. Uncomplicated cases show a tendency to go on to recovery after the fifteenth to the seventeenth day, and convalescence is usually uninterrupted. Mild forms of the disease are not attended with grave symptoms during any part of their course, and in these cases convalescence is often established by from the eighth to the twelfth day. In those countries in which typhus fever is common we find a mortality-rate of from 10 to 20 per cent. This rate is influenced largely by the frequency of complications in a certain epidemic.

Relapses.—Relapses are extremely rare, and in practically all cases one attack establishes immunity for life.

MALTA FEVER

(Mediterranean Fever; Undulant Fever).

Definition.—An acute infectious disease caused by the micrococcus melitensis. No characteristic pathologic lesions have thus far been identified with this disease, although splenic enlargement and enlargement of the mesenteric lymph-nodes have been found.

Clinical Remarks.—Irregular fever, muscular pains, marked prostration, profound sweating, and a tendency to relapses are the clinical characteristics of this disease.

Clinical Varieties.—(1) **Pernicious Malta fever** is a type of the disease that is somewhat unusual in Mediterranean districts, but when it occurs, usually tends toward a fatal termination.

(2) The **undulant type** is characterized by a repetition of exacerbations of fever that develop at irregular intervals.

(3) The **continued type** of Malta fever, in which the febrile period persists for weeks and even months without well-marked intermissions.

Exciting and Predisposing Factors.—Bacteriology.—The micrococcus melitensis is found in the blood and other tissues of those suffering from Malta fever. The spleen particularly contains may of these cocci. Pure cultures of micrococcus melitensis, when introduced into apes, is capable of producing toxic symptoms. European students have also found this micrococcus in the blood, milk, and urine of the goats in infected regions. A residence along the shores of the Mediterranean Sea appears to be the most

potent predisposing factor. The disease has also been found on the shores of the Gulf of Mexico and in the West Indies. It is occasionally transported along the lines of commerce, Musser and Sailer having studied a case in Philadelphia which originated in Porto Rico. The exact mode of infection is thus far somewhat questionable. The majority of investigators believe it is transmitted by using the milk of infected goats. The urine of those affected has been shown to contain the coccus. The theory has been offered that the disease may be transmitted to man through the bites of a certain mosquito.

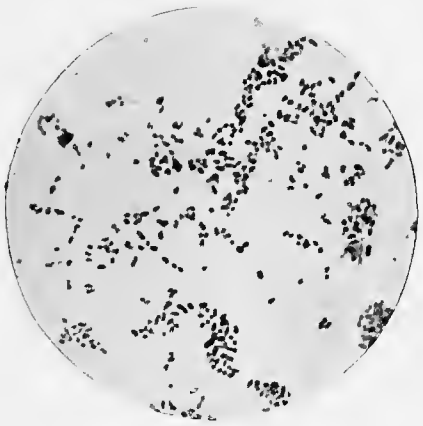


Fig. 282.—Micrococcus Melitensis (Jordan).

Period of Incubation.—The length of the incubation period fluctuates greatly, and varies between that of a few days to twenty or even thirty days.

Principal Complaint.—The symptoms develop gradually, and, indeed, the early stage of Malta fever resembles closely that of beginning typhoid. There are headache, malaise, moderate fever, complete anorexia, occasionally slight chilly sensations, and mild attacks of shivering. Epistaxis may be an early symptom. There is, as a rule, well-marked constipation, and the stools may be streaked with blood. Diarrhea may occur in those cases that show marked prostration.

Relapses are frequent, and, as a rule, last for from five to six weeks. The afebrile periods last for one or two weeks, during which the patient enjoys fair health. In each attack rheumatic pains may be sufficiently severe to prevent movement of any kind. Following the first relapse the condition may go on to recovery, or within a period of one or more months there may be another repetition of the febrile exacerbation.

Thermic Features.—The fever is of the remittent type, and persists for one, two, or probably three weeks, when there is an absence of fever for two or more days, the period of apyrexia being followed by a relapse, when the fever continues high (100° to 103° F.), as in the initial paroxysm. In grave cases the temperature may be continuous rather than remittent, and when hyperpyrexia occurs, the outlook is grave. In selected cases the temperature may be decidedly irregular, in which case its diagnostic significance is lost.

Physical Signs.—Inspection.—The expression is somewhat anxious, the movements are sluggish, and when the muscular pains are severe, the patient may remain in one position.

Palpation.—The spleen is tender, and may even be painful upon firm pressure. During the early stage of the disease the pulse resembles that of typhoid fever.

Percussion shows the area of splenic dullness to be enlarged.

Laboratory Diagnosis.—A differential leukocyte count shows the polymorphonuclear elements to be increased. With pure cultures of the micrococcus melitensis the serum from persons suffering from Malta fever will be found to give a typical agglutination reaction. (See Serum-reaction, p. 343.) Blood culture will give a pure growth of the organism.

Summary and Differential Diagnosis.—This is based largely upon the residence of the patient in districts known to be infected with Malta fever. The insidious onset, remittent temperature, and the tendency toward relapses serve to differentiate this condition from typhoid fever, although in the early stage of the disease a differential diagnosis is quite impossible, except upon application of the agglutination test. Polyarthritis and, at least, soreness in the region of the articular surfaces are features that strongly support the existence of Malta fever.

Clinical Course.—The disease is of long duration, the febrile periods alternating with periods of apyrexia for two, three, or four months.

RELAPSING FEVER

(FEBRIS RECURRENS).

Pathologic Definition.—An acute infectious disease caused by Spirochæta recurrentis, and characterized by cloudy swelling of the heart, liver, and kidneys when death occurs during the febrile period. There may be hemorrhagic infarction of the viscera and extravasations into the serous sacs. The spleen is, as a rule, enlarged, but varies greatly in size in different cases. There is hyperplasia of the lymphoid elements of the bone-marrow, and occasionally the viscera, skin, and mucous surfaces are jaundiced.

General Remarks and Parasitology.—In 1873 Obermeier first detected in the blood of man, then suffering from relapsing fever, a peculiar organism which has since been named the Spirochæta recurrentis. Originally, the spirillum of Obermeier was regarded as a true bacterium, but of recent years Schaundinn has classed it among the protozoa.

Clinical Varieties.—Relapsing fever will be found to vary greatly in different individuals, a feature that is probably explained by the peculiar type of infection in a given case; consequently very mild cases occur, that consist of only one or two brief febrile periods.

The so-called **"bilious typhoid"** is the other extreme in the clinical types of relapsing fever. In this variety the symptoms are unusually severe, and the patient is likely to fall into the typhoid state, and, in addition, to develop jaundice, hemorrhage from the stomach and the bowel, and the symptoms of uremia with sudden collapse. Septic and pyemic processes, including the deposit of septic emboli in different portions of the body, may be observed.

Exciting and Predisposing Factors.—Relapsing fever is due to an infection with Spirochæta recurrentis (Spirochæta duttoni). Dutton and Todd found the horse-tick (Ornithodorus moubata) (Murray) (Fig. 283), to be the intermediate host of the spirilla causing this disease. These observers permitted the horse-tick to bite infected human beings, and subsequently these infected ticks were found capable of transmitting the disease to monkeys. The above observations have been confirmed by Koch, Ross, and others. **Age** serves as quite a prominent predisposing factor, since the majority of those attacked are found to be between the twelfth and the twenty-fifth year. The disease is also more common in males than in females.

Principal Complaint and Symptoms.—Following the period of incubation, the symptoms develop abruptly, with a distinct *rigor*, or, in mild cases, a succession of *mild chills*. Extreme *frontal headache* is constantly complained of, and there are also *pains* in the back, loins, and limbs, and *extreme prostration* and vertigo. The *throat* is often sore, and there may be considerable difficulty in swallowing. *Anorexia* becomes complete early

during the course of the disease, and nausea, vomiting, and inordinate thirst are common. *Constipation* obtains during the prodromal stage.

After the crisis the patient's general vitality is at a low ebb, consequently profuse sweating, menorrhagia, and intestinal hemorrhage may occur. In favorable cases all annoying symptoms disappear with great rapidity after the crisis is passed.

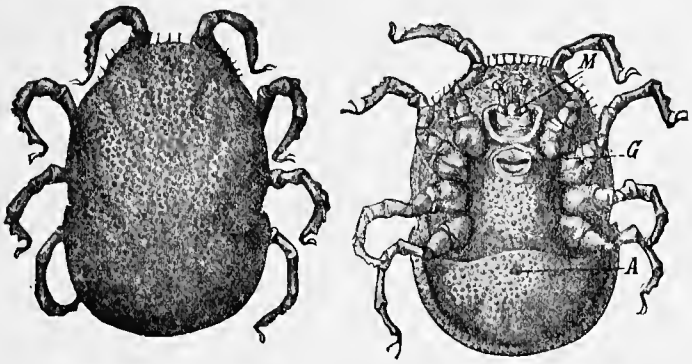


FIG. 283.—ORNITHODORUS MOUBATA. TICK THAT TRANSMITS AFRICAN RELAPSING FEVER. *a*, Viewed from above; *b*, viewed from below (Murray and Doflein).

Nervous Manifestations.—These are, as a rule, mild in character, although headache may persist for several days, during which time the patient is more or less stupefied. Delirium is not usual in uncomplicated cases, although it may set in just prior to the crisis. In the average case the patient may remain conscious throughout the attack.

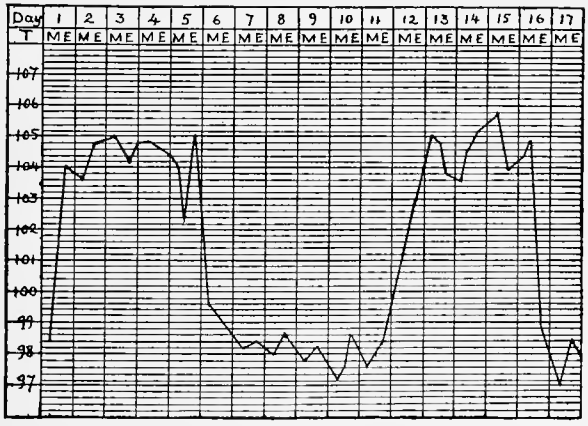


FIG. 284.—RELAPSING FEVER (Register).

After an *afebrile period*, lasting about one week, all the symptoms previously outlined are repeated, but with lessened severity than during the initial paroxysm.

Thermic Features.—The temperature rises somewhat rapidly after the chill, reaching an elevation of 102° to 106° F. by the end of the first or during

the second day. The temperature is of the continued type for a period of about six days, when, just prior to the crisis, the fever may suddenly rise one or two additional degrees. This hyperpyrexia is a common precursor of the crisis, and is soon followed by a rapid fall to, or in many instances below, the normal. (See Chart, Fig. 284.) Each succeeding attack of fever is of shorter duration and milder in character than the preceding attack, and four or five such exacerbations may occur.

Physical Signs.—Inspection.—The *skin* becomes pigmented and dusky in appearance and sooner or later acquires a dirty yellow tint, a condition that is frequently referred to as bronzing. The cheeks are flushed during the febrile period, the eyes appear somewhat sunken, and the face is often beaded with perspiration; indeed, in certain cases sudamina may be an annoying feature. Peculiar cutaneous eruptions are occasionally present, but are in no way characteristic of relapsing fever. Herpes labialis may be a common feature in certain epidemics, whereas in others it is practically absent.

The *tongue* is at first covered with a moist, yellowish fur, but as the fever rises it becomes brown, dry, and fissured, and sordes accumulate about the teeth. Numerous small ulcers may be seen along the margins of the gums and upon the tongue, and congestion of both the tonsillar and the pharyngeal mucous membrane is likely to be present. The frequency of the chest movements may be somewhat increased, and the chest-wall may be seen to pulsate violently as the result of cardiac palpitation.

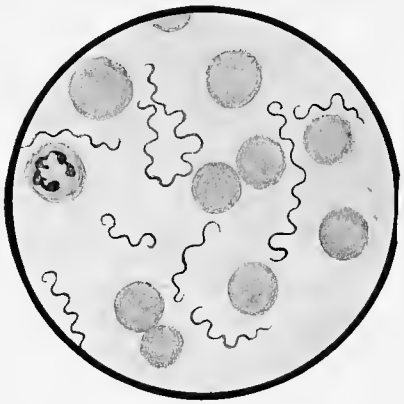


Fig. 285.—Spirochete of Relapsing Fever in Human Blood (× 1000) (Boston).

Palpation.—The pulse is found to be increased in frequency while the temperature is rising, although this increase is seldom in direct proportion to the heightened fever. At first the pulse is full and strong, the beats numbering from 100 to 120 or even 140 a minute. In severe types of infection the pulse may exceed 140 beats a minute, and become weak, irregular, and even dicrotic.

During the febrile paroxysm there is usually tenderness over the epigastrium, and even gentle pressure over the trunk elicits tenderness; certain portions of the cutaneous surface also, as well as certain groups of muscles, may be hyperesthetic. On placing the hand over the precordium, the heart may often be felt to pulsate violently, and moderate excitement may induce an attack of palpitation. Palpation over the region of both the liver and the spleen may show these viscera to be moderately enlarged.

Percussion.—The area of hepatic and of splenic dullness may be moderately increased.

Auscultation.—The sounds of the heart are, as a rule, full and strong, even though the pulse may be 120 to 140. In severe cases an appreciable portion of the muscular element of the first sound of the heart may be missing at the time of the crisis. A systolic murmur, probably hemic in character, is occasionally audible over the heart. The physical signs of bronchitis are commonly present, and lobar pneumonia and hypostatic congestion are among the usual complications.

Laboratory Diagnosis.—The vomitus is, as a rule, greenish in color, although at times it is black, and contains large quantities of bile; rarely do we find red blood-cells.

The *urine* is highly colored, of high specific gravity, and may contain a trace of albumin. When jaundice is associated, which is quite a common occurrence, the urine is rich in bile and displays a heavy yellow froth.

Blood obtained from the finger-tip or from the ear during the febrile period will be found to contain the spirochete. (See Fig. 285.) The spirochæta may be readily studied in both the fresh blood and in stained preparations.

Summary of Diagnosis.—A history of an epidemic, or of the patient having recently resided in the tropics, together with the sudden onset, the continued type of fever with an abrupt termination on the seventh day, would strongly support the existence of relapsing fever. Again, the characteristic interval separating the febrile periods is known to no other disease. Examination of both the fresh and the stained blood during the febrile period makes the diagnosis positive through the detection of the organism.

Clinical Course.—The duration of relapsing fever depends entirely upon the number of paroxysms the patient may have; hence should there be a single relapse, the febrile period will continue for but from twelve to sixteen days. In complicated cases, and particularly when lung and kidney complications are present, convalescence may be greatly delayed and continue over several weeks. A fatal termination is seldom witnessed unless one or more serious complications are present.

YELLOW FEVER.

Pathologic Definition.—An acute infectious disease (epidemic and endemic), transmitted to man through the bites of infected mosquitos. The liver is anemic, although when death occurs early, the organ may be congested. It is pale yellow in color, and at times may have an orange hue. Punctate extravasations make the organ appear mottled. Fatty degeneration of the hepatic cells is the rule, although sections made from certain portions of the organ may be practically normal. The gastro-intestinal mucous membrane is the site of an acute catarrhal inflammation. The serous surface may display punctate hemorrhages, and hemorrhagic infarctions are frequently present in the solid viscera. The kidneys show the pathologic changes characteristic of acute diffuse nephritis (see p. 661) and the heart muscle is the seat of fatty degeneration. There is extensive fatty degeneration of the walls of the blood-vessels, and the red blood-cells are often disintegrated and are found to have given up their hemoglobin.

Clinical Varieties.—Several clinical varieties of yellow fever have been described, and each of these is characterized by the presence of one or more of its prominent features. Finley's classification of the disease permits of the following distinctive clinical types: (1) **Acclimation fever** or **non-albuminuric yellow fever;** (2) **plain albuminuric yellow fever;** and (3) **melano-albuminuric yellow fever,** characterized by the presence of blood or "black vomit" in the stomach or intestines.

Predisposing and Exciting Factors.—Among the predisposing factors, **season** figures most prominently, yellow fever prevailing chiefly during the summer months; epidemics are, as a rule, arrested by the approach of frost.

Age is also an important factor, since children are more susceptible to

the disease than are adults, because in a yellow fever district the adults are immune on account of an attack of the disease during childhood.

Sex has a feeble influence, males being more often attacked.

Race.—The disease is more common in white than in colored individuals. One attack establishes permanent immunity, so that the natives of a district in which yellow fever is endemic are less likely to develop the disease than a newcomer. The suggestion has been made that the native children develop yellow fever early in life, and consequently have established an immunity before puberty.

Exposure to Mosquitos.—In 1881 C. J. Finley pointed out that this disease was transmitted through the agency of the mosquito. It remained for the commission of the U. S. Army, made up of Drs. Reed, Carroll, Lazear, and Agramonte to furnish incontestable experimental proof that yellow fever is a mosquito-borne affection. These observers have shown that the *Stegomyia calopus* is probably the only carrier of the infecting agent. Twelve days after biting a yellow fever patient the bite of the mosquito will infect a non-immune person. The insect is capable of infecting man for a period of several weeks. There is some evidence to show that the mosquito (once infected) is capable of transmitting the parasite for the remainder of its life. The patient's blood infects the mosquito only during the first three days of the disease. The clothing, vomitus, urine, and feces are believed to be non-infectious.

The *Stegomyia calopus* has been found as far north as Philadelphia, and southward to the Rio de la Plata river; it is prevalent in Cuba. The larvæ develop only in artificial collections of comparatively clean water, and this mosquito seldom breeds far outside a city's limits. Yellow feevr is thus a domiciliary infection. Both insects and larvæ are killed by freezing. They inflict their bites principally late in the afternoon. They are not capable of long flights. Not all mosquitos that bite a yellow-fever subject become infected. They either fail to secure the parasite, or the parasite does not subsequently develop.

Period of Incubation.—This varies from two to five days, and possibly a longer period may be required. During the stage of incubation headache, languor, and a poor appetite may be present.

Clinical Stages.—Stage of Invasion.—*Principal Complaint.*—Given an average case of yellow fever, the onset is abrupt, being ushered in by a *chill*, which is seldom severe, and practically never prolonged. Following the chill the patient complains of feeling hot, of *headache*, of distressing *pains* in the loins and legs, and later he becomes extremely restless, with some confusion of ideas. *Photophobia* is an early annoying symptom, and vomiting is common. The patient usually complains of a burning sensation and of marked oppression in the region of the epigastrium. The *duration* of the initial symptoms will be found to vary between six and eight hours in ordinary cases, although it may continue for two or three days, and a longer period has been observed. The stage of invasion is often unusually long in mild types of the disease. With the termination of this stage there is an appreciable subsidence of the fever and of all the symptoms and signs presented by the patient.

Nervous Symptoms.—In addition to the restlessness previously mentioned, the patient may manifest well-marked delirium, and maniacal outbreaks occasionally develop.

Thermic Features.—Following the initial chill the temperature rises somewhat abruptly to 103°, 104°, or 105° F. After the temperature has attained

its greatest height, it declines by lysis, showing slight evening exacerbations and morning remissions.

Physical Signs.—Inspection.—The face is flushed, and soon gives evidence of jaundice, yellow pigmentation of the skin being the most characteristic sign of the disease. The eyes are markedly congested. The tongue may be furred, although this is by no means a constant finding.

Palpation.—Abdominal tenderness is present, and firm pressure may elicit pain over the epigastrium. The relation of the pulse-rate to the temperature is an important diagnostic feature of yellow fever. This relation is seen uniformly in no other disease. As the temperature rises the pulse falls, so that it is frequently observed that a patient with a temperature of 104° F. or over will have a pulse-rate of 80 or lower. As convalescence progresses the pulse may fall to 50 a minute or below. In fatal cases there is either a progressive rise in the pulse-rate over several days, or a sudden rise for a few hours before death.

Stage of Remission.—Following immediately upon the termination of the initial stage, convalescence may begin, and go on to recovery without interruption. In the majority of cases, during this stage the patient displays certain symptoms and signs of impaired health, *e. g.*, prostration, jaundice, and choluria, all of which are likely to continue during the first twenty-four hours, at which time, unless convalescence is established, another more serious exacerbation—the stage of secondary fever—begins.

Stage of Collapse (Secondary Fever).—Here the patient's general condition is that of extreme prostration, the various signs of collapse being manifest.

Nervous Symptoms.—Grave nervous symptoms, convulsions, coma, and the general clinical picture of uremia may be seen during this stage of the disease, and when present, are usually attributed to an associated nephritis.

Physical Signs.—Inspection.—The features are pinched, the skin has a peculiar yellow or bronzed tint, and there may be numerous minute cutaneous hemorrhages. The expression is dull, the tongue is dry, brown, and often blackish in appearance; but in some cases the surface of the tongue may be smooth, bright red, and deeply fissured. The teeth and lips are covered with sordes.

Palpation.—The surface of the body is cold. The pulse becomes weak, rapid, soft, irregular, and compressible. In certain instances in which the degree of infection is unusually severe the pulse may be slow and the beats not exceed 20 to 30 a minute.

Laboratory Diagnosis.—During the initial stage the *vomitus* may be blood-streaked, or contain chocolate-colored particles of blood-clot. Rarely, indeed, does the patient vomit pure blood before the stage of remission.

During the stage of collapse there is likely to be hemorrhage into the stomach, when the blood is ejected with the gastric secretion—the so-called "black vomit." Occasionally pure blood that has been unchanged by the gastric secretion is vomited.

Blood is also expelled by the bowel, and, as a result, the stools are tarry. In severe types of yellow fever hemorrhage from the nasal and uterine mucous membranes is not unusual.

The quantity of *urine* voided is usually decreased, even during the initial stage, and the fluid is of high color and of high specific gravity. During the stage of collapse the quantity excreted will be found to be much smaller than normal. The urine is bile-stained, displays a rich yellow froth, and may con-

tain albumin, red blood-cells, and casts. In those cases complicated by nephritis, anuria may be present.

Summary of Diagnosis.—In formulating a diagnosis during the initial stage of yellow fever the following points should be carefully weighed: The existence of an epidemic, a sudden onset with a mild chill, pain in the back and loins, cephalalgia, slight yellowing of the skin, nausea with the vomiting of bile-stained material, and the characteristic pulse (*i. e.*, a gradual decrease in the rate of frequency while the fever continues to rise); all these strongly favor the existence of yellow fever.

During the third stage the diagnosis is comparatively easy, and is based upon the presence of severe jaundice, black vomit, the high color and diminished quantity of the urine, together with the signs of collapse.

In a mild type of yellow fever the diagnosis is often made with great difficulty, since the febrile period may last but one day.

Differential Diagnosis.—See Differential Diagnosis of Dengue, p. 886.

LOBAR PNEUMONIA

(CROUPOUS OR FIBRINOUS PNEUMONIA; PNEUMONITIS; LUNG FEVER).

Pathologic Definition.—An acute infectious disease, usually excited by the micrococcus lanceolatus, which produces a specific inflammation resulting in consolidation of the lung. This inflammatory process is divided pathologically into three stages: (1) Stage of congestion; (2) stage of red hepatization; and (3) stage of gray hepatization.

(1) **Stage of Congestion.**—Upon opening the chest, the portion of lung involved is seen to be dark red in color and somewhat firm to the feel, although it does crepitate. From the cut surface blood-stained serum oozes, and the capillaries are engorged with blood. Excised pieces of the lung float when placed in water.

Microscopically, the alveolar epithelium is swollen, the capillaries are markedly distended, and the air-cells contain alveolar epithelium, red corpuscles, and a few leukocytes. The smaller bronchi may also contain some exudate. The duration of this stage is ordinarily from twelve to twenty-four hours.

(2) **Stage of Red Hepatization.**—Here the involved area of the lung is solid to the feel, resembling in this respect liver tissue (Fig. 286). Incised portions of the diseased organ do not crepitate and sink when placed in water. The cut surface of the lung is reddish-brown or of a mahogany color, and its surface is dry and somewhat mottled. The diseased part of the lung is larger than normal, and when the consolidation extends to the surface of the organ, indentations corresponding to the ribs are seen. During this stage the diseased lung is airless, nor can it be inflated from a bronchus. Thin slices of the diseased organ break readily, and the broken surface is more or less irregular and finely granular in character; by scraping the back of a knife over this irregular surface, minute plugs, composed of the inflammatory exudate, are dislodged from the alveoli. The dry exudate occupying the air-cells soon softens, and in such instances a viscid fluid flows from the cut surface of the organ.

The visceral pleura overlying the affected lung is covered with a fibrinous exudate. Effusion into the pleural sac is not unusual.

Microscopically, the air-spaces are found to be filled by a fibrinous exudate which contains red blood-corpuscles, leukocytes, and alveolar epithelial

cells. At times the interlobular connective tissue shows infiltration. The pneumococcus is invariably present, and staphylococci and streptococci may also be seen.

(3) **Stage of Gray Hepatization.**—With the beginning of this stage the fibrinous exudate loses its mahogany color and becomes grayish or granite-like in appearance (Fig. 287). There is now extensive fatty and granular degeneration, with softening of the inflammatory exudate, and from the cut surface of the diseased lung a yellowish-white, and at times almost purulent, frothy liquid exudes. The pleura overlying the diseased lung is covered with fibrinous exudate.

Microscopically, the air-cells are found to be almost entirely filled with leukocytes, which is in striking contrast to the second stage of the disease, in which they contain red blood-cells, white blood-cells, and fibrin.

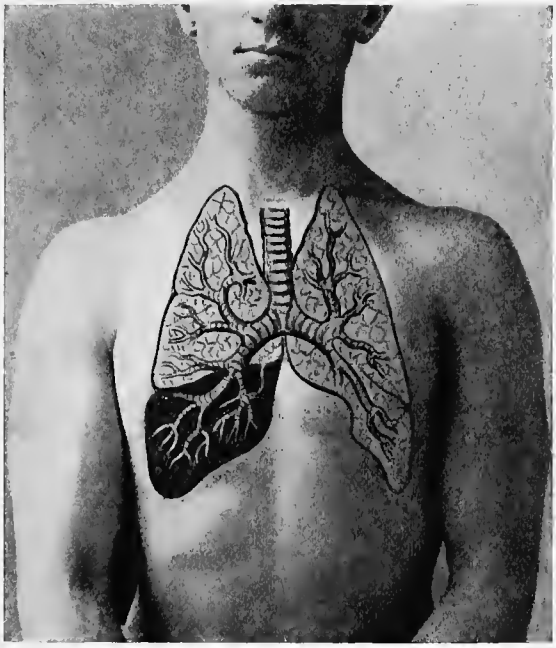


Fig. 286.—Acute Lobar Pneumonia (Second Stage).

In unfavorable cases there may be purulent infiltration of the lung tissue with extensive necrosis, or, as is occasionally seen, an abscess may develop. Resolution may be delayed, and the pulmonary condition remain unaltered for a variable time. Fibroid induration (see Chronic Interstitial Pneumonia, p. 113) may also be present in some cases. Certain pneumonic conditions in which resolution is delayed may be found later to be of a tuberculous nature. Pericarditis is present in a small proportion of cases, but endocarditis is more common.

Clinical Varieties.—(*a*) The **usual type** of the disease will be described in detail.

(*b*) **Typhoid pneumonia** is a serious form of the disease in which typhoid symptoms are present. It has nothing to do with typhoid fever. It often

complicates low fevers, septicemia, diabetes, and chronic nephritis, and is the variety seen in alcoholic subjects and persons of low resisting power. The *onset* is gradual, and the *physical signs* may be somewhat ill defined, but the general features are often more or less characteristic.

Prostration is extreme, and there are often *delirium* and *stupor*. The *temperature* is not characteristic, and the *respirations* and *pulse* are, as a rule, much increased. The skin is dry and may be jaundiced. The tongue is dry and brown, and gastric irritation with vomiting is fairly common. The sputum is blood-streaked, and in some cases almost clear blood is expectorated. The *spleen* is generally enlarged.

(*c*) **Latent Pneumonia.**—In this class are included the *central pneumonias*. The leading clinical feature is the detection of many *pneumococci*

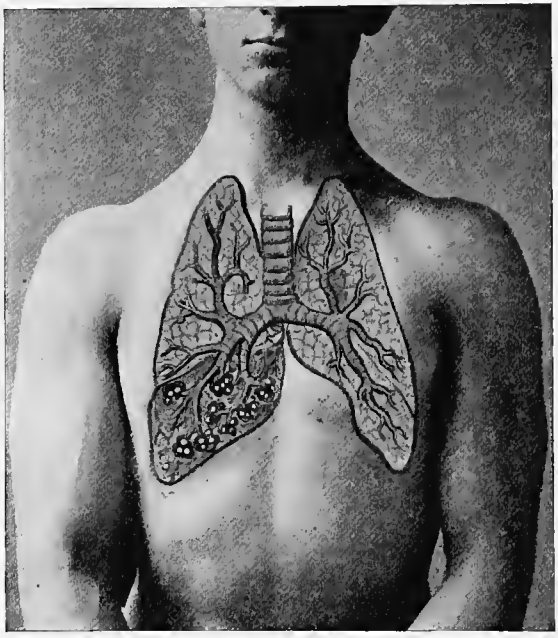


Fig. 287.—Acute Lobar Pneumonia (Third Stage).

in the sputum. If the lung was emphysematous prior to the development of pneumonia, the characteristic *physical signs* of pneumonia, *e. g.*, dullness and bronchial breathing, may be absent throughout the entire course of the disease. Evidences of consolidation, when present, depend upon the extension of the inflammatory process to the surface of the lung.

(*d*) **Migratory Pneumonia.**—In this variety the specific inflammation extends to other parts of the lungs. Such extension may prevent the occurrence of the usual crisis, and often causes an exacerbation of the general pneumonic features. We have frequently studied at autopsy cases in which the pneumonic process showed three distinct stages of development in the same lung.

(*e*) **Epidemic Pneumonia.**—This is often of the malignant type, and the symptoms display noticeable variations, dependent upon the etiologic

factors; they may vary, too, in different epidemics. The pneumonias seen with *epidemic influenza* are complicated with or preceded by an attack of general bronchitis. Owing to failure of the heart, pulmonary congestion is likely to occur, and the *physical signs* are, as a rule, indefinite.

The so-called "*streptococcus pneumonia*," likewise seen to follow influenza, is frequently referred to as *serous pneumonia*. The *physical signs* often resemble those of bronchopneumonia, although large areas of consolidation may sometimes be detected. The sputum may be mucopurulent throughout, or it may be blood-streaked late in the disease. The termination of this type varies greatly in different cases, the fever falling at times by *lysis*. *House* and *institution epidemics* are quite common, and have been referred to under the Etiologic Factors of Pneumonia.

(*f*) **Senile Pneumonia.**—In the *aged* the initial chill is often absent or replaced by attacks of chilliness. Nausea and vomiting may be present; *prostration* is profound early, and the *fever* is low and irregular in type. *Cough*, *expectoration*, and *pain* may be absent, and the nervous symptoms may be mild or, at times, severe. The *physical signs* are not characteristic, although in selected cases dullness and exaggerated breathing (possibly bronchial) may be detected. This type of pneumonia is highly fatal.

(*g*) **Pneumonia of Children.**—Here the onset is often marked by a convulsion, and cerebral symptoms (delirium, stupor, coma) may appear early. The apices are more frequently attacked. *Pain* may be reflected over the abdomen, and when the right lung is involved the pain may be in the region of the gall-bladder or of the appendix. The *sputum* is not characteristic.

(*h*) **"Abortive pneumonias"** last only from twenty-four to forty-eight hours. The *general features* are rigor, high fever, and defervescence by crisis, with profuse sweating. The *sputum* is rarely characteristic, and the physical signs are variable; typical tubular breathing is rare, whereas râles and pleural involvement are common.

(*i*) **Bilious Pneumonia** (**"Malarial Pneumonia"**).—In pneumonia occurring in malarial subjects the initial chill is prolonged, and the *fever* is frequently remittent. *Jaundice* and *tympanites* are common features.

(*j*) **Terminal Pneumonia.**—Many cases of pneumonia are diagnosticated only at autopsy. These arise in advanced cases of chronic pulmonary tuberculosis, organic heart disease, chronic nephritis, diabetes, and other chronic maladies. The only features to call attention to pneumonia in such cases are slight elevation of temperature and moderate increase in the number of respirations. *Physical signs* may be absent in this class of cases.

(*k*) **Ether Pneumonia.**—"Opinions are divided as to the frequency of occurrence of lobar pneumonia after ether narcosis. The aggregate number of cases from all sources (57,842) gives a percentage of 0.07." In our statistics, embracing 12,842 cases, giving a percentage of 0.23, we have called special attention to certain etiologic factors in ether pneumonia as follows:

The *principal causes* are: "(*a*) *Season.*—According to my investigations, over 80 per cent. of the cases occur during the winter and spring months. The patient is sometimes carried from a heated operating-theater through a cold corridor to a room or ward with a lower temperature."

"(*b*) 'Catching cold,' or exposure, as may obtain during protracted operations.

"(*c*) Bronchitis, coryza, or other morbid state of the respiratory mucosa at the time of anesthesia.

"(*d*) Dried secretions or incrustations of foreign matter that are loosened by the ether and drawn downward into the lungs.

"(*e*) Abdominal operations give the highest percentage of cases, and my studies show that this may be due to the more protracted etherizations" (Anders).

Certain authors have suggested that ether pneumonia is often due to septic emboli. The *general clinical picture* of ether pneumonia is practically identical with that of catarrhal pneumonia. The physical signs are often indistinct, yet we would advise that a physical examination of the lungs and heart be conducted in every instance in which there is an elevation in temperature following ether anesthesia.

(*l*) **Relapsing Pneumonia.**—This type is rare, although recurrences, as stated under Predisposing and Exciting Factors, are quite common.

Exciting and Predisposing Factors.—Bacteriology.—The micrococcus lanceolatus (pneumococcus) is now generally conceded to be the exciting cause of the disease, and this organism is found in the sputum throughout the entire course of the disease. (See Laboratory Diagnosis, p. 781.) Other bacteria may also be present, and, indeed, the rule is to find more than one bacterium in the sputum of pneumonia. We have studied at postmortem a few cases dead of lobar pneumonia in which the predominant bacterium in the consolidated lung tissue was either the streptococcus or the staphylococcus, and in two such cases studied at the Philadelphia Hospital only streptococci and staphylococci were found in the sputum during life, and only streptococci were found in the lung tissue at autopsy and were developed in cultures from the lung. In this class of cases it cannot be asserted that the streptococcus excited the inflammatory process, for, indeed, the streptococcic or staphylococcic infection may have been secondary.

In pneumonia there is frequently a bacteriemia in consequence of which inflammatory processes are set up by the pneumococcus in certain of the serous sacs, *e. g.*, the pleura, meninges, and the various synovial membranes. Ulcerative endocarditis may also follow the bacteriemia, and, indeed, frequently antedates infection of the serous surfaces.

Among the *predisposing factors* are unsanitary surroundings; thus we find **epidemics** that may be serious in nature, affecting especially the inmates of barracks, tenement-houses, and public institutions. Laboratory experiments on animals have shown that pneumonia is a common disease among young mice, and the fact that mice are frequently found in the kitchens, cupboards, and even upon receptacles in which food is kept may, in a measure, help to explain the occurrence of house epidemics. Rodman reports an epidemic developing in a prison with 735 inmates, of whom 118 developed pneumonia, 25 succumbing to the disease. Epidemic influences are not without effect, and during certain years, and particularly at certain seasons, the disease prevails both in cities and in rural districts.

Season appears to figure somewhat prominently as a predisposing factor, as is shown by the statistics of Anders, who studied the mortality list of the city of Philadelphia, covering the decade from 1894 to 1903: "January, 4210; February, 3717; March, 3490; April, 3039; December, 2860; May, 2238; November, 1936; October, 1269; June, 1165; July, 913; September, 826; August, 800." The period showing the greatest number of cases is sometimes found to correspond with the period displaying the greatest variations in temperature and humidity. Seitz, of Munich, collected 5905 cases, and of these, 36.8 per cent. developed during the spring; 32 per cent. in winter; 15.7 per cent. in autumn, and 15.3 per cent. during the summer months.

Age and Sex.—Lobar pneumonia is somewhat common at all decades of life, yet during the first and second years it is comparatively infrequent. During the third and fourth decades and after the sixtieth year it is unusually common. Males are attacked more often than are females.

Traumatism to the chest may be followed by pneumonia, and *unhygienic surroundings* favor the development of the disease.

Pneumonia Complicating Other Maladies.—In a person whose resisting power to infections is lowered by the excessive use of alcohol, by exposure, by recent acute disease, or by present chronic disease, such as nephritis, cardiac disease, diabetes, carcinoma, and the anemias, pneumonia is especially likely to occur and to result fatally. A fatal attack of pneumonia occurring during the course of one of the above-mentioned chronic diseases is known as a *terminal pneumonia.* A *prior attack* of pneumonia appears to render the patient especially susceptible to the disease, so that repeated attacks are quite common.

Influenza.—According to the statistics of Wells, pneumonia has steadily increased during the last century, and the disease has been unusually common during the last fifteen years, a fact that has been attributed by some clinicians to the effect of attacks of influenza upon the respiratory tract.

Principal Complaint.—In those cases in which pneumonia does not develop during the course of another malady the clinical history is in itself quite characteristic, but if the disease develops in conjunction with either a chronic or another acute infection, many of the leading features may be masked by the preëxisting disease; consequently in this particular class of cases the physical signs may furnish the first evidence of the nature of the complaint.

Prodromal Symptoms.—These may not be absent, but when present, consist of slight indisposition, which lasts for a day or so. *Cough, anorexia, oppression,* profuse sweating on exertion (six to twelve hours before initial chill), and slight discomfort in the chest may, in exceptional cases, be present. In those displaying distinct prodromes the disease may not develop so abruptly as usual, and there may be symptoms referable to the intestinal tract, as one or more liberal bowel movements.

Invasion.—In the typical cases this is *abrupt,* and is characterized by a distinct chill, lasting for from one-half to one hour. The patient always complains of feeling extremely ill during the rigor, and following the chill high fever almost immediately supervenes. *Oppression* is more or less pronounced, and headache is a common complaint. When seen at this period, the patient is always restless, appears to be greatly disturbed, and his expression and general attitude call for immediate relief.

Within the course of a few hours, and probably by the time the chill has terminated, the patient complains of intense *stabbing pain* in the side, and this is especially severe if he attempts to inspire deeply. *Cough* is an annoying symptom, and aggravates the pain. All these features of pneumonia may develop so rapidly that by the time the physician reaches the patient's side, slightly *blood-streaked expectoration* will have accompanied the cough for an hour or more.

Following the chill the patient develops intense thirst; there is no desire for food, and *vomiting* often occurs as the result of an attempt to take nourishment. Constipation is the rule, although diarrhea occurs in certain cases.

Leading Symptoms and Signs in Detail.—Pain develops within a few hours after the chill, except in those especial cases in which the pneumonic process is centrally located and does not come in relation with the

visceral pleura. The pain continues for a period of two, three, or more days, is stabbing in character, and is usually situated immediately below the nipple or in the axilla of the affected side. Pain is aggravated by deep inspiration, and may be reflected over the upper portion of the abdomen; in selected cases it will extend well into the flanks. *Cough* also increases the pain, and during the act of coughing the patient usually inclines toward the affected side, and frequently grasps that side of the chest to prevent chest movements. Pain is dependent entirely upon involvement of the pleura, and, as previously stated, in pneumonic processes that are centrally located, and in the pneumonia of the aged, pain may be absent during the greater part of the disease. Pain is also alleviated by the patient lying upon the affected side, although when the disease is well advanced and pain but moderate or absent, the patient commonly assumes the supine position.

Cough.—Cough, in a measure, is characteristic, frequent, short, dry, and to some extent under the control of the patient. In certain cases, as in pneumonia of the aged, of alcoholics, and when the pneumonic process does not cause inflammation of the pleuræ, cough may be absent. Following the chill cough is, as a rule, accompanied by slight, viscid, and extremely *tenacious sputum*, which is streaked with blood. (See Laboratory Diagnosis, Rusty Sputum, p. 781.) In sthenic cases the cough is likely to be harsh, and following each cough there is a distinct expiratory grunt that may be heard at some distance from the patient.

Respiratory Phenomena.—See Physical Signs, p. 778.

Nervous Phenomena.—*Headache* develops early,—often during the chill,—and may be persistent for several days. In children lobar pneumonia may be ushered in by a convulsion, and less often by a series of convulsions.

Delirium in its various forms is common at the height of the disease. In exceptional cases acute mania may develop, and this form of delirium may be continued for several days. In mild cases delirium may be absent during the greater portion of the twenty-four hours, and present only during the night. Patients who have been addicted to the excessive use of alcoholic stimulants are likely to develop delirium tremens. Special attention has been called to the "'walking pneumonia' seen in alcoholics, in which the patient goes about until excitement gives way to a coma that deepens into death" (Anders).

In the so-called "cerebral pneumonia" the nervous symptoms are unusually prominent, and in this respect the disease may simulate acute cerebrospinal meningitis. This type of the disease is characterized by high fever, unless it develops in the aged, when the fever may be moderate. Many writers point to the fact that apical pneumonia is likely to assume the cerebral type. It must be remembered that during the course of a pneumonic process in children the nervous symptoms are conspicuous. When the pneumonic process involves a portion of both lungs, cerebral symptoms are usually prominent, yet we have seen cases in which they were absent.

Gastro-intestinal Symptoms.—The patient always complains that his mouth is dry, and that the food he eats has practically no taste. There is also an inordinate thirst, and anorexia is present until after the crisis. Vomiting, as previously mentioned, may develop with the onset of the disease, and attacks of vomiting during the febrile period, and even after the crisis, may be seen. Constipation is, as a rule, obstinate early during the disease; exceptionally, this may alternate with diarrhea.

Pain in the epigastrium is frequently present, and when the base of the lung is involved, the pain is commonly below the costal border. Pain in

the region of the appendix and the gall-bladder is often misleading, and the clinician should always eliminate any pulmonary involvement of the right side in analyzing the significance of this type of pain. Rigidity of the muscles

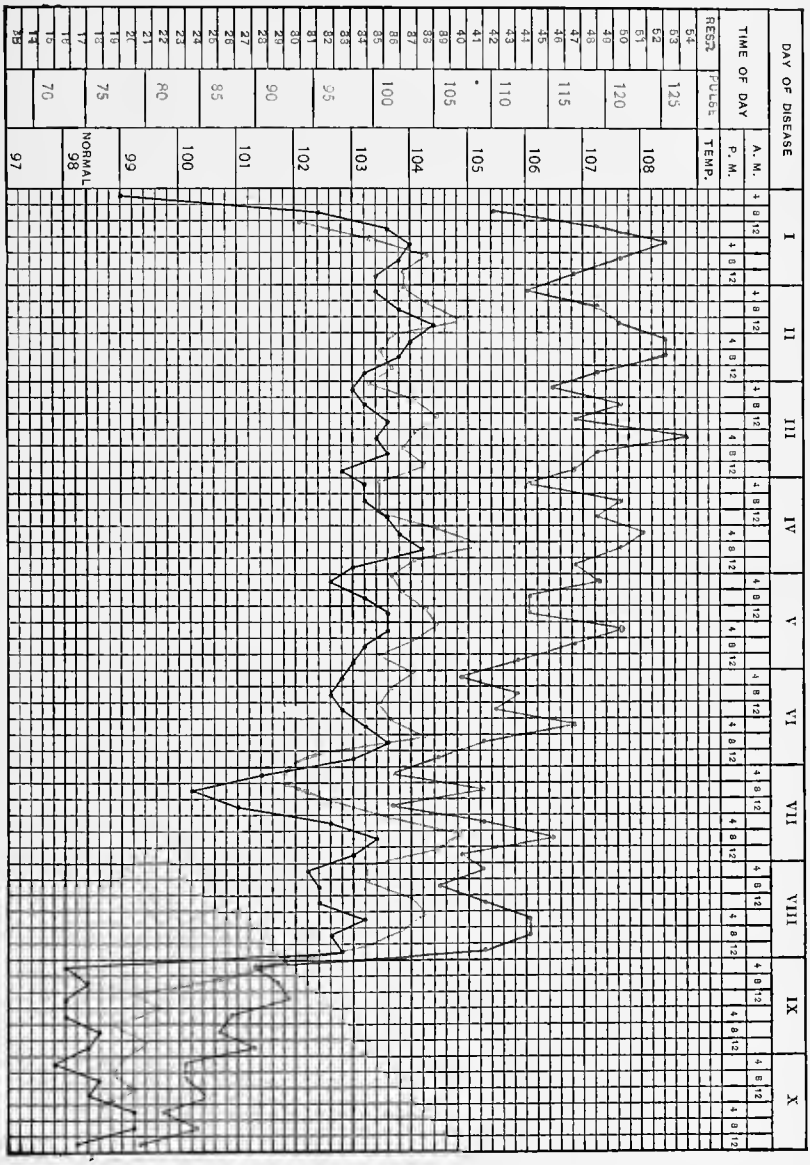


Fig. 288.—Chart of a Case of Lobar Pneumonia with Favorable Course (Anders).

A. T., aged thirty-two years; lower right lobe affected. Black, temperature; red, pulse; blue, respiration.

of one side of the abdomen may be present in pneumonia, and this is especially true when the pain is reflected over that portion of the abdominal surface.

Thermic Features.—The fever rises rapidly during the initial chill, so that in from eight to ten hours the temperature reaches 102° to 105° F. It then remains high until the crisis, pursuing the continued type, with slight nocturnal remissions of one or more degrees. In children, following the convulsion that commonly ushers in an attack, the fever rises rapidly, but seldom becomes so high as it does in the adult. The temperature has a lower range in the debilitated, in aged persons, and in alcoholics than it has in previously healthy adults. On the fifth to the seventh day of the disease a pronounced fall of temperature may be observed,—the pseudocrisis (Fig. 288),—but the temperature again rises to its former height. This clinical feature may be seen before the fifth day, however. In rare instances a pseudocrisis may be seen more than once, and the temperature-curve bears a strong resemblance to the remittent or the intermittent type of fever. The temperature may be unusually high,—106° or 107° F.,—and such striking elevation is at times a precursor of the crisis. In certain cases hyperpyrexia may be a danger-signal. The characteristic fever of pneumonia terminates by crisis. The crisis may occur at any time from the end of the third to the fourteenth day, but in the majority of instances it occurs on the seventh or the ninth day. The temperature usually falls during the night, and the drop is accompanied by copious perspiration, so that by the following morning the thermometer is found to register normal, or more often a subnormal point—98° to 95° F.

The duration of the period of decline is usually from eight to twelve hours. It may be much shorter, but is commonly longer. When the temperature terminates by lysis, the clinician should suspect the existence of some complication. Following the crisis, in uncomplicated cases the respirations and the pulse-rate soon return to the normal.

Cardiovascular Phenomena.—A careful study of the heart and pulse is an important point to be observed in pneumonia. The average pulse-rate in typical cases varies between 100 and 110 beats a minute, and when the beats exceed 120 a minute, there is sufficient cause for alarm. A marked increase in the pulse-rate within the course of twenty-four hours is believed to depend upon the action of toxins upon the heart or upon the presence of some complication. The pulse is, as a rule, small at first, a feature commonly seen when there is extensive consolidation; later, however, it becomes full and bounding, although at this time the pulse tension may be low. Dicrotism, together with an irregularity in both volume and rhythm, is an unfavorable feature, and may develop either before or after the crisis. The pulse will be found to vary greatly, depending upon the individual characteristics of the patient; *e. g.*, in feeble individuals the general characteristics may be absent throughout the course of the disease. The heart-sounds are in themselves fairly characteristic of this disease. (See Auscultation, Physical Signs, p. 779.)

Physical Signs.—For convenience of study, the physical signs have been arranged in the order in which they are presented during the successive pathologic stages of the disease.

Stage of Congestion.—In the majority of cases the physician probably does not see the patient until after this stage has passed. When the patient is seen early, however, the following physical signs may be elicited:

Inspection.—The movements of the affected side of the chest are often slightly restricted, especially if the base of the lung is involved, and upon mensuration it is found that expansion of the affected side is limited. If the patient is suffering from a double pneumonia, the costal type of breathing

may be seen, and pronounced movements of the abdominal muscles are also present. The face is flushed, and the so-called mahogany flush may involve the cheek of the affected side; the conjunctivæ are also at times suffused. The lips may, though rarely, show the beginning of herpes. The nostrils play violently, even though the degree of consolidation be but slight, and the patient frequently breathes with his mouth open.

Palpation.—It may be possible to detect slight diminution in the movements of the affected side of the chest, and tactile fremitus is somewhat increased over the congested area. If the area of congestion is localized at the center of one lung, palpation gives negative results.

Percussion.—The note may be normal over both lungs; as a rule, however, it is slightly shorter than normal, somewhat higher pitched, and, surrounding the congested area, it is tympanitic.

Auscultation.—It is quite common for the inflammatory products to occupy the smaller bronchi; consequently subcrepitant râles are usually audible over the area of congestion. The crepitant râle is also heard during the latter part of this stage. The breath-sounds, while usually weak, are at times bronchovesicular in character, and especially so when the patient is directed to inspire deeply. The breath-sounds are slightly exaggerated over the affected lung.

Stage of Consolidation.—*Inspection.*—If a large area of one lung is involved, the movements of the chest on the affected side are greatly diminished, whereas on the unaffected side they are appreciably increased. Exceptions to this general rule are quite common, and this is especially noticeable when the patient rests upon his back. If the greater portion of the base of one lung is involved, *mensuration* will show that side of the chest to be larger than the opposite, despite the fact that there is compensatory emphysema of the healthy lung. There may be abdominal distention.

The face may still be flushed, the nostrils play violently, and the respirations may number between 40 and 60 a minute in the adult, and between 60 and 80 in children. In persons older than sixty years suffering from pneumonia the respirations may not be greatly increased. At this time herpes labialis, involving the angles of the nose and lips, is quite common, and the lips are often dry and fissured. The tongue is heavily coated, and late during this stage it may become deeply fissured; its center is covered with a heavy yellowish or brown coat. Swelling and redness in the region of the large joints may be present, and indicate involvement of the synovial sacs.

Palpation confirms inspection with reference to the expansion of the two halves of the chest. Tactile fremitus is increased in the majority of all cases, although in exceptional instances fremitus is diminished, and in the case of massive pneumonia, or when there is considerable pleural effusion, it may be absent. A distinct friction fremitus is often detected during this stage of the disease, and is due to involvement of the pleura. The abdomen is rather tense. (See Abdominal Tension, Figs. 201, 225.)

The *pulse* is full, the beats numbering from 100 to 110 a minute, although great variation will be found in different cases. (See Cardiovascular Characteristics, p. 778.)

Percussion.—Dullness will be found to vary at different times during this stage; *e. g.*, before the lung is completely consolidated a somewhat tympanitic note is obtained, but after complete consolidation has occurred, the note is flat. When the base of the lung is involved, absolute dullness is more commonly found posteriorly and in the axilla than upon the anterior surface of the affected side. Indeed, it is not uncommon to find dullness over the

posterior portion of the lung, and a varying degree of tympany at the same level anteriorly. Considerable importance attaches itself to the degree of resistance offered to the pleximeter finger. If pleural effusion is present, the note over the affected side may be flat, especially at the base of the chest, but the sensation offered to the finger placed against the chest-wall is decidedly different in the case of fluid in the pleural sac and when there is only consolidation of the lung present. In the case of a pneumonia developing at the center of the lung and advancing toward the periphery, the lung tissue surrounding the consolidated area is emphysematous, and may be interposed between the hepatized tissue and the chest-wall, in consequence of which a hyperresonant note is obtained upon feeble percussion, but upon deep percussion over the same area dullness may be elicited.

Dullness appears to be a less conspicuous feature in the pneumonia of the aged. When there is an extensive pneumonia at the base or apex of one lung, the uninvolved portions of the lung may display skodaic resonance. The abdomen is moderately tympanitic.

Auscultation.—The characteristic breath-sound is that of bronchial or tubular breathing, and is heard over the consolidated lung, especially when consolidation extends to the visceral pleura. When the large bronchus leading to the consolidated portion of the lung is plugged with exudate, as is the case in the so-called *massive pneumonia*, bronchial breathing is absent. The voice-sounds are exceptionally well transmitted over the consolidated tissue, consequently bronchophony is obtained over the pneumonic area, but, as in the case of bronchial breathing, it, too, may be absent in some cases (plugging of the bronchus). In certain instances the sound conveyed to the ear is egophony. The whispered voice is also transmitted well over the consolidated area, consequently pectoriloquy here resembles the sound obtained over a pulmonary cavity.

Subcrepitant râles are present, and probably depend upon the associated bronchitis, and the crepitant râle may be audible at the end of inspiration, although this is more commonly heard during the first stage of the disease. A friction murmur may be present at any time during the stage of consolidation.

Stage of Gray Hepatization.—*Inspection.*—With beginning resolution the exudate present in the alveoli begins to liquefy, so that air now enters them; consequently upon inspection the movements of the two sides of the chest gradually become alike. In this stage the playing of the nostrils diminishes progressively unless some complication is present. The lips, which were probably cyanosed during the stage of consolidation, gradually assume their natural color, and by this time herpes labialis tends to disappear rapidly.

Palpation.—Tactile fremitus diminishes gradually from day to day until the normal is reached.

Percussion.—The alterations in the percussion-note (*e. g.*, dullness surrounded by a hyperresonant area and hyperresonance over the unaffected lung) gradually disappear, and are replaced by the normal percussion-note. It is important for the clinician to bear in mind that the percussion-note returns to the normal more slowly than do other physical signs previously outlined, and, indeed, a variable degree of impairment over the original area of consolidation may be present after convalescence is well established and after the patient is up and about his work. In those cases in which there has been extensive involvement of the pleura during the pneumonic process, the percussion-note may be impaired for an indefinite period.

Auscultation.—With the beginning of this stage, the crepitant râle may reappear; the subcrepitant râle, owing to the liquefaction of material in the air-cells, is also heard both on inspiration and on expiration (râle redux). Coarse bubbling râles are heard over the bronchi. In certain cases râles are universally numerous during this stage. Bronchial breathing disappears gradually, and in its stead bronchovesicular breathing is heard, and later the normal breath-sounds appear.

In those cases in which the toxemia has been pronounced, the first sound of the heart is lacking in muscular quality, whereas the accentuation of the second pulmonic sound has gradually diminished. In certain cases the heart is unusually rapid, especially after the crisis, which invariably marks the beginning of the stage of gray hepatization, and in such instances arrhythmia is common and may continue for days or even weeks. We have studied cases in both hospital and private practice in which a pericardial friction murmur was audible during the greater portion of this stage of pneumonia. Endocardial murmurs are not common unless disease of the endocardium existed prior to the development of the pneumonia. An exception to this rule is found in those cases that develop acute endocarditis as a complication. (See Acute Endocarditis, p. 251.) The pleuropericardial friction-sound is also occasionally heard. (See Physical Signs of Pleurisy, p. 139.)

Laboratory Diagnosis.—Sputum.—The patient expectorates a small quantity of extremely tenacious, blood-streaked sputum. So marked is the tenacity of the sputum, that the cup containing it may be inverted without the contents escaping. Microscopically this sputum is found to contain the pneumococcus, which sometimes occurs in dense aggregations. Gram-positive diplococci, which are in all probability pneumococci, are sometimes found in normal sputum. In cases of so-called Friedländer's pneumonia Friedländer's bacillus (Fig. 34) may also be present. We have examined the sputum of several cases displaying the clinical characteristics of pneumonia in which the predominant organism was the streptococcus. Many red blood-cells are present, and leukocytes and alveolar epithelium may be seen. During the third stage of the disease the quantity of sputum may be increased, but the rule is for the sputum to be scanty. Korelkin emphasizes the importance of albumin in the sputum as a valuable feature in the recognition of centrally located pneumonia.

The *hematologic* changes are quite characteristic, leukocytosis developing early and varying between 12,000 and 40,000 leukocytes in a cubic millimeter. In uncomplicated cases, following the true crisis, there is a marked diminution in the number of leukocytes in a cubic millimeter. A differential leukocyte count shows the increase in the number of white cells to be due chiefly to the polymorphonuclear elements. Eosinophilia is common after the true crisis. It is very important to remember that in extremely malignant types of the disease there may be a high leukocytosis, or, on the other hand, we may find that the number of leukocytes in a cubic millimeter is below that of the normal. In average cases the hemoglobin and the red cells are but slightly, if at all, altered. When the pulmonary consolidation is extensive and there is embarrassment of the circulation, with cyanosis of the lips, ears, and finger-tips, the number of red cells will be found to be between 5,000,000 and 10,000,000 per c.mm. The same condition—cyanosis—may cause the hemoglobin to register above the normal —from 90 to 120 per cent. During the third stage of the disease, and especially after the circulation has become nearly normal, there is an ap-

preciable diminution in the number of red cells in a cubic millimeter, a reduction of 500,000 to 600,000 being common.

Serum from persons suffering from pneumonia has been found to agglutinate the pneumococcus, but not in higher dilution than 1 : 60. In those cases showing pneumococcic septicemia and bacteriemia, the pneumococcus may be cultivated from the venous blood.

The *urine* is that characteristic of the acute fevers; *e. g.*, the quantity is somewhat diminished, the color is high, and the specific gravity is moderately increased—1.020 to 1.025. A small amount of albumin is commonly present —the so-called febrile albuminuria. If nephritis develops as a complication, the urine becomes rich in albumin and contains both hyaline and granular casts, and at times red blood-cells are present. In those cases in which meningitis complicates pneumonia, glycosuria may be present. During the second stage the chlorids may be absent.

Feces.—Rutz* has demonstrated that both the pneumococcus and the bacillus of Friedländer are present in comparatively large numbers in the feces of persons suffering from lobar pneumonia. The pneumococcus is commonly found after the third day of the disease.

The *serous sacs* may become infected with the pneumococcus during the course of this disease, and in such cases the exudate obtained from the serous membrane (pleura, meninges, and synovial sacs) contains pneumococci.

Illustrative Case of Lobar Pneumonia.—Family History.—Father living at the age of fifty-four; mother healthy at fifty-eight; three brothers and a sister living in apparently perfect health. No history of pulmonary disease, renal disease, or carcinoma in ancestors for three generations.

Previous Medical History.—The patient had the diseases of childhood, including diphtheria at the age of twelve, since which time he has enjoyed good health and does not recall having consulted a physician during the past five years.

Social History.—The patient was a male, aged twenty-eight years; he was married at the age of twenty-two and had two children living, who, with his wife, were in good health. He used no alcoholic stimulants, but drank one cup each of tea and coffee, daily. He was a traveling salesman by occupation, consequently his work demanded considerable exposure to wet and cold; he did not take systematic exercise, had paid no attention to diet, and had always eaten hurriedly.

Present Illness.—Two days before admission to the hospital he complained of a slight "cold," accompanied by moderate coryza. Twelve hours before admission he experienced a severe chill, which lasted for approximately one-half hour, and following which he stated that he felt feverish and was unusually thirsty. There was lancinating pain in the left chest, and deep respiration intensified the pain. The cough was an annoying symptom; it was persistent, although voluntarily restrained, and was accompanied by a slight amount of mucoid expectoration. Later, the sputum became tenacious in character, and presented the usual streaks of blood which are characteristic of rusty sputum.

The temperature remained at 102° F., displaying but slight morning remissions until the seventh day, when, within the course of a few hours, it fell to 97° F., and remained subnormal for a period of four hours. Following the crisis the temperature did not rise above the normal limit during convalescence.

Physical examination upon admission, and twelve hours following the chill, showed the following:

General Examination.—The patient rested in bed upon his back and gave an expression of pain whenever he was moved from side to side; he tended to incline toward the left side. The left side of his face was flushed, and there was distinct playing of the nostrils unless the patient breathed with his mouth open. Herpes of the lips and of the alæ nasi was present.

Local Examination.—At the time of the first examination *palpation* and *percussion* of the chest gave negative results, whereas upon *auscultation* a few fine crackling râles were audible over the left base posteriorly, and the respiratory murmur was somewhat exaggerated (bronchovesicular breathing).

* New York Med. Jour., July 20, 1912, p. 113.

During the second day of the disease, and approximately forty-eight hours following the chill, characteristic physical signs were present; *e. g.*, the patient still elected to rest upon his back, and there was an appreciable diminution in the movements at the base of the left chest. The nostrils played freely, and the movements of the chest were rapid—approximately 40 a minute. The impulse of the heart was seen at its normal site, but the area of pulsation was greatly increased.

Palpation confirmed inspection with reference to the cardiac pulsation and the rapidity and character of the respirations. The expansion of the two sides of the chest appeared to be nearly the same, except for a slight diminution over the left base. There was a distinct increase in the tactile fremitus over the base of the left lung posteriorly. The *pulse* was full and bounding, the beats numbering 110 to 120 a minute, and remained at this rate until the crisis occurred, although immediately after the pulse became weak, more rapid, and tended toward dicrotism.

Percussion.—There was dullness over the lower third of the left lung posteriorly, and this dull area extended anteriorly as far as the midaxillary line. A hyperresonant note was obtained over portions of the left lung not involved in the pneumonic process, and over the area of the right lung it was moderately hyperresonant. The area of cardiac dullness was not altered, and it was impossible to detect an increase in the area of splenic and of hepatic dullness.

Auscultation.—The increase in the frequency of respiration was distinctly apparent over both the right and the uninvolved portion of the left lung. Posteriorly, at the left base (limited to the area of dullness), the breath-sounds were markedly increased and bronchial in character. During each respiratory act a peculiar grunting sound was also audible over this area, and as the disease advanced this sound was so pronounced as to be heard when standing at the bedside. A distinct friction murmur was heard over the left base on the morning of the third day of the disease, but this disappeared within the course of a few hours, and was not detected later. The heart-sounds were rapid, of good volume, and there was an appreciable accentuation of the second pulmonic sounds.

Laboratory Findings.—The sputum was scanty, highly tenacious, and streaked with blood. Upon microscopic study the field displayed numerous dense aggregations of pneumococci. Red blood-cells and leukocytes were present.

The urine gave a negative reaction for chlorids. An estimation of the leukocytes gave 21,200 in a cubic millimeter, and a differential count showed 82 per cent. of the white cells to be of the polymorphonuclear variety.

Diagnosis by Induction from Clinical Data.—The history of the patient, showing that he had been healthy for some years, the age, the record of having suffered for the first few days from an acute cold, and the description of a severe chill twelve hours before admission to the hospital were of great clinical importance in formulating the diagnosis. Pain in the region of the nipple, and the fact that such pain was intensified by deep inspiration and by cough, gave positive evidence of an associated involvement of the visceral pleura. The cough in itself was somewhat characteristic, and was accompanied by a slight amount of tenacious, blood-streaked sputum that, upon microscopic study, was found to contain great numbers of diplococci. The temperature, which rose suddenly following the chill and pursued a continuous type to the seventh day, when it fell by crisis, was a feature practically characteristic of pneumonia. The fact that the urine did not give a reaction for chlorids during the febrile period was considered to support strongly the diagnosis of pneumonia, as did also the leukocyte count, which gave 21,200 cells in a cubic millimeter. Among the physical signs supporting the diagnosis were the following: (*a*) Impaired resonance over the area of lung involved; (*b*) bronchial breathing over the same area; and (*c*) transmission of both the whispered and spoken voice-sounds over the area of consolidation.

Differential Diagnosis.—The existence of a cold, together with the occurrence of a severe chill, the degree of prostration, cough, and fever, suggested the possible existence of influenza, from which condition pneumonia was distinguished by the following clinical evidences: (*a*) Blood-streaked, tenacious sputum containing diplococci; (*b*) leukocytosis; and (*c*) the physical signs of pulmonary consolidation.

Course of the Disease and Change in Physical Signs Following Crisis.—During the night of the seventh day of the disease the patient's temperature fell, within the course of twelve hours, to normal, and immediately following this crisis the pulse rose to 140 a minute; his respirations remained rapid, there was distinct cyanosis of the lips, face, and finger-tips, and the body became beaded with perspiration. All these features disappeared within the course of a few hours following the judicious application of external heat and of cardiac stimulants. Upon the eighth day of the disease the physical signs were materially altered, respirations numbered 26 a minute, and the pulse was 106. The area over which consolidation had been present still showed dullness, and tactile fremitus was less increased. Upon auscultation numerous subcrepitant and bubbling

râles were heard over the base of the left lung, and the breath-sounds were not moderately intensified, in striking contrast to that of bronchial breathing, which had been present since the second day of the illness. Uninterrupted convalescence followed.

Summary of Diagnosis.—When the disease attacks the robust and previously healthy, it is characterized by the severity of onset; thus there are chill, rapid rise in temperature, pain in the side, and distressing cough. The frequency of the respirations—30 to 60 a minute—and the disturbance of the pulse-respiration ratio are quite characteristic of the disease, and especially so when this clinical phenomenon is coupled with the previously mentioned mode of invasion and symptoms. The sputum is an important feature in the diagnosis of pneumonia, and it is practically the only disease in which the quantity of sputum is small, and the expectoration itself is extremely tenacious and blood-streaked. A microscopic study of the sputum, with detection of many diplococci, is confirmatory of the other features, but is not essential to a diagnosis in typical cases. The continued type of the fever, with termination by crisis, and the signs of consolidation of a segment of one lung, or rarely of both lungs, are highly valuable diagnostic features. Playing of the nostrils and the peculiar expiratory grunt are also important, and the absence of chlorids from the urine and a leukocytosis of from 12,000 to 40,000 leukocytes in a cubic millimeter further support the diagnosis.

Differential Diagnosis.—Lobar pneumonia is to be distinguished first from **acute pneumonic phthisis.** The distinctive features between these two maladies are shown in the accompanying table (modified from Anders):

TABLE SHOWING THE POINTS OF DIFFERENTIATION BETWEEN PRIMARY LOBAR PNEUMONIA AND ACUTE PNEUMONIC PHTHISIS.

Primary Lobar Pneumonia.	Acute Pneumonic Phthisis.
1. There may have been prior attacks.	1. Inherited predisposition or previous tuberculous disease.
2. Onset sudden, with severe rigor and rapid rise of temperature.	2. Onset usually more gradual; repeated chilly sensations (rarely, severe rigor), often following exposure or "cold."
3. Fever of continued type, terminating by crisis.	3. Fever of remittent type, often becoming intermittent, without crisis.
4. No drenching sweats, except at time of crisis.	4. Drenching sweats common.
5. Herpes common.	5. Herpes unusual.
6. Emaciation slight or absent.	6. Rapid, progressive emaciation.
7. Sputum rusty, viscid, and sticky; may contain pneumococci.	7. Sputum may be blood-tinged; is more purulent and copious, and may contain tubercle bacilli and elastic tissue.
8. Duration of febrile stage, seven to nine days, terminating by crisis.	8. Duration longer, and may become intermittent. No crisis.
9. Physical signs, as a rule, first referable to base of lung, except in apical pneumonia.	9. Physical signs first referable to apex.
10. Usually limited to one lobe or the lower segment of one lung.	10. Commonly extends from apex to base.
11. Signs of consolidation, followed by resolution.	11. Signs of consolidation, followed by characteristic signs of cavity formation.
12. Apex of opposite lung not involved.	12. Apex of opposite side generally attacked.
13. Leukocytosis of 12,000 to 40,000 by end of the first forty-eight hours.	13. Leukocytosis after cavity formation.

Typhoid Pneumonia.—When the patient suffering from lobar pneumonia presents the so-called "typhoid state," it is to be distinguished from pneumotyphoid (see Typhoid Fever, p. 745), and in this connection an examination of the blood is of inestimable value, since in typhoid pneumonia *leukocytosis* is present, whereas in pneumotyphoid *leukopenia* is the rule. After the end of the first week the Widal reaction is present in pneumotyphoid (typhoid fever simulating pneumonia), but absent in typhoid pneumonia (pneumonia displaying the typhoid state), unless the patient has at some previous time suffered from an attack of typhoid fever. After the first week other symptoms of typhoid fever, *e. g.*, eruption, tympanites, and diarrhea, are likely to develop in pneumotyphoid.

Pneumonia may at times simulate **acute meningitis,** a type of the disease more commonly seen in children than in adults. In children the initial symptom may be a convulsion, and frontal headache is also common. In pneumonia accompanied by meningeal symptoms the pulse-respiration ratio is disturbed early, and the physical signs of pneumonia are, as a rule, present, although at times it is difficult to detect pneumonia in children by a physical examination. Lumbar puncture serves as a positive means of differential diagnosis. (See Epidemic Meningitis, p. 841.)

A table showing the distinctive features between lobar pneumonia and **pleurisy with effusion** will be found under Differential Diagnosis of Pleurisy, p. 151.

Lobar pneumonia is also to be distinguished from **bronchopneumonia,** and this task is extremely difficult in those cases in which, in bronchopneumonia, numerous small areas of consolidation unite to form one solid mass that may involve the greater portion of one lobe. Of further clinical importance is it to remember that lobar pneumonia is more common during early adult life, whereas bronchopneumonia is frequent in children and in the aged. (The distinctive features between lobar pneumonia and bronchopneumonia will be found in a differential table under the Diagnosis of Bronchopneumonia, p. 112.)

Clinical Course and Complications.—The clinical course, as well as the severity of the disease, is in a measure dependent upon the severity of the type of infection and the individual characteristics of the patient. They survive the disease best who have no organic changes in the heart or the kidneys, and in whom complications are absent. In hospital practice, 25 per cent. of cases go on from bad to worse until a fatal termination is reached, whereas in private practice approximately 15 per cent. terminate unfavorably. In sthenic cases a severe type of infection is manifested by a severe rigor, high temperature, and marked nervous symptoms, whereas in milder types of infection all these features are likewise mild. Irrespective of the area of lung involved, in typical cases the temperature falls between the fifth and ninth days (usually, the seventh or the ninth day) and following the crisis the respirations soon become normal.

Pain and *cough* are aggravated symptoms early during the disease, but become less prominent after the second day, and are usually absent following the crisis. The strength of the heart, as indicated by the pulse, is probably the most important clinical feature in pneumonia, and in the average uncomplicated case the pulse remains fairly strong and regular at about 110 until the time of the crisis, when the number of beats a minute may reach 120 or more; at this time the pulse is likely to become dicrotic, compressible, and intermittent. The greater the area of lung involved, the less completely is the patient's blood oxidized; consequently in that class of

cases in which a large portion of the lung texture is consolidated, severe nervous symptoms are present, and practically all types of delirium may be seen, including coma, which, in unfavorable cases, may continue until death. The respirations, however, although always rapid, are of less importance than is the strength of the heart. With extensive involvement of the lung, cyanosis of the lips, face, ears, and fingers is common, and seldom exists for any great length of time without an appreciable weakening of the pulse. Following the crisis, convalescence becomes established in those cases that terminate favorably, although, when complications are present, convalescence is greatly delayed.

Typhoid pneumonia, previously referred to under Clinical Types, p. 771, presents no symptoms that are in themselves distinctive, yet the clinical course is often longer than is that of typical lobar pneumonia, and convalescence is usually prolonged. In the absence of complications, ordinary cases go on to recovery, the patient being able to leave his bed by the end of the third or fourth week; singularly enough, however, complications arise in 50 per cent. of cases, and, regardless of the character of the complication in question, there is usually an increase in the pulse-rate and there may also be alterations in the fever and respirations.

Pleurisy with effusion is a somewhat common complication, but if pleurisy is limited to the side affected by pneumonia, it is less serious than when the opposite pleural cavity becomes filled with fluid. Pleurisy always retards convalescence, and the patient is usually confined to bed for from six to ten weeks.

Empyema not infrequently follows lobar pneumonia, and although it is a serious complication, it often terminates in recovery. It is a more common complication in children than in adults. In practically every case of pneumonia there is a certain degree of bronchitis, especially of the larger tubes, but if an extensive bronchitis develops the symptoms are intensified, there is marked tendency to heart weakness and cyanosis, and the clinical course is greatly prolonged.

Pericarditis may result from direct extension of the inflammatory process through the pleura to the pericardial surface, and this complication materially lessens the chances of recovery. It is also accompanied by the characteristic physical signs of pericarditis. (See p. 238.)

Ulcerative endocarditis is probably the most serious complication known to pneumonia, since the peripheral vessels in the various viscera may become plugged with thrombi and bacteria, and septic phenomena supervene. When endocarditis develops, the clinical course is that of a bacteriemia. (See Ulcerative Endocarditis, p. 255.)

Pulmonary abscess (see p. 121) and **pulmonary gangrene** (see p. 118) are occasionally seen to follow lobar pneumonia, and both these conditions appreciably retard convalescence, although they are not of necessity fatal.

Pulmonary edema may develop at any stage during the course of pneumonia, and this complication is characterized by increased rapidity of the heart, with a weak, thready, and irregular pulse, profound cyanosis, and rapid respirations.

Acute nephritis, when present, may subside at the time of the crisis, although in unfavorable cases the albuminuria persists. A mere trace of albumin in the urine is not to be regarded as of serious moment, but when, in addition to a large amount of albumin, granular casts and both red and white blood-cells are present, the patient's condition is alarming. The

presence of nephritis delays convalescence, although many patients have come under our observation in whom the kidney condition completely disappeared during their stay in the hospital.

THE PLAGUE

(Bubonic Plague; Black Death).

Pathologic Definition.—An acute infectious disease, due to bacillus pestis, and characterized pathologically by enlargement of the superficial lymph-nodes, with the formation of buboes, isolated areas of gangrene of the cutaneous and subcutaneous tissue, or pneumonia. There may also be minute hemorrhages into the mucous surfaces (stomach, intestines, lungs). Both the discharge obtained from the lesions and sections of the pneumonic organs will be found to contain the Bacillus pestis.

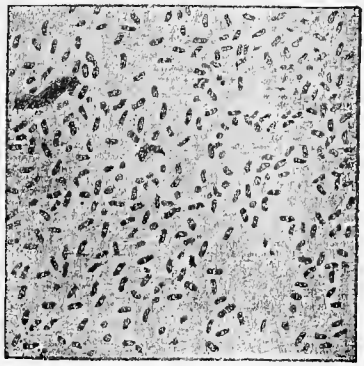


Fig. 289.—Bacillus of Bubonic Plague (Yersin).

Exciting and Predisposing Factors.—Bacteriology.—In 1894 Kitasato and Yersin both discovered the presence of the bacillus pestis in plague, and this organism is now conceded to be the exciting cause of the disease. The bacillus stains more deeply at its extremities than at its central portion, which gives it an imperfect, coccus-like appearance.

The point of entrance for bacillus pestis is through the punctures made by fleas and probably bedbugs in biting their human hosts. In some cases of primary plague pneumonia the bacillus may gain access to the lung with the inspired air. D. T. Verjbitski* has conducted a series of experiments through which he has permitted the bedbug, *Cimex lectularis*, to feed upon animals suffering from plague, and to later bite uninfected guinea-pigs. The cimex was known to convey the disease through its bite to uninfected guinea-pigs for a period of five days after sucking blood from infected animals, while the flea communicates the disease for a period of approximately three days.

If the bacillus gains access to the body through the puncture made by a flea in biting, the nearest lymph-nodes become involved. The infection may stop here, and the bubo thus produced may suppurate, and, after a prolonged period of discharge, the sinus may close and the patient recover. In more than half the cases, however, a general septicemia results and the patient dies.

Recent studies have shown that the human epidemic of plague is preceded by an epizoötic among the rats of the locality.

Clinical Varieties.—There are three clinical types of plague: (1) The bubonic form; (2) the septicemic form, and (3) the pneumonic form.

(1) *The Bubonic Form.*—After an incubation period of from two to eight days the disease begins suddenly with fever and prostration. The face is

* Manning, Medical Record, July 27, 1912.

said to be peculiarly drawn and swollen in appearance; sometimes there is a look of horror. Nause, vomiting, and diarrhea are noted in some cases. There is loss of co-ordination (staggering gait), and thick and stammering speech. Delirium, on the one hand, and stupor, on the other hand, may occur. The bubo is first noticed usually within the first twenty-four hours; sometimes it does not appear until the fourth or fifth day. In 70 per cent. of the cases the saphenous lymph-nodes are affected; in about 20 per cent. of cases the bubo is in the axillary region; and in about 10 per cent. of cases it is found in the cervical region. The buboes are usually single; they vary in size from that of a walnut to that of a goose-egg. Hemorrhages into the skin, epistaxis, hematemesis, hemoptysis, melena, and hematuria are common symptoms. In favorable cases the constitutional symptoms improve after the formation of the bubo. The fever ends by lysis; the bubo softens and opens spontaneously, or it may be incised, and a chronic suppurating sinus results. Death usually occurs between the third and fifth days from exhaustion, hemorrhage, heart failure, convulsions, or coma (Manson).

(2) In the *septicemic form* there is no bubo formed. The disease is septicemic from the start. A general enlargement of the lymph-nodes has been demonstrated at autopsy, however. The temperature is not very high; there are marked toxicity, hemorrhage, stupor, coma, and death.

(3) In the *pneumonic form* there is a consolidation of the lungs, indicated by the usual physical signs, with the presence of large numbers of bacillus pestis in the sputum. This form is very fatal. Abortive cases of plague are called *pestis ambulans*, and Choksy has described a cellulocutaneous form, characterized by local necrosis of the skin and the subcutaneous tissue.*

Laboratory Diagnosis.—In cases of bubonic plague the puncture of the bubo with a sterile hypodermic syringe and examination of smears made from the contents of the lesion will give short, Gram-negative bacilli showing polar staining. In septicemic cases blood culture will show bacillus pestis. In pneumonic cases examination of the sputum will show large numbers of the short, polar-staining bacilli, which at first look like diplococci. The Gram stains will differentiate micrococcus lanceolatus, which is Gram-positive, from bacillus pestis, which is Gram-negative.

Summary of Diagnosis.—The history of an epidemic or of the patient having resided in tropical or infected districts should always be given careful consideration. The rapidity of the onset, the increasing fever, with tendency to an early formation of a bubo, invasion of the lymphatics, should, at least, suggest the possibility of plague. Hemorrhage from the various mucous surfaces and petechiæ, both of the skin and of the mucous membranes, together with a high grade of prostration, are cardinal features of bubonic plague. The detection of the bacillus pestis in the sputum, in the fluid obtained from puncture of the involved glands, or in pus recovered from abscesses makes the diagnosis positive.

INFLUENZA

(La Grippe; Epidemic Catarrhal Fever).

Pathologic Definition.—An endemic and epidemic, acute infectious and transmissible disease, said to be excited by the bacillus influenzæ, and characterized by a catarrhal inflammation of the respiratory and alimentary tracts. There is a special tendency toward the development of the patho-

* Trans. Amer. Soc. of Tropical Medicine, 1909.

logic changes known to acute bronchitis, bronchopneumonia, and myocarditis.

Predisposing and Exciting Factors.—Practically all persons are likely to contract the disease. **Age** exercises but moderate influence. The greater number of cases, however, develop in young adults between the ages of twenty-five and thirty-five years. The disease is far less common before the tenth year. Influenza is a disease that commonly attacks those in whom the general nutrition is impoverished, consequently it is prone to arise as a secondary condition in the ill nourished.

Bacteriology.—In 1892 Pfeiffer described the influenza bacillus at length. This organism is obtained from the sputum and nasal secretions during the febrile period of the disease, and Pfeiffer declares that the bacillus enters the bronchial tissue and may even penetrate the pulmonary coverings and enter the pleural sac. (See Laboratory Diagnosis, p. 792.) The relation that probably exists between influenza, ordinary colds, measles, diphtheria, scarlet fever, and other acute infections, when seen in children, is by no means clearly understood, yet the fact remains that influenza is a frequent complication in such infectious conditions.

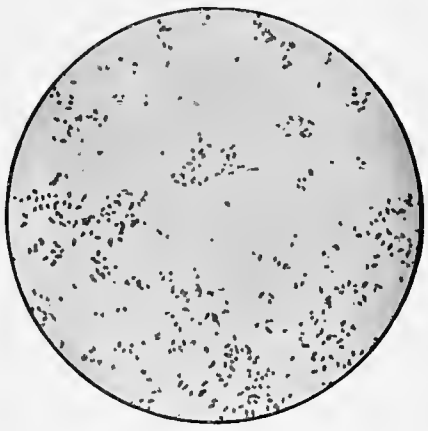


Fig. 290.—Bacillus of Influenza, from a Gelatin Culture (× 1000) (Itzerott and Niemann).

Modes of Infection.—The various modes by which the disease may be transmitted from one person to another are not well understood, although several theories have been offered in explanation.

A single attack does not bestow *immunity*, and, indeed, subsequent attacks are quite common, the disease recurring from every one to five years. Certain individuals contract the disease with the appearance of each epidemic; therefore, it is not unusual to encounter those who have suffered from two or more attacks.

Clinical Varieties.—(1) **Respiratory Type.**—In this variety the early and more prominent symptoms are those referable to the respiratory tract—*e. g.*, coryza, pharyngitis, laryngotracheitis, and bronchitis. A more or less general aching of the throat and prostration are also present. When the respiratory symptoms predominate, it is not unusual to find bronchopneumonia as a coexisting condition. Persistent cough is a constant symptom, and violent paroxysmal coughing may be present.

(2) **Gastro-intestinal Type.**—The initial symptoms of this type may be practically identical with those of the respiratory form, except that they are milder in degree and are somewhat masked by the severe symptoms referable to the gastro-intestinal tract, namely, abdominal pain, nausea, vomiting, and profuse watery diarrhea. *Prostration* is well marked in this as in practically all types of influenza.

(3) **Typhoid Type.**—In a small percentage of all cases continued fever, with delirium, may be present. The lips are brown and fissured, the tongue is parched and heavily coated, and the general condition of the patient is

that known as the typhoid state. The fever may be remittent or even intermittent, and this feature, together with the repeated occurrence of chills or of chilly sensations, often suggests to the clinician the possible existence of malarial infection.

(4) **Cardiac Type.**—Certain cases of influenza are seen in which the cardiac features constitute the most prominent symptoms. The pulse is rapid, feeble, and irregular, and there is a tendency toward heart failure with cardiac dilatation.

(5) **Nervous Type.**—The nervous symptoms are fairly prominent in most clinical varieties of influenza, but cases are occasionally encountered in which they are unusually prominent; for example, atrocious headache, muscular pains, stiffness of the joints, and the early development of delirium may serve as the leading features in this type of the disease. In the cerebral type of influenza symptoms quite identical with those of meningitis develop rather suddenly and persist for a period of one, two, or more days when, in favorable cases, they disappear suddenly.

(6) **Rheumatoid Type.**—The rheumatoid type differs from the foregoing one in that the predominant feature is extreme muscular pain involving the greater portion of the body.

(7) **Apyretic Form.**—As the name implies, this variety of influenza may simulate in general any one of the previously described clinical pictures, and is that of the respiratory type of the disease, except that fever is absent.

(8) **Ambulatory Type.**—This form is of special importance, because of its tendency to spread the disease.

Period of Incubation.—This is, as a rule, brief, and does not exceed two or three days.

Principal Complaint and Symptoms.—In the majority of cases the *onset* is sudden, the disease being usually ushered in by a rigor or at least by a repetition of **chilly sensations**. **Profound prostration** is characteristic, and develops early in the majority of all cases. In typical forms of the disease the initial symptoms may be vertigo, bilious vomiting, and epistaxis.

Dyspnea is often a conspicuous symptom, and may persist even for days in uncomplicated cases, and profuse **sweating** is at times troublesome. The nature and degree of severity of this condition are decidedly variable; at times the symptoms are so severe as to advance rapidly to a state of collapse, while within the course of a few hours the symptoms of bronchopneumonia may be displayed. **Complications** may arise and escape observation until they are well advanced, being masked by the severe symptoms already present. Congestion of the various viscera and edema of the extremities (see Cardiac Complications) are not uncommon.

In an average case of the pulmonary type of influenza the symptoms of severe **bronchitis** are present—cough, with possibly slight expectoration, coryza, with sneezing, and increased secretion of the lacrimal glands.

In children the **gastro-intestinal manifestations** of the disease are often prominent, and frequent attacks of vomiting are likely to occur; rarely there is hemorrhage from both the stomach and the bowel, the general clinical picture being that of severe acute gastro-intestinal catarrh.

Nervous Symptoms.—Probably the most constant nervous symptom is perineuritis, which may be so severe as to induce profound suffering.

Pain.—Headache is almost constant in the early stage of influenza, and may be frontal, temporal, or occipital. In some patients the pain is most severe in and between the eyes. Muscular pains, affecting the loins, lumbar

region, back, arms, and limbs, are intensified by the slightest movement. Neuralgic pains are occasionally present, and sharp, lancinating, or stitch-like pains may be felt over various portions of the chest and abdomen. The pain may at times be referred to as burning in character, and here it is limited chiefly to the cutaneous surfaces, whereas in the same patient there may also be deep, boring pain in the back.

Hysteric outbreaks are occasionally seen in those of a neurasthenic temperament. In grave forms of the disease there is at times active delirium, but such severe nervous manifestations seldom appear unless complications are present. The nervous symptoms are greatly intensified in those cases in which such complications as bronchopneumonia, nephritis, and otitis media develop. Pleuritic pain may be present for one or more days, when the patient's complaint is quite identical with that given for acute pleurisy, although the other features of pleurisy are absent.

Thermic Features.—Following the chill, the temperature usually rises somewhat abruptly to 102° to 104° or 105° F., depending upon the severity of the type of infection, and may assume a remittent course. The temperature is in no way characteristic, and in uncomplicated cases will be found to reach the normal by the end of the first week. It is to be borne in mind that the temperature is influenced largely by the presence of complications, and we are inclined to regard influenza as an infection in which complications are most common.

Physical Signs.—Inspection.—The face is somewhat flushed at the onset, but later, and after cardiac failure, there may be pallor. Cyanosis involving the face, lips, tongue, and extremities is present. The tongue is coated, and should the fever continue high for several days, the lips are likely to be fissured. The movements of the chest are increased even in mild cases, and are further increased in proportion to the severity of the bronchitis or of the pulmonary complications present. Swelling of the feet and of the hands is present only when heart failure or renal complications occur. Jaundice, as the result of duodenal catarrh, may develop at any time during the course of influenza. In those cases of the disease in which the clinical picture resembles that of cerebrospinal meningitis, there may be inequality of the pupils and a fixed position of the head, and one or more of the extremities may be held firmly in one position.

Palpation.—Upon making firm pressure over the loins, shoulders, and arms, the muscles may be found to be slightly tender, although muscle soreness is usually produced by movements of the patient. Edema of the extremities may be detected, but is a feature only of complicated cases. The apex-beat is at first strong, but is soon found to weaken, and when the toxic symptoms are pronounced, the cardiac impulse is feeble and diffuse. In cardiac dilatation the apex impulse may be almost imperceptible. Areas of increased tactile fremitus may be found at the bases of the lungs posteriorly, and indicate the existence of the so-called "grip" pneumonia. The pulse is increased in frequency in proportion to the elevation of temperature. Certain writers, however, describe bradycardia as a feature in many cases. In those cases in which the toxic substances appear to affect the heart, the pulse is likely to become rapid and irregular as to both time and force. In the aged the pulse is readily compressible, and tends to become dicrotic as the disease progresses.

Percussion.—In uncomplicated cases percussion is negative.

Auscultation.—The heart-beats may be increased somewhat as to frequency, although in the early stage the heart action may be comparatively

slow in proportion to the degree of prostration. In severe and complicated cases the heart action is rapid, and the first sound appears to have lost its muscular quality. Auscultation of the chest reveals the signs of acute bronchitis. (See p. 88.) Bronchopneumonia is a common pulmonary complication. (See Bronchopneumonia, p. 107.)

Laboratory Diagnosis.—The *urine* may contain a trace of albumin, and in those cases in which true nephritis exists, the urine will be found to be rich in albumin and to contain leukocytes and casts. In the gastro-intestinal type of grip the urine may be rich in indican, and occasionally stained with bile.

In uncomplicated and mild cases the number of *leukocytes* is not increased, although occasionally a leukocytosis of from 10,000 to 15,000 may be seen. Leukopenia may be present for a short period.

The *sputum*, saliva, and nasal secretions, when studied in stained preparations, contain numerous slender bacilli. These have received careful study by Pfeiffer, who suggests that they may be the cause of influenza. Special attention must be called to the striking resemblance between the laboratory characteristics of the bacillus influenzæ and those of the organism described by Koch and Weeks as the exciting cause of acute conjunctivitis. (See special works upon Bacteriology.)

Illustrative Case.—C. H. C., male, aged twenty-three years; clerk. Has enjoyed perfect health during the past ten years. Two days prior to his admission to the hospital he noticed some loss of appetite and languor, and sneezed several times during the day. Upon admission he stated that he had suffered from chilly sensations during the day, violent headache, marked pain in the orbits, and aching in the back, loins, and muscles of the limbs. Severe cough, dyspnea, and a temperature of 103° F. were present.

Physical examination revealed nothing beyond that the respirations were hurried and that there were numerous fine râles over both lungs. The pulse was weak but regular, although the degree of prostration appeared to be extreme. For a period of four days the temperature fluctuated between 99° and 102° F., reaching the higher elevation during the evening hours. The respirations became more and more hurried during this time, until he was breathing approximately 30 times a minute. The urine contained a trace of albumin, and constipation was sufficiently obstinate to necessitate the administration of a laxative. By the end of the first week the fever had fallen to the normal, and all symptoms were greatly ameliorated; from this day convalescence was not interrupted.

Summary of Diagnosis.—This is seldom difficult except in ill-defined, sporadic cases. Great importance attaches itself to the presence of an epidemic, an abrupt onset, with alternating flashes of heat and mild chills, the short duration of the febrile period, the intensity of the headache, with severe pain in the eyes and orbits, and muscular pains. In addition to the foregoing clinical peculiarities, if the prostration is out of proportion to the catarrhal manifestations, the diagnosis is practically assured. A microscopic study of the sputum, having for its object the detection of the bacillus influenzæ, although essential to a diagnosis only in sporadic and atypical forms of the disease, should always be undertaken.

Clinical Course.—The duration of the attack is brief, although special cases show great variations. In the milder forms severe symptoms exsit for but from two to four days, whereas in the more severe types of infection they are present for from seven to ten days or possibly two weeks. Influenza, when it attacks those who are already suffering from some acute or chronic malady, may continue for a longer period than it does in previously healthy subjects. Epidemic influenza usually continues over a period of

from four to eight weeks, after which there may be, for a considerable period of time, more sporadic cases than usual.

Complications and Sequelæ.—**Bronchopneumonia** is probably the most common and severe complication known to influenza. Hyperpyrexia may rarely develop, and diarrhea with hemorrhage from the bowel is also an occasional complication. Nephritis, pleurisy, and severe diffuse bronchitis, should they develop at any time during the disease, are to be regarded as of serious moment. Meningeal symptoms with maniacal delirium that is followed by coma seldom appear in uncomplicated cases of influenza, and these features are suggestive not only of a grave type of the disease, but of the existence of other serious complications.

Among the *sequelæ* known to this affection are pulmonary tuberculosis, pulmonary abscess, pulmonary gangrene, and chronic bronchitis. Following an attack of influenza the heart may remain irritable, and tachycardia is common, whereas true angina of the precordial region is but an occasional feature. Subacute and chronic catarrh of the stomach and intestinal tract may continue for months, and following a somewhat protracted attack of influenza, we occasionally encounter chronic cystitis. Nephritis may continue after the febrile period. Among the annoying nervous sequelæ should be mentioned insomnia, headache, melancholia, suicidal tendencies, peripheral neuritis, and ascending myelitis.

Otitis media and even mastoid abscess may be seen to follow influenza, and ocular sequelæ—*e. g.*, choroiditis, acute glaucoma, and conjunctivitis—are somewhat more common.

TUBERCULOSIS.

Pathologic Definition.—An acute, subacute, or, more commonly, chronic infectious disease, caused by *bacillus tuberculosis.* The disease is characterized anatomically by the formation of a lesion called a tubercle. Tubercles, which may be formed in all the tissues of the body, are at first small—the so-called miliary tubercles; these tend to fuse, forming larger tuberculous masses of varying sizes, which undergo caseous degeneration, softening, and ulceration. With the softening of the tubercle the pyogenic organisms invade the lesion, provided it is so situated that they may gain access to it, and then a mixed infection results, with the production of pus. When the pyogenic organisms do not invade the softening tubercle, the purulent contents of the lesion is apt to be sterile. The tendency of the tubercle is to limit itself by the formation of a capsule of connective tissue, *sclerosis,* with inspissation and subsequent *calcification* of the contents. In cases of disseminated infection a disease known as *miliary tuberculosis* is produced, in which discrete tubercles of the size of a pinhead or less are found in nearly every organ of the body.

Caseation is the term employed to denote a process of coagulation necrosis of the cells forming the tubercle, by which the cells are converted into a yellowish, structureless substance, like cream cheese. This process, which begins at the center of the tubercle and gradually extends toward the periphery, is probably due to the local action of the toxins of the bacillus. If a tubercle is properly situated, the caseous process may extend until an opening into a natural cavity is produced, such as a bronchus or a joint, and a *tuberculous cavity* results. Such a tuberculous cavity is very likely to become infected with the pyogenic cocci and bacilli. If a caseous nodule

becomes encapsulated with fibrous tissue (*sclerosis*), it may be converted into a hyaline, fibrous material in which lime salts are subsequently deposited. Such a calcareous tubercle is harmless.

The dissemination of tubercle bacilli is effected principally through the lymphatics; but in some instances the organisms are widely distributed by the blood-stream, in which case *miliary tuberculosis* results. In some cases the disease extends by contiguity, as when a tubercle on the visceral layer of the peritoneum produces a tubercle on the parietal layer of the peritoneum just opposite to it. Also, in cases of tuberculous peritonitis the disease is disseminated along the surface of the peritoneum by the peristaltic movements of the intestines.

Distribution of the Lesions.—In the adult the lung is most frequently the seat of tuberculous new-growths.

Next in the order of frequency follow the larynx, intestines, peritoneum, genito-urinary organs, brain, spleen, liver, and heart. The pleura, meninges, and synovial membranes are frequently attacked. In children, the favorite sites of origin are the lymph-nodes, intestines, bones, and joints. Here the distribution, if we except the bronchial and mesenteric lymph-nodes, corresponds quite closely to that of surgical tuberculosis.

The Elementary (Nodular) Tubercle.—This may develop in any tissue in which the tubercle bacillus has become lodged, and the presence of the bacillus is the sole distinguishing feature, since apparently identical growths are produced by other microörganisms—*e. g.*, the actinomyces, Aspergillus glaucus, Aspergillus fumigatus—and even by irritation of foreign bodies—for example, podophyllum. Pseudotuberculosis is caused by organisms other than the tubercle bacillus.

The **stages in the development of a tubercle** are:

(1) A *proliferation* of the fixed tissue elements (connective-tissue cells, endothelium of the capillaries, etc.) of the part infected, due to the local specific irritative action of the bacillus. These anatomic structures are transformed into epithelioid and giant-cells. The epithelioid cells assume various shapes, chiefly rounded and polygonal, and sometimes contain tubercle bacilli in their cytoplasm. As the result of increase in their size and a repeated division of their nuclei, or from the union of contiguous cells, a certain number of the epithelioid cells are transformed into giant-cells. The giant-cells occupy the center of the tubercle and often contain bacilli.

(2) *Diapedesis* of leukocytes occurs around the site of infection. It is of the nature of a defensive inflammatory process. At first the leukocytes are of the polymorphonuclear variety; these are quickly destroyed; later, however, mononuclear leukocytes appear. The granular elements described are immediately surrounded by a reticular stroma.

Fully developed tubercles are small, nodular bodies, having a diameter of from $\frac{1}{2}$ to 2 or 3 mm. At first they are almost transparent, but as the result of further changes, they soon lose this quality. Tubercles invariably undergo degenerative changes, such as caseation and sclerosis.

Bacteriology.—The bacillus tuberculosis (Plate I) is the sole exciting cause of the disease. The organism is a straight or slightly bent, non-motile rod, rather slender, with rounded extremities. It varies in length from one and one-half to five microns, or from one-fourth to one-half the diameter of a red blood-corpuscle. It is about 0.3 micron thick. It belongs to a group of organisms which are known as the *acid-fast bacilli*. These organisms have the peculiarity of retaining fuchsin in spite of sub-

sequent treatment with a solution of a mineral acid. The tubercle bacillus is also alcohol-fast.

Biology.—The bacillus tuberculosis is cultivated with difficulty. It will not grow at all on the ordinary laboratory media; but on various special media growth can be produced, although but slowly. Glycerin bouillon and glycerin-agar are the two media habitually employed for the cultivation of this organism.

The other members of the group of acid-fast bacteria are bacillus lepræ, bacillus smegmæ, the bacillus of timothy grass, and the butter bacillus of Rabinowitch. The following points differentiate bacillus tuberculosis from the other members of this group: (1) The tubercle bacillus grows very slowly upon glycerin-agar, glycerin bouillon, or glycerin potato. (2) Bacillus lepræ and bacillus smegmæ cannot be cultivated upon artificial media. (3) The bacillus of timothy grass and the butter bacillus grow readily on the ordinary culture-media.

A method for determining the presence of the tubercle bacillus is to inoculate a guinea-pig with a portion of suspected tuberculous tissue that has been emulsified, or with scrapings from suspected tuberculous lesions. The animal develops tuberculosis within the course of from four to six weeks, and always succumbs to the disease; its body will be found to contain many tubercles. If the animal should die at an earlier date, its death cannot be attributed to the action of the tubercle bacillus.

Bovine Tuberculosis.—Tuberculosis is common among cattle, as has been shown by the work conducted by the various health departments in practically all parts of the world. Ravenel, of Philadelphia, has performed conclusive and elaborate experiments with both bovine and human tubercle bacilli. His conclusions are, briefly, as follows:

"(1) That the tubercle bacillus from bovine sources has in culture fairly constant and persistent characteristics of growth and morphology by which it may tentatively be distinguished from that ordinarily found in man.

"(2) That cultures from the two sources differ markedly in pathogenic power, affording further means of differentiation, the bovine bacillus being much more active than the human for all species of experimental animals tested, with the possible exception of swine, which are highly susceptible to both.

"(3) The tuberculous material from cattle and from man corresponds closely in comparative pathogenic power to cultures of the tubercle bacillus from the two sources for all animals tested.

"(4) That it is a fair assumption, from the evidence at hand, and in absence of evidence to the contrary, that the bovine tubercle bacillus has a high degree of pathogenic power for man also, which is especially manifest in the early years of life" (Anders).

The sputum, feces, urine, and pus from ulcers and sinuses of tuberculous persons are among the various sources of infection, and in a series of experiments conducted by us at the Philadelphia Hospital we found that during the acts of coughing, sneezing, laughing, and talking patients suffering from advanced tuberculosis ejected a fine spray from the mouth that was found to contain virulent tubercle bacilli. Food-stuffs handled by persons suffering from tuberculosis of the respiratory tract are likely to become contaminated with tubercle bacilli, and are probably a potent factor in the spread of the disease.

CHRONIC TUBERCULOSIS.

Exciting and Predisposing Factors.—In all types of the disease the exciting factor is the tubercle bacillus, first described by Koch in 1881, and found in the blood-stream (in 10 per cent. of cases) and in the diseased tissues.

Incubation Period.—In tuberculosis produced experimentally it has been found that the guinea-pig and other laboratory animals develop the disease in from two to four weeks and die from it in from four to eight weeks.

Race and Nationality.—Sears, in a study of 200 cases, found that 50 per cent. represented either the first or the second generation of Irish immigrants. We have also observed that American children whose parents are of different nationalities show a marked inclination to develop pulmonary tuberculosis, this being especially true of immigrants from the Latin countries who marry natives of Ireland, England, and Scotland.

Race also exercises a decided influence, the African being especially susceptible to the disease when he takes up his residence in the northern States. We have also observed that an unusually high proportion of mulattos acquire the disease. Tuberculosis is common among the American Indians, a fact that may possibly be explained, in part, by their habit of eating uncooked meats. Some observers claim that an attempt to civilize the American Indian always ends in an increase in the number of cases of tuberculosis among them. The Mongolian race—the Japanese and Chinese—is prone to develop the disease on coming to America.

Age.—Tuberculosis may develop at any age, but certain forms of the disease are especially frequent among children—*e. g.*, meningeal, peritoneal, lymphatic, and bone (hip-joint and spinal) tuberculosis. Pulmonary tuberculosis is most commonly encountered between the ages of twenty and thirty and is quite rare during childhood and in old age.

Sex.—Females are said to be more susceptible to the disease than are males, yet this statement is not borne out by the statistics of Boston and Blackburn. When women afflicted with the disease become pregnant, the tuberculous process is likely to run an unusually acute clinical course. The fact that females take less outdoor exercise than males may, in a majority of instances, explain the frequency with which the former are afflicted with this disease.

Previous Diseases.—Tuberculosis not infrequently develops as a sequel of some one of the acute infections, although it is impossible to say definitely that in such instances some obscure tuberculous lesion did not exist prior to the development of the acute infection. Influenza, measles, pneumonia, whooping-cough, colds, acute bronchitis, and typhoid fever are not infrequently followed by the development of pulmonary tuberculosis. Tuberculosis of the lung may occur during the course of such chronic maladies as hepatic cirrhosis, diabetes mellitus, chronic interstitial nephritis, and the anemias.

Climate.—Humidity and excessive atmospheric moisture appear to increase the prevalence of the disease. Sudden variations in temperature probably predispose to the development of the disease more markedly than does any one other climatic condition, since such changes increase the susceptibility to contract acute colds. Altitude produces immunity in the native born inhabitants, as is shown in Colorado and New Mexico, where tuberculosis among the natives is rare. An exceptionally dry climate of

uniform temperature, such as that of southern California, is also unfavorable to the propagation of the disease.

Heredity.—This is estimated by different authors as being a causal factor in between 10 and 40 per cent. of cases. A child born of and reared by a tuberculous parent may be free from the disease at birth, but the environment certainly predisposes the child to infection. Children and other members of the same family are alike exposed to the disease. Tuberculosis is most likely to develop among the members of a family when the cooking is done by the afflicted person, since those suffering from pulmonary tuberculosis are likely to carry tubercle bacilli to the food they handle. Again, those afflicted with both pulmonary and laryngeal tuberculosis are continually clearing their throats, and are likely, in this way, to contaminate the food of others. It has been shown conclusively, as previously stated, that such patients disseminate a spray containing tubercle bacilli during the acts of coughing, sneezing, laughing, and talking; hence it is fairly reasonable to suppose that food prepared by tuberculous cooks will be contaminated with tubercle bacilli. As an illustration of the truth of the foregoing, the following case, which came under our observation, may be cited: Seven resident physicians in a hospital in Philadelphia developed pulmonary tuberculosis within the course of two years, and investigations made to ascertain the source of such infection disclosed the fact that the colored cook who prepared the food for the resident staff was suffering from a chronic form of pulmonary tuberculosis, both his saliva and his nasal secretion showing the presence of tubercle bacilli.

An *inherited* tendency to tuberculosis is more often transmitted through the mother than through the father, yet this statement, with reference to the transmission of the disease by the mother to her offspring, must be made guardedly. Women suffering from ill health at the time of conception and during the period of gestation are likely to produce offspring that show a special predisposition to develop tuberculosis as well as other infectious conditions. The children of syphilitics are very susceptible to the development of tuberculosis.

Pulmonary tuberculosis developing during childhood is, as a rule, unusually mild in form and is probably often overlooked until puberty or some later day, when the patient's general health becomes impaired, and permits the at one time encapsulated tuberculous process to assume an active stage, following which the clinical phases of pulmonary tuberculosis rapidly develop.

Acute Catarrh.—Acute catarrh of the respiratory tract provides a fertile soil for the development of the tubercle bacillus, and in consequence of this tuberculosis frequently follows acute colds and attacks of acute bronchitis, pharyngitis, and laryngitis.

Local Irritants and Wounds.—Factory employes are constantly exposed to the inhalation of particles of dust, and hence are prone to develop pulmonary tuberculosis; *occupation*, therefore, bears an etiologic relationship to this disease. As the result of the inhalation of irritating substances, glass-blowers, brass and metal workers, and coal-miners display a subacute or chronic bronchitis, following which tuberculosis is likely to develop. A single tuberculous focus may appear in the lung, and for a time many of the clinical features characteristic of either bronchopneumonia or lobar pneumonia may be present, tuberculosis being distinguished from these conditions largely by the fact that resolution does not occur. Pleurisy may be the exciting cause of a beginning tuberculous process that may, sooner or later,

become more or less general. (See Pleurisy.) Local tuberculosis of the mediastinal and abdominal lymph-nodes is occasionally encountered in children, and is rarely seen after middle life. Traumatism, sufficient to cause abrasion of the skin, may be followed by local tuberculosis, and it may also follow injury to the articular surfaces of certain bones and to the synovial membranes.

Modes of Infection.—(1) The *ingestion of the meat of tuberculous animals*, either beef or pork, is generally conceded to be one of the chief sources of infection. The milk of cows suffering from tuberculosis is a common source of infection in both children and adults.

(2) The *sputum and dejecta of tuberculous patients* are a prolific source of infection, the bacilli being carried directly from them by flies and other insects and deposited on the food of healthy individuals. Again, dried and partially pulverized sputum is blown about by the wind and may settle upon the food or upon open wounds. There is some question as to the virulence of the tubercle bacilli present in dry sputum and dejecta.

(3) As previously stated, the air of a room occupied by a person suffering from an advanced form of tuberculosis has diffused through it a fine spray that contains virulent tubercle bacilli. The inhalation of such spray is, in our opinion, capable of exciting tuberculosis.

(4) In those suffering from tuberculosis of the kidney or of any other portion of the genito-urinary tract the urine may contain many tubercle bacilli, and this excretion, when it falls upon vegetation, may serve as a means of infecting herbivora. A great number of tuberculous patients have taken up residence in the southwestern prairie-lands and in the far west, and have lived out of doors on the frontier. It is possible that the urine and feces of such patients serve as the source of infection for the cattle of those sections.

(5) *Direct Inoculation.*—Tuberculosis may result from the introduction of material containing the bacillus into open wounds, and this is the probable explanation of the origin of lupus. Ravenel has reported three cases of accidental inoculation of bovine tuberculosis in man.

(6) *Direct hereditary transmission* of the disease to the fetus *in utero* is exceptional, although authentic reports of such cases have been published. In the light of our present knowledge it is impossible to assert that any one of the previously mentioned modes of conveyance is the chief source of infection in man, although it is at least fair to assume that the ingestion of tuberculous meats or of milk containing tubercle bacilli is one of the more common sources of tuberculous infection in man.

Illustrative Case of Pulmonary Tuberculosis.—C. A., male, single, aged twenty-four years. An only child. Height, 5 feet 9 inches; weight since twenty years of age, 154 pounds, until the past four months, during which time his weight has fallen to 137 pounds.

Family History.—Parents living and in good health. No history of tuberculosis in ancestors.

Previous History.—The patient had the usual diseases of childhood before the age of twelve years; but between the twelfth and twentieth years he does not recall having consulted a physician. Since the age of twenty he has had repeated "colds," which, he states, he contracted readily, although he was not compelled to remain indoors at any time.

Social History.—The patient is a clerk by occupation, although he states that it has been his custom to take abundant outdoor exercise. He is not addicted to the use of alcoholic liquors or narcotics. He states that he has been irregular in his habits as regards the taking of food, and that, for the past year, he has not felt any inclination to eat in the morning, but that he would be able to take a fairly good meal by the middle of the day or during the evening.

Present Illness.—This probably dates from an attack of influenza, from which he suffered four months ago. Since this attack he has never fully regained his usual vigor, but, on the contrary, becomes easily exhausted, and although he is able to follow his usual vocation, he finds work extremely irksome. Violent exercise is followed by well-marked dyspnea, and for the past month he has been unable to walk against a strong breeze without suffering from extreme shortness of breath. Since the attack of influenza he has experienced a variable degree of dyspnea; he has hiccoughs; feels uncomfortable after the taking of food, and there is a decided distaste for fats.

Pain and Cough.—There has been no distinct pain at any time, although he experiences a variable degree of soreness at the base of the chest, and at times complains of a sensation of tightness in the thorax. Cough is more or less constantly present, and exposure to wet and cold, as well as exercise, always aggravates this symptom. The expectoration has been scanty, thin, and glairy, and when collected in a glass, showed a heavy, beaded froth. Irrespective of actual cough, the patient is continually clearing his throat, and a change from a warm to a cold room appears to aggravate this annoying symptom. Changes in posture, that is, upon retiring at night or upon rising in the morning, give rise to a paroxysmal attack of coughing.

Nervous Phenomena.—The patient declares that he is nervous and readily excited, and that he worries considerably over his physical condition.

Thermic Features.—At present, the night and morning temperature, taken over a period of six days, is normal or subnormal during the morning hours, but shows a rise of one to one and one-half degrees as evening approaches.

Physical Examination.—*General.*—The skin and mucous membranes are pale, and, as the result of emaciation, which appears to affect both muscles and adipose tissue, the skin is wrinkled.

Local Examination.—Inspection.—The ears project quite prominently; there is distinct playing of the nostrils; the cheeks are sunken; the patient usually breathes with his mouth open, and at the angles of the mouth a small quantity of saliva can be seen while the patient is talking. The typical so-called "phthisical" chest is not present, but there is a distinct depression at the right apex, and the supraclavicular and infraclavicular spaces of the right side are much deeper than those on the left. The shoulders are drooped, somewhat rounded, and the right is approximately one inch lower than the left.

Palpation.—At the apices of the lungs the two sides do not expand equally, there being some restriction on the right side. Tactile fremitus is slightly increased as low as the third rib at the right apex, and a distinct increased vibration is also felt over the right lung, between the vertebral column and the inner border of the scapula. The pulse is full and of fair volume, numbering 75 beats a minute, but becomes greatly accelerated upon slight exertion.

Percussion.—There is slight impairment of resonance over the apex of the right lung anteriorly, extending as low as the upper border of the third rib. No appreciable difference in the percussion-note is obtained over the two lungs posteriorly.

Auscultation.—The breath-sounds over the left apex anteriorly appear to be normal, but over the right apex, and as low as the third rib, breath-sounds are slightly bronchovesicular and roughened, and, when the patient is directed to clear his throat, a few, fine crackling râles are audible. The spoken voice-sound is abnormally well transmitted over the right apical area anteriorly, and along the inner border of the right scapula.

Laboratory Findings.—Repeated examinations of the sputum over a period of two and one-half months gave negative results as to the presence of tubercle bacilli. A guinea-pig to which a portion of the sputum was fed developed tuberculosis and died of the disease six weeks later, and at about the same time tubercle bacilli were first detected in the patient's sputum.

Calmette's ophthalmotuberculin reaction was positive four weeks before it was possible to confirm the diagnosis through the feeding experiment, and von Pirquet's needle-track tuberculin reaction was also positive.

Diagnosis by Induction from Clinical Data.—Certain facts set forth in the clinical history figured prominently in reaching a correct diagnosis; among these are: a young individual who had previously been in good health lost 17 pounds in weight within a comparatively short period. He had also been subjected to repeated attacks of what he termed "acute colds," the present illness dating, in the patient's estimation, from an attack of influenza. He was unable to eat at the morning meal, had a distaste for fats, and was often nauseated upon rising after a night's sleep. He further complained of a sense of fullness after the ingestion of food, and, indeed, symptoms of gastro-intestinal catarrh were present. Dyspnea had developed gradually, and was often accompanied by violent coughing. The fact that he coughed after a night's sleep and

on change of position was also suggestive of tuberculosis. Again, importance was attached to the fact that during exacerbations of coughing he frequently vomited. Although tuberculosis was suspected early, it was impossible to confirm such a diagnosis by microscopic examination of the sputum, but when the sputum was fed to a healthy guinea-pig, tuberculosis was produced in the animal, a piece of evidence that confirmed the diagnosis; later, the detection of tubercle bacilli in the sputum made the diagnosis positive.

Differential Diagnosis.—The symptoms of gastro-intestinal catarrh were prominent during the early course of the disease, and, indeed, practically overshadowed the other features of beginning pulmonary tuberculosis. A correct diagnosis was attained by the following facts: (*a*) Progressive loss in weight; (*b*) such loss following an attack of influenza; (*c*) almost constant cough and clearing of the throat; (*d*) the patient displayed an intolerance for fats; and (*e*) healthy animals fed upon the sputum developed tuberculosis, and later tubercle bacilli were found in the patient's sputum.

Course of the Disease.—This was influenced entirely by judicious treatment, the patient being immediately removed from the city to the mountains, and there placed under the care of a physician. One year later this patient was rid of all annoying symptoms, cough, weakness, shortness of breath, etc., and weighed 160 pounds. Physical examination still showed impairment of the percussion-note at the right apex, and over this area the breath-sounds were still slightly bronchovesicular, yet there was no evidence of an active inflammatory process in the lung. The patient returned to the city and has remained in good health.

ACUTE TUBERCULOSIS.

General Remarks.—A fact to be emphasized in connection with this variety of tuberculosis is that an old tuberculous focus is present within the body. Apart from this primary lesion, the pathologic lesions consist of widely disseminated tubercles. Their most frequent seats are in the lungs, liver, and spleen, and less commonly in the marrow of the bones, the heart, the kidneys, the choroid, and the meninges. This form of tuberculosis is characterized by the rapid development of miliary tubercles in many and widely separated parts of the body. In certain cases the tubercles are quite evenly distributed throughout all the organs of the body, manifesting the symptoms of an acute general infection. In other instances there is a tendency to localization of the tuberculous lesions, *e. g.*, in the lungs (pulmonary variety) or in the meninges (meningeal variety).

The fact that miliary tubercles may exist in different organs of the body (liver, heart, etc.) without giving rise to definite symptoms is a clinical fact of considerable moment.

MILIARY TUBERCULOSIS

(ACUTE GENERAL TUBERCULOSIS; ACUTE DISSEMINATED TUBERCULOSIS).

General Remarks.—This type of the disease probably results, in the majority of cases, from the rupture of a tuberculous nodule into a vein (rarely into a lymphatic vessel), following which tubercle bacilli are disseminated by the blood-stream to all parts of the body. Irrespective of the site of origin, the condition is at first an acute, generalized infection, and may continue as such for an indefinite period; but later in the course of the disease the symptoms pointing toward tuberculosis become more or less localized—that is, meningeal or pulmonary symptoms are likely to develop. In some cases meningeal symptoms may be the first positive evidence of the existence of tuberculosis, whereas in others pulmonary symptoms may be pronounced from the onset.

Clinical Varieties.—(1) General or typhoid form; (2) acute tuberculous meningitis (see Diseases of the Meninges, p. 1125); (3) acute miliary pulmonary tuberculosis; (4) chronic tuberculosis, and (5) tuberculous adenitis.

GENERAL MILIARY TUBERCULOSIS (Typhoid Form).

Clinical Features.—The general condition of the patient points conclusively to a severe grade of infection, which resembles a severe type of typhoid fever. The patient complains first of malaise, weakness, chilly sensations, impairment of appetite, and may occasionally experience one or more mild attacks of epistaxis. At times the disease develops *abruptly*, the patient becoming very ill by the end of the first forty-eight hours. There may be *cough*, with shortness of breath upon exertion, but expectoration is, as a rule, scanty or absent. *Prostration* soon becomes extreme, and within the course of a few days there may be diarrhea, mental dullness, delirium, and stupor.

Thermic Features.—The fever rises more rapidly, as a rule, than in typhoid, although there may be a gradual daily elevation until the temperature reaches 103° to 104° F., when there is likely to be well-marked morning remissions, although an evening remission with a morning exacerbation is occasionally observed. Rarely, indeed, the fever is not a conspicuous feature, and a subnormal temperature may be present.

Physical Signs.—Inspection.—The face is flushed; the patient, as a rule, lies upon his back; the tongue is heavily coated, and as the disease progresses it becomes brown, fissured, and often bleeds. Sordes accumulate about the teeth and lips. The respirations become somewhat hurried as the disease progresses, and late during its course there is evidence of cyanosis of both the mucous surfaces and the extremities. Jaundice is rarely seen, as is also a petechial eruption. An ophthalmoscopic examination may reveal the presence of choroidal tubercles. If the peritoneum becomes studded with miliary tubercles, abdominal distention follows, and a cyanotic areola may be seen surrounding the umbilicus.

Palpation.—The *pulse* increases gradually with the progress of the disease, reaching 120 to 160 a minute, and becomes weak, dicrotic, and compressible. The spleen is moderately enlarged, and can usually be felt beneath the costal border. Abdominal tenderness is, in typical cases, conspicuous by its absence.

Percussion shows the area of splenic dullness to be increased, and there may be a moderate increase in the area of hepatic dullness.

Auscultation.—Numerous fine crackling and moist râles are usually audible over the base of the lungs posteriorly. The heart's action is rapid, and, late in the disease, the muscular tone is deficient.

Laboratory Diagnosis.—With the progress of the disease there may be a moderate amount of sputum in which tubercle bacilli are rarely present. Tubercle bacilli may also be present in both the feces and the urine throughout the entire course of the disease. As a rule, they are commonly present in the circulating blood, a clinical fact that can best be proved by inoculating a guinea-pig with a small quantity of the patient's blood.

Differential Diagnosis.—It is at times extremely difficult to distinguish between *acute ulcerative endocarditis* and this form of miliary tuberculosis. The extreme irritability of the heart during the early stage of the illness favors the existence of endocarditis, whereas a cultural study of the blood makes the diagnosis of endocarditis positive in the event of pathogenic bacteria being cultivated from the venous blood. The venous blood in miliary tuberculosis is capable of producing tuberculosis in animals. Leukocytosis is commonly found in endocarditis, and is unusual in uncomplicated cases of tuberculosis. The distinctive features between the typhoid

form of miliary tuberculosis and typhoid fever have been discussed at length under the Differential Diagnosis of Typhoid Fever. (See p. 754.)

ACUTE TUBERCULOUS MENINGITIS.

This type of tuberculosis may be localized to a certain portion of the meninges, or there may be a miliary tuberculous involvement of the greater portion of the meningeal surface. The symptoms, however, vary in direct ratio with the degree of pathologic change that takes place in each given case. (For clinical characteristics of tuberculous meningitis see p. 1125.)

ACUTE MILIARY PULMONARY TUBERCULOSIS.

Pathologic Definition.—A pathologic condition resulting in the production of disseminated tubercles, principally throughout one or both lungs in a generalized infection.

Clinical Picture.—In those cases in which the miliary infection is chiefly localized to the pulmonary tissue the *onset* may be sudden. More commonly, however, the symptoms of a general infection are present, to which are added *cough, increased respiration,* and *pleural pain.* The *sputum* becomes mucoid and mucopurulent quite early in the disease, and in certain cases rusty and blood-streaked sputum is seen in which tubercle bacilli are present.

Thermic Features.—The fever develops early, and usually fluctuates between 102° and 103° F., although more marked remissions and exacerbations are occasionally shown.

Physical Signs.—Inspection.—The patient displays extreme prostration and profound dyspnea, and cyanosis appears upon even the slightest exertion. The respirations are rapid, panting in character, and usually number between 40 and 80 a minute in children.

Palpation confirms inspection in regard to the frequency of respirations. The pulse is rapid, even during the first few days of the illness, and continues to increase in frequency as the disease progresses, reaching 120 to 140 or even 160 beats a minute.

Percussion.—In typical cases percussion reveals no definite signs as to the pulmonary condition, but in those cases in which small areas of consolidation are present percussion may show localized areas in which the note is impaired, and it is customary to find a hyperresonant zone immediately surrounding the area of impairment. The area of splenic dullness is enlarged, and may even extend below the costal border, and in some cases the area of hepatic dullness is also increased. It is possible, at times, to have effusion into the pleural cavity as a coexisting feature of this type of tuberculosis, in which event the physical signs are confusing and demand careful analysis. (See Pleural Effusion, p. 136.)

Auscultation.—At first the breath-sounds are merely increased in intensity, and there is but little change in character, but as the disease progresses numerous dry and moist râles are heard. If the pleura is involved, a distinct pleural friction murmur is audible, and occasionally a pleuropericardial murmur, synchronous with both the heart and the respiratory sounds, is present.

Laboratory Diagnosis.—The sputum may or may not contain tubercle bacilli; but the bacilli are more likely to be found late than early in the course of the disease, and they may also be present in the feces, the urine, and in the peripheral blood. (See Tuberculin Reactions, p. 806.)

Summary and Differential Diagnosis.—The severity of the dyspnea, the rapid respirations (60 to 80 a minute), and the extreme cyanosis, when present in adults, point strongly to the existence of acute miliary tuberculosis of the lungs. Failure of the fever to terminate by crisis differentiates this condition from lobar pneumonia, and an absence of the pre-existing conditions known to antedate bronchopneumonia is also serviceable in differentiating these diseases. The detection of tubercle bacilli in the sputum, urine, or feces should always be regarded as conclusive evidence of the existence of tuberculosis of the lung.

Clinical Course.—The disease progresses from bad to worse, ending in a fatal termination in from eight to twelve weeks.

ACUTE PULMONARY TUBERCULOSIS

(ACUTE PNEUMONIC PHTHISIS).

Varieties.—Two clinical varieties are recognized: (1) The *pneumonic form*, in which the clinical features of the disease are those of an extensive lobar pneumonia, the disease running its course, in typical cases, in from two to six weeks, although rarely it may be protracted to from twelve to sixteen weeks.

Diagnosis.—This particular type of tuberculosis is to be distinguished from **lobar pneumonia**; the distinctive clinical features between these two conditions have been set forth in the table on p. 784.

Bronchopneumonic Type.—*Clinical Picture.*—Here the patient is likely to have a *chill*, or possibly a series of chills, followed by *high fever* of an irregular type. Coincidentally with the development of the chill and fever the *pulse-beats* and *respirations* are greatly increased. *Hemoptysis* may be an early symptom, and is soon followed by extreme prostration, rapid loss in weight, and profuse night-sweats (*galloping phthisis*). Early during the disease *cough* may be an annoying symptom, but it is not accompanied by expectoration. Later, however, the expectoration may become profuse, and may contain both elastic tissue and tubercle bacilli.

Physical Signs.—These are indefinite during the early stage of the disease, and, indeed, only the signs of bronchitis may be present until the disease is well advanced, when, as a rule, there are evidences of the formation of small areas of pulmonary consolidation—impairment of the percussion-note, bronchial and bronchovesicular breathing, accompanied by numerous râles. The physical signs just mentioned may be unilateral or bilateral, the latter distribution existing in a large proportion of all cases.

As the disease progresses the evidences of softening and cavity-formation appear, and the patient may go into the so-called typhoid state.

Differential Diagnosis.—**Bronchopneumonia** simulates closely the bronchopneumonic form of tuberculosis, and the distinction between these conditions is made, first, from a clear history of the absence of tuberculosis in other members of the family, or of association with those afflicted with the disease; and, second, upon the detection of tubercle bacilli in the sputum or excreta. The signs of cavity-formation, when they develop, are almost conclusive evidence of the existence of pulmonary tuberculosis.

Typhoid Fever.—When this disease is accompanied by an unusual degree of bronchial irritation, tuberculosis may be suspected. The presence of the Widal reaction, the absence of tubercle bacilli in the sputum, and the

prominence of abdominal symptoms (iliac tenderness, gurgling, diarrhea, and tympanites), together with the history of an existing epidemic, would warrant a diagnosis of typhoid fever. During the past year we have studied three cases (all negroes) in our hospital service in which typhoid fever and acute pneumonic phthisis were present in the same individual; in each instance the diagnosis was confirmed at autopsy, and in each the ophthalmo-tuberculin reaction and the Widal reaction were present.*

CHRONIC PULMONARY TUBERCULOSIS

(CHRONIC PULMONARY PHTHISIS).

Remarks.—In this form of the disease the onset is gradual, and at times insidious. In exceptional instances one of the acute types of tuberculosis previously described may merge into the chronic form.

Clinical Varieties.—(1) **Initial** or **incipient phthisis,** in which both the physical signs and the symptoms are indefinite until tubercle bacilli appear in the sputum. This clinical form may continue for months or even years, and if judicious treatment is instituted, may terminate in recovery.

(2) **Advanced tuberculosis** is a condition that prevails after the development of definite physical signs referable to the pulmonary system, although during this stage many symptoms more or less characteristic of the disease are displayed.

INCIPIENT PHTHISIS.

Clinical Picture.—The patient usually complains of an increased sense of languor, weakness, and moderate but progressive loss in weight. He may also experience chilly sensations at different times during the disease. The appetite is poor, and fatigue and dyspnea follow even slight exertion, and the patient has for months, and probably years, been unable to take food rich in fats. A distaste for fats is a highly significant symptom. Not infrequently gastric disturbances—*e. g.*, anorexia, with nausea and possibly vomiting after a night's sleep, epigastric distress, eructations of acid substances, and flatulency—are among the early symptoms, and, indeed, it is often for these that the patient consults the physician, believing that he is suffering from gastric disease. Jacob's report† of 92 cases shows gastro-intestinal symptoms to be present in 91 per cent. of his series. We have studied a large number of cases of beginning pulmonary tuberculosis in which the gastric symptoms practically overshadowed all evidence of disease of the respiratory tract, and in which the associated anemia and laboratory investigations made the diagnosis possible. Occasionally slight pleuritic pains are experienced, and if prostration is well marked there is profuse sweating at night. Cough is a common symptom, although in many cases it is but slight, and may be accompanied by but a moderate amount of glairy expectoration. In selected cases the first symptom to arouse the patient's alarm is the expectoration of blood and blood-streaked material, a symptom that, in North America, is highly suggestive of the existence of pulmonary disease.

Thermic Features.—Slight fever is ordinarily present at some time during the twenty-four hours, and in certain cases chills, followed by exacerba-

* See also "The Relation of Typhoid Fever to Acute Tuberculosis," J. M. Anders, Amer. Jour. Med. Sci., May 4, 1904.

† New York Med Jour., Feb. 8, 1913.

tions of temperature and profuse sweating, are present; it is in this particular class of cases that the disease in a measure simulates malaria.

Physical Signs.—These may be indefinite at the onset, but within the course of a comparatively short time—a few weeks or months—they become plainly manifest.

Inspection.—The characteristic phthisical chest, which is abnormally long and narrow, with widened interspaces, altered angle of the ribs, a conspicuous degree of flattening at the anterior surface of the chest, is often present; but by no means do all those developing tuberculosis display this type of thorax. In the African negro and in mulattos and quadroons the chest is generally very flat. Expansion is usually limited to one or other apex, and depression of the supraclavicular and infraclavicular spaces is often present early, and, as a rule, more marked upon one side (Fig 291). The mucous membranes may be anemic, and evidences of emaciation, with extreme pallor, may also be manifest.

Fig. 291.—Chest of Individual Predisposed to Pulmonary Tuberculosis.

Palpation.—In those cases in which the initial site of pulmonary inflammation is at one or the other base, nothing positive is revealed by palpation until a later stage in the disease is reached. If the apex of one lung is involved, the tactile fremitus is appreciably increased early, especially if the involved area is near the anterior surface of the chest-wall, or situated near that portion of the chest posteriorly that is uncovered by the scapulæ. As the disease progresses the fremitus may be increased over a large portion of the surface of the chest. Tactile fremitus may be increased as the result of pleural adhesions, a clinical fact of considerable moment, especially when the increase is over distinctly localized areas at different portions of the chest.

Mensuration.—In the vast majority of cases the chest expansion is below that of the normal; this is true even of the earliest cases, and later deficient expansion of one side of the chest becomes marked. Chronic pleurisy also influences the measurements of the chest. (See Pleurisy, p. 156.)

Percussion.—Resonance is impaired over the affected area, and as the disease progresses distinct dullness will be elicited when sufficient consolidation has resulted. Impairment of the percussion-note is more likely to be detected at the right than at the left apex. In selected cases the impairment may first become manifest along the spine and just within the inner border of the scapulæ when the arm is drawn well across the chest.

Auscultation.—Extremely fine, moist, crackling râles are audible directly over the area of involved lung, and are heard most distinctly at the end of inspiration. Slight prolongation, roughening, or hoarseness of the expiratory murmur are among the earliest signs of pulmonary involvement.

Later the expiratory murmur becomes harsh, and may even be rasping in quality. The respiratory movements are at times interrupted and jerking in character, giving rise to the so-called "cog-wheel" breathing. As the disease progresses the harshness of the breath-sounds gradually increases, until they become bronchovesicular in character. The expiratory murmur becomes high-pitched and is distinctly prolonged. Both the spoken and the whispered voice-sounds are increased immediately over the affected lung.

Caution.—In those cases in which tuberculosis begins in the form of pleurisy, the signs previously outlined as characteristic of incipient pul-

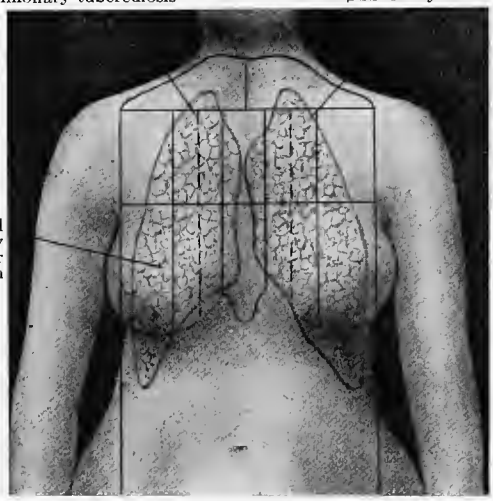


FIG. 292.—NORMAL POSITION OF LUNGS. VARIOUS AREAS ATTACKED BY TUBERCULOSIS.

monary tuberculosis are masked to a greater or lesser extent, depending upon the degree of pleuritic change that resulted from the initial attack. (See Fibroid Pleurisy, p. 137.)

Laboratory Diagnosis.—The sputum is glairy, often of watery consistence, depositing but a slight amount of sediment at the bottom of the fluid upon standing. The presence of tubercle bacilli confirms the diagnosis. Later in the disease the sputum may become profuse, thick, and mucopurulent in character. Tubercle bacilli may be found in the blood, feces, and urine. The total acidity of the gastric contents is normal or increased, as shown by Jacob's report of 50 cases. A persistent albuminous sputum is highly significant of a tuberculous lesion of the lung, and may exist for a definite period antecedent to the detection of tubercle bacilli.

Cutaneous Tuberculin Reaction.—In May, 1907, von Pirquet* communicated to the Berlin Medical Society the discovery that the application of a small quantity of tuberculin to a denuded surface of the skin produced in persons suffering from tuberculosis a characteristic reaction that was absent in healthy individuals.

When tuberculin is injected hypodermatically in a tuberculous subject,

* Wien. med Woch., July 6, 1907, p. 1, 369.

three reactions take place: (1) The focal reaction—a congestion in tuberculous lesions; (2) pyrexia; and (3) the needle-track reaction. By introducing tuberculin into the superficial layers of the skin, as in vaccination, the first two reactions are minimized, and slight redness and swelling, corresponding to the third reaction, alone result. Of 700 vaccinated cases, pyrexia occurred in but 3.

This method is quite similar in its application to that of vaccination against smallpox: The skin of the arm is first cleansed thoroughly with ether, after which two drops of Koch's old tuberculin, diluted with 1 part of a 5 per cent. solution of phenol in glycerin and 2 parts of normal salt solution, are applied to the surface of the skin drop by drop. At the point at which the tuberculin comes in contact with the skin the cutaneous surface is punctured with a platinum-iridium needle that has been sterilized in a flame. The surface of the skin is now covered with cotton-wool, the covering being allowed to remain for five minutes; further dressing is unnecessary.

Reaction.—In a tuberculous subject the skin at the site of inoculation becomes red and swollen within twenty-four hours, and a small papule develops. The diameter of the papule averages 10 mm. The color, at first, is a bright red, which fades, leaving a pigmented spot that is visible for weeks. In children the stages are passed through more rapidly, so that no trace of a positive reaction may remain on the sixth day. The intensity of the reaction varies considerably, and in weak children the hyperemia may be slight and the papule hardly visible. In exceptional cases vesicles form. The typical reaction reaches its height in from twenty-four to forty-eight hours. A delayed reaction occasionally occurs in older children, who show no evidence of tuberculosis clinically; this possibly indicates that tuberculous foci were present, but are now healed.

Significance.—Von Pirquet found this reaction positive in all but 11 of 80 cases of tuberculosis in children. It has been further found that, late during the course of tuberculosis, both adults and children may not display a characteristic reaction. In 113 healthy infants the reaction was positive in but 5, and 3 of those showing a positive reaction subsequently died, the autopsy revealing tuberculous lesions. The real clinical worth of von Pirquet's reaction in the diagnosis of obscure tuberculosis cannot as yet be estimated.

Ophthalmotuberculin Reaction.—This reaction was described by Calmette in "La Presse Medicale," June, 1907, and has since been confirmed by observers from all parts of the civilized world.

Technic.—(1) Place one drop of a 1 per cent. solution of dried tuberculin in sterile water or in normal salt solution in the eye of the suspected patient. If the patient is suffering from tuberculosis, the following local reaction will be observed: (2) Within about three hours following the application of tuberculin to the eye the conjunctiva of the eye thus treated becomes reddened, the congestion increasing until, within the course of several hours, an acute mucopurulent inflammation of the conjunctiva occurs (typical positive reaction). (3) The maximum reaction is usually seen within from six to seven hours after the application of the tuberculin solution to the conjunctiva. (4) All traces of the conjunctival inflammation disappear within two or three days. (5) Ordinarily, the patient suffers little or no discomfort as the result of such treatment. (6) In non-tuberculous subjects, the application of a 1 per cent. solution of dried tuberculin to the conjunctiva gives a negative reaction.

Clinical Significance.—(1) Following the application of a 1 per cent.

solution of tuberculin to the eye a positive reaction favors the existence of tuberculosis. (2) A negative result does not disprove the presence of tuberculosis, since approximately 50 per cent. of advanced cases of the pulmonary type of the disease do not display the reaction. Ordinarily, mild forms of tuberculosis give a typical reaction. (3) Following the introduction of a solution of tuberculin into the eye there is produced a hypersensitiveness of the treated conjunctiva to tuberculin; this occurs even in non-tuberculous persons. (4) In tuberculous individuals, following the ophthalmotuberculin reaction, there is apt to be hypersensitiveness to tuberculin in both eyes. (5) During the course of typhoid fever, and especially throughout convalescence from this disease, the ophthalmotuberculin reaction may be positive.

The diagnostic value of Calmette's reaction has been tested by many observers, but the results differ so widely that positive deductions regarding the clinical or diagnostic significance of this convenient reaction cannot be drawn.

Tuberculin Test.—Tuberculin, given in carefully graduated doses and administered by a skilled clinician, may be said to be entirely harmless, its only danger lying in indiscriminate and careless administration. If administered in the presence of slight elevations of temperature, it possesses but little clinical significance, a temperature of over 99° F. being sufficient, in many cases, to interfere with diagnostic interpretations. The *temperature* of the patient should be taken at least twice daily (morning and evening) for a period of not less than three days before the tuberculin test is applied, and whenever possible, a longer period of observation is desirable. Still better, the patient should be placed on a two-hour temperature observation for forty-eight hours before the tuberculin is injected. If, during this period, the thermometer registers above 99.8° F., the injection should not be made.

Dilution.—The administration of tuberculin demands the utmost care in adjusting the size of the dose. It is well to dilute the dose at the time of administration, as the product may become inert after being diluted for more than forty-eight hours. A recognized method of dilution is to use 0.5 per cent. of phenol in distilled water. Preceding each injection everything used in the administration—the syringe, tubes, pipets, etc.—should be sterilized by boiling.

Dose.—The first injection should approximate one milligram, although certain investigators employ as much as 3 milligrams as an initial dose.

If no reaction follows the initial administration, a second dose may be given after the lapse of two days, during which time the temperature should be recorded every two hours. If the patient fails to react to smaller doses, the dose may be gradually increased to 5 milligrams.

Inoculation.—The skin should be thoroughly cleansed with alcohol or ether, rendered aseptic, and a sterile pad be placed over the point of inoculation and allowed to remain for at least twenty-four hours. Most clinicians prefer injecting deeply into the muscle, although some advocate subcutaneous inoculation.

The Reaction.—The reaction is attended with an elevation in the temperature ranging between one and four degrees, a feature which in itself explains the necessity for definite knowledge of the patient's thermic condition prior to the application of the test. The reaction usually takes place in about ten hours, but may be delayed to as late as the second day. In selected cases symptoms of a more or less profound constitutional disturbance, of one or two days' duration, may take place; such as chill, headache, malaise, restlessness,

pain in the back, limbs, and joints, nausea, and vomiting. The symptoms of the reaction usually subside within the course of a few hours.

A local reaction, when it can be recognized, is of clinical importance. (See von Pirquet's Reaction, p. 806.)

Clinical Significance.—The subcutaneous tuberculin test has a limited scope of diagnostic usefulness, confined to a small percentage of cases in which other methods of diagnosis have not given definite clinical results. Except in the hands of those thoroughly skilled in its use, this test is of questionable value.

A positive reaction must be attended by an elevation in temperature, constitutional disturbance, and the local manifestations previously detailed. The positive reaction may in rare cases be absent in infected individuals, and an equally positive reaction may be obtained in those apparently not tuberculous, but who, nevertheless, may have concealed inactive foci.

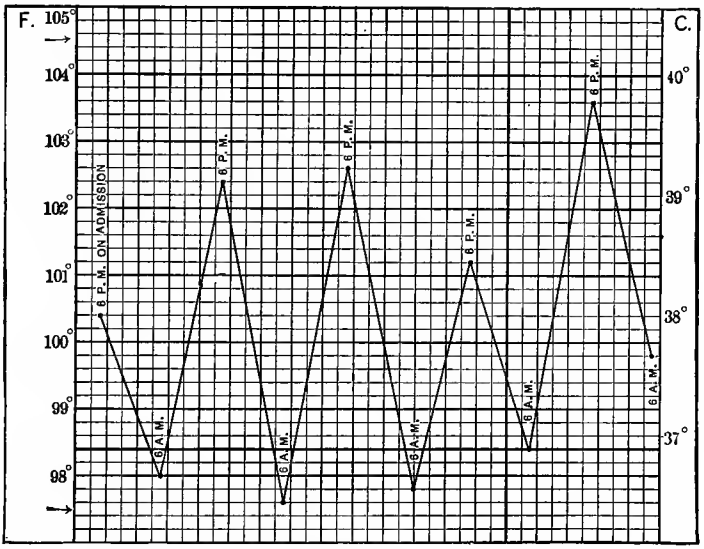


FIG. 293.—TEMPERATURE-CHART OF A CASE OF PHTHISIS.
Cavity in left apex, giving cracked-pot sound, Wintrich's sign, etc. George C———, aged twenty-two years; glass-worker.

Again, the fact that a quiescent tuberculous focus is commonly found at autopsy in those apparently not tuberculous materially detracts from the value of this test in the recognition of incipient pulmonary tuberculosis. In our experience no deleterious effects have followed the conservative application of this test.

Inhalation Tuberculin Test.—Tuberculin has been administered by inhalation through the agency of a vaporizer, but we have no knowledge of the efficacy of this method of administration.

ADVANCED TUBERCULOSIS.

Remarks.—Here practically all the symptoms that have been detailed under the incipient stage of the disease are intensified. The **cough** is markedly increased, and may keep the patient awake at night; it is often paroxysmal in character, the paroxysms being induced by changes in the patient's

posture—as from the erect to the recumbent, or vice versâ. *Pain* is a common and annoying symptom, and is especially severe in those cases in which the pleura is also involved. It may be limited to the upper portion of the chest, but is more likely to extend to the base of one lung. **Emaciation** is a prominent feature, and in those cases in which the loss of weight has been great, drenching night-sweats are common.

Among the **gastro-intestinal disturbances** are: anorexia, nausea, vomiting, flatulence, and constipation alternating with attacks of diarrhea. **Amenorrhea** is commonly present in young females, and may even antedate serious loss in weight; **menorrhagia** is rarely seen. **Pregnancy** is said by some observers to arrest the development of the tuberculous process, although the weight of professional opinion is to the effect that the disease assumes an unusually rapid course following childbirth. *Dyspnea* is a constant, and in many cases a most annoying, symptom, being present even in the early stage of the disease.

Nervous Symptoms.—As the disease progresses the patient becomes more and more convinced that his malady is curable, and not infrequently he attributes his condition to gastric or other disturbances. This hopeful attitude is a conspicuous feature of this stage of the disease. Delirium is not observed until a late stage is reached, and generally occurs during the afternoon or at night.

Thermic Features.—In advanced tuberculosis the fever, which was of the continued type in the incipient stage, becomes remittent or intermittent, and becomes a fairly reliable symptom after cavity-formation has taken place. Owing to infection of the lung tissue with pyogenic bacteria the temperature is generally intermittent, being characterized by afternoon and evening exacerbations (from 102° to 105° F.), with morning intermissions, during which it falls to normal or subnormal. In certain cases in which other evidences of extensive disease of the lung are present the temperature will be found to remain at or near the normal, and in still a smaller number the temperature may remain subnormal for days or weeks. A remittent temperature may be observed throughout the greater portion of this stage of cavity formation. That form of fever most commonly seen—normal or subnormal in the morning and markedly elevated in the evening—is generally referred to as septic, suppurative, or the so-called "hectic" fever (Fig. 293).

Physical Signs.—**Inspection.**—In practically all cases there is pallor of the skin and of the mucous surfaces, with undue prominence of the bony structures, the result of emaciation. The clavicles are apparently elevated, and the scapulæ stand out prominently, while the supraclavicular and infraclavicular spaces are greatly depressed, the depression being most marked on the affected side. In the African negro, owing to the anatomic formation of the clavicle, no great depression of supraclavicular and infraclavicular spaces can take place, even though the destructive changes at the apex of the lung are extensive. The characteristic phthisical chest, previously referred to under the incipient stage of the disease (p. 804), may or may not be observed. In those cases in which the lesion is situated in the left apex the impulse of the heart may extend over an unusually large area—from the second to the fifth interspace. The greater portion of the anterior surface of the heart may be uncovered, owing to the tuberculous process having caused sclerotic changes with retraction of the pulmonary tissue. The two sides of the chest may expand unequally, and late in the disease the movements of the chest may be vertical rather than expansile. There is

distinct playing of the nostrils concomitant with respiration, and the patient usually breathes with his mouth open.

Late in the disease the extremities and mucous surfaces may become decidedly cyanotic, and the skin shows yellowish-brown patches which are especially well marked over the anterior surface of the chest. The fingers are often clubbed (Fig. 121) during the incipient stage of the disease, and such clubbing becomes more conspicuous as the disease progresses. Edema of the feet and ankles is a late sign, and is a precursor of a fatal termination.

Palpation confirms inspection in regard to the movements of the chest and the area of diseased lung, even though such area be occupied either by consolidation or cavity-formation, since in cavity-formation there is a peripheral area of consolidation through which vibrations are well transmitted. The vibrations of the voice are diminished in those cases in which an abnormal thickening of the pleura or pleural effusion is present. The *pulse* is increased in frequency, and will become rapid, and later weak and irregular, depending upon the extent of the pulmonary lesion and the character of the bacteria with which it has become secondarily infected. A friction fremitus is present at times over certain portions of the affected side of the chest.

Percussion.—The entire chest, the healthy as well as the diseased portions, should be percussed, and special attention should be directed to percuss carefully over both the anterior and the posterior portion of the apex of the lungs, comparing the note of the two sides as obtained over the supraclavicular, supraspinous, infraclavicular, and interscapular spaces. When examining the interscapular regions, the patient should be directed to bend forward and fold his arms well across the chest, thus separating the scapulæ. It will be remembered that under normal conditions the percussion-note obtained over the right apex anteriorly is a trifle higher in pitch than that elicited over a corresponding portion of the left lung, but in well-developed and healthy individuals these differences are slight, and any deviation from the normal may be readily appreciated. When cavity-formation is present at one or other base, the upper portions of the chest may be apparently normal. In those cases in which extensive fibrinous changes are present in the pulmonary structure the percussion-note displays a peculiar wooden quality. If the pleuræ are markedly thickened, the note may approximate that of absolute dullness, and in such cases there is usually decided retraction of the affected area. Extensive consolidation of the lung also causes a variable degree of dullness, which is modified to a great extent by the amount of healthy or partially solidified lung that is interposed between the consolidated tissue and the chest-wall. Pleural effusion when encapsulated gives a distinctly flat note, and is to be distinguished from that resulting from consolidation. (See Pleurisy, p. 142.)

A tympanitic (dull tympanitic) note obtained upon making firm percussion over the upper portion of the lung may point to two pathologic conditions—(1) pulmonary cavity and (2) pulmonary consolidation—occupying the space between a large bronchus and the chest-wall—a rare, although possible, condition.

Myoidema is a condition of the muscular tissue of the chest in which, upon delivering a somewhat firm, quick blow with the percussing finger to a muscle near its tendinous insertion, a localized contraction of such muscle-fibers takes place, and by striking over the belly of a muscle, contraction may be seen to follow along its tendinous insertion. Myoidema was at one time considered a valuable sign in diagnosing pulmonary tuberculosis, but at present it is believed to indicate muscular irritability, and it is observed

most commonly when the tissues of the body are undergoing rapid emaciation. In the last stage of pulmonary tuberculosis myoidema is an almost constant feature.

Among the characteristic signs to be elicited over a pulmonary cavity should be mentioned Wintrich's sign, Gerhardt's sign, and Friedreich's sign. (See p. 62.)

Cracked-pot Sound.—This peculiar note is elicited by making firm percussion over a pulmonary cavity, provided the following conditions exist: (1) The cavity must be located near the chest-wall; (2) the wall of the cavity must be thin; (3) the cavity must communicate directly with a bronchus; (4) the chest-wall must be thin and relaxed; (5) the patient must be breathing with the mouth open at the time percussion is made. (See p. 61.)

Auscultation.—Both pleural and pericardial friction murmurs may be present at any time during this stage of the disease. The characters of the breath-sounds vary from a prolonged expiratory murmur through the

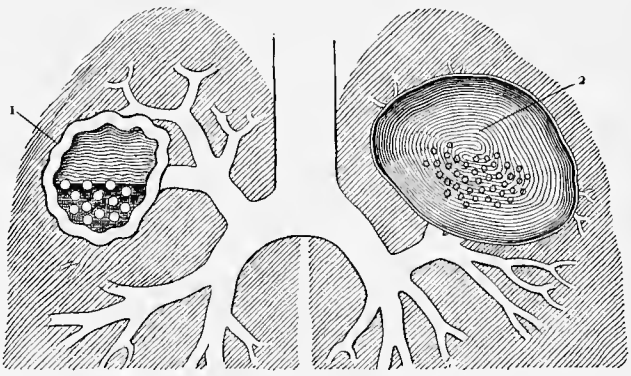


FIG. 294.

1, Small cavity near periphery, with thick relaxed walls, containing secretion and communicating with a bronchus; 2, large parietal cavity, with thin, tense, smooth walls, communicating with a bronchus (Anders).

successive stages of bronchovesicular and bronchial breathing. Irregular, jerking (cog-wheel) breathing is a highly significant symptom. Mention was made of the fine crackling râles heard during the incipient stage of the disease, and which may also be present over certain portions of the lung late in the affection. Râles are generally heard best at the end of inspiration, and practically all varieties of both dry and moist râles (see p. 69) are heard in this stage of tuberculosis. After cavity-formation has taken place the breath-sound immediately over such a cavity may display an amphoric element that is almost entirely dependent upon the tension of the wall of the cavity and the amount of consolidated tissue that exists between the cavity and the visceral pleura. The voice-sounds are transmitted with unusual intensity through the diseased lung and also through a pulmonary cavity. The whispered voice (pectoriloquy) will be found to vary greatly in different cases, but is more or less distinctly audible in the presence of pulmonary consolidation (consolidation surrounding a cavity). The most reliable signs of pulmonary cavity are a tympanitic percussion-note, amphoric breath-sounds, and amphoric voice-sounds, with or without râles.

Laboratory Diagnosis.—The **sputum** is mucous in character at first, and as the disease progresses it may become grayish or greenish-gray

in color. After cavity formation has occurred the sputum is often ejected in mouthfuls, and when expectorated into a vessel containing water masses about the size of a dime or even larger may be seen floating upon the surface; this is the so-called "nummular sputum." Albuminous sputum is common in tuberculosis and in other organic inflammatory processes of the lung, independent of whether or not there be blood in the sputum.

Hemoptysis is present in from 60 to 80 per cent. of all cases of pulmonary tuberculosis, depending, as it does, upon pulmonary congestion and upon erosion of a pulmonary vessel. Some writers claim that hemoptysis at times results from an aneurismal dilatation of a pulmonary artery. Frequent and profuse hemorrhages may occur, and, indeed, after extensive cavity-formation has occurred, fatal bleeding may ensue.

Microscopically, the sputum will be found to contain tubercle bacilli (Plate I[A]), pus-cells, leukocytes, alveolar epithelial cells, and yellow elastic tissue. Red blood-cells are present in those cases in which hemorrhage has occurred. Many pathogenic bacteria may also be present, the staphylococcus and the streptococcus being somewhat common. Both large and small diplococci, bacilli, and fungi may also be seen in the sputum after cavity-formation has taken place.

The **hemic** changes are those of secondary anemia, both the hemoglobin and the red cells becoming greatly reduced. As the result of infection of the pulmonary lesion with staphylococci and streptococci, leukocytosis is frequently present. Rarely, tubercle bacilli are found in the circulating blood, and when the venous blood is ejected into animals (guinea-pigs), such animals may develop general tuberculosis.

In tuberculous meningitis the cerebrospinal fluid may contain tubercle bacilli, although, as a rule, but few bacilli are present in this fluid, and a positive diagnosis is more often attained by injecting the spinal fluid into the pleural or the peritoneal cavity of a guinea-pig or a rabbit, the animal succumbing to the disease in from four to six weeks if tubercle bacilli were present in the injected serous fluid. During the advanced stage of pulmonary or of laryngeal tuberculosis the nasal secretions generally contain tubercle bacilli.

In tuberculosis of the skin the bacilli may be found in scrapings from the lesion. The pus from tuberculous sinuses, as a rule, contains but few bacilli, consequently bacilli are found with difficulty in this secretion. We have found tubercle bacilli in the semen of a case of tuberculous orchitis in which tuberculosis of other portions of the genito-urinary tract was also suspected.

The **urine** will be found to contain tubercle bacilli in all cases in which there are ulcerative changes of a tuberculous nature along the genito-urinary tract. In selected cases of pulmonary tuberculosis tubercle bacilli will be found in the urine, while autopsies performed on such patients do not reveal the presence of tuberculosis of the genito-urinary organs. In tuberculosis of the kidney and of the bladder the bacilli are usually found in clusters containing from six to twenty or more organisms. The diazo-reaction is present in from 7 to 12 per cent. of advanced cases.

Feces.—Tubercle bacilli are always present in the feces of those suffering from tuberculous enteritis, and are quite commonly found in the stools of incipient and advanced cases of tuberculosis. Rosenberger, in a series of more than 600 examinations of supposedly non-tuberculous patients at the Philadelphia Hospital, found tubercle bacilli present in the feces of many of them. His work raises considerable doubt as to the actual significance of tubercle bacilli in the feces, but nothing definite can be stated regarding this, as the work is of too recent date.

Tubercle bacilli may, in certain cases, be present in the discharge from the external auditory canal.

X-Ray Diagnosis.—There is probably no other field within the scope of internal medicine in which the application of the Röntgen rays is of greater service to the clinician than in pulmonary tuberculosis. Nevertheless, although this means of diagnosis serves to determine accurately the exact location and extent of the pulmonary lesions, it is not to be employed to the exclusion of other methods of physical diagnosis. By means of the Röntgen rays it is possible to detect the presence of minute tuberculous foci long before they can be recognized by other present methods of physical diagnosis. Without it, therefore, the clinician can never determine positively the number of foci present in the pulmonary tissue.

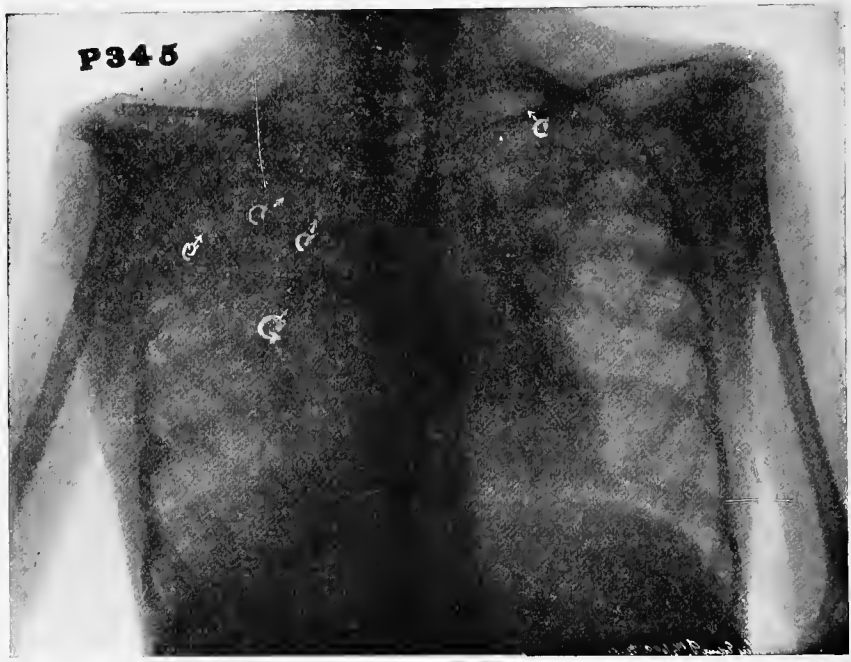


Fig. 295.—Tuberculosis in a Child (Pfahler).
Advanced disease of the right lung and of the left apex. *C*, Cavities; *G*, an old enlarged gland. Notice consolidation elsewhere.

Areas of consolidation 1 cm. in diameter may be recognized in emaciated subjects, and since such consolidations seldom occur singly, a more or less mottled appearance is produced. After these foci coalesce (Fig. 295), larger areas of consolidation, or their shadows, may be recognized and the degree of involvement of the affected lung may readily be appreciated by making a comparison with the picture of the opposite side and with uninvolved portions of the same lung. The true clinical significance of the *x*-ray plate can be interpreted only by one thoroughly skilled in Röntgenography, since areas of consolidation resting at different planes are brought upon the plate in one plane, a fact that tends to deceive the untrained eye and conveys the impression that the involved area is unusually large.

Calcified Tubercles.—These cast a decided shadow, and are readily distinguished from the shadow produced either by areas of consolidation or by scar tissue (Fig. 296). Areas of thick fibrous tissue (*e. g.*, old scars) (Fig. 296) cast a less dense shadow than calcified tubercles; consequently a large area is required for the production of a definite shadow, and here again skill in the interpretation of the plate is required to determine the actual nature of the lesion. It is highly important to recognize the exact nature of the last two described pulmonary lesions, and in the hands of a thoroughly skilled examiner such deductions provide invaluable data as regards both prognosis and treatment.

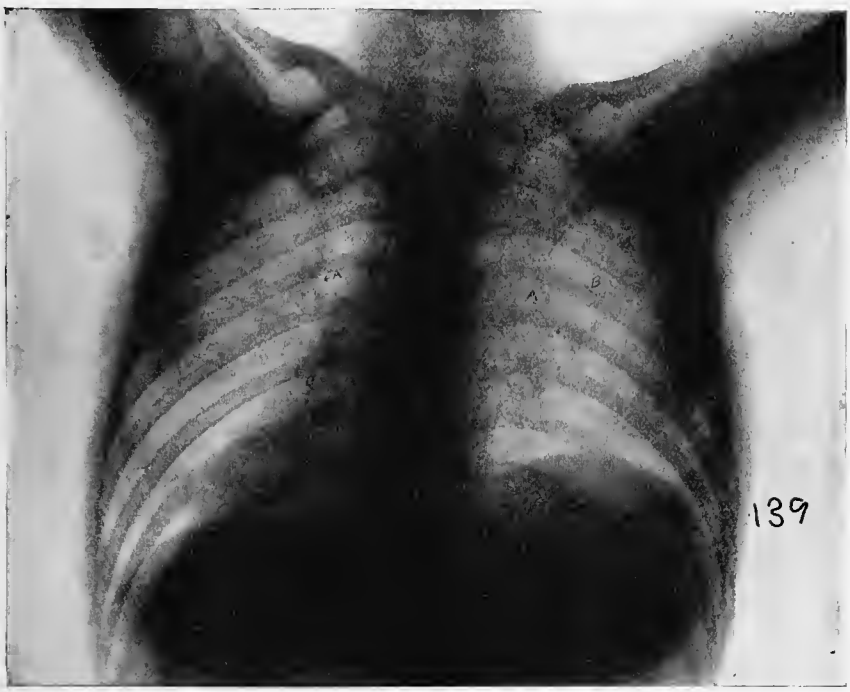


FIG. 296.—INCIPIENT TUBERCULOSIS (Pfahler).

Notice very small consolidations, detected with difficulty at autopsy. Followed tubercular peritonitis. *A*, Areas of congestion; *B*, small tubercles the size of a pinhead; C, branching pulmonary vessels.

Cavities.—These can always be recognized by their marked transparency and the surrounding shadow of consolidation (Figs. 295 to 297). Pfahler states that a large cavity can readily be recognized, but that a small cavity, surrounded by a comparatively large area of consolidation or covered by thickened pleura, is less easily recognized. Under favorable conditions, however, a cavity of 1.5 cm. may be detected.

Emphysematous Lung Tissue.—Emphysematous areas also show a noticeable degree of transparency, and this condition must be distinguished from cavity-formation (Fig. 297). "A dense shadow may be found on one side of this area, but it is likely to have the other side continuous with more or less healthy tissue, and, therefore, can be differentiated from a cavity" (Pfahler).

Summary of Diagnosis.—In the incipient stage of pulmonary tuberculosis the diagnosis is based on the following findings:

(1) A history of tuberculosis in the family or of association with those suffering from the disease. The patient may have suffered from an attack of pleurisy, influenza, or a severe cold during the past year.

(2) Prominent gastro-intestinal symptoms, accompanied by progressive anemia and emaciation.

(3) Before definite physical signs can be elicited, positive reactions may

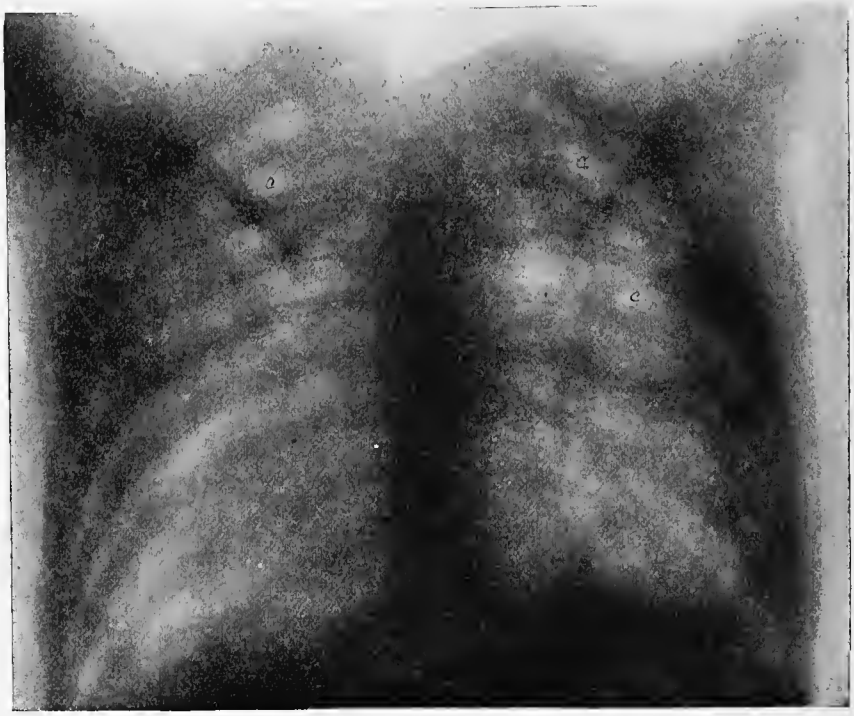


FIG. 297.—OLD TUBERCULOSIS OF THE LUNGS (Pfahler).
C, Cavities surrounded by consolidation.

be obtained from the use of tuberculin; when these are present, they strongly support the diagnosis. (See Calmette's Reaction, p. 807; Koch's Tuberculin Reaction, p. 808; von Pirquet's Reaction, p. 806.)

(4) The detection of localized impairment of resonance on percussion over some portion of the lung, and the evidence revealed by the *x*-rays (see p. 814), make the diagnosis reasonably certain before bacilli are to be found in the sputum.

(5) The detection of the tubercle bacilli in the sputum is conclusive evidence of the existence of a tuberculous lesion communicating with some portion of the respiratory tract. The sputum is, as a rule, rich in albumin, and this may be a conspicuous feature at an early date and before tubercle bacilli are detected.

During the second stage the diagnosis is rarely in doubt, the charac-

teristic features being: (1) The degree and character of the expectoration and the presence of tubercle bacilli in the sputum. (2) Cough is an important diagnostic symptom, especially when it is excited by change of position of the patient; at times it becomes paroxysmal, when it is likely to be accompanied by vomiting. (3) Progressive emaciation, night-sweats, and hectic fever are highly suggestive symptoms. (4) The presence of the characteristic physical signs further support the diagnosis. (5) Localized impairment of the respiratory movements over the affected area, depression of the chest-wall over the diseased part, localized dullness on superficial and deep percussion, a somewhat tympanitic or wooden sound upon making deep percussion if cavity-formation, surrounded by a comparatively thick

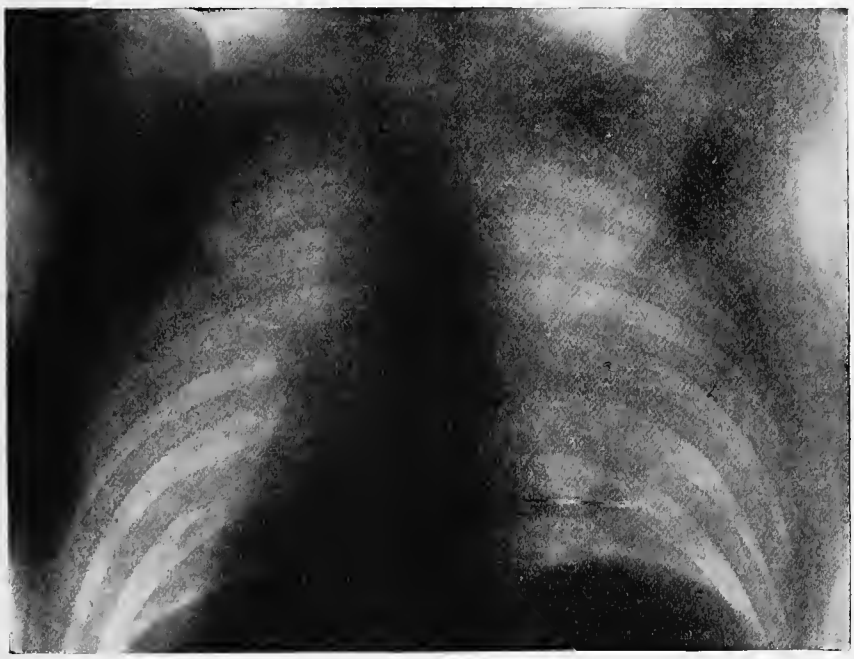


FIG. 298.—PLEURISY ON THE RIGHT SIDE (Pfahler).
A, Old tubercle; *B*, disease at the left apex.

wall of consolidation, is present. Wintrich's change of note (see p. 62) and the cracked-pot sound, if a superficial cavity communicates with a bronchus or is partially filled with fluid, are valuable signs.

(6) The presence of tubercle bacilli in the feces and urine support the diagnosis, although their recovery from these excretions should not be regarded as sufficient evidence on which to base a diagnosis of pulmonary tuberculosis, but would favor a diagnosis of a tuberculous lesion either of the alimentary or of the genito-urinary tract.

(7) After cavity-formation has occurred the *x*-ray diagnosis gives positive information. (See p. 815.)

Differential Diagnosis.—In the incipient stage the differential diagnosis is of serious moment, and, as a rule, its making is attended with

considerable difficulty. Here the clinical history, as previously mentioned under Diagnosis, together with a careful analysis of the signs and symptoms presented by the case in question, must be obtained in order that definite deductions may be drawn. The recovery of the tubercle bacillus from the sputum makes the diagnosis positive, and in the absence of this clinical evidence, the recovery of many tubercle bacilli in the feces should suggest, at least, the existence of tuberculosis. The employment of the cutaneous and ophthalmotuberculin reactions is to be considered in obscure cases.

Bronchial catarrh (apical catarrh) may simulate advanced tuberculosis in certain respects, the distinctive features of the former being that râles are heard over both apices, that tubercle bacilli are absent from the sputum, that there is but little fever, which is never hectic in character, and that emaciation and prostration are mild.

Bronchiectasis.—It is to be remembered that if a portion of a bronchus is expanded, the physical signs of pulmonary cavity are present; consequently the differentiation is based entirely upon the symptomatology and course. (1) The naked-eye appearance of the sputum in bronchiectasis (see p. 101) is quite different from that of pulmonary cavity, and again in bronchiectasis tubercle bacilli are absent. (2) Nutrition is comparatively good in bronchiectasis, whereas emaciation is progressive in phthisis. (3) Elevated temperature is rare in bronchiectasis, whereas in tuberculosis with cavity-formation hectic fever is the rule.

Streptothricosis (pseudotuberculosis) may rarely be confounded with pulmonary tuberculosis, and the distinctive features between these two conditions have been detailed on p. 925.

Actinomycosis.—This rare pulmonary disease may at times simulate tuberculosis, the points of difference between the two being discussed in detail on p. 922.

Clinical Course.—In incipient tuberculosis the clinical course may not only be materially modified by judicious treatment, but recovery may follow when such treatment is instituted and continued for a sufficiently long period. Those cases that go on to the second stage of the disease will be found to linger over an indefinite period, the length of which is somewhat influenced by the age of the patient—the younger the individual, the more rapid and the shorter the course of the disease. When pulmonary tuberculosis develops after the age of fifty, the patient's life may be but slightly, if at all, shortened as the result of the pulmonary condition, whereas in early adolescence—from fourteen to twenty-five—a more virulent type of infection is present and destructive changes in the lung develop early.

Complications.—**Pulmonary hemorrhage** is probably the most frequent complication, and is always to be feared during the course of the disease. When it develops during the incipient stage, it may not be of serious prognostic import. On the other hand, it is followed at times by decided relief from local symptoms.

Rupture of a cavity into the pleura, with subsequent development of pyopneumothorax, is a serious complication, and likely to terminate fatally, although in two instances coming under our observation recovery from the pneumothorax followed.

Tuberculosis of the larynx complicating the pulmonary type of the disease is a precursor of an early fatal termination, since nutrition is interfered with.

Enteritis is followed by severe depletion of the patient, and materially shortens the course of the disease.

Acute miliary tuberculosis may develop at any time, even during the incipient stage. (See Varieties of Miliary Tuberculosis, p. 800.)

Tuberculous pleurisy may have antedated tuberculous disease of the lung, and is likely to recur as a complication after cavity-formation has taken place, when there may be added the characteristic signs and symptoms of pleural effusion. (See p. 154.)

Amyloid disease of the liver, spleen, kidneys, and intestines is occasionally found at autopsy.

Peripheral neuritis and **acute endocarditis** are rarely seen to complicate this type of *tuberculosis.*

FIBROID PHTHISIS.

Pathologic Definition.—A type of chronic phthisis, characterized by the extensive formation of fibrous tissue at one and possibly at both apices. This fibroid change may continue for an indefinite period, until great deformity of the chest over the portion of the lung affected results.

Clinically, expansion over the affected portion of the lung is limited or absent; the percussion-note is dull, and the heart may be uncovered as the result of extensive sclerotic changes, with retraction of the lung. In other cases the heart may be drawn well to the right side of the body from contracture of fibrous bands. The respiratory murmurs are bronchovesicular or bronchial in quality, and the various types of râles (see p. 69) are likely to be present at different stages of the disease. Vocal resonance is, as a rule, diminished, although in a certain proportion of cases the voice sounds may be increased in intensity.

Diagnosis.—This is based largely upon the history of long-standing disease, with gradual deformity, and the detection of tubercle bacilli in the sputum.

Clinical Course.—The case seldom, if ever, terminates in less than ten years, and cases of twenty years' duration are far from uncommon.

TUBERCULOSIS OF THE SEROUS MEMBRANES.

Tuberculous Meningitis.

Pathologic Definition and Remarks.—A disease characterized by the development of tubercles on the pia-arachnoid of the brain and the spinal cord and by an increase in the quantity of cerebrospinal fluid, in which, in certain cases, tubercle bacilli may be found. (See Tuberculous Meningitis, p. 1125.)

Tuberculous Pleurisy.

Remarks.—In the majority of cases of acute pleurisy the tubercle bacillus figures as an etiologic factor. (See Predisposing and Exciting Factors, p. 154.)

Tuberculous Peritonitis.

Pathologic Definition.—A type of subacute or chronic inflammation of the peritoneum caused by the tubercle bacillus, and characterized by the development of tubercles of varying size, and by the accumulation of fluid in the peritoneal sac, in which tubercle bacilli may be demonstrated. The fluid will produce tuberculosis when inoculated into susceptible animals. (See Tuberculosis of the Peritoneum, p. 560.)

TUBERCULOUS ENDOCARDITIS AND PERICARDITIS.

See Pericarditis, p. 237.

TUBERCULOSIS OF THE LIVER.

Pathologic Definition.—A disease of the liver in which tubercles of varying size are found disseminated throughout the hepatic tissue. Owing to both its anatomic and its physiologic relations, the liver serves as a nidus for the lodgment of tubercle bacilli that have gained entrance to the arterial, venous, or lymphatic channels; consequently tuberculosis of the liver is, as a rule, a secondary condition.

Predisposing Factors.—Those suffering from any form of tuberculosis are especially likely to develop tuberculosis of the liver. Hepatic tuberculosis not infrequently follows a similar condition of the alimentary tract, particularly when the lesion is situated in the colon. Bone tuberculosis with caries serves as a marked predisposing factor to both the hepatic and the pulmonary types of the disease.

Clinical Features.—Among the constitutional features of hepatic tuberculosis are emaciation, prostration, and fever; the last may be but slight, and of such nature as not to indicate the existence of tuberculosis. There is symmetric enlargement of the liver, which continues throughout the entire course of the affection. *Pain* is uncommon until the peritoneal covering of the viscus becomes involved, when the symptoms will usually simulate those of chronic peritonitis. (See Tuberculosis of the Liver, p. 595, and Tuberculosis of the Peritoneum, p. 560.)

RENAL TUBERCULOSIS.

This form of tuberculosis has been discussed at length under the heading Disease of the Genito-urinary Tract. (See p. 685.)

TUBERCULOSIS OF THE BLADDER, PROSTATE, AND URETERS.

Remarks.—These forms of the disease, while less common than tuberculosis of other portions of the body, are often discovered at autopsy. They are more common in adults than in children. Their clinical characteristics have been detailed in the section on Genito-urinary Diseases, p. 705.

TUBERCULOUS ORCHITIS.

The testicles may be the site of tuberculous disease, which occasionally occurs simultaneously with involvement of the peritoneum and the kidney. In tuberculosis of the testicles the epididymis is appreciably enlarged, and may equal or exceed the remainder of the gland in size. Pressure may excite pain.

Tuberculosis of the testicle is a purely surgical condition, and the reader is referred to works upon surgery and genito-urinary diseases for a complete description of this condition.

TUBERCULOSIS OF THE OVARIES AND FALLOPIAN TUBES.

In this condition there is usually a family history of tuberculosis, but the other diagnostic measures within the scope of this volume are not sufficient on which to base a diagnosis of the condition. Later the symptoms become masked, as it were, by an associated tuberculous peritonitis, when the disease is treated by the physician as peritonitis.

Tuberculous Arthritis.

Pathologic Definition.—A subacute or chronic inflammation of the synovial membranes, due to the bacillus tuberculosis. Tuberculous arthritis is a surgical disease, and the reader is referred to special works upon surgery for an extended description of the subject. Some of the distinctive features between tuberculous arthritis and acute articular rheumatism, however, will be found on p. 896.

Tuberculosis of the Mesenteric Glands.

These glands are, as a rule, involved secondarily to tuberculosis of the intestines, although in children the glandular type of the disease appears to equal, and possibly may exceed, in frequency the truly intestinal type.

Tuberculosis of the Tracheobronchial Lymph-nodes.

Pathologic Definition.—A condition excited by infection of these structures by tubercle bacilli.

Predisposing Factors.—This disease is more common in children than in adults, although it may be met quite frequently after middle life in those suffering from pulmonary tuberculosis. Tuberculosis of other lymph-nodes, such as the cervical and the axillary, is usually present.

Symptomatology.—The most annoying symptoms are the *mechanical* ones, arising, as they do, from pressure excited by the lymph-nodes when they have become sufficiently enlarged. The mediastinal lymph-nodes are the ones usually affected, and if they should press upon the recurrent laryngeal nerve, paroxysmal cough, aphonia, and paroxysms of dyspnea follow. Pressure upon the superior vena cava is followed by extreme cyanosis, with swelling and edema of the face. Edema of the lower extremities may also result from pressure upon the circulatory apparatus. Fig. 133, p. 323, will serve to demonstrate the effect of pressure from mediastinal growths, as seen in a patient under our care.

In most instances the patient complains of *distress in breathing* upon exertion, and in some cases there is *pain* in the chest; *cough* with free expectoration may be a constant feature in those cases in which paroxysmal cough is absent. When cyanosis has continued for months, or even years, the parts of the body affected become distinctly pigmented.

Physical Signs.—It is usually possible to elicit the physical signs of consolidation in the mediastinum, and these signs are often equally prominent, or even more marked, in the interscapular space, between the second and fifth thoracic vertebræ. The breath-sounds are at times well transmitted along the borders of or over such enlargements; consequently the respiratory sounds are of but limited clinical value.

Laboratory Diagnosis.—The cough may be accompanied by the expectoration of an extremely tenacious sputum, although in those cases in which a bronchus is partially occluded as the result of pressure, copious expectoration is seen, and in these the symptoms may closely resemble those of bronchitis. (See p. 88.) Occasionally an enlarged lymph-node may break and rupture into a bronchus, such an accident being followed by the copious expectoration of caseous, semiliquid, blood-streaked material.

Summary of Diagnosis.—Paroxysmal cough, hoarseness, swelling of the face and neck, with undue prominence of the superficial veins of the

chest, and at times of the abdomen and extremities, are sufficient to suggest intrathoracic obstruction to the venous circulation. Tuberculosis of the lung is likely to follow, and may even precede, involvement of the bronchial lymph-nodes; consequently a history pointing to the existence of the former disease is to be considered. Tubercle bacilli are but rarely detected in the sputum, but their presence strongly supports the diagnosis of tuberculosis of the mediastinal lymph-nodes.

Differential Diagnosis.—Aneurism is to be distinguished from enlarged mediastinal lymph-nodes, a distinction that may be made from the following clinical facts: In aneurism there are, in addition to the signs and symptoms resulting from enlarged bronchial lymph-nodes, inequality of the radial pulses, hypertrophy of the heart, pulsation with shock over the area

FIG. 299.—TUBERCULOUS ADENITIS.
Female, aged sixteen years, treated at Philadelphia Hospital. Diagnosis confirmed by section of small gland which showed tubercle bacilli four months before death. Autopsy not permitted.

of consolidation, and a bruit, all of which features are unknown to glandular enlargement.

TUBERCULOUS CERVICAL ADENITIS.

Pathologic Definition.—Chronic enlargement of the cervical lymph-nodes resulting from infection with the tubercle bacillus.

Clinical Picture.—In typical cases the submaxillary lymph-node (Fig. 299) is the first to give evidence of disease, but subsequently there is enlargement of the lymph-nodes in the postcervical, supraclavicular, and scapular systems, and enlargement of the bronchial lymph-nodes may also follow. Tuberculous adenitis is, as a rule, a bilateral disease, although generally the lymph-nodes of one side enlarge in advance of the corresponding structures of the opposite side. All the involved organs enlarge slowly, and when such enlargement is first detected, it may vary in size from that of a pea to that of a hazel-nut; after the disease has progressed for months or years, however, it is unusual to see cases in which the lymph-nodes attain the size of a hen's egg. These enlargements, as a rule, occur in

clusters, are smooth, and seldom attach themselves firmly to the skin until just prior to rupture externally. If one or more lymph-nodes break down after undergoing caseation and liquefaction, fluctuation will be present.

Diagnosis.—Fluid obtained from an enlarged lymph-node may contain but few tubercle bacilli, although animals either fed upon or inoculated with such fluid are likely to develop tuberculosis. The characteristic growth of the structures, combined with the presence of slight fever, anemia, and progressive loss in weight, strongly favors a diagnosis of tuberculosis. There is also likely to be chronic catarrhal disease of the pharynx and larynx, and enlargement of the tonsils in those suffering from tuberculous adenitis. Suppurative otitis media, keratitis, and chronic conjunctivitis are also commonly present. Finally, chronic eczema of the external auditory canal and of the lips is not infrequent in connection with tuberculous involvement of the superficial glandular system. (See Special Tuberculin Reactions, pp. 806–809.)

Differential Diagnosis.—**Pseudoleukemia,** particularly at the onset, is more readily confused with tuberculous adenitis than is any other disease. Although acute tuberculous adenitis may closely simulate Hodgkin's disease and make a diagnosis between the two almost impossible, more often the lymph-nodes in tuberculous adenitis enlarge more slowly than they do in Hodgkin's disease. The extension of the lymphatic enlargement of tuberculosis is rare as compared with pseudoleukemia, and, again, tuberculous adenitis is more common in the young, is commonly unilateral in the neck, and attacks the submaxillary lymph-nodes more often than the structures along the sternomastoid muscle. Periadenitis, adhesion, and suppuration of the lymph-nodes are seen in tuberculosis, and tuberculous foci may also be found in other organs. Intermittent attacks of fever favor the presence of Hodgkin's disease. Some one or more of the tuberculin tests (see p. 806) should be employed in making a differentiation between these conditions, and, if necessary, a portion of a diseased organ may be removed for laboratory diagnosis. A hematologic study is necessary in every instance since chronic glandular enlargement may be a feature of true leukemia and the latter condition is recognized only by making a differential count of the leukocytes.

Fig. 300.—The Two Spirochetes in the Center are Treponema Pallidum; the Three Others, Spirochæta Refringens (Schaudinn and Hoffmann).

The therapeutic test serves as the most practical method for distinguishing between tuberculous adenitis and syphilitic adenitis.

SYPHILIS.

Pathologic Definition.—A chronic infectious disease, believed to be excited by treponema pallidum.

(*a*) **Primary Lesions.**—These consist of an infiltration of the connective tissue, chiefly with round-cells, of the same type as those seen in recent granulations. There is sclerosis of the smaller blood-vessels involving the adventitia of the arterioles, and the neighboring lymph-nodes undergo hyperplasia and induration.

(*b*) **Secondary Lesions.**—Macular, maculopapular, papular, and pustular lesions are seen on the cutaneous surfaces, and, with the mucous patch, show round-cell infiltration with plasma cells and leukocytes and changes similar to those found in chancre. The favorite sites for the appearance of mucous patches are the mucocutaneous junctions, *e. g.*, the mouth, anus, and vulva (Fig. 301). Other lesions appearing in this stage are general adenopathy, alopecia, and pharyngitis.

(*c*) **Tertiary Lesions.**—These consist of circumscribed inflammatory masses, known as gummata. They appear in the connective tissue, bones, periosteum, skin, muscles, brain, liver, lungs, kidneys, heart, testes, etc. Although usually sharply circumscribed, gummata may be diffuse, and may vary in size from that of a pin-point to a hen's egg. They tend to form ulcers. They are grayish in color, and on section show a caseous, semiopaque center, with a fibrous, translucent periphery.

Fig. 301.—Mucous Patches on Lip (Schamberg). Lesions are deeper and rather more excavated than usual.

Microscopically, the gumma consists of lymphoid cells, plasma cells, leukocytes, and epithelioid cells, in which fatty degeneration and softening result in the formation of a pasty mass. The mass thus formed may either be absorbed or persist; in most instances, however, coagulation necrosis, due to local anemia, takes place in the center, with conversion of the peripheral zone into fibrous tissue. The central caseous material may be absorbed, or may remain as a calcareous mass with a cicatrix. Destructive ulceration and sloughing may follow. Syphilis shows a special predilection to attack the nasal and frontal bones, the palate, and the tibiæ.

Varieties.—(1) Acquired syphilis; (2) congenital syphilis; (3) malignant syphilis; (4) visceral syphilis.

Predisposing and Exciting Factors.—Bacteriology.—Schaudinn and Hoffmann have described an organism which is now known as treponema pallidum (Fig. 300) which is found in the lesions of syphilis during all stages of the disease.

Acquired syphilis occurs only as the result of inoculation, a break in the cutaneous or mucous surfaces, *e. g.*, a slight abrasion, fissure, or laceration, particularly of the genital mucosa, being essential to infection. The lips and hands may be the seat of the lesions.

Susceptibility is universal. *Reinfection* is exceedingly rare, but may occur.

Contagion of Syphilis.—The blood of a syphilitic during the secondary period and the secretion from the chancre or any of the secondary lesions are contagious, the lesion at the point of inoculation always being a chancre. The physiologic secretions—saliva, sweat, milk, and urine—do not convey the virus unless they become contaminated by admixture with

the discharge from some of the lesions of the primary or the secondary stage. The semen is capable of infecting the embryo.

Modes of Infection.—(1) **Direct Inoculation.**—In approximately 70 per cent. of cases syphilis is transferred by sexual intercourse.

(2) **Accidental inoculation** most frequently results from kissing. "In Russia from 75 to 80 per cent. of cases is acquired in this manner from popular customs" (Anders).

The mouth and tonsils may be the site of inoculation, the virus being conveyed during the practices of sexual perverts. The wet-nurse may infect the suckling babe, or vice versâ. Fournier cites forty instances of chancre of the hand. Humanized vaccine virus may in rare cases transmit the disease.

Unusual Modes.—Accidental infection has, at times, though very rarely, taken place as the result of handling infected rags, clothing, drinking-cups, pipes, cigars, and the like; it has also been known to follow tattooing.

"Krafft-Ebing found that out of 3455 cases, 15.6 per cent. were of extragenital origin. The lesion was upon the lip in 51 per cent" (Anders).

(3) **Hereditary Transmission.**—Paternal transmission (through the semen) is more common than maternal. Appropriate treatment of a syphilitic parent greatly lessens the danger of transmission, and in such instances the danger of transmission after the third year of treatment is slight. A syphilitic father or mother may beget healthy offspring, the infants having acquired an immunity that protects them from infection by the mother (Profeta's law). Infected women not uncommonly bear syphilitic children. A woman who has become infected after conception may bear a syphilitic child, although, on the other hand, the infant may escape infection.

Fig. 302.—Pustular Syphilid (Welch and Schamberg). During an epidemic of variola this patient was sent into the smallpox hospital under erroneous diagnosis.

Immunity.—Those born of syphilitic parents may possibly possess a certain degree of natural immunity. A woman that has borne a syphilitic child may enjoy perfect immunity (Colles' law). One attack of syphilis usually bestows immunity.

ACQUIRED SYPHILIS.

Clinical Stages.—(1) **Primary Stage.**—The typical lesion—the chancre—makes its appearance in approximately three weeks after exposure, and within the course of a few days, or at most a fortnight, is followed by swelling and induration of the surrounding lymph-nodes. The primary lesion appears as a red papule that soon increases in size and shows a tendency to undergo central necrosis with the formation of a small ulcer. The tissue immediately surrounding the ulcer becomes appreciably hardened and of cartilage-like consistence—hence the term, "hard chancre." A single lesion is the rule, although two distinct chancres may be present. The chancre may be situated within the urethra, near the meatus, but is most likely to occur at or near the junction of the mucous membrane with the skin. We have seen a number of cases in which the initial chancre made its appearance on the skin, in the mouth, and in one instance upon the cervix uteri. The primary lesion of syphilis may be so slight as not to attract the attention of the patient, and, as a consequence, we not uncommonly see females in whom no initial lesion was observed display the secondary manifestations of the disease.

Fig. 303.—Serpiginous Syphilid; only Patches Present (Schamberg).

(2) **Secondary Stage.**—The manifestations of this stage occur within about six weeks after the appearance of the chancre, although a much longer interval may intervene between the appearance of the chancre and that of the secondary stage. The patient complains of *languor, a sense of indisposition, aching of the bones, anorexia* or *impaired digestion*, and a *moderate degree of prostration. Soreness of the throat* may be present, and at times the patient complains of *pronounced angina.*

Thermic Features.—Moderate fever is the rule, the temperature usually fluctuating between 100° and 101° F., although in exceptional cases it may run higher.

Skin and Lymph-nodes.—The *cutaneous manifestations* are polymorphous, although an erythematous or roseolar eruption is commonly the earliest cutaneous manifestation of this stage of the disease. The eruption is, as a rule, profuse upon the trunk, chest, back, buttocks, thighs, and forearms. Papules may also appear early, and will be found to vary greatly in size. The papular form of the eruption is frequently conspicuous on the face, trunk, and flexor surfaces of the extremities. The lesions are rounded and symmetrically distributed on the two sides of the body. These lesions are, as a rule, symmetric, their outlines being more or less perfectly rounded, and in color they resemble that of a slice of beef, displaying a slight coppery hue. These lesions are usually not accompanied by either itching or pain.

The *lymph-nodes* are appreciably enlarged, especially in the cervical and epitrochlear regions.

Mucous Membrane.—Mucous patches are seen to occur upon the mucous surfaces, especially at the angles of the mouth, on the tongue, upon the tonsils, pharynx, and vulva, and about the anus.

Late in the secondary stage true pustules may be seen (Fig. 302), and the cutaneous lesions show a decided tendency to become agglomerated in certain portions of the body; they are seldom diffuse, and are not so likely to be symmetrically distributed as the earlier secondaries. After the secondary stage of the disease has advanced for a period of weeks or months, the patient may complain of certain other symptoms, among which falling of the hair is most common (Fig. 304). Pharyngitis may be annoying, and the patient may state that his finger-nails are becoming unusually brittle. Various ocular manifestations, such as iritis, choroiditis, and retinitis, may develop during this stage.

Duration.—This stage usually continues for from two to three months, although it may run a much longer course—from eight months to one year. The interval between this stage and the onset of the tertiary stage varies greatly in different cases, and may be from a few months to many years.

The symptoms displayed during the secondary stage vary greatly in severity, and no satisfactory cause for such variation can be given.

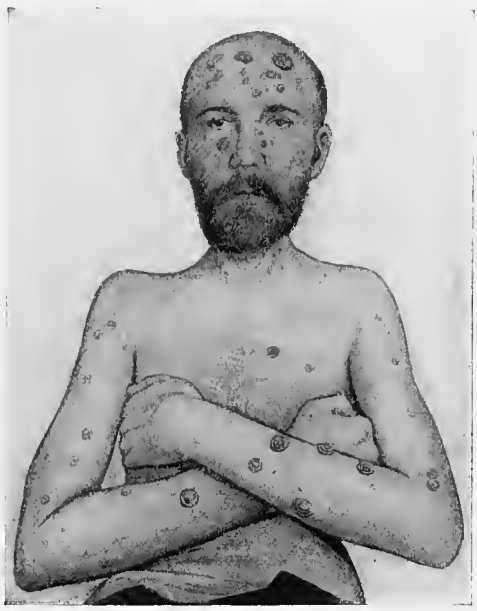


FIG. 304.—SYPHILIS.

This case had originally been quarantined as one of smallpox, but was later admitted to the venereal wards of the Philadelphia Hospital.

(3) **Tertiary Stage.**—Occasionally tertiary symptoms may develop while the late secondary lesions of syphilis are still visible.

In this stage the cutaneous manifestations are important; among these are the characteristic rupia, which appears first in the form of pustules that later break down with the formation of ulcers that become covered with true, laminated crusts—the so-called "oyster-shell" lesion (Fig. 304). The tubercular variety of lesion is generally seen upon the face, back, and extremities. The cutaneous lesions just described affect only the true skin, and leave distinct scars on healing. These lesions are not believed to be infectious or contagious, and are at times attended by itching. The detection of treponema pallidum in gummata, however, would seem to throw doubt on the accuracy of this belief. True gummata may be seen to develop in the skin, and may involve the subcutaneous tissue; these lesions later tend to break down, with the formation of reniform ulcers that, in many instances, show a tendency to suppurate. Healing of syph-

ilitic ulcers takes place somewhat slowly, and scar formation may be extensive.

Mucous Membrane.—Gummata are to be seen upon the mucous membranes, where they pass through the successive stages of ulceration and cicatrization. If the gummatous lesion should involve the rectal mucosa, diarrhea may be present, and, following the healing process, stricture may result.

Muscles.—Gummata may appear within the muscle substance in the form of small, hard tumors.

Bones.—The osseous structures are not infrequently attacked, and periostitis, followed by necrosis, is occasionally seen. As previously stated, syphilis shows a special predilection to attack the frontal (Fig. 304), the nasal, and the palate bones, and the tibiæ, although other bones may be attacked. Bone lesions, as a rule, give rise to pain, this symptom being most marked at night, and increased even upon making slight pressure over the affected organ.

Lymph-nodes.—Glandular involvement is common; the affected structures show but slight tendency to go on to suppuration. In certain cases, also, the testicles are attacked.

Viscera.—Gummata are occasionally seen in the viscera, a condition that will be discussed further on. Amyloid degeneration of the liver and of the other structures may follow tertiary syphilis.

Malignant syphilis is a rare but unusually virulent and fatal type of the disease. The successive clinical stages appear early and in rapid succession, a feature that is especially true of the development of tertiary lesions.

Malignant syphilis is further characterized by the fact that it resists treatment. A. E. Roussel has described a case that terminated fatally within the course of one year.

Laboratory Diagnosis.—Scrapings from the initial lesions and from the mucous patches, moist papules, and other cutaneous manifestations may show the presence of the treponema pallidum. Recent investigations have shown that syphilis may be diagnosed from the detection of antibodies in the blood by the method of complement binding. Antibodies are asserted to be present in the blood of syphilitics irrespective of the length of time that has elapsed since the appearance of the initial chancre. (See special works on Laboratory Diagnosis. See also Wasserman Reaction, under Locomotor Ataxia, p. 1121.) After the secondary stage is well advanced, marked secondary anemia is generally present, this condition improving after the administration of specific treatment.

Justus's blood-test consists in a distinct, though transient, reduction in the percentage of hemoglobin following the administration of mercury, given either by inunction or hypodermatically, but it must be remembered that a similar, though possibly less well-marked, reduction may be seen to occur in non-syphilitic individuals.

Cobra Venom Reaction (Weil's).—Dr. R. Weil* gives in detail the hemolytic change following his experiments with the blood from various types of disease. An extraordinary degree of resistance to the action of cobra venom is exhibited in blood from syphilitics following the primary stage. In case the cells show a lesser degree of resistance the disease in question is not syphilis. The possible exception is cancer, where the hemolytic changes resemble those of syphilis.

Summary of Diagnosis.—Great importance attaches itself to a clear history of the initial chancre in those cases seen after the disappearance

* Jour Inf. Diseases, Nov., 1909.

of the primary lesion. In the absence of a positive history of chancre, followed by the appearance of secondary symptoms, such as malaise, slight fever, and the characteristic eruption, the diagnosis may be made only with difficulty. When, however, the symptoms of the secondary stage are present, syphilis can scarcely be mistaken for any other disease. During the tertiary stage, if the manifestations of the first and second stages have been unusually slight, the condition may give rise to confusion. On the other hand, the presence of the characteristic lesions of the tertiary stage renders the diagnosis clear, but atypical cases are by no means uncommon, and in such the therapeutic test, when followed by improvement in the patient's general condition, confirms the diagnosis. The recovery of treponema pallidum from the lesions is positive evidence of the existence of the disease.

Differential Diagnosis.—Numerous affections and conditions, both local and general, are likely to be confounded with syphilis. Only a few of these will be mentioned here:

(1) **Epithelioma.**—The primary sore of the lip has been repeatedly mistaken for epithelioma. The history and symptoms of syphilis, together with the therapeutic test, will clear up any doubt. In one case coming under our care a chancre of the cervix was diagnosticated as carcinoma by two gynecologists and an operation advised.

(2) **Skin Eruptions.**—Lichen, psoriasis, papular eczema, measles, etc., may be mistaken for the eruption of secondary syphilis, and for their differentiation the reader is referred to special works on Diseases of the Skin.

(3) The **specific eruptive fevers,** and particularly the pustular stage of smallpox, have been mistaken for secondary syphilis.

(4) Syphilitic arthritis, which may develop at the beginning of the second stage, is to be distinguished from *rheumatic arthritis.* This is best accomplished by making a careful study of the history of the primary lesion, and the characteristic secondary manifestations of syphilis.

(5) The tertiary stage of syphilis may simulate **chronic gout** or **rheumatism,** and unless there is definite evidence of the presence of syphilis, on the one hand, or typical rheumatic symptoms and history on the other, the diagnosis may remain indefinitely uncertain. The therapeutic test, however, may lend assistance.

(6) Periosteal nodes, similar to those occurring in syphilis, may follow **acute infections,** *e. g.*, smallpox and typhus and typhoid fevers, and here again the diagnosis is attained only by making a careful study of the clinical history, and as the result of the therapeutic test (see Justus's Test, p. 828), as well as by the detection of the treponema pallidum in portions of the nodule.

(7) Enlargement of the tonsils may result from syphilis, and such enlargements may be confounded with **epithelioma** of these organs.

(8) During the febrile period of syphilis the clinical picture may resemble, in many respects, that seen in **tuberculosis.** The therapeutic test, a careful examination of the blood, Calmette's ophthalmotuberculin reaction (p. 807), and von Pirquet's needle-track reaction (p. 807) will usually serve as means for differentiating between tuberculosis and syphilis.

HEREDITARY SYPHILIS.

Clinical Features.—These may, in rare instances, resemble those previously described under Acquired Syphilis; in the hereditary form, however, chancre is absent. In certain cases the characteristic symptoms are present

at birth, although in the vast majority positive symptoms make their appearance between the first and fourth months of life. "Kassowitz states that one-third of all children procreated of syphilitic parents are born dead, and of those born living, 24 per cent. die within the first six months of life" (Anders). Inherited syphilis may further be classified according to the time at which symptoms make their appearance.

In the new-born there is a lack of physical development. The child is greatly emaciated, "snuffles" is present, and hiccough occasionally appears soon after birth. Cutaneous eruptions are rare, but pemphigus neonatorum may attack the palmar surfaces of the hands and the soles of the feet. Among the rare skin phenomena are gummata around the radiocarpal articulations, palmar psoriasis, and roseola. Ulcers and fissures may be seen about the mouth and the anus. The bony skeleton may show hyperostoses of the long bones. Enlargement of the liver and spleen is common. Pseudoparalysis has also been observed.

Early Postnatal Symptoms.—Many subjects of hereditary syphilis are well developed at birth and exhibit no manifestations of the disease, symptoms appearing, in the majority of cases, not later than the third month.

Coryza (syphilitic rhinitis) is often the initial symptom, being accompanied by a seropurulent and at times a bloody discharge—a peculiar form of obstructed breathing rendering nursing difficult. Coryza may be preceded by singultus, and *ulcers* may form in the nose, leading to *necrosis* of the bones and ultimate deformity of the organ. *Coryza, otitis media,* and *deafness* are the chief symptoms, and the skull may be asymmetric in conformation.

The *cutaneous symptoms* appear early. The skin has a tawny hue; the nates and genitalia are the seats of an erythematous eruption. Onychia is seen at times, and the lips and angles of the mouth may show well-marked fissures. There may be a moderate degree of glandular enlargement, and falling of the hair may occur.

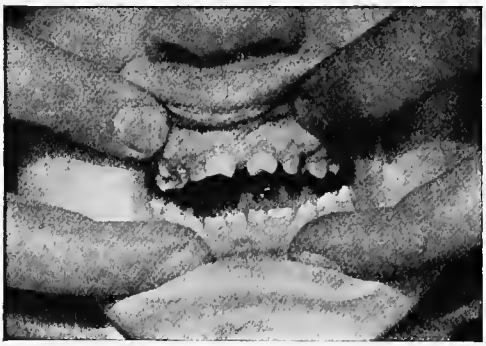


Fig. 305.—Hutchinson's Teeth in a Child with Hereditary Syphilis (Schamberg).

Splenic enlargement is a common feature, and when seen before the third month, is of great diagnostic importance.

The *liver* may also be enlarged, but this symptom is of less diagnostic importance than is enlargement of the spleen.

Syphilitic infants at times display a marked tendency to develop hemorrhage, and at birth, or within the course of a few days, there may be bleeding from the cord and hemorrhage into the subcutaneous tissues and from the mucous surfaces—*e. g.,* the vagina, stomach, and mouth. Syphilitic children are generally restless and sleepless, and may give utterance to a peculiar, harsh, shrill cry. Both anemia and cachexia are to be seen.

Late Symptoms.—The manifestations of congenital syphilis that develop later in life have been grouped under the following subheadings:

(1) Those cases in which the general appearance of the child is a prominent feature, and is indicated by a retarded development both of the bony

structures and of the muscles. This feature may be so pronounced that a child from four to twelve years of age may resemble an infant in size and form. The skin of such children has an earthy tint, and the hair is scanty and poorly nourished.

(2) Those cases in which lesions of the skeleton are prominent show the so-called "natiform skull"—a transverse enlargement, lateral bulging, and flattening in the middle. At times the skull may be hydrocephalic or asymmetric, and deformity of the nasal bones, as previously stated, is by no means unusual. Thickening or deformity of the tibiæ and of the sternum may also be seen.

(3) *Cicatrices.*—These are seen upon the cutaneous surface, and also about the nose, mouth, soft palate, and genitals.

Hutchinson's Teeth.—In some syphilitic children the teeth are erupted late, and often present various irregularities. (See Fig. 305.) The dental arch may be deformed. In rare instances the child may be born with one or more teeth. The incisors, especially the superior median of the second dentition, are notched, and show a thinness of the free edge, atrophy of the summit, and crescent-shaped erosions (Fig. 305). The absence of one, two, or more teeth is an occasional feature of inherited syphilis.

Auditory Manifestations.—Otorrhea, previously referred to, is, as a rule, secondary to syphilitic disease of the nasopharynx, and an increasing form of deafness appears at about the time of puberty.

Ocular Manifestations.—Interstitial keratitis and iritis are to be seen in inherited syphilis.

The testicle may fail to develop at puberty, showing the condition known as "infantile testicle."

VISCERAL SYPHILIS.

Syphilis of the Nervous System.—See chapter on Diseases of the Nervous System, p. 1118.

Syphilis of the Liver.

Gummata develop in the substance of the liver, and, following their absorption, the organ becomes distinctly lobulated. The viscus may also be further deformed as the result of chronic inflammation, which appears to affect mainly the capsule of Glisson. True perihepatitis may be present, and serve later to cause lobulation of the organ. (See Perihepatitis, p. 595.) Amyloid degeneration of the liver may be seen to follow syphilitic lesions of the bones and syphilitic ulceration. (See Amyloid Liver, p. 593.)

Symptomatology.—The clinical picture may be that of hepatic cirrhosis, with ascites, gastro-intestinal disturbances, and slight jaundice. In certain cases the liver may be appreciably enlarged, and there may be enlargement of the spleen even in those cases in which sclerotic changes are present in the liver.

Distinct gummatous tumors may be responsible for enlargement of any portion of the liver, although the left lobe is more commonly involved. The symptoms may arise both as the result of contractions from the formation of cicatricial tissue and from pressure by gummata upon the portal circulation.

Summary of Diagnosis.—Unless there be a clear history of syphilitic infection, the diagnosis is made with difficulty. In those cases in which ascites is present, the actual size of the liver can be ascertained only by re-

moving the ascitic fluid. Well-marked indentations along the edge of the liver are highly suggestive of syphilis of the organ. In obscure cases the administration of antisyphilitic remedies may be necessary in order to formulate a diagnosis.

Syphilis of the Lung.

Syphilitic changes occasionally attack the pulmonary structure, in which case the general clinical picture is that of chronic pulmonary tuberculosis. (See Pulmonary Tuberculosis, p. 796.)

The **symptoms** of chronic bronchitis are present. In doubtful cases of fibroid tuberculosis of the lung accompanied by slight fever, in which the sputum does not contain tubercle bacilli, a therapeutic test must be made in order to arrive at a diagnosis.

Diagnosis.—The features that point strongly to syphilitic infection of the lung are extensive amyloid disease involving the viscera, the presence of a well-marked secondary anemia, and arteriosclerosis.

Syphilis of the Rectum.

When syphilis appears in the form of more or less diffuse, submucous gummata located within the external sphincter, the most characteristic symptoms are those pointing to progressive stricture of the rectum. The patient may complain of a more or less constant dysentery, which is chronic in nature and not accompanied by pain or tenesmus. An examination of the rectum is necessary, and usually reveals the presence of a firm fibrous ring. Ulceration of the mucous surface may also be present.

Syphilis of the Testicles.

There are two clinical forms of syphilis of the testicle: (1) Atrophy of the gland, which may be either irregular or uniform in outline, is not accompanied by pain, and commonly involves one organ more than the other; (2) gummata of the testicle.

Differential Diagnosis.—Syphilis produces an irregularity in the body of the testicle, which serves to distinguish it from *tuberculosis* of this organ, which attacks the epididymis. *Malignant disease* of the testicle develops more rapidly than does syphilitic tumor, and is, as a rule, painful, a feature which is uncommon in syphilis.

Syphilis of the Kidney.

Syphilis of the kidney is sometimes found at autopsy, but concerns us but little with reference to antemortem diagnosis. The changes are chronic in nature, the most common being amyloid degeneration.

Syphilis of the Heart.

Gummata may develop in the wall of the left ventricle. Fibroid sclerotic myocarditis is also seen, this process beginning in the perivascular tissue, and proceeding outward from the vessel-walls. These changes are more or less diffuse, and in time cause narrowing of the lumen of the coronary arteries and their branches, although in some cases aneurismal expansion of the arteries has been observed. Syphilitic endocarditis is also of the fibroid sclerotic type. The diagnostic clinical features of syphilis of the heart are the same as those described under Chronic Myocardial and Endocardial Changes. (See Diseases of the Heart, pp. 261 and 297.)

Syphilis of the Arteries.

Clinical Forms.—Two varieties are recognized: (1) **Obliterating endarteritis,** in which there is a proliferation of the subendothelial tissue, which in time encroaches upon the lumen of the artery. (2) **Gummatous periarteritis** is the name given to a condition in which the arteries at the base of the brain are most often attacked. (See Syphilis of the Brain, p. 1118.) During the course of syphilis a variable degree of arteriosclerosis is likely to develop, and following such atheromatous changes aneurism (p. 311) is common.

LEPROSY.

Pathologic Definition.—A chronic disease, due to infection by the lepra bacillus, and characterized by the presence of clusters of bacilli in

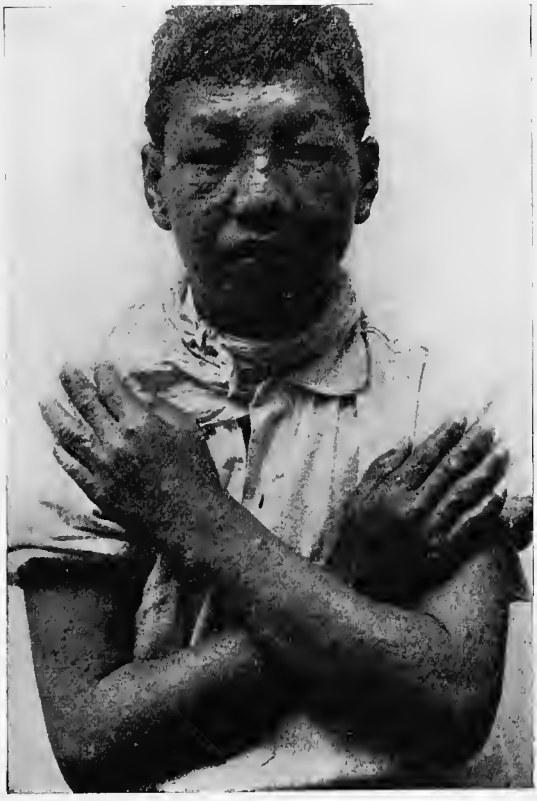


Fig. 306.—Tubercular Leprosy.
Patient, aged fifteen. Disease of three years' duration. Began as pinhead-sized nodules on face (Sandwich Island case).

the skin, these being surrounded by a tuberculous nodule. There may also be areas of cutaneous pigmentation, with the deposition of bacilli in the epithelial cells and leukocytes. Each granulomatous mass is surrounded by a layer of connective tissue; certain of the lymph-nodes become enlarged, and bacilli are deposited within them. These bacilli are also present

in the liver, spleen, and blood drawn from the lesions, and rarely in the circulating blood. Secondary infection with pus-producing organisms may hasten destructive changes in the lesions. When the lepra bacilli are deposited around the nerve-sheaths, irritation and hyperplasia are set up, leading to atrophy and degenerative changes in such nerves.

Varieties.—(*a*) **Tubercular leprosy** is characterized by cutaneous manifestations, such as erythema, macules, tuberculous nodules, and cutaneous pigmentation. (*b*) The **anesthetic form** is marked by local symptoms, such as pain, hyperesthesia, and anesthesia.

Exciting and Predisposing Factors.—**Bacteriology.**—The bacillus lepræ, which was first described by Haynes, resembles in certain respects the bacillus of tuberculosis. Upon sectioning the lesions, the bacilli are found within the tissue.

Age figures prominently as a predisposing factor, the majority of cases being seen between the twentieth and fortieth years; the disease is extremely uncommon among children.

The influence of **heredity,** although questionable, is a conspicuous factor in approximately 25 per cent. of cases; there are many investigators, however, who believe that the disease is not inherited.

Fig. 307.—Macular Lesions in Leprosy.

Immediate **surroundings and environment** are prominent influences in certain localities in which the disease prevails, lepers being seen, as a rule, more commonly in the rural districts than in the large cities.

Latitude is usually recognized as a predisposing factor, although the disease may be found in different, more or less isolated localities, extending from the equator to Norway and Iceland. From recent observations we learn that leprosy is extremely common among the natives of Peru and Ecuador. The natives of the Hawaiian and other of the Pacific Islands are also frequently victims of the disease. There are isolated regions in the United States, of which New Orleans is the center, in which cases of leprosy occur.

Modes of Infection.—Different investigators hold widely divergent views as to the modes of infection, although practically all agree that the disease is probably transmitted by contact. Morrow suggests that leprosy, like syphilis, is generally transmitted by sexual intercourse, whereas other observers hold that the disease is propagated by insects. As a matter of fact,

the exact mode of infection is unknown. Sticker believes the primary site of infection to be the nasal mucous membrane.

Incubation Period.—This, according to Hansen, is from three to five years. Some writers believe this stage to occupy a much shorter period, but there is evidence to show that, in certain cases at least, incubation may occupy several years. Such *prodromes* as chilliness, recurrent attacks of fever, excessive sweating, mental dullness, drowsiness, and debility may be experienced for years before the characteristic symptoms become manifest.

Clinical Picture.—Tubercular Leprosy.—In the *first stage* there are areas of cutaneous erythema, with a slight hyperesthetic elevation of the affected skin, seen on the face or upon the extensor surfaces of the arms, hands, legs, back, buttocks, abdomen, and chest. These may vanish after a time, leaving the skin pigmented and anesthetic; later the pigmented areas may disappear, leaving in their stead white spots of corresponding size (lepra alba). Such areas lose the hair that is normally present—a characteristic feature.

Tubercular nodules that tend to fuse and form irregular masses, dusky red or almost brown in color, develop in addition to the anesthetic patches. These tubercles may soften and become absorbed, or they may ulcerate. The skin is greatly thickened, presents a scaly surface, and there is loss of substance in certain parts, whereas other portions are markedly enlarged (eyebrows, nostrils, lips). Ozena, hoarseness, and aphonia are present as the result of involvement of the respiratory mucosa, and there may be extensive ulceration of the larynx, pharynx, and nose.

Anesthetic Form.—Here the *local symptoms* are referable to implication of the nerves, and as a consequence *pain* and areas of *hyperesthesia* constitute the prominent early symptoms. As the disease progresses minute bullæ may be seen, and evidences of trophic changes, with wasting of muscles that are supplied by the involved nerve-trunks, appear. Distinct nodules along the course of the nerves may also be present. *Anesthesia* is a characteristic feature of this type of the disease, and areas of vasomotor congestion usually precede the anesthetic stage. Yellowish-white patches, which are dry and scaly and may at any time become anesthetic, are distributed over the body. Extensive ulceration of the skin is most likely to occur upon the extremities, and, depending upon the degree of ulceration, wasting and necrosis, with extensive deformity, will result. An example of such deformity is the so-called "claw hand."

Laboratory Diagnosis.—Blood withdrawn through an incision into a tubercle may contain lepra bacilli. We have found bacilli free in the circulating blood in one case, but other instances of such finding are recorded. Lepra bacilli may be present in the exudate collected from ulcerated surfaces, and where the nasal mucous membrane is involved, they are commonly present in the nasal secretion. The sputum of cases suffering from ulceration of the pharynx and larynx will also be found to contain the bacilli.

CEREBROSPINAL MENINGITIS

(SPOTTED FEVER; CEREBROSPINAL FEVER).

Pathologic Definition.—An acute infectious disease, caused by the micrococcus (diplococcus) intracellularis, and characterized by an acute inflammation of the cerebrospinal meninges, with the formation of pus and of an excessive quantity of seropurulent cerebrospinal fluid.

The entire meninges of both the brain and the spinal cord are covered with a somewhat thick, cream-like or yellowish exudate, which is composed principally of pus-cells. The meningeal arteries are engorged with blood.

Predisposing and Exciting Factors.—Bacteriology.—The exciting cause is the diplococcus intracellularis, which may be found in microscopic examinations of the cerebrospinal fluid. The meningococcus may also be recovered from the synovial fluid of the larger joints, from the nose, and we have obtained a pure culture from a myocardial abscess.

Age.—Most cases occur during childhood and early adult life, although the disease may be seen at practically any age.

Climate is an important predisposing factor, epidemic meningitis being unknown in the tropics, whereas both epidemic outbreaks and sporadic cases have developed in practically all parts of the temperate zone.

Season.—The disease is said to prevail more commonly during cold weather, but a statistical study of its epidemiology shows that epidemics are likely to develop at any season. Boston* showed, in an analysis of the post-mortem findings in 80 cases of sporadic meningitis that came to autopsy at the Philadelphia Hospital from April 4, 1894, to October 26, 1898, that 13 of these were due to infection with the diplococcus intracellularis. The accompanying table shows the influence of season, sex, age, race, and nativity as predisposing factors in sporadic purulent meningitis:

Month.	Sex.	Age.	Color.	Nationality.
April	Male	6 months	White	American
June	"	1 week	"	"
October	"	13 months	"	"
"	"	26 years	"	"
April	"	64 "	"	Irish
June	"	26 "	"	German
March	"	31 "	Black	American
May	Female	7 months	White	"
August	Male	37 years	Black	"
October	"	52 "	White	German
January	"	26 "	Black	American
July	"	26 "	White	"
September	Female	13 "	"	"

Environment appears to exercise a prominent influence, since epidemics are common in homes, schools, barracks, etc., where the inmates are overcrowded in ill-ventilated apartments. Extensive epidemics may be seen in country districts, but are somewhat more common in towns and cities, and, indeed, they are often limited to a small section of a town. The mode by which the specific infection is conveyed from one patient to another is not definitely understood, and although several theories have been advanced, none of these have proved their claims conclusively.

Clinical Varieties.—(*a*) **The Malignant or Apoplectic Form.**—The symptoms characterizing this fulminating type of meningitis are not constant. There may be a severe chill, followed almost immediately by headache, loss of consciousness, and death—the entire clinical course occupy-

* "Etiology of Sporadic Purulent Meningitis," Med. News, May 20, 1899.

ing a period of but a few hours. As a rule, however, the disease is not so rapid in its course, but continues for two or more days following the initial symptom, which is an intense rigor. Headache, vertigo, obstinate vomiting, extreme prostration, rigidity of the muscles of the neck, stupor, and coma develop in rapid succession. The fever may not be high, and the pulse-beats may not exceed 40 to 60 beats a minute; as the disease progresses all the symptoms become intensified, and coma ends the scene. The fulminating type of meningitis is a characteristic of certain epidemics, whereas in others such cases are not observed.

(*b*) **The Mild Form.**—The disease may be so mild that the patient may be able to walk about or even to follow his usual occupation, complaint being made merely of headache, languor, nausea, occasional vomiting, and pain in the muscles at the back of the neck. In this type of meningitis, fever is not an essential symptom, but a mild febrile movement is the rule.

(*c*) **Intermittent Form.**—In this variety of the disease the symptoms intermit or remit every two or more days, the fever being decidedly intermittent, and resembling somewhat that of chronic sepsis.

(*d*) **Abortive Form.**—Here the initial attack is quite as intense as in the ordinary type of the disease described further on, but a decided amelioration occurs in the course of two or three days, and the patient goes on to convalescence without interruption.

(*e*) **Ordinary Type.**—During the course of an epidemic there appears to be no accurate means of ascertaining the incubation period. Certain *prodromal symptoms*, however, will be found to be present, and to vary widely in different epidemics, although even in severe types of the disease these may be wanting. A patient previously in vigorous health may be suddenly stricken down as though he had received a blow upon the head. In those cases that tend to be rapidly fatal there are lassitude, headache, muscular pains, joint pains, nausea, and obstinate vomiting. The prodromal symptoms are at times present for but a few hours, whereas in other, milder cases they may persist for from three to six or more days, during which time *occipital pain* serves as the most conspicuous feature. Irrespective of the character of the prodromal symptoms, the disease may follow these without an initial chill; mild cases are likely to complain only of languor, debility, headache, pain in the loins, vomiting, and possibly diarrhea prior to the development of typical symptoms.

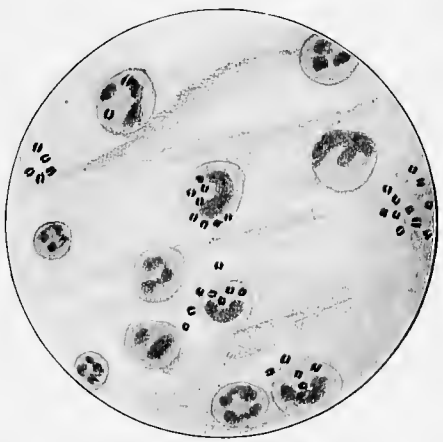


Fig. 308.—Diplococcus Intracellularis (Boston).
Meningeal pus obtained by lumbar puncture from case of epidemic cerebrospinal fever observed at Philadelphia Hospital (obj. Spencer one-twelfth oil-immersion).

In those cases in which the disease begins abruptly there is likely to be a *severe chill*. In children *convulsions* are common, and are usually followed by *intense pain* in the back and cervical regions. Movements of the head increase the pain in the neck, and bending the back intensifies the spinal

pain. Early during the disease the patient may refuse to swallow because of the extreme pain excited by deglutition.

There may be cough and difficulty in breathing, although in uncomplicated cases the respirations may be free and not greatly increased. Owing to exhaustion of the respiratory center the respirations become frequent and irregular and there may be marked dyspnea. In unfavorable cases Cheyne-Stokes breathing may develop. Myalgic pains are often intense, involving both the extremities and the abdominal region.

Gastro-intestinal Phenomena.—As previously stated, vomiting is the most common among this particular group of symptoms, and is present in 75 per cent. of cases, and, from its character, appears to be of cerebral origin. The appetite may be normal until the initial chill appears, or it may be impaired during prodromal symptoms. After the disease has become well established, anorexia obtains. *Constipation* usually exists throughout the entire course of the disease, although diarrhea may occasionally be present.

The patient complains continually of intolerance to light (*photophobia*), and intolerance to sound is also present during the early stage of the disease; later, however, a variable degree of deafness is prone to occur, which may continue even after convalescence is well established. Tinnitus aurium may be an early and annoying symptom, and abscess of the middle ear is often an alarming complication.

Nervous Symptoms.—In addition to the nervous symptoms already detailed there are often active delirium and hallucinations, during which the patient may shout loudly, and restraint may be necessary to keep him in bed. Paroxysmal outbreaks of delirium are by no means uncommon, and occur most often during the night; in young females the nervous symptoms are hysteric in character. The maudlin delirium of alcoholics is occasionally seen, but sooner or later the patient becomes somnolent, and eventually passes into a state of coma, which may continue from the time the disease develops, although it is occasionally a temporary condition. In those cases displaying a high grade of hypersensitiveness of the cutaneous surface, priapism may be an annoying symptom. Catalepsy is rarely present. Holmes* believes that his sign—analgesia or partial or complete anesthesia of the conjunctivæ and cornea—is present in a large proportion of all cases.

Thermic Features.—The fever of epidemic meningitis is not characteristic, and may be found to vary between 100° and 105° F.; in the average case it will be found to fluctuate between 100° and 103° F. The fever continues to run an irregular course until defervescence, which takes place gradually by lysis, occurs. We have seen cases in which the temperature rose to 106° and even to 108° F. during the last few hours of life. Under ordinary conditions the fever is lower in children than in adults, although we have seen cases of epidemic meningitis in persons after the age of fifty in whom the temperature did not exceed 102° F., and in whom the range was from 99° to 101° F.

Physical Signs.—**Inspection.**—Immediately following the onset of active symptoms the cheeks are flushed, although pallor and lividity are equally common; pallor of the lips is regarded as a fairly constant sign in this disease. In children conjunctivitis may be present. Keratosis, corneal ulcer, strabismus, ptosis, inequality of the pupils (see p. 1104), iridochoroiditis, and temporary, as well as permanent, blindness are among the ocular manifestations of epidemic meningitis. Coma-vigil is not uncommon, and the patient may lie for hours, and even for days, without moving the eyelids. As the disease progresses there may develop, at any time during

* Jour. Amer. Med. Assoc., January 25, 1908.

its course, an eruption which is not characteristic, and which may be a rather generalized subcuticular mottling (10 per cent. of cases) which disappears within a few hours. The eruption may be erythematous, macular, maculopapular, or petechial. In the case of blondes the eruption is cherry red. In brunettes a brownish-red or raw-ham color. In negroes it is slightly darker than the surrounding skin. A bullous eruption is rarely seen. A fact ever to be borne in mind is that in some cases the eruption is absent. Herpes facialis is also quite common and was present in 58 per cent. of cases in a 1911 to 1912 epidemic, and it should be remembered that this condition is frequent in but two other infections—lobar pneumonia and malaria. Among the unusual cutaneous manifestations seen in meningitis are sudamina, ecthyma, erysipelatous reddening, urticaria, and gangrene, although none of these is of diagnostic importance. Bed-sores may develop, and the abdomen is usually scaphoid.

The *tongue* is slightly coated at first, but as the disease progresses, this coating becomes heavier and heavier, and in those cases that go on to the so-called "typhoid state" the tongue becomes dry, brown, and fissured, the teeth and lips being covered with sordes.

In the course of one or more days *localized paralyses* may develop; these are the result of motor irritation, and consist of strabismus or paralyses of the face or of the extremities. Muscular contractions, particularly of the lower extremities, may develop. Twitching of a group of muscles is by no means uncommon. Later during the course of the disease tonic spasm of certain groups of muscles holds the patient in a fixed position; thus we have frequently seen the forearm flexed upon the arm, and the arm fixed firmly against the side of the chest. (See Fig. 418.) In another frequent form of contracture the thumb is coiled tightly within the palm of the hand. The illustration (Fig. 418, on page 1126) is taken from the photograph of a patient seen in our service at the Philadelphia Hospital. The position of the lower extremities and of the left hand and arm was permanent and the result of spasm. Spasmodic contraction of the muscles at the back of the neck causes the occiput to be drawn backward, and the child is unable to bring the chin forward upon the chest. The patient usually assumes the dorsal posture, and complains when he is turned from side to side. Contraction of the muscles of the back, limbs, and neck may be sufficiently well marked to produce *opisthotonos*, a condition in which the patient rests only upon his occiput and his heels, the entire body being arched—a feature characteristic of but one other disease, namely, tetanus.

Palpation.—Tenderness is usually present over the muscles of the back of the neck and along the course of the spine. Areas of hyperesthesia are common, and hypersensitiveness of the skin may be general. Localized areas of anesthesia also constitute one of the valuable signs of meningeal involvement. The joints, especially the larger of these, may be swollen and tender, and movement of parts controlled by certain groups of muscles often excites great pain.

The *pulse* is at first moderately accelerated, but in those cases that progress from bad to worse the pulse finally becomes weak, rapid, thready, and irregular.

According to Head, *Kernig's sign* is present in 84 per cent. of all cases of spinal meningitis, but it may be confined absolutely to epidemic types of the disease. It may be absent during the early stages of the disease, and, in our opinion, if the sign is not present as an early feature of the disease, the patient should be examined for it daily throughout the entire course of the

illness. Kernig first pointed out the impossibility of obtaining complete extension of the leg on the thigh when the patient is sitting and the thigh is flexed at a right angle to the trunk. The sign is produced as the result of irritation of the meninges of the lower portion of the spinal cord and of the nerve-roots that constitute the cauda equina, although it is no indication of a distinct lesion of these structures. This irritation, which is augmented by the stretching effect of the sitting posture, increases the tonicity of the flexor muscles of the leg, and, as a consequence, complete extension of the leg becomes impossible. If the patient is lying in bed, the thigh may be flexed

Fig. 309.—Kernig's Sign, Showing the Strong Contraction of the Flexors on Attempting to Exteno the Leg (Ruhräh, from Osler).

upon the abdomen (Fig. 309), when, if meningitis is present, complete extension of the leg will be prevented by contraction of the flexor muscles (Fig. 309). Should Kernig's sign be doubtful, force the chin forward on the chest and then try for Kernig's sign. This procedure often makes a questionable Kernig's sign positive. Whenever Kernig's sign is typical, its production excites spasm and pain. Kernig's sign may rarely be present in one leg and absent in its fellow.

Brudzendski's Sign.—By flexing the head on the chest there is some bending at the joints of the lower extremities (knees and hips). Flexion of one leg on the abdomen is accompanied by a lesser degree of flexion of the opposite leg.

MacEwen's sign is designed to indicate increased pressure of the fluid present in the ventricles by giving an increased resonance. Place the bell of the stethoscope against the forehead, and at the same time tap with the finger over the squamous portion of the opposite temporal bone.

Laboratory Diagnosis.—**Blood.**—The percentage of hemoglobin and the number of red blood-cells are but slightly altered during the active stage of the disease, except when well-marked cyanosis is present, in which case both the hemoglobin and the red cells give unusually high readings. Leukocytosis is present early, and continues throughout the febrile period. A differential leukocyte count shows an increase in the polymorphonuclear elements.

The quantity of *urine* voided during the twenty-four hours is approximately normal, although it may fluctuate as the result of cerebral irritation. A trace of albumin may be present, and, rarely, the urine contains a trace of glucose. It is frequently necessary to obtain the urine by catheterization, as both retention and incontinence are symptoms of meningitis.

Spinal Puncture.—*Aspiration* of the subarachnoid space results in the recovery of a turbid exudate, which, when smeared and stained, will be found to contain many diplococci that resemble the gonococcus in appearance. The diplococcus intracellularis is found within the pus-cells, although it is customary to find many extracellular cocci also present. Another characteristic feature of the meningococcus is that it does not stain by Gram's method. Cultures on Löffler's blood-serum made from the cerebrospinal fluid will, when kept at body-temperature, develop colonies of the meningococcus in from twenty-four to forty-eight hours. The Bacillus proteus, the bacillus of influenza, Bacillus typhosus, and Bacillus coli are at times present. The pneumococcus and pyogenic cocci may be found.

The *nasal secretion* is increased, and both cultural studies and stained specimens will show the presence of the diplococcus intracellularis. Slataper's analysis of 210 cases gave coryza as an early feature in 97 per cent. of his series. In a case studied by us at the Philadelphia Hospital, in which there was an associated purulent ophthalmia, the Diplococcus intracellularis was recovered from the conjunctivæ.

Illustrative Case of Epidemic Meningitis.—C. C. D., female, colored, aged nine years.

Family History.—Mother died of pneumonia at the age of twenty-seven. Father died as the result of an injury sustained by falling a distance of 20 feet.

Previous History.—No further evidence could be obtained than that the child had enjoyed good health during the past year.

Social History.—Admitted to a children's home in Philadelphia one year ago, and has remained in the institution ever since. Performed the usual school work given to the children of the home, the attendant stating that, until the present illness, she has been in excellent health.

Two weeks ago a child suffering from epidemic meningitis was removed from this institution. Twelve years ago there was an outbreak of epidemic meningitis in the same home, 16 children being afflicted with the disease; since this epidemic there was no return of the disease in the institution, the case here narrated being the first to occur since that time. Following the removal of the child suffering from meningitis no other cases have developed until the present one appeared.

Present Illness.—During the past three days the child has shown an indisposition to play. Anorexia is present, and the child complains when compelled to move her head. There is obstinate constipation. She awoke during the night and complained of feeling chilly, and a few hours later, after an apparently sound sleep, she awoke, uttering a harsh, shrieking cry. There is pain in the muscles of the neck, back, and limbs. Pain is aggravated by lifting of the head, and becomes intense when an attempt is made to tip the chin forward on the sternum. There is slight cough.

Pain is increased upon swallowing, and becomes severe when the child attempts to move her head. Late during the disease she would shriek with pain whenever moved about the bed. She also exhibited an intolerance for sound, and would often cry out whenever any harsh noise was made. Photophobia developed early, and strabismus, ptosis, inequality of the pupils, and paralysis of the extremities were observed during the fourth to the eighth days of the disease. Distinct convulsions did not occur, although muscular twitching was frequently seen. Spasmodic contraction of the muscles frequently followed when a draft of cool air was permitted to come in contact with the patient. During the first day the temperature rose to 101° F., and subsequently pursued an irregular type, ranging between 99° and 102.5° F.

Physical Examination.—*General.*—When seen during the second day of the illness, the child was resting in bed, and when lying upon the back, the chin was well elevated and the back slightly arched. When turned upon the side, it immediately became apparent that the head was markedly retracted, and that there was some curv-

ing of the spine. The limbs remained in one position when the child was turned from her side to the supine position. The expression was anxious, the mental condition was markedly dulled, the muscles of the nape of the neck were spastic, and paresis of the right arm and strabismus were also present. There was a petechial eruption on the extremities and over portions of the body, but these petechiæ were not numerous. By the fourth day of the disease distinct arching of the body was seen when the child was resting upon her back.

Local Examination.—Palpation.—The head could not be brought forward so that the chin would touch the sternum unless *force* were used, and the mere act of pushing the head forward elicited an expression of pain. The lower limbs and the arms were more or less spastic.

Auscultation.—At first the heart-sounds were strong and slightly increased in frequency; as the disease advanced, the first sound lost its muscular, booming quality, and the heart became rapid—120 to 140 beats a minute being recorded.

Laboratory Findings.—The urine was scanty, and by the third day was voided involuntarily. A specimen obtained by catheterization on the fourth day of the disease contained a trace of albumin, and gave a feeble reaction for glucose.

The spine was punctured, and about three drams of turbid fluid were recovered from the canal. Microscopically, the fluid contained pus; and in many of these cells diplococci were seen. Extracellular diplococci were also present. Cultural studies of the meningeal fluid showed the presence of the diplococcus intracellularis. Cultures made from the nasal mucous membrane also revealed the presence of the Diplococcus intracellularis, as well as of other bacteria.

Diagnosis by Induction from Clinical Data.—Two weeks ago a child found later to be suffering from epidemic meningitis was removed from the institution; hence suspicion was at once aroused as to the nature of the condition in question. The early development of rigidity of the muscles of the neck, together with an expression of pain upon moving the head from side to side and upon swallowing, further increased the probabilities of meningeal inflammation. At the onset photophobia was present, and as the disease progressed, strabismus, inequality of the pupils, ptosis, and paralysis of the extremities followed—features that are all practically characteristic of meningitis. The detection of the Diplococcus intracellularis in the spinal fluid left no room for doubt as to the true nature of the disease.

Course of the Disease.—The rigidity of the muscles progressed until distinct opisthotonos existed. By the fourth day of the disease respirations became hurried, and continued to increase in rapidity and to become more and more shallow. The nervous symptoms subsided slightly after lumbar puncture, but within the course of twenty-four hours they were again severe, and the general condition became decidedly unfavorable about the fifth day, and continued so until the twelfth day, when death terminated the scene.

Summary of Diagnosis.—In typical cases the diagnosis is made with extreme ease, as has been outlined by the clinical picture in our description of the ordinary type of the disease. We would call special attention to headache, rigidity of the muscles of the neck, and to hysteric and maniacal outbreaks, as features that are common in even atypical forms of the disease. The eruption, unless it should be petechial in character, carries with it but little diagnostic significance. Kernig's sign, when present, is positive evidence of meningeal irritation only. Lumbar puncture serves as the only positive means of diagnosis, and in certain cases this method alone enables us to obtain an accurate knowledge of the character of the disease in question. Inequality of pupils, strabismus, ptosis, and rigidity of certain groups of muscles are valuable clinical factors in formulating a diagnosis. While the typical cases of meningitis are always referred to, in our experience the so-called "text-book cases" do not include the majority.

Differential Diagnosis.—In the light of our present knowledge of laboratory methods meningitis is to be regarded as one of the few diseases in which this means of diagnosis is the only reliable one. A microscopic study of the cerebrospinal fluid obtained by lumbar puncture is a positive means of diagnosis and of differential diagnosis; consequently other tedious methods are unnecessary. The Widal reaction is also valuable in distin-

guishing between typhoid fever and meningitis, being negative in the latter condition.

Clinical Course.—The fulminating type of the disease usually terminates in death during the first few days. Mild and other atypical forms may continue over a period of one or more weeks before convalescence is established, whereas in the usual type favorable cases show improvement in two or three weeks, although convalescence is generally protracted.

Complications.—Eye and ear complications are quite common, and always cause an aggravation of the existing symptoms. Bronchopneumonia and arthritis are probably the most frequent complications.

ACUTE ANTERIOR POLIOMYELITIS.

(INFANTILE SPINAL PALSY.)

Pathologic Definition.—In the acute stages there is usually found an acute congestion of the blood-vessels, especially in the gray matter of the anterior horns, with round-cell infiltration and hemorrhages, some of which are quite large, destroying the cells of the anterior horns. Occasionally the hemorrhages or areas of inflammation involve the surrounding white matter, especially of the motor columns. Rarely a slight round-cell infiltration is found in the meninges. Early in the disease lumbar puncture may sometimes detect an increased amount of fluid. In most cases the acute congestion with hemorrhages disappears, but in those cases in which the cells in the anterior horns have been fully destroyed the paralysis is permanent—the so-called residual palsy.

Contributing and Exciting Factors.—The disease usually appears in a child previously healthy, and rarely in the course of or following infectious diseases. It occurs singly, although several members of the same family may become diseased.

Season.—The greatest number of cases are seen during the summer months, although the disease is not unusual throughout the year. Rosenau* gives a preliminary report upon the transmission of poliomyelitis by the common stable fly (stomoxys calcitrans), and Anderson and Frost† report having confirmed Rosenau's work. These investigators find that where stomoxys were permitted to bite monkeys that had been previously infected with poliomyelitis by intercerebral inoculation, these same flies were capable of transmitting the disease to uninfected monkeys.

Langhorst‡ cites 2 interesting cases, 1 following the bite of a dog, and the other developed after a dog (suffering from paralysis of the hind legs) had been permitted to lap his master's hand upon which there were a few slight wounds. Haywood (1913) reports a case where fourteen days after receiving a bite from a rabid dog a boy of fourteen developed the disease.

Flexner has called attention to the probability of the infection being carried through the mucous secretion from the nose, while Langhorst's unique experience emphasizes the danger of infection through the secretion of the buccal cavity.

* Congress on Hygiene and Dermography, Oct 5, 1912. The Jour. Amer. Med. Assoc., Nov. 2, 1912, p. 1627.

† Public Health Report, Oct. 25, 1912, p. 1733.

‡ Jour. Amer. Med. Assoc., Dec. 28, 1912, p. 2312.

Manning* has emphasized the probability of the disease being at times transmitted through the bites of the bedbug (cimex lectularis), while other writers have reported instances where acute myelitis has developed following the bite of this parasite. The habits of the cimex, as is well known, are such as would explain the outbreak of epidemics in man during the winter months.

Townsend†, of Rutland, Vt., has examined the spinal cord of a horse dead from paralysis, during an epidemic of poliomyelitis. Townsend's findings were as follows: "Section of the lumbar portion of the cord showed a granular degeneration and pigmentation of the ganglion cells of the anterior cornua, and atrophy of the nerve-roots." During an epidemic of 1000 cases in Minnesota in 1909 several members of the equine family were attacked. There are at least four recorded epidemics throughout the United States, where domestic animals and man were at the same time afflicted with acute epidemic paralysis. Among the animals and fowl thus afflicted were horses, hogs, sheep, dogs, cats, chickens, and ducks.

Manning cites the epidemic of 1911 in Brazil,‡ where a heretofore unknown disease of this country caused the death of approximately 4000 cattle and 1000 horses. During the past summer there have been 13 cases of acute poliomyelitis in man reported from this district. Extensive epidemics have prevailed in Scotland and various sections of England, and there was in these localities at the same time an epidemic among the sheep, goats, and other domestic animals.

ACUTE PARALYTIC DISEASE AND DEATH AMONG DOMESTIC ANIMALS OCCURRING COINCIDENTALLY WITH EPIDEMIC POLIOMYELITIS IN MAN (Manning).

Reported by	Locality	Year	Horse	Sheep	Dog	Cat	Hog	Fowls	Total
Caverly	Vermont	1894	Horse		Dogs			Chickens	Many.
Wickman	Sweden	1905			Dogs				And other animals.
Free	Dubois, Pa.	1907					Pigs	Chickens	
Manning	Wisconsin	1907–08	Colts	Sheep		Cats		Ducks	Many.
Lovett	Massachusetts	1911			Dog	Cats		Hens	39 in all.
Hill	Minnesota	1909	Colts						Three.
Snow	California	1910	Colts		Dog	Cat		Chickens	Many.
Kelly	Washington	1910*							
Williams	Wash., D. C.	1910						Chickens	Many.
Bierring	Iowa	1910				Cat	Hog	Chickens	Many.
King	Indiana	1911†							
Batte	Ohio-Ky.	1911						Chickens	
Krause	Westphalia	1910						Chickens	Many.
Gregor & Hopper	Cornwall, England	1911‡							
Carina	Sao Paulo, Brazil	1910–11§							

* Dog paralyzed one week before onset in child.
† Eighteen animals (1 cow) paralyzed among 102 cases of poliomyelitis.
‡ Horse, one week before onset of paralysis in boy.
§ One thousand horses and 4000 cattle dead with "symptoms of rabies," coincidental with 13 cases of human poliomyelitis at Sao Paulo.

From the evidences furnished by the various reporters it is only fair to suppose that probably more than one biting insect is capable of transmitting epidemic myelitis from man to man as well as from man to domestic animals, and *vice versâ*. Thus far we are cognizant of proof of the transmission of the disease by the ordinary stable fly, yet there are recorded

* Medical Times, April, 1912, p. 112
† Jour. Amer. Med Assoc., Jan 4, 1896.
‡ Annales de Institute Pasteur, Paris, Nov., 1911.

several instances where bites from the ordinary bedbug and the saliva of the dog appear to have been the source of the infection.

Proescher* has detailed a method for the staining of the organisms in poliomyelitis virus, as well as in smears, from the diseased portions of the nervous system. This writer described spirilli coccus and bacillus forms as being present in poliomyelitis.

Varieties and Symptoms.—The disease usually appears in the infantile period, generally between the ages of one and three, although it may occur later in life, especially in epidemics. Rarely it may be seen in adults.

It is ushered in by fever, with its accompanying symptoms of malaise and chilliness, or the child may feel sick for a day or so with at times an indefinite eruption, when the weakness or paralysis is discovered. At

FIG. 310.—PARALYSIS OF THE LEFT UPPER AND TO A LESS EXTENT OF THE LEFT LOWER LIMB, SHOWING ATROPHY, IN ACUTE ANTERIOR POLIOMYELITIS.

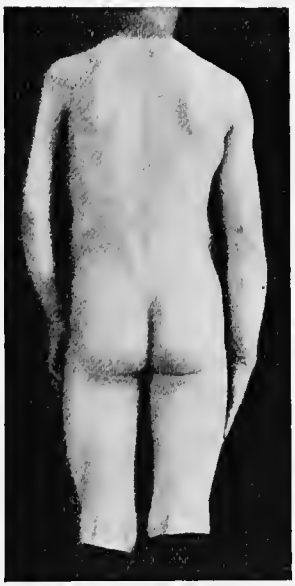


FIG. 311.—PARALYSIS OF THE LEFT UPPER AND TO A LESS EXTENT OF THE LEFT LOWER LIMB, SHOWING ATROPHY IN ACUTE ANTERIOR POLIOMYELITIS (POSTERIOR POSITION).

first it is quite extensive and may affect all of the limbs, but, as a rule, it involves by preference one or both lower limbs. Within a few days to four or five weeks the extent of the paralysis gradually lessens, and there remains what is called a residual palsy. All the muscles of the limb are never paralyzed, but there seems to be a predilection for certain groups, as, for instance, in the leg, the anterior tibial, and peroneal. Because of this unequal paralysis contractures of various types result. The paralysis is always flaccid in type and it is possible to passively move the limbs freely. Rarely the cells of the anterior horn in the thoracic part of the cord are diseased, this causing weakness or paralysis in the abdominal, lumbar, and thoracic muscles. This sometimes produces inability to sit up or to stand properly. Following the loss of power atrophy develops, the degree depending upon the extent of the destruction of the cells in the anterior horn.

* New York Med. Jour., April 12, 1913.

The tendon reflexes, as well as the normal electric reactions, will be lost in those parts in which the reflex arcs have been destroyed or interfered with (Figs. 310 and 312).

Not only will there be an atrophy of the muscles, but there also will be an atrophy of the bones of the involved limb. Because of the fact that the cells in the anterior horn are trophic in function there will also be lessened nutrition of the skin, which sometimes becomes dry, and the hair may not grow.

It is not at all uncommon in the onset of the disease to have a rigidity of the head, neck, and limbs, with pain in the back and neck and considerable tenderness in the limbs. This is due to an early meningeal involvement, which usually does not last very long and subsides within two or three days or a week. In rare instances, however, the pains may persist for a month or longer. There are never disturbances of sensation or of the bladder and rectal functions. When the disease appears in adults, the onset and clinical symptoms do not differ from those already described. This, however, is rare.

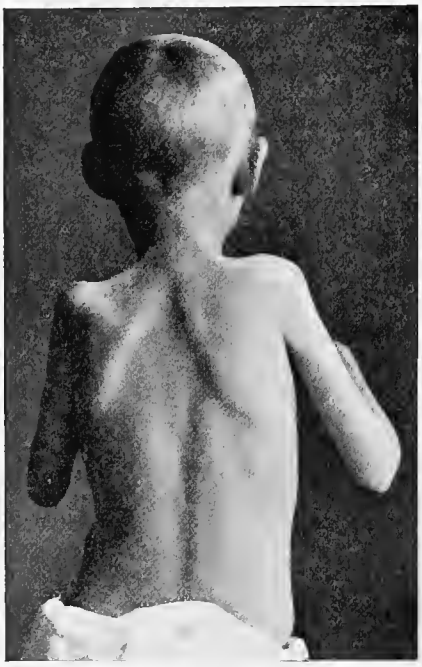


Fig. 312.—Acute Anterior Poliomyelitis, showing Atrophy of the Muscles of the Back, Shoulders, and Upper Limbs.

Summary of Diagnosis.—A previously healthy infant of from one to three years of age, with or without fever and its accompanying symptoms, sudden paralysis of one or both upper or lower limbs, flaccid in type. There is gradual diminution of the paralysis in the course of from one to seven weeks, followed by atrophy with contractures, loss of tendon reflexes, and electric reactions of degeneration. Bladder and rectal functions normal.

Differential Diagnosis.—The disease must be distinguished from acute myelitis and multiple neuritis. In acute myelitis the onset and the early clinical picture may be the same, but in acute myelitis there are always sensory symptoms with involvement of the bladder and rectum and greater and more general paralysis. From multiple neuritis the disease can be distinguished by the absence of sensory disturbances and persistent pain on pressure over the nerve-trunks, and the paralysis in multiple neuritis is limited to the distribution of certain peripheral nerves. Again, multiple neuritis in children is very rare.

Acute anterior poliomyelitis is sometimes difficult to differentiate from the symptom-complex, known as Landry's paralysis. In the latter, however, the paralysis is rapid, death usually resulting in a few days.

Clinical Course and Complications.—The course of the disease is chronic, and, with exception of the improvement of the paralysis in the first few weeks, there is no recovery of function.

Sometimes the pathologic process involves not only the spinal cord, but also different parts of the brain, and we may have, in addition to the symptoms already described, those resulting from involvement of the medulla, pons, or cerebrum. These have been previously discussed.

Sometimes in adults in whom there is an old acute anterior poliomyelitis there may develop a chronic degeneration of those anterior horn-cells which were at one time involved by the pathologic process, but in which recovery ensued. This degeneration caused gradual wasting with fibrillary tremors, and loss of power.

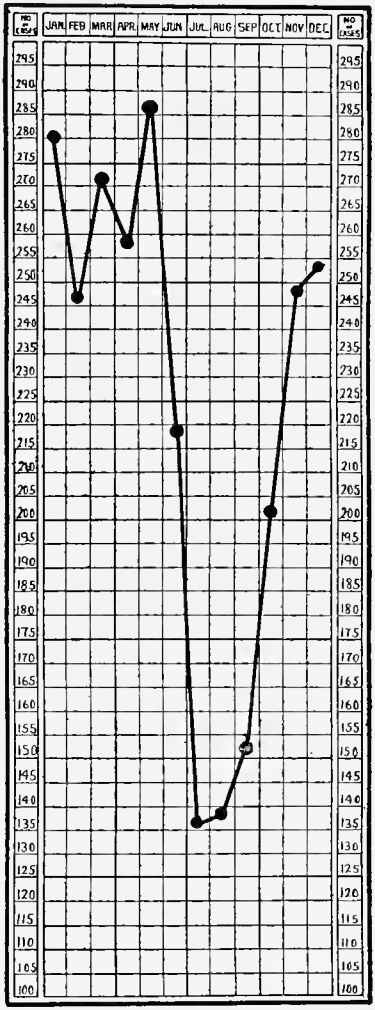


FIG. 313.—AVERAGE NUMBER OF CASES OF SCARLET FEVER OCCURRING DURING EACH MONTH OF THE YEAR. STATISTICAL ANALYSIS OF 32,317 CASES (A. K. Sallom, in "Medical Record").

SCARLET FEVER.

Pathologic Definition. — An acute infectious disease, characterized by high fever, marked angina, and a diffuse erythematous dermatitis.

Varieties.—Clinically, three types of scarlet fever are seen: (1) The *average type*, in which all the symptoms are active; (2) the *mild type*, in which all the symptoms are mild; and (3) the *malignant type*, in which all the symptoms are severe, and which may terminate fatally within the first twenty-four hours, before the appearance of the eruption. Usually, however, death does not take place until several days have passed.

Predisposing and Exciting Factors.—The presence of an epidemic or exposure to the contagium serves as the most important predisposing factor.

Age figures prominently in the predisposition to scarlatina, the disease being most prevalent between the first and sixth years, and practically unknown during the first six months of life. It is rare between the sixth and twelfth months, and the tendency diminishes after the tenth year, although an occasional case of scarlet fever is encountered after the thirtieth year.

Women are particularly liable to contract scarlet fever during the puerperium, and the infection may be conveyed to them by the physician or nurse. During the winter of 1878 an epidemic of scarlet fever occurred in the maternity wards of the Philadelphia Hospital.

Climate.—The disease is more common in temperate than in tropical districts.

Season.—Statistics show that the greatest number of cases develop during the winter, fall, and spring months. Cold weather, therefore, appears to exercise some influence on the spread of scarlet fever, but, as in diphtheria, it is possible that the fact that children are congregated in schools and homes during the cold weather may account for the increased number of cases seen during the winter months. Caigere, in his analysis of 1008 cases of scarlatina, gives October as the month in which the highest mortality of cases developed, and Whiteleege's analysis of 6000 cases confirms Caigere's observation. The result of A. K. Sallom's analysis of 32,317 cases reported in Philadelphia is shown by Figs. 313 and 314.

Bacteriology.—Many varieties of bacteria have been recovered from the skin, mucous membranes, urine, and blood of persons suffering from scarlet fever, but thus far the exciting microörganism has not been dis-

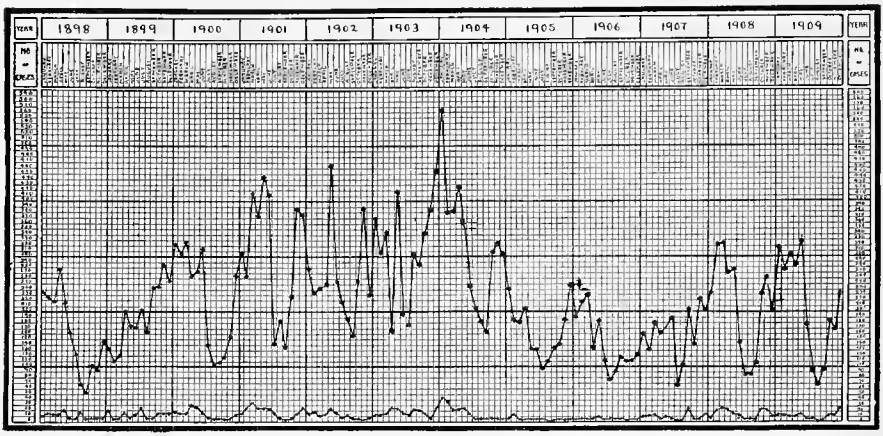


Fig. 314.—Chart Showing Number of Cases and Mortality of Scarlet Fever by Months. Statistical Analysis of 32,317 Cases (A. K. Sallom, in "Medical Record").

covered. Streptococci, diplococci, atypical pneumococci, and streptobacilli have all been isolated from the throat of scarlatinal patients. In 1899 Class isolated a special diplococcus (diplococcus scarlatinæ) from the mucous membrane of the throat, from the blood, and from the urine of scarlet fever patients. Bodies have been described by Mallory and by Duval which were found in the epithelial cells and in the lymphatic vessels of the skin in cases of scarlet fever, and which they believed were protozoa; these they considered might bear some etiologic relation to the disease.

Scarlet Fever in Hogs.—Behele reported an epidemic of scarlet fever in swine. There are at present a few complete records of epidemics of scarlet fever following a peculiar sickness affecting the hogs of the district in which the epidemic prevailed. Both streptococci and diplococci isolated from scarlatinal patients will be found highly toxic for swine.

Modes of Infection.—Scarlet fever is a highly contagious and infectious disease, although not so markedly so as smallpox and measles. The infection is generally believed to be conveyed from the sick to the well by means of the fine epithelial scales that are given off during convalescence, and this probably explains why the virus of scarlatina is so often conveyed

by clothing, carpets, furniture, toys, and the like. Examples are recorded in which the disease has unquestionably been conveyed by the nurse or by the physician. The secretion from the throat and nose are infectious, as is also the urine.

Billington's observations, made among 26 families residing in the tenements of New York city, where there was practically no attempt at isolation, showed 43 cases of scarlet fever, and, further, that 47 other children, who resided in the same tenements at the same time, and who were unprotected by previous attacks, did not contract the disease. Johannessen reported that of 158 children who were exposed to scarlet fever, 28 per cent. contracted the disease. Johannessen also observed that of 314 adults exposed, 5 per cent. developed scarlet fever.

Domestic animals, particularly cats and dogs, are likely to spread the disease, and, as previously stated, there is authentic evidence to show that hogs may suffer from scarlet fever. The fact that several investigators, as well as one of the present writers, have been able to produce scarlatinal symptoms in rats and other laboratory animals would seem to indicate that rodents may be instrumental in spreading the infection of scarlet fever. Persons afflicted with open wounds are especially likely to become infected. As a result of the careful investigations recently made by the various boards of health throughout the United States, a number of epidemics of scarlet fever would appear to have had their origin in the milk-supply, and were traceable to cases of mild scarlatina occurring in some one who handled the milk supplied to a certain district.

Immunity.—One attack protects against subsequent attacks of the disease.

Incubation Period.—Holt, in his tabulation of 113 cases in which the period of incubation could be accurately determined, gives the following figures:

24 hours or less	6 cases	8 days	2 cases
2 days	15 "	9 "	5 "
3 "	28 "	11 "	1 case
4 "	25 "	14 "	1 "
5 "	6 "	21 "	1 "
6 "	15 "		
7 "	8 "		

Clinical Varieties of Scarlet Fever.

Ordinary Type.—As a rule, the initial symptoms of scarlet fever are pronounced, and, generally speaking, may be said to appear suddenly. At first a variable degree of lassitude is present for a few hours, during which period the child feels uncomfortable, is drowsy, and may complain of some soreness in the throat. These prodromata are followed by an abrupt chill, with anorexia, nausea, *vomiting*, and, in small children, there may be a *convulsion* or a series of convulsions. By the time the physician is summoned the child complains of intense headache and nausea, and the vomiting may be obstinate. Marked *angina* and sharp pains over the muscles of the back and limbs are also present. At this time the *pulse* is found to be between 110 and 160 a minute, and is full and bounding, of high tension, and out of all proportion to the amount of fever present.

Thermic Features.—Within a few hours after the chill the temperature rises to 102° to 104° F., and continues to rise steadily until the eruption is completely developed, when it may reach a maximum of 104° to 106° F.

With the fading of the eruption there is a remission in the fever toward the close of the first week, and in uncomplicated cases the temperature may then fall to near the normal line. With the onset of such complications as nephritis, otitis media, and bronchopneumonia the temperature may be present for an indefinite period, and whenever the fever continues for more than seven days, complications are to be suspected.

Cutaneous Manifestations.—A diffuse rose-red or scarlet punctiform erythema ordinarily develops within the first twenty-four hours, and never appears later than the thirty-sixth hour. It is seen first about the clavicles and neck, but rapidly spreads over the chest, back, and extremities, so that within four or five hours the entire surface of the skin is of a scarlet hue.

Exception.—The face is the last part to become involved in the dermatitis; the forehead, nose, chin, and lips are pale, being in striking contrast to the

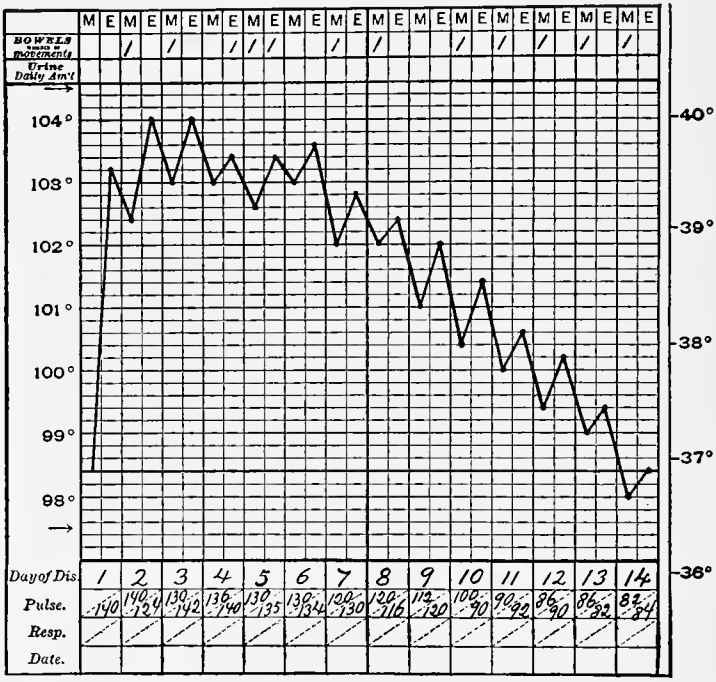


FIG. 315.—TEMPERATURE-CURVE OF A CASE OF SCARLATINA WITH FAVORABLE COURSE. PATIENT AGED SEVEN YEARS (Anders).

cheeks, which are an intense scarlet. Filatoff has called special attention to the unusual pallor of the chin, which is in striking contrast with the degree of redness of the cheeks. Pastia has likewise called attention to a somewhat distinctive eruption that occurs at the bend of the elbows, but which is by no means a constant feature of scarlatina.

Pressure over the skin of the chest or of any other portion of the body causes a decided pallor, the scarlet hue reappearing as soon as the pressure is removed. So marked is this pallor of the skin that one may write upon the child's chest or back. The rash of scarlet fever is particularly fine, and can scarcely be confounded with the eruption of any other disease, although

a not dissimilar reddening of the skin is occasionally seen to occur in some persons after the ingestion of shell-fish or of certain vegetables. In the ordinary type of scarlet fever the eruption is manifest for from thirty-six to forty-eight hours, being present, as a rule, during the greater portion of the second day; in mild cases, however, to be described later, the eruption disappears after the first twenty-four to thirty-six hours.

A close inspection of the skin shows that the eruption is made up of innumerable fine red points (puncta), each of which surrounds a hair-follicle. At the margin of these spots there is a zone of intense redness, which blends with adjacent red areas, thus producing the general erythema. In atypical forms of eruption the reddened areas surrounding the hair-follicles are not surrounded by the erythematous blush.

Blotches of scarlatinal eruption interspersed with healthy skin are occasionally seen, but are by no means common; these are more characteristic of malignant scarlet fever. We have seen cases in which the eruption was distributed in blotches and developed after death, and at this time there may also be detected fine hemorrhages into the skin.

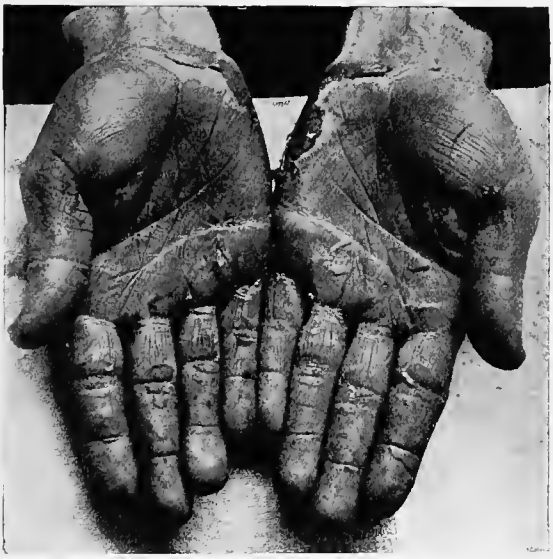


Fig. 316.—Scarlatiniform Erythema: Desquamation upon the Hands (Welch and Schamberg). Patient, a man of twenty-nine years, has had two attacks every year of his life.

Rumpell-Leede Phenomenon.—By the application of a bandage immediately above the elbow, pressure excreted between 45 and 60 mm. of mercury, for a period of from five to twenty minutes, minute hemorrhage will be seen to form on the anterior surface of the elbow. This same result is obtained by lifting a portion of the skin of the chest and pressing it rather firmly between the thumb and index-finger. This cutaneous sign is of some value in distinguishing scarlatina from other eruptive fevers. It is to be remembered that in measles, and at times in small-pox, a slight irritation of the skin is followed by a tendency to hemorrhages.

Desquamation.—In cases of scarlet fever of average severity desquama-

tion begins within a few days after the eruption has fully developed; the severity of the exfoliation is in direct relation to the degree of eruption, and likewise to the height of the fever. The entire integument is involved in the desquamation and the epithelium is thrown off in large and small flakes. A similar desquamation takes place in the throat and in the mucous membrane of the soft palate, uvula, pharynx, and tonsils. The tongue, the entire mucous surface of the mouth, and the nasal fossæ may exfoliate. Following the desquamation of the tongue its dorsal surface becomes a bright red, and its papillæ become prominent, thus giving rise to the so-called *strawberry tongue,* which is said to be characteristic of scarlet fever.

The duration of the desquamation is variable, the palms of the hands (Fig. 316) and the soles of the feet being the last parts to be involved. Health boards maintain that so long as there is any evidence of this process the child is capable of transmitting the disease to unprotected individuals. We have repeatedly seen cases of the ordinary type of scarlet fever in which desquamation continued for from six to eight weeks after the fever had subsided,

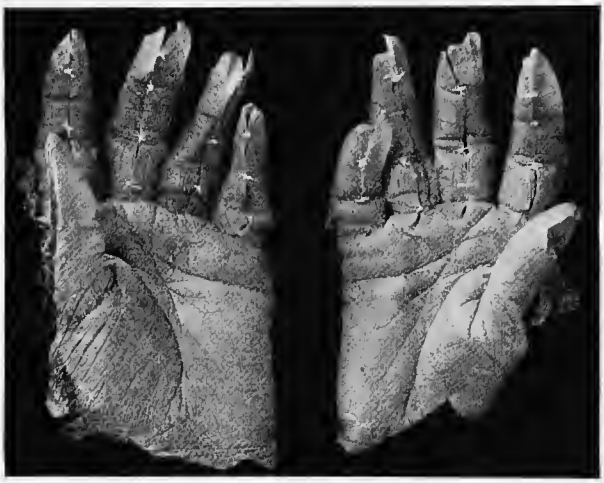


Fig. 317.—Epidermal Casts of the Hands Shed from a Fatal Case of Scarlet Fever (Welch and Schamberg).

and our experience has been that, in the majority of cases, the process requires about three weeks for its completion.

Ocular Manifestations.—In scarlet fever the conjunctivæ are markedly congested early and the eyelids are often swollen.

Nasal Symptoms.—Coryza develops together with the eruption, and may even precede it by a few hours, although the nasal secretion is but slightly increased after the first thirty-six hours. In some cases, however, a diminished secretion is present throughout the course of scarlet fever.

Gastro-intestinal Symptoms.—Vomiting is an early symptom in the majority of cases. During the first forty-eight hours anorexia occurs, but after this time, in uncomplicated cases, the appetite improves, as a rule, and by the end of the first week the child relishes its food. Constipation is the rule, and unless treated, usually obtains during the first week.

Local Symptoms.—With the onset of the disease the entire mucous membrane of the throat becomes reddened, and at times covered with a

thick, yellowish, tenacious mucus that serves to make a differential diagnosis between tonsillitis, measles, and scarlet fever difficult. The child complains of intense pain in the throat on swallowing or even on talking, and of lancinating pains shooting to the ears. He holds his head in one position, as though it were fixed, and utters a cry of pain on being compelled to move his chin.

Nervous Symptoms.—As previously stated, convulsions may occur early in scarlet fever, and mild delirium is not unknown, even in uncomplicated cases. It must be remembered that scarlet fever is one of the few diseases that show a predilection to attack the serous surfaces; consequently the physician should be ever alert for meningeal, joint, pericardial, endocardial, and pleural symptoms.

Illustrative Case of Scarlet Fever.—C. K. C., male, aged nine years.

Family History.—Parents and three sisters living and in apparent good health.

Previous History.—Developed measles at the age of four years and later had whooping-cough and mumps. Has enjoyed good health during the past two years.

Social History.—Breast-fed until one year of age. Began to go to school three months ago.

Present Illness.—While at school he complained of chilliness and of headache and was dismissed for the day. Upon reaching home he vomited, the headache became more severe, and he complained of intense pain in the throat. The child appeared to be feverish, and refused to take nourishment.

When seen, the child rested quietly in bed, with the chin slightly elevated. He complained of pain in the throat and neck whenever disturbed. Angina was increased by swallowing and by turning the head from side to side. He would complain occasionally of a shooting pain in the right ear.

When seen four hours later, the temperature was 102° F.; it continued to rise during the night, reaching 104° F. the following morning. The temperature continued high for a period of four days, when it fell gradually, reaching the normal by the ninth day.

The mother stated that, soon after returning from school, the child appeared to be greatly exhausted, showed some nervous twitchings of the hands and face, which were followed by a distinct convulsion. Mild delirium was present during the night for a period of three days, and at the time when the temperature was highest.

Physical Examination.—*General Examination.*—Twenty-four hours after the first symptoms were noticed the entire body was covered with a rose-red erythematous eruption. There was paling of the skin at those parts where pressure was made, and dermographia was well marked. The head was somewhat fixed, and the chin elevated.

Local Examination.—Inspection.—There was distinct swelling at the angles of the jaw, and by the end of the second day this was so prominent that the neck was greatly distorted. After the fifth week there was puffiness beneath the eyes, and swelling of the backs of the hands and of the feet and ankles. The erythematous eruption, which appeared at the end of the first twenty-four hours, faded by the end of the third day, and about one week later there was a distinct, scale-like desquamation, which continued until the beginning of the eighth week; the palms of the hands and the soles of the feet were the last to show desquamation. The mucous membrane of the tonsils and pharynx was intensely congested, and the tonsils were moderately swollen. The tongue was heavily coated at the beginning of the illness, and by the third day the typical strawberry appearance was present.

Palpation.—There were distinct enlargement and hardening of the lymph-nodes of the neck, and pressure upon them and in the vicinity of the angles of the jaw elicited pain. The pulse was rapid—130 beats a minute—and of high tension.

Auscultation.—The heart action was rapid, and the sounds were loud and clear. A few moist râles were present at the bases of both lungs posteriorly from the third to the fifth days of the disease.

Laboratory Findings.—Cultures from the throat showed the presence of streptococci and of diplococci. By the end of the second day the urine was scanty, not more than eight ounces being excreted during the twenty-four hours. The urine showed the presence of albumin, granular casts, leukocytes, and a few red cells until the tenth day of the disease, when the quantity of urine excreted was increased to from 20 to 30 ounces a day. Albuminuria continued, however, until the end of the fifth week, when epithelial casts and few granular and hyaline casts were present. From the fifth to the tenth weeks the amount of albumin gradually diminished, and the quantity of urine excreted became slowly greater until, by the twelfth week, the evidence of renal involvement

had disappeared. Early during the disease the only conspicuous blood change was a mild leukocytosis. A blood examination made during the fourth week showed: red cells, 3,150,000; hemoglobin, 70 per cent. The general characteristics of the stained blood were those known to secondary anemia.

Diagnosis by Induction from Clinical Data.—The sudden onset, developing with a chill followed by nausea, vomiting, angina, and a somewhat rapid rise in the temperature to 104° F., all strongly suggested the existence of scarlet fever. By the end of the first twenty-four hours the characteristic eruption appeared and confirmed the diagnosis. The later development of albuminuria, together with the characteristic desquamation, further supported the diagnosis.

Differential Diagnosis.—The sudden onset, headache, nausea, and vomiting somewhat resembled an attack of acute gastritis, from which scarlet fever was distinguished by: (1) Soreness of the throat; (2) the characteristic eruption by the end of the first thirty-six hours; (3) the fact that the high temperature continued for a longer period than is characteristic of gastritis; (4) the presence of albuminuria; (5) the appearance of a scaly desquamation at the end of the second week, which continued in a manner characteristic of scarlet fever.

Course of the Disease.—By the end of the third week the glandular enlargement of the neck had nearly disappeared, and desquamation was now taking place, and continued until the eighth week, when there was still some scaling of the palms of the hands and the soles of the feet. Albuminuria continued from the first to the tenth weeks of the disease, and from the fifth to the eighth weeks of the illness edema of the feet, ankles, and backs of the hands and puffiness beneath the eyes were conspicuous. The child was unable to leave his bed until the twelfth week following the initial symptoms, after which time convalescence progressed somewhat slowly.

Laboratory Diagnosis.—*Urinary Phenomena.*—Even in uncomplicated cases the urine is high colored, of high specific gravity,—1.020 to 1.030,—and diminished in quantity, from 15 to 30 fluidounces being excreted in a day; it contains a trace of albumin and is rich in solids. Casts, renal epithelium, and red blood-cells are by no means uncommon. During the first week of the disease the diazo-reaction is positive in from 15 to 20 per cent. of all cases.

The Blood.—Early during the course of scarlet fever the number of leukocytes is decidedly increased, ranging between 12,000 and 20,000 per c.mm. In uncomplicated cases the hemoglobin and red blood-cells are relatively decreased in number at the beginning of convalescence.

Nicoll and Williams* have reviewed the work done in reference to the **inclusion bodies of Döhle,** and at present there appears to be sufficient observations confirming the work of Döhle to regard the presence of these bodies in the blood of those suffering from scarlatina as a point of diagnostic value. These bodies are usually present in the blood of scarlatinal patients during the first week of the disease; they have been detected before the appearance of the eruption, and may persist after the sixth day of the disease, but in such event they are not present in great numbers.

Method.—Blood-smears are made after the usual method (see page 327), fixed and stained with Loffler's methylene-blue solution, or Manson's stain (borax methyl-blue), for a period of several hours.

Inclusion bodies are chiefly located in the polymorphonuclear leukocytes. These bodies vary greatly in size and form, from that of a small coccus to irregular masses, approximating one-fifth the size of the normal red blood-corpuscle. Certain of the bodies are elongated (so-called bacillary forms).

Manson's stain gives to the nuclei a deep blue cloor; the cytoplasm a very faint blue, and the inclusion bodies a tint varying between these two shades.

Caution.—Inclusion bodies have been found in the blood of patients suffering from measles, erysipelas, and syphilis.

*Archives of Pediatrics, May, 1912.

Cultures from the throat show streptococci, staphylococci, and bacilli, but no diphtheria bacilli, unless both diseases are present at the same time.

Malignant Scarlet Fever.—In this type of the disease there may be no prodromal symptoms; the attack is usually ushered in by a decided rigor or a convulsion. Angina is intense, and vomiting is, as a rule, uncontrollable.

The temperature rises abruptly to 105° or 107° F., and, as a rule, remains high until death occurs. Indeed, the temperature may reach its highest point one hour after death. In cases that tend to go on toward recovery the temperature becomes remittent after the first twenty-four hours, but may continue at a high point for several days, and, if the patient survive the first forty-eight to seventy-two hours, the fever-curve generally becomes septic in character.

Physical Signs.—*Inspection.*—In malignant cases the throat and, more particularly the tonsils, are usually covered with a thick, tenacious membrane that resembles in a measure the pseudomembrane of diphtheria.

Within the first twelve hours following the chill the child either becomes stupid or, in some instances, at least, restless, and there may be evidence of cyanosis, which often increases rapidly. The accumulation of mucus in the throat and mouth is very annoying, and the discharge from the nose is profuse. Before the end of the first day the glands of the neck are greatly enlarged, and a true acute cellulitis may exist.

Following the chill the skin may become very hot to the touch, although it may be livid in color; if the child survive until the end of the first twenty-four to thirty-six hours, a profuse scarlatinal eruption will appear. In the most severe types of scarlet fever the child succumbs to the disease before the appearance of the eruption, which is first detected after death. In the hemorrhagic type of malignant scarlet fever the eruption is petechial, and these minute hemorrhages frequently coalesce to form large ecchymoses. The hands and feet become pale and cold, and there is evidence of embarrassed circulation. Indeed, many of these cases fall into a state of circulatory collapse during the first twenty-four hours, and death soon follows.

Cases tending to go on toward recovery show, after the third day, a septic temperature, extensive necrosis and sloughing of the tonsils, ulcerations of the face, glandular enlargement, purulent rhinitis, and purulent otitis media.

If the case have assumed the hemorrhagic type of malignant scarlet fever, hemorrhages from any of the mucous surfaces may occur; the commonest of these is hematuria, although epistaxis and melena may appear.

Complications are far more common in the malignant types of scarlet fever that go on to convalescence than in the milder or ordinary forms of this disease.

Desquamation begins by the end of the first week, and is similar to that seen in other types of scarlet fever, except that the scales given off are larger, and may measure one, two, or even three inches in length. A complete cast of the finger or of the hand may be exfoliated. Following a profuse eruption, the hair and nails may fall out. Nephritis is common.

Mild Scarlet Fever.—When the disease is not marked by any decided symptoms, the invasion is of short duration and the child is, comparatively speaking, ill but a few hours. Among the symptoms are nausea, vomiting, headache, and fever, the temperature reaching 101° to 103° F. If examined carefully at this time, congestion and reddening of the pharynx, tonsils, uvula, and soft palate will be seen. The eruption is not profuse, and may occur only about the neck and chest, all evidence of it disappearing by

the third day. We have seen many cases of this type of scarlet fever in which the child played throughout the entire course of the illness.

In one epidemic occurring in Pennsylvania the eruption faded by the second day, and nearly all the cases seen were extremely mild and free from complications. The following year an epidemic broke out in the same vicinity, and one case of malignant scarlet fever was seen among every six children suffering from the disease. It must be remembered that a child may contract malignant scarlet fever from one who is suffering from a mild case. Holt states that in his dispensary service in New York city he has repeatedly seen children in the desquamation stage of scarlet fever who had never remained from school a day during the entire attack. The mild cases of scarlet fever are doubtless responsible for the majority of epidemic outbreaks.

Relapses.—Relapses are rare in scarlet fever; they occur most frequently during the stage of desquamation—seldom earlier than the tenth and not later than the twenty-fifth days. A relapse may be accompanied by most of the symptoms characteristic of scarlet fever, but, as a rule, they are all milder than those of the initial infection. In rare cases the relapse may assume a severe type and terminate fatally.

Summary of Diagnosis.—The characteristic symptoms and signs of scarlet fever are: The presence of an erythematous eruption; rapid, wiry pulse; vomiting; angina; strawberry tongue; and a characteristic, scale-like desquamation. Among the symptoms suggestive of scarlet fever should be mentioned an abrupt onset with a chill, possibly convulsions and fever, and the occurrence of certain serious complications.

Differential Diagnosis.—The various clinical forms of scarlet fever must be distinguished from **acute follicular tonsillitis,** the characteristic features of which have been tabulated below. The eruption of scarlet fever is to be distinguished from those rashes that follow the ingestion of large doses or the prolonged use of such drugs as quinin, belladonna, and the like. The characteristic pulse of scarlet fever and the angina are absent in **drug rashes,** and the eruption is rarely so diffuse as is that of scarlet fever.

Lastly, the eruption of scarlet fever is to be distinguished from that associated with acute **gastro-intestinal irritation,** such as that following the ingestion of certain vegetables, strawberries, and shell-fish. In the latter condition urticaria and intense itching are usually present, two features unknown to scarlet fever during the first twenty-four hours.

The following table sets forth the distinctive features of scarlet fever and of acute follicular tonsillitis:

Scarlet Fever.	Acute Follicular Tonsillitis.
1. There may be a history of exposure to the disease.	1. Exposure to cold and wet common.
2. Entire mucous membrane of the throat is intensely red.	2. Membrane of tonsils congested; small yellowish patches distributed over its surface. Often unilateral.
3. Nausea and vomiting follow the chill.	3. Vomiting rare.
4. Extensive scarlatinal eruption appears during the first thirty-six hours.	4. Eruption uncommon and never extensive.
5. Albuminuria develops by the end of the first week.	5. Albuminuria seldom present.
6. Characteristic desquamation begins by the end of the first week.	6. Desquamation not characteristic.

Acute Pharyngitis.—In this condition the initial symptoms are less severe, the eruption is absent, and there is but little likelihood of the occurrence of grave complications. (See table, p. 857.)

Measles.—Since both measles and scarlet fever are common among the diseases of childhood, a careful discrimination between the symptoms presented by each individual case must be made. Many workers among contagious diseases assert that scarlet fever and measles frequently develop in the same individual at or about the same time—an unfortunate condition that would render the making of an accurate diagnosis very difficult. For convenience of study we have tabulated the distinctive differential features of acute pharyngitis, scarlet fever, and measles.

Acute Pharyngitis.	Scarlet Fever.	Measles.
1. Usually follows a cold affecting the head and throat.	1. Follows exposure to the disease. Child healthy prior to the initial symptom.	1. Follows within ten to fourteen days after exposure.
2. May be ushered in with a slight chill or a series of chilly sensations.	2. Decided rigor may be the initial symptom.	2. Chill is less decided than in scarlet fever.
3. Temperature usually ranges between 99.3° and 101° F., although a high temperature is not impossible.	3. Temperature reaches 102° to 105° F., immediately following the chill.	3. Temperature rises steadily until the second day, and then remits until the fourth day, when with the appearance of the eruption, it again rises. Fever declines after the eruption has developed.
4. Vomiting unusual.	4. Vomiting an early symptom and may be persistent.	4. Vomiting not common.
5. No eruption.	5. A diffuse, erythematous rash, with red points. Appears within the first thirty-six hours.	5. Eruption does not appear until the fourth day. It appears in the form of blotches, first upon the neck and cheeks, and then spreads over the entire body.
6. Koplik's spots absent.	6. Koplik's spots absent.	6. Koplik's spots present.
7. No desquamation.	7. Scale-like desquamation begins at end of a week.	7. Branny desquamation.
8. There is scratching of the throat, with some pain upon talking, and swallowing.	8. Angina a most annoying symptom.	8. Moderate soreness of the throat.
9. There is a continuous desire to relieve the throat of mucus, but cough is seldom present unless the inflammation extends to the larynx.	9. The child makes no effort to clear the throat, but often places the hand to the throat when attempting to swallow.	9. There is a marked acute bronchitis, which begins early and continues throughout the course of the disease.
10. Photophobia and conjunctivitis absent.	10. Photophobia absent. Conjunctivitis may develop late.	10. Photophobia is an early symptom, and conjunctivitis is also common.
11. Albuminuria absent.	11. Albuminuria appears early and may continue throughout convalescence.	11. Albuminuria uncommon except in complicated cases.
12. Examination for plasmodium negative.	12. Examination for plasmodium negative.	12. Rosenberger's plasmodium may be recovered from the blood and from the secretions of the throat.

Clinical Course and Duration.—In moderately severe types of infection convalescence is well established during the third week, and if no complications set in, the child is able to leave the house as soon as desquamation is completed. In malignant scarlet fever death ensues, as a rule, by the fourth or fifth day, and many cases die during the first forty-eight hours. Complications of whatever nature increase the severity of the disease, and render the prognosis more unfavorable.

Complications and Sequelæ.—Pseudomembranous Angina.—This throat condition may be mistaken for diphtheria, which it greatly resembles; the only distinctive feature is obtained by making a bacteriologic study. Pseudomembranous angina is due to infection with a virulent streptococcus. It is possible, however, to have a mixed infection, both streptococcus and Bacillus diphtheriæ being present. Pseudomembranous angina develops either early during the course of scarlet fever or at the height of the disease. The membrane usually covers the tonsils, may extend to the soft palate, pharynx, nose, mouth and Eustachian tube, and may even invade the middle ear. The color of the membrane resembles that of diphtheria, and may be of a grayish-black or a grayish-brown hue. Pseudomembranous angina is also characterized by marked infiltration of the cellular tissue of the neck, swelling of the lymph-nodes, general edema of the throat, and difficulty in swallowing. After the condition has persisted for two or more days the expectoration and the discharge from the nose and mouth emit a fetid odor. There is some evidence of nasal obstruction, and occasionally laryngeal obstruction with associated croup ensues.

In practically all cases of membranous angina the constitutional symptoms are severe, and the general condition of the patient is of the nature of a profound streptococcus infection. The lymph-nodes may suppurate.

Gangrenous angina is known to complicate only the severest types of scarlet fever. The gangrenous process may be seen to involve the throat during the first forty-eight hours of the disease, and in rare instances it may be detected almost with the development of the infection. The tonsils and other affected mucous surfaces are grayish-black in color, and masses of necrotic tissue may be seen hanging from the involved areas. The gangrenous process may extend to the cellular tissue of the cheeks or neck.

The odor of the breath is characteristic of gangrene. The blood-vessels of the throat, and particularly those of the tonsils, are likely to be encroached upon by the gangrenous process, and death may result from hemorrhage. There is a rapidly progressing anemia, septic in nature, which is accompanied by the general symptoms of asthenia. Most cases of gangrenous angina terminate fatally between the third and seventh days.

Cellulitis.—Involvement of the cellular tissue of the neck may complicate severe cases of scarlet fever, but does not develop, as a rule, until the end of the first week. There is a somewhat rapid infiltration of the tissue, the head is held in a fixed position, respirations are often labored, and the skin of the neck becomes tense and presents a brawny appearance. The infiltration may be localized to the lymph-nodes, or, less often, it may be diffuse. Death usually results from septicemia, thrombosis of the jugular veins or of the lateral sinuses, meningitis, or pyemia.

Pulmonary Complications.—*Bronchitis* occurs less frequently during the course of scarlet fever than in either measles or diphtheria. *Bronchopneumonia* is the commonest pulmonary complication, and is most likely to

develop in those cases in which there are laryngeal stenosis, high fever, and delirium. Bronchopneumonia seldom develops until after the third day of the disease, and in many instances gives rise to no definite symptoms or signs by which it may be recognized until the condition is well advanced. *Empyema* may occur as a sequel of scarlet fever, and if permitted to run its course without treatment, is likely to result in general pyemia. *Edema of the lungs* is seldom seen unless scarlatinal nephritis, of which pulmonary edema may be the terminal stage, is present at the same time.

Cardiac Complications.—*Endocarditis and pericarditis* are said to be uncommon in the scarlet fever of children, although they may appear during convalescence. Endocarditis is sometimes seen after cases of scarlet fever in which sepsis has been a complication, and it is fairly common as a sequel of scarlatinal nephritis.

During convalescence a systolic murmur is frequently heard over the base of the heart (hemic), but with general improvement in the condition of the blood, the murmur disappears. Malignant endocarditis is seen only in those cases in which extensive suppuration is present. Associated with endocarditis there may be embolism of the brain, hemiplegia, and other paralyses, all of which are to be differentiated from post-diphtheritic paralysis. In the more severe forms of scarlet fever there is a variable degree of myocardial degeneration, manifested by diminished volume and force of the pulse, irregularity, and a tendency toward dicrotism. It is exceptional to find acute dilatation following the myocardial changes.

Involvement of the Serous Membranes.—As has previously been stated, the pleura, pericardium, and endocardium may become involved during the course of scarlet fever, and in particular is this true of the serous sacs of the joints, those of the wrists and hands suffering most frequently, although any of the other joints may be attacked. Carslaw collected the reports of 533 cases of scarlet fever, and found involvement of the articulations in 60 of them. Synovitis develops at the end of the first or the beginning of the second week of the disease. It is characterized by redness, swelling, tenderness of the joints, and a moderate elevation of temperature.

Involvement of the serous surface of the joints closely resembles that seen in acute articular rheumatism, except that it does not pass from joint to joint and seldom, if ever, causes a fatal termination. Septic arthritis, often associated with extensive throat involvement and pyemic symptoms, is rarely met in severe and fatal cases. Occasionally tuberculous invasion of the joints occurs as a sequel.

Auditory Complications.—Of these, the most common, and by far the most serious, is *otitis media*, which results from the direct extension of the infection from the pharynx through the Eustachian tube to the middle ear. Some writers assert that otitis media is the most frequent complication of scarlet fever, and that the younger the patient, the more likely is he to develop this disease.

Season is not without its influence, the greater number of ear complications occurring during the winter months. The type of the infection in a given epidemic also influences the number of ear complications during such an epidemic. Holt cites as an instance an epidemic occurring during the spring and summer of 1889, in which, in 73 cases, not one developed ear complications. In another epidemic in the same locality occurring during the winter months, of 43 cases of scarlet fever, one in every five developed ear complications. Finlayson collected 4339 cases from the literature, and found that otitis media occurred as a complication in 10 per cent. of them. Craig,

in an analysis of 1008 cases, found otitis media present in 13 per cent. When severe throat symptoms are present, ear involvement will be found to follow in from 20 to 70 per cent. of the cases.

Otitis media manifests itself by extreme pain in the ear and by an abrupt rise in temperature. Within a few hours rupture of the tympanum occurs, and purulent or bloody material is discharged from the ear.

The time at which involvement of the ear takes place may vary considerably in different cases and in different epidemics. Both ears are seldom attacked at the same time, and in the majority of cases the first evidence of otitis media is detected after the disease has reached its height. Ear complications seldom develop after convalescence is well established.

Deafness.—The pathologic changes present in the ear are usually of a suppurative nature. The hearing is generally markedly impaired, even if the attack of otitis media has been comparatively mild. The number of patients that become permanently deaf as a result of this complication is very high, and varies with different epidemics. May, in the study of 5613 mutes, found that 572 attributed their affliction to ear complications following scarlet fever.

Renal Complications.—*Nephritis* is the most serious and, therefore, the most important complication of scarlet fever. There is very often a slight albuminuria during the height of the fever, and it is possible that no more serious consequences will follow it than occur after other cases of febrile albuminuria. Two serious forms of nephritis are seen in scarlet fever, and although the symptoms of each are distinct, they have been confounded with each other.

(*a*) *Septic nephritis* is seen in those cases in which the angina is particularly severe, with sloughing tonsils, involvement of the soft palate, and general adenitis. In this form of nephritis the urine contains a large amount of albumin, but little or no blood and but few casts. The renal symptoms, if present, are masked by the manifestations of septicemia. Dropsy and uremia are rare, and the fatal termination occurs at the end of the second week of the process. The autopsy shows a kidney riddled with small metastatic abscesses.

(*b*) *Post-scarlatinal nephritis* is believed by the majority of writers to be the result of an inflammation of the epithelium lining the uriniferous tubules, similar to that covering the surface of the body. The renal disease may begin at any time from the end of the second to the end of the fourth week of the attack. The onset is insidious: a trace of albumin is first seen; then the amount of albumin increases, blood appears, and blood-casts and epithelial casts are found in the sediment. Fever returns, the amount of urine passed in twenty-four hours is diminished, the pulse is rapid and hard, and edema of the face appears; later there is edema of the feet and ankles, hands, and scrotum; vomiting occurs, and the patient is seriously ill of an acute nephritis. The complication may terminate in recovery or in uremia and death. A fatal termination may result from heart failure, due to dilatation following the high blood-pressure, from endocarditis with embolism, or from pericarditis. *Sudden death* is often the result of acute dilatation of the heart.

Gastro-intestinal Complications.—As is to be expected, in all cases of scarlet fever there is some interference with digestion, but true organic changes are unusual. Catarrhal stomatitis occasionally complicates the severer types of scarlet fever, but this condition seldom increases the gravity of the prognosis. Obstinate vomiting, probably nervous or uremic in origin,

may be an early and troublesome complication. Diarrhea, although uncommon, tends to deplete the child, and renders the prognosis less favorable.

Nervous Complications.—These are extraordinarily rare, except in severe types of infection. Convulsions, which are often present and may constitute the initial symptom, can hardly be regarded as a complication, but when seen late in the disease they are, as a rule, uremic in origin. Peripheral neuritis is occasionally observed, as is also meningitis. Hemiplegia and monoplegia may accompany either meningitis or ulcerative endocarditis.

DIPHTHERIA

(ANGINA MALIGNA; DIPHTHERITIS).

Pathologic Definition.—An acute, endemic, infectious, and transmissible disease, caused by the bacillus diphtheriæ. It may be sporadic or epidemic. It is characterized anatomically by the development of a grayish-

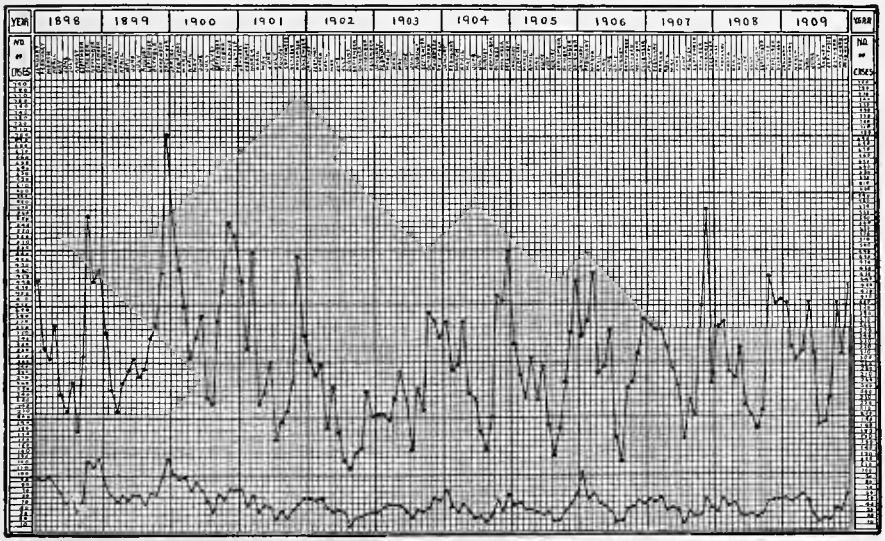


FIG. 318.—CHART SHOWING THE NUMBER OF CASES AND MORTALITY OF DIPHTHERIA BY MONTHS. STATISTICAL ANALYSIS OF 43,997 CASES (M. Sallom, in "Medical Record").

white pseudomembrane in the throat, which shows a special tendency to spread to the nose and the larynx. In severe cases there is a marked tendency for cardiac failure, postdiphtheritic paralysis, otitis media, conjunctivitis, bronchopneumonia, and acute nephritis to develop.

Varieties.—Among the varieties are to be considered: (1) *Tonsillar diphtheria* (mild diphtheria), in which the formation of the pseudomembrane is limited to the surface of the tonsil; (2) *malignant diphtheria*, in which all the symptoms are severe; (3) *pharyngeal diphtheria*, in which the pharynx appears to be the initial point of involvement; (4) *nasal diphtheria*, in which the pseudomembrane first develops in the nares, and then spreads to the pharynx; (5) *laryngeal diphtheria*, which first attacks the mucous membrane of the larynx; (6) *conjunctival diphtheria*, in which the pseudomembrane

develops on the conjunctiva; and (7) *wound diphtheria,* which results from infection of an open wound by the bacillus diphtheriæ.

Exciting and Predisposing Factors.—The exciting cause of diphtheria is the bacillus diphtheriæ. Isolation of the bacillus diphtheriæ is necessary in order to determine whether the membrane present is or is not a true diphtheric membrane, because other microörganisms are capable of causing similar pseudomembranes upon the mucous surface of the pharynx and the upper air-passages.

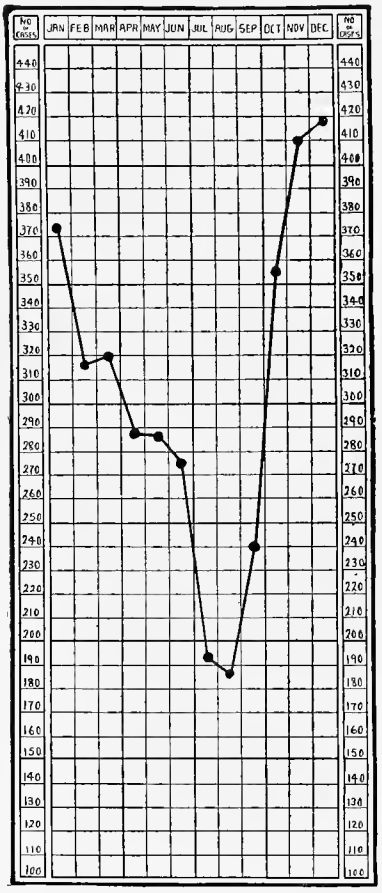


FIG. 319.—AVERAGE NUMBER OF CASES OF DIPHTHERIA OCCURRING DURING EACH MONTH OF THE YEAR. STATISTICAL ANALYSIS OF 43,997 CASES (M. Sallom, in "Medical Record").

(*a*) Prominent among the predisposing factors are **age,** the disease usually appearing between the second and the tenth years of life, as is shown by the following table taken from Billings, covering 14,688 deaths from diphtheria occurring in the city of New York:

Under one year	1,214
One to five years	9,622
Five to ten years	3,212
Ten to fifteen years	311
Over fifteen years	329
	14,688

Diphtheria rarely occurs after the age of fifty, although it may develop at practically any age.

(*b*) A history of **exposure** to the infection or of an epidemic is of great importance, for it is not uncommon for a large percentage of children attending a certain school to develop diphtheria within the short period of from two to four weeks.

(*c*) **Season.**—The greatest number of cases are seen during cold weather (see Fig. 318), as shown by Bosworth's analysis of 18,688 cases from the records of ten years of the Bureau of Health of New York city, where 10,769 deaths were recorded from October 1st to March 1st inclusive, whereas only 7,919 deaths occurred between April and September of the same years. This is due, in part, at least, to the fact that during the winter months large numbers of children are congregated in schools. M. Sallom's analysis of 43,997 cases reported in Philadelphia is given by Figs. 318 and 319.

(*d*) Many writers hold that chronic **irritation of the throat, nose,** and **pharynx** predisposes to the development of diphtheria; and it cannot be disputed that such acute conditions as tonsillitis, pharyngitis, influenza, measles, and scarlet fever enhance the tendency toward its development.

(*e*) **Previous attacks** predispose to subsequent infection. Instances

have recently been recorded in which epidemics of diphtheria have broken out among families supplied with milk from a certain dairy; in one case one of the workmen who handled the milk supplied to the affected district had suffered from a mild attack of diphtheria but a short time previous to the outbreak of the epidemic. There are numerous authentic reports of epidemics that have been traced to an infected milk-supply. In one epidemic, occurring in Philadelphia, 23 cases of diphtheria developed among families that obtained their provisions from a certain dealer, investigation proving that a child ill with diphtheria was then residing in the rear of this man's store. The mother, who nursed the child, at the same time handled the provisions supplied to customers.

(*f*) **Unhygienic surroundings** predispose to diphtheritic infection, for besides a lack of cleanliness, there is no attempt made to isolate the sick. Defective drainage and damp cellars have not been shown to bear any direct relation to epidemic outbreaks of diphtheria, although they may, by lowering the resistance of the individuals, serve as predisposing factors. On the other hand, epidemics of diphtheria often develop in rural districts, where sanitation is apparently perfect.

(*g*) **Climate** figures prominently as a predisposing factor, diphtheria being less common in tropical than in temperate zones. Excessive humidity and wet weather seem to promote the spread of diphtheria, probably because in inclement weather the children are closely housed; but they certainly do not exercise any influence upon the development of the diphtheria bacillus.

Transmission and Mode of Infection.—In almost all cities in the temperate zone the disease is endemic, an occasional case developing throughout the year; in such cities, where the population is greatly congested, periodic outbreaks or epidemics are to be expected. Diphtheria often develops in the most remote localities and in rural districts, and its transmission in these districts is, as a rule, inexplicable, but they may usually be traced to the fact that a case has not been recognized until several children have been exposed to contagion. Every case of diphtheria has its origin, either directly or remotely, in some previously existing case, although it is often difficult, and at times impossible, to trace such mode of infection. It has long been held that the bacillus diphtheriæ may enter the system with the inspired air or with the inhalation of the breath of a person suffering or convalescent from the disease. In view of our present knowledge of infection, however, this theory is questionable, and infection in this way probably does not occur, unless atomized particles of sputum or mucus from the throat of the patient are inhaled.

In the majority of cases infection probably takes place through the introduction of the diphtheria bacillus into the mouth; thus a child playing with toys that have been used by one suffering from diphtheria is likely to contract the disease by carrying them or his bacillus-laden fingers to his mouth. It must be remembered that the saliva contains the bacilli in great numbers throughout the course of the disease, and that they are also present in the secretions from the nose.

We have known diphtheria to spread from a single case to four other children, all of whom used a pencil that had been infected by the original patient. These five children were the only ones who developed diphtheria in a school of 46 pupils, during the entire school term of nine months. We believe that the disease is very often spread in a similar manner.

In laryngeal diphtheria, where coughing is a prominent symptom, so that much spray is atomized in the room, contagion is more likely to take

place than in cases that show no laryngeal involvement. During the period of convalescence the throat contains virulent diphtheria bacilli for weeks, and sometimes for months, and there is little doubt, therefore, that diphtheria is often spread by convalescents. "It has been shown that a person may harbor virulent bacilli in his nose and throat, and may even communicate the disease to others, without himself suffering from diphtheria at any time" (Holt). As a rule, virulent diphtheria bacilli may be recovered from the throat for from ten days to three weeks after the membrane has disappeared. In a study of 321 cases we found that the average time at which diphtheria bacilli could not be obtained from cultures after the membrane had disappeared was eleven days. The infection may be spread by means of clothing, rugs, and carpets, that have been soiled by the expectoration or the vomitus of the patient. Family epidemics are often the result of children playing upon the floor and contaminating their hands, and eventually conveying the bacilli to their lips. Again, epidemics have been known to follow the use of a drinking-cup or of a tooth-brush previously used by an infected individual. According to the researches of Rosenberger, the ordinary clinical thermometer is often the means of carrying diphtheria from the infected to the healthy.

Domestic animals may suffer from diphtheria, as has been demonstrated by the researches of Ravenel and others. The disease is found in pigeons, chickens, turkeys, and other fowl. Cats may suffer from diphtheria and spread the disease, but more often this is occasioned by a child suffering from the disease playing with the animal, and contaminating its fur with bacilli. Animals handled by sick children may come in contact with pets belonging to other families in the neighborhood, and the bacilli may be conveyed to these animals and thence to other children.

Incubation.—The period of incubation may vary greatly in different children and in different epidemics; *e. g.*, when most of the cases of a given epidemic are severe from the onset, the period of incubation is comparatively short,—two to four days,—whereas, on the other hand, in epidemics in which the majority of the cases is of a mild type, the incubation stage varies between four and twelve days.

Tonsillar Diphtheria (Mild Diphtheria).

Principal Complaint.—In this type of diphtheria there are no prodromal symptoms characteristic of the disease, although in the majority of cases the child may have refused to play or have stated that he felt indisposed on the day preceding the development of symptoms. Mild headache and soreness of the muscles of the limbs, particularly those of the back and shoulders, may be experienced, and the child complains of feeling cold at various intervals throughout the day. Convulsions are rare in this type of diphtheria except in young subjects.

Thermic features are not pronounced, an elevation of temperature of from one-half to one and one-half degrees being the rule. There may be soreness of the throat during the first twenty-four to forty-eight hours, and a mild degree of discomfort on swallowing is to be expected.

Physical Signs.—**Inspection.**—During the first day the surface of the tonsils, uvula, and soft palate is congested, and presents a livid appearance; by the second day a false membrane may be seen upon the tonsils, which may later spread to any portion of the throat or nares. In this mild type of diphtheria the membrane tends to remain localized, and the child may not seem to be extremely ill; in fact, he may not refuse to take his food throughout the entire course of the disease.

It is through the medium of these mild cases of tonsillar diphtheria that epidemics are started, for in many instances the physician is not consulted until other children have been exposed. It must not be forgotten that these mild forms of diphtheria often discharge highly virulent bacilli with the mucus and expectoration from the throat, and, according to the investigations of the New York Board of Health, virulent bacilli are frequently cultivated from such cases.

Although many of these cases go undetected, a fairly large percentage of them develop albuminuria during the stage of convalescence, and, indeed, this may prove to be a true acute parenchymatous nephritis that subsequently becomes chronic. Postdiphtheritic paralysis, affecting the muscles of the throat or of the extremities, may follow mild types of diphtheria, but the complications common to the severer forms are, as a rule, absent.

Pharyngeal Diphtheria.

Principal Complaint.—In diphtheria localized to the pharynx the symptoms develop insidiously, and often several days elapse before the patient experiences any decided annoyance. During the development of pharyngeal diphtheria, however, such prodromata as languor, chilliness, and lack of energy are complained of.

Thermic Features.—The temperature usually fluctuates between 99° and 100° F., and may be normal during the morning hours. It must be borne in mind that even mild cases of diphtheria may be accompanied by a high temperature or even by hyperpyrexia; such decided elevation in temperature is, as a rule, due to the presence of complications, *e. g.*, bronchopneumonia, acute nephritis, and otitis media.

Cardiac Symptoms.—There is a slight acceleration of the pulse-beats and as the disease progresses the pulse-rate may be out of all proportion to the mild degree of fever. The pulse may be weak, dicrotic, and intermittent, the quality being dependent upon the degree of intoxication.

Physical Signs.—Inspection and Palpation.—The lymph-nodes beneath the jaw are swollen and painful. In the milder types of pharyngeal diphtheria there may be only slight evidence of glandular enlargement. Glandular involvement, accompanied by a chill, even if the other symptoms are mild, should be regarded as strong evidence of the existence of diphtheria.

Examination of the throat discloses the fact that the mucous membrane of the pharynx is red and swollen, and in certain areas a variable degree of lividity is present. In this type of diphtheria the initial lesion is generally seen upon the mucous membrane of the tonsil, and the character of this exudate closely resembles that described under simple tonsillar diphtheria. The pseudomembrane spreads rapidly over the pharynx, soft palate, and uvula, and may involve the entire pharyngeal wall. It must be remembered that in true pharyngeal diphtheria the tonsils are also swollen and congested, and are not infrequently the site of the false membrane.

Nasal Diphtheria.

Principal Complaint.—In severe cases of pharyngeal diphtheria the process is likely to extend to the nasal mucosa, and sometimes the initial lesion is situated upon this membrane, whence it may spread to the pharynx, tonsils, and, less often, to the larynx. It must be stated that every case of nasal diphtheria is not accompanied by well-marked constitutional symptoms, although the majority of them are severe in character. In mild types of

nasal diphtheria the chief complaint is of inability to breathe freely through the nose and of coryza. *Inspection* of the nares with the head-mirror and speculum should always be made.

Thermic Features.—The thermic features of nasal diphtheria closely resemble those of the pharyngeal type, and are in no way characteristic. In a virulent type of infection the false membrane may extend from the nares to the conjunctiva.

Clinical Course and Duration.—In nasal diphtheria the course of the disease should be watched carefully, for there is, as a rule, special liability to the development of bronchopneumonia. Other complications common to pharyngeal diphtheria are also likely to develop in this type of the disease. Conjunctival involvement always renders the prognosis less favorable.

Laryngeal Diphtheria.

Laryngeal diphtheria is distinguishable from the types previously described in that the pseudomembrane forms upon the mucous surface of the larynx. Involvement of the laryngeal mucosa may occur without decided extension to the pharynx, nose, or tonsils, although, as a rule, the soft palate and the tonsils are involved. Laryngeal diphtheria differs in its symptomatology from the other types of diphtheria by the existence of a metallic cough and a peculiar harsh tone of voice.

The **thermic features** of laryngeal diphtheria are not characteristic, and mild constitutional symptoms generally occur unless the disease makes such progress as to interfere with respiration. In practically every case of laryngeal diphtheria the local manifestations give rise to alarm because of the obstruction and the fact that the toxic symptoms resulting from imperfect oxidation of the blood are added to the toxemia of diphtheria. The accessory muscles of respiration are brought into action—one of the earliest alarming symptoms in laryngeal diphtheria.

Clinical Course and Duration.—The prognosis rests upon the degree of laryngeal obstruction and the intensity of the cyanosis. When the patient is unable to rest in the recumbent posture, but sits bent forward, grasping some object firmly with both hands, relief is indicated and is absolutely essential to recovery. Death may result either from a portion of the detached membrane obstructing the larynx, or from the lodgment of particles of membrane in the bronchi, with the production of pneumonia.

Mild cases of laryngeal diphtheria terminate in recovery in from ten to fifteen days. In the severer forms convalescence is more or less protracted, and recovery is not complete for weeks or months. Complications are unusually frequent in laryngeal diphtheria. Before the introduction of the antitoxin treatment the percentage of deaths from laryngeal diphtheria was extremely high, and even now probably exceeds that of any other type of the disease.

Wound Diphtheria.

Infection of wounds with the bacillus diphtheriæ is decidedly uncommon, yet a few such cases have been seen. In this connection it may be well to state that the diphtheria bacillus does not live upon the normal human skin. The factors necessary for the development of the bacillus diphtheriæ appear to be heat and moisture, and when it is introduced into open wounds, it finds a fairly good soil for its development. Wounds that have become infected with diphtheria bacilli yield readily to treatment. Diphtheria of certain mucous surfaces, *e. g.*, the vagina and the conjunctiva, is very uncommon.

Further Considerations in the Prognosis of Diphtheria.

Illustrative Case of Diphtheria.—Charles A., aged seven years.

Family History.—The patient is the youngest of a family of five children all of whom suffered from diphtheria during an epidemic two and one-half years ago. Except for this illness, these children have enjoyed fair health.

Previous History.—The patient had measles and mumps at the age of four years, and diphtheria at the age of five years. Following his attack of diphtheria he had a permanent enlargement of the tonsils; he has been subject to attacks of acute tonsillitis, and on one occasion the tonsil was incised for peritonsillar abscess. There has also been impairment of the auditory function of the left ear since his first attack of diphtheria.

Social History.—He has attended school during the regular school year since he was six years of age. He resides in the city, but is permitted to play upon the street and in the parks the greater portion of each day. Three days before the patient was seen his mother learned that he had been playing with a child suffering from diphtheria.

Present Illness.—The child complains of feeling chilly, of headache and sore throat, and shows no disposition to play, but prefers to lie upon a couch the greater portion of the day. The pulse is weak—110 beats a minute; respirations, 30 a minute. There is slight soreness on swallowing; the voice is husky; and on the fourth day acute pain in the left ear developed.

When first seen by the physician, the child's temperature was 99.8° F., and upon the second day it had risen to 100.4° F. in the morning, and 101° F. during the evening. The temperature varied greatly during a period of four days, when it rose suddenly to 104° F. At this time the child complained of intense pain in the left ear and the other clinical manifestations of otitis media were present.

Physical Examination.—*General.*—The expression was unusually anxious. The patient breathed with his mouth open; there was some playing of the nostrils and little or no inclination to move about the bed. There was swelling at the angles of the jaw, and the glands of this region were extremely hard to the touch. The patient's body was perfectly nourished, but his face, fingers, and lips were pale.

Local Examination.—Inspection.—The mucous membrane of the tonsils and pharynx was markedly congested, and a distinct silvery membrane covered the greater portion of the left tonsil and a small area of the soft palate. Upon removing a portion of the membrane from the tonsil a bleeding surface remained, and the membrane reformed within the course of twelve hours.

Palpation.—The glands in the region of the neck were moderately enlarged, this being more marked upon the left side. (See General Examination, p. 865.) The pulse was extremely weak and thready, and showed a tendency toward dicrotism. As the disease progressed the pulse continued irregular, and both force and tension were diminished. Following the administration of circulatory stimulants the pulse became less frequent and its tension markedly improved. Within the course of twenty-four hours, following the administration of antitoxin, the pulse continued to improve.

Auscultation.—The breathing was somewhat labored, and the breath-sounds were distinctly audible at some distance from the bed. Numerous râles were heard over the entire surface of both lungs posteriorly.

Laboratory Findings.—A smear made from the false membrane removed from the tonsil showed the presence of great numbers of bacilli, which, morphologically, were identical with the bacillus diphtheriæ. Cultures from the throat developed bacillus diphtheriæ and streptococci. The urine obtained on the third day of the disease showed a trace of albumin, and repeated examinations revealed the presence of a moderate amount of albumin until the fourth week; this disappeared during convalescence. Microscopically, the urine contained many leukocytes and a few cylindroids.

Diagnosis by Induction from Clinical Data.—Of great importance is the clinical history that the child had been in the company of a playmate suffering from diphtheria three days before his physician declared that he was suffering from the same disease. Mild soreness of the throat, the presence of a distinct false membrane on the tonsils, and the high degree of prostration made the diagnosis fairly positive. The irregular character of the temperature and the additional fact that the false membrane, when removed from the tonsil and examined microscopically, showed the presence of bacilli morphologically identical with the bacillus of diphtheria, left but little room for doubt as to the nature of the disease. Cultures from the throat revealed the presence of colonies both of bacillus diphtheriæ and of streptococci, the finding of the first being sufficient evidence on which to base the diagnosis. Albuminuria and the general course of the disease bore out the findings.

Differential Diagnosis.—On account of the degree of angina and the marked reddening of the tonsillar mucous membrane the question as to the possibility of tonsillitis being present arose. The clinical evidence that eliminated the possibility of tonsillitis and confirmed that of diphtheria was: (*a*) The presence of a false membrane

in the throat, which when removed, left a bleeding surface behind; (*b*) the membrane contained bacilli resembling the diphtheria bacillus, and on culture true diphtheria bacilli were found; (*c*) the disease developed less abruptly than does tonsillitis, and the chill was less pronounced; (*d*) the temperature rose slowly to its maximum elevation, whereas in tonsillitis a more abrupt rise is the rule. Albuminuria, which was well marked and quite persistent, is uncommon in tonsillitis.

Course of the Disease.—The false membrane disappeared from the throat upon the third day of the disease, although repeated cultures showed the presence of diphtheria bacilli until the beginning of the third week. The temperature fell about one degree, and the pulse-rate was diminished 10 beats a minute within twenty-four hours following the administration of antitoxin and cardiac stimulants. The agonizing pain in the left ear was promptly relieved by puncture of the drumhead. During the fifth week of the disease, at a time when convalescence was progressing most favorably, the mother observed that the child experienced difficulty in swallowing, and that liquid foods were regurgitated through the nostrils (paralysis of the palate), but this somewhat frequent complication improved rapidly, and complete recovery ensued.

Laboratory Diagnosis of Diphtheritic Disease of the Throat.—Cultures made from bits of false membrane taken from the different areas and from the saliva develop colonies of the bacillus diphtheriæ. It must be remembered that the diphtheria bacillus grows best upon a special medium made from blood-serum. It is unusual to obtain a pure culture of the bacillus diphtheriæ from the patches in the throat or the nose, but colonies of other bacilli, cocci, and spirilla are likely to be present in the same culture. Smears made from the culture should be stained with Löffler's alkaline methylene-blue in order to demonstrate the presence of bacillus diphtheriæ.

In the majority of instances a diagnosis of diphtheria is made from a cultural study of the false membrane, but it must be remembered that other bacilli, whose cultural and tinctorial properties are similar to those of bacillus diphtheriæ, are also present in the throat. The only positive evidence to be had that we are not dealing with the bacillus pseudodiphtheriæ (bacillus xerosis) is obtained by injecting a portion of a bouillon culture of the suspected organism into a rabbit or a guinea-pig. The injection of such a culture will kill the animal in twenty-four or forty-eight hours if the organism is the bacillus diphtheriæ, but bacillus pseudodiphtheriæ will not kill the animal. Although this precaution is not taken in the routine work of health boards in making a diagnosis of diphtheria, we believe that this experiment should be performed whenever a doubt arises, and before rigid quarantine is established.

Early during the course of diphtheria the quantity of urine excreted is greatly diminished; its specific gravity is increased; its color is high; and, as the disease progresses, albuminuria is likely to develop. During the first week of the disease the diazo-reaction will be found positive in from 10 to 15 per cent. of all cases. Microscopically, the urine contains few red blood-cells, many leukocytes, degenerated renal epithelium, and casts. Conradi and Bierast have recovered diphtheria bacilli from the urine of infected children.

Summary of Diagnosis.—The detection of the characteristic membrane in the throat, the pronounced circulatory symptoms, mild fever, albuminuria, and prostration, all point strongly toward the existence of diphtheria. The diagnosis is confirmed by the finding of the bacillus diphtheriæ in the cultures.

The hemoglobin content falls to between 75 and 65 per cent. during the first week of diphtheria, and the red cells are greatly decreased in number by the beginning of the third week. Leukocytosis is an early feature of diphtheria, and its degree is dependent, as a rule, on the severity of the infection.

Differential Diagnosis.—Infections of the throat that might readily be mistaken for diphtheria are: *Acute follicular tonsillitis, acute pharyngitis, streptococcus angina, and scarlet fever.*

Streptococcus angina is a condition in which there is an extensive formation of pseudomembrane on the throat, due to infection with the streptococcus. It is to be distinguished from diphtheria by the intense pain, abrupt development, and the absence of albuminuria and of glandular involvement. The accompanying table shows the distinctive features of acute follicular tonsillitis, diphtheria, and scarlet fever:

Follicular Tonsillitis.	Diphtheria.	Scarlet Fever.
1. Premonitory symptoms are mild sore throat, headache, and constipation.	1. Premonitory symptoms often absent or indistinct.	1. There is usually lassitude for a few hours, and nausea and vomiting may be the cardinal symptoms.
2. Onset with a chill, which may be either mild or severe.	2. No rigor or chilly sensations may be experienced.	2. Chill may be the initial symptom.
3. Temperature reaches 102° to 104° F., within a few hours after the chill, and remains high for a few days unless reduced by the administration of sodium salicylate.	3. Temperature, 99° to 101° F., by the end of the first day. Not influenced by salicylates, but falls after injection of diphtheria antitoxin.	3. Temperature ranges between 102° and 106° F., immediately following the chill. Fever is in proportion to the amount of eruption, and usually declines as this disappears.
4. Pulse, 90 to 110 a minute, bounding, but not wiry. The frequency is dependent upon the temperature.	4. Pulse may be rapid, but beats are less forcible than in either tonsillitis or scarlet fever. Tends to become irregular and compressible.	4. Pulse is greatly accelerated—120 to 140 a minute, bounding, hard, wiry, and out of proportion to the temperature.
5. Eruption unusual and not characteristic.	5. No characteristic eruption, although varied forms of eruption are occasionally seen.	5. Eruption appears during the first thirty-six hours. It is diffuse, dusky red, with an occasional slightly raised spot. It is seen first in the region of the clavicles and chest, but spreads to all parts of the body. The skin is intensely hot, and pressure over the back or chest causes a decided paling. Eruption fades within forty-eight to seventy-two hours.
6. Lymphatic glands of throat and neck are greatly enlarged, except when complicated by peritonsillar abscess.	6. Glands of the neck enlarged early, and have a peculiar, stony feel.	6. Glands not enlarged at first, but later show enlargement.
7. Tongue is heavily coated with a whitish or yellowish fur. Later, the tongue may be intensely red and show marking of the teeth along its edges.	7. Tongue slightly coated, but not characteristic.	7. Tongue is coated with a thick white fur, which peels from the edges on the fourth day, leaving a bright-red surface. Small red elevations (strawberry tongue) are seen scattered here and there.
8. Redness localized to the tonsils. Small elevated yellowish spots over the surface of the tonsils, which, when removed, leave only a reddened surface.	8. Grayish or silvery membrane on the tonsils, but more often on the pillars of the fauces and the soft palate. When removed leaves a bleeding surface.	8. Fauces are either a slight or intense dusky red. Marked swelling of the throat, and in some instances isolated white spots, are to be seen.

Follicular Tonsillitis.	Diphtheria.	Scarlet Fever.
9. Membrane, when removed, seldom reforms.	9. A new pseudomembrane reforms after the initial one has been removed.	9. Membrane absent.
10. Albuminuria absent.	10. Albuminuria occurs as early as the second or third day, and may continue throughout convalescence.	10. Albuminuria seldom appears until after desquamation has begun.
11. No desquamation.	11. Desquamation absent.	11. Extensive desquamation, beginning from the sixth to the tenth day, and continuing for from one to several weeks. Desquamation is pronounced upon the palms of the hands and the soles of feet.
12. Complications are rare.	12. Ear and eye complications are quite common. Pneumonia and postdiphtheric paralysis frequent.	12. Otitis media, with consequent deafness, is the most frequent complication. Arthritis, endocarditis, and suppuration of the submaxillary glands are less frequent. Pericarditis, epilepsy, endocarditis, and mental weakness have been observed.
13. Cultures from the surface of the tonsil and from pus from the abscess do not show diphtheria bacilli, but contain other organisms.	13. Both smears and cultures from the false membrane show diphtheria bacilli.	13. Cultures from the throat show streptococci and diplococci, the latter being pathogenic for hogs, rats and other animals.

Clinical Course and Duration.—In every case of diphtheria the course of the disease is dependent upon the following conditions: (1) The virulence of the type of infection; (2) the age of the patient—the older the child, the more favorable the prognosis; (3) the institution of proper treatment within the first twenty-four hours, antitoxin being of limited value unless administered early; (4) the presence or absence of complications (see Complications, p. 870); (5) history of previous attacks—in repeated attacks each subsequent infection tends to be more mild than the preceding one, although exceptions to this rule are fairly common.

The mortality rate for diphtheria has lately been reduced from 40 to about 20 per cent., and this change coincides closely with the introduction of serum therapy. The prognosis should always be given guardedly, for even the mildest cases may develop serious complications, *e. g.*, nephritis and multiple neuritis.

The prognosis is also governed by the rapidity and strength of the heart-beats. In unfavorable cases the pulse becomes weak, rapid, and dicrotic. If the pulse is irregular and the heart action extremely slow, the prognosis is grave.

Cases of nephritis usually recover from the initial attack, and the patient is able to get about the house in from six to ten weeks, but the nephritis is likely to recur within the course of a year or more.

During the acute stage of diphtheria death may result from laryngeal stenosis, the inspiration of false membrane into the bronchi, with a resultant bronchopneumonia, septic infection, and cardiac paralysis.

Complications.—The most frequent complication occurring in

diphtheria is **bronchopneumonia,** and this is most common in cases of diphtheria of the larynx, and when interference with either respiration or deglutition is present. The presence of bronchopneumonia aggravates all the symptoms of diphtheria, and, in addition, is responsible for many of the symptoms known to this infection. (See Bronchopneumonia, p. 107.)

Dysphagia occasionally complicates diphtheria, and its presence, because of the prolonged strain it imposes upon the general economy, is an unfavorable accompaniment.

Neuritis seldom appears until the third or fourth week of the disease, and often not until convalescence is apparently well established. The child suddenly notices that upon attempting to swallow food or liquid it is regurgitated through the nose. Paralyses are most likely to involve the muscles of the soft palate, yet paralysis of the extremities is ordinarily seen, when the knee and biceps reflexes are absent. Rarely, indeed, is paralysis of the muscles of the trunk observed.

Albuminuria, when marked, should be considered under the head of complications, although a mild degree of albuminuria is a symptom of practically all forms of diphtheria, and, in the absence of a bacteriologic study, is of great importance in formulating a diagnosis. When the amount of albumin excreted is very high, the gravity of the disease is enhanced. In *acute nephritis* with anuria the prognosis is unfavorable.

Ocular Complications.—The false membrane may extend to the conjunctiva, in which case the prognosis is grave. *Strabismus* is occasionally seen, and still less often there is paralysis of the ciliary muscles, with dimness of vision and disturbed accommodation.

Otitis media is a frequent complication, and with its development all the constitutional symptoms of diphtheria are aggravated. Impairment of hearing, or even total deafness, may follow, and there may also be a chronic otorrhea.

Myocarditis.—The *heart* muscle is perceptibly weakened, and this weakness is characterized by a distinctly feeble pulse. The heart-sounds are weak, and in extreme cases the muscular quality of the systolic sound is absent. A moderate degree of cardiac dilatation is present. Not infrequently death results from cardiac failure, the final change being that of acute dilatation.

The more marked the myocarditis, the more pronounced is the *anemia* during convalescence; it is quite impossible to restore the blood to its normal tone unless the heart be well fortified. Both the anemia and the myocarditis are due to the diphtheria toxin.

MEASLES.

Pathologic Definition.—An acute infectious disease, characterized by the presence of a catarrhal inflammation of the respiratory mucous membrane. The only lesions that are constant are those on the skin and mucous membranes. The cutaneous manifestations consist of a superficial inflammation, accompanied by congestion and by an exudation of round-cells in the region of the smaller blood-vessels and around the sweat-glands. The skin, particularly of the face, is somewhat edematous. The mucous surfaces are involved quite as commonly as is the skin, consequently catarrhal conjunctivitis, rhinitis, pharyngitis, and tracheobronchitis are present.

Exciting and Predisposing Factors.—Bacteriology.—The cause of the disease is unknown. The lesions of the respiratory tract show the presence of staphylococci and streptococci, and in some cases one organism may predominate, whereas in others, almost equally severe, the reverse con-

dition obtains. Both streptococci and staphylococci may be present in the sputum, and the pneumococcus and other diplococci may also be recovered. In those cases in which bronchopneumonia complicates measles, the bacteriologic findings are practically identical with those described under Bronchopneumonia. (See p. 110.)

Parasitology.—Rosenberger and others have studied a doubtful parasite (protozoön?) found in the blood of those suffering from measles. Certain observers assert that they have found specific bacteria in the blood and on incising the lesions of this disease.

Measles is usually spread by *direct contagion*, although the disease is occasionally transmitted by clothing and furniture. Goetze* succeeded in transmitting the disease to swine by injecting the patient's blood during the eruptive stage of measles.

Immunity.—One attack of measles generally establishes permanent immunity.

Age.—Age figures prominently as a predisposing factor, the majority of cases being seen during childhood and after the third year of life. The disease rarely attacks children under one year of age, and the infant members of a family in which all the older children are affected frequently escape infection. The aged who have not suffered from a previous attack of measles are less likely to develop the disease than are those in the first, second, and third decades of life. Experience has shown that measles is highly contagious by direct exposure, especially when children are permitted to associate with those ill with the disease.

Period of Incubation.—Holt, in an analysis of 144 cases in which the incubation period could be definitely determined, gives the following table:

Incubation of	less than nine days	3	cases
"	" nine or ten days	22	"
"	" eleven to fourteen days	95	"
"	" fifteen to seventeen days	19	"
"	" eighteen to twenty-five days	5	"

It will be seen from the preceding table that in 66 per cent. of cases the incubation period varied between eleven and fourteen days; that in but a single instance was it less than one week; and in but three cases did it develop before the ninth day.

Duration of the Infective Period.—This is short as compared with scarlet fever, the average time being placed at four weeks, but instances are recorded in which, apparently, a child has conveyed the disease to another thirty days after the appearance of the rash. Those ill of the disease are capable of transmitting it to others after the appearance of the catarrhal symptoms referable to the respiratory tract, and there are apparently authentic records of cases in which the disease was transmitted to others two to four days before the eruption developed.

Principal Complaint.—There are, as a rule, marked prodromal manifestations, the patient complaining of headache, malaise, constipation, soreness and aching of the muscles, and photophobia for two or three days.

Catarrhal Stage.—The early symptoms resemble those of a cold in the head. The child has fever, marked coryza, lacrimation, and a dry *cough*, and sneezes frequently. The symptoms of a catarrhal laryngitis and bronchitis are also present, and there is an abundant secretion from the respiratory mucous membrane, as well as from the conjunctivæ. There is aching of the back and limbs.

* Jahrbuch für Kinder, August, 1912.

Thermic Features.—There is usually an elevation of temperature of from one to two degrees during the evening hours, with decided morning remissions.

Physical Signs.—Inspection.—The mucous membrane of the fauces, tonsils, and pharynx is congested, and an eruption may be seen upon the mucous membrane of the cheeks, palate, and the lips. This eruption, which is known as *Koplik's spots*, is composed of bluish-white specks surrounded by a red areola. It is found on the buccal mucous membrane two or three days before the rash appears on the skin. These spots should be sought for in strong daylight, since artificial light does not bring out the colors nor properly illuminate the spots. Very frequently they are seen only opposite the molar teeth, although they may be present on any part of the buccal mucous membrane.

Eruptive Stage.—*Principal Complaint.*—This is highly characteristic. With the appearance of the eruption there may be a slight amelioration in certain of the constitutional symptoms. (See Thermic Features.) The cough may now become metallic or ringing in character, and in certain cases this constitutes the most annoying symptom. Headache, which has been persistent during the preëruptive stage, may now ameliorate or subside.

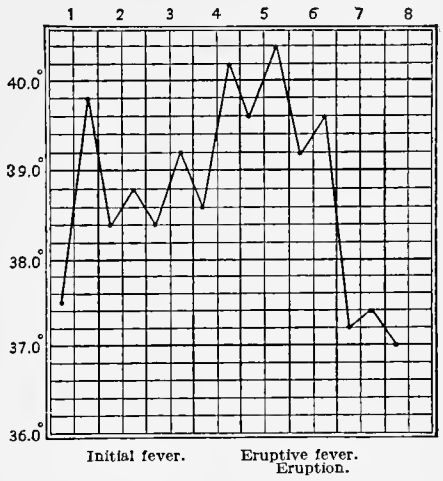


FIG. 320.—TEMPERATURE-CURVE OF A CASE OF MEASLES) (J. M. Anders).

Thermic Features.—The temperature, which during the preëruptive stage may have registered as high as 103° or 105° F., falls with the development of the rash, and remains at a much lower level during the eruptive stage (Fig. 320). At about the fifth or sixth day there is a decided abatement in the severity of all the symptoms, particularly those referable to the respiratory tract and to the eyes.

Physical Signs.—Inspection.—Initial Eruption.—At the onset of the disease, and within the first twelve hours after the child shows some indisposition and headache, there is an almost universal reddening of the skin resembling a faint scarlatinal eruption. This initial eruption disappears within a few hours, and at the time of its disappearance Koplik's spots make their appearance on the buccal mucous membrane. The physician seldom detects the initial rash because the patient is not seen until this feature of the disease has disappeared. Experience has shown that it is practically impossible to distinguish between the pre-eruptive measle stage and the mild attack of scarlet fever, and the diagnosis is usually made at the time of the appearance of Koplic's spots or of the typical eruption. The typical rash makes its appearance during the fourth day of the disease, and is seen first upon the face, neck, and forehead, spreading thence to the trunk and extremities. As the result of the swelling the features are somewhat distorted, and those portions of the face not affected by the eruption are intensely red. There is always a distinct discharge from the conjunctivæ,

which are greatly congested. The eruption of measles is composed of small, disk-like papules, which show a tendency to coalesce, giving to the skin a more or less blotched appearance. The eruption becomes more and more profuse, and within the course of two or three days the trunk and extremities are well covered. The eruption is rarely papular. By the end of the fifth or the sixth days the eruption has attained its height (Fig. 321),

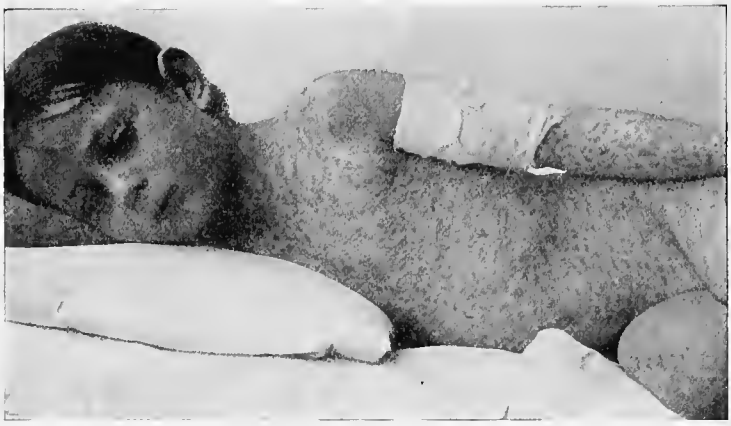


FIG. 321.—WELL-MARKED MEASLES ERUPTION ON THE FIFTH DAY OF THE DISEASE (Welch and Schamberg).

and an appreciable fading now begins, first involving the face and neck, and then extending over the body and extremities. Following this a fine bran-like desquamation spreads over the body in the same topographic manner, the skin now displaying a flushed or somewhat mottled appearance.

Laboratory Diagnosis.—The **sputum** secreted is increased, and contains many microörganisms, staphylococci, and streptococci. Czajkamski

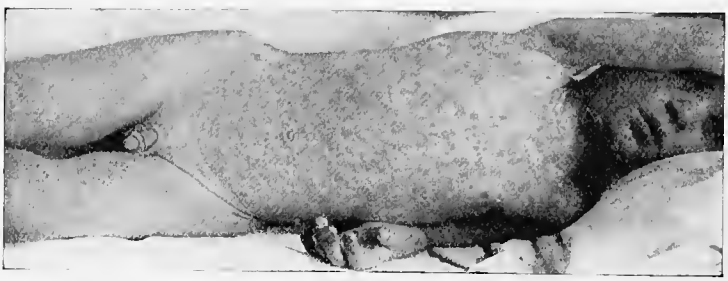


FIG. 322.—MEASLES IN A CHILD (Welch and Schamberg).

has described a special motile bacillus, which was decolorized by Gram's method. Schottelius has isolated the staphylococcus pyogenes aureus from the conjunctivæ, and pathogenic bacteria may also be discovered in the viscera in fatal cases.

The *urine* is decreased in quantity and high in color and in specific gravity, and during the height of the febrile period may contain a trace of albumin. In those cases complicated by nephritis both albumin and casts are present, and occasionally hematuria is observed. The *diazo-reaction* is found in nearly all cases of measles, at least in the earlier stages. It is not

present in German measles, and its occurrence may serve as a means of diagnosis between the two conditions.

Constipation obtains during the febrile period, but after the eruption has fully developed, and even during convalescence, diarrhea often forms one of the annoying complications.

Illustrative Case of Measles.—James L., aged five and one-half years.

Family History.—Parents two older sisters, and a brother are living and in good health.

Previous History.—Had mumps at the age of three years, tonsillitis and scarlet fever a year ago, since which time he was well.

Social History.—His playmates have been members of families in which unques-

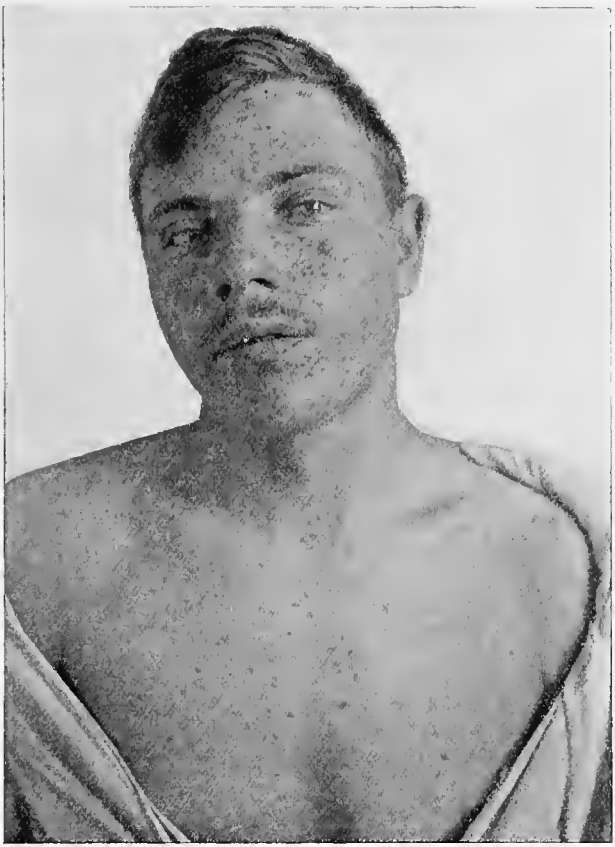


Fig. 323.—Measles of the Papular Type in an Adult (Welch and Schamberg). Mistaken, during a variolous epidemic, for smallpox.

tionable cases of measles have occurred during the past few months. There is, however, no history of direct association with children who have recently suffered from the disease.

Present Illness.—This began quite abruptly, with distinct chilliness, followed by vomiting. He complained of feeling ill, and showed an intolerance to light, and no disposition to play. During the first twenty-four hours there was intense headache, soreness of the limbs, arms, and back, and distress on being moved by his nurse. The throat was sore, and there was pain upon swallowing.

Cough developed early, and was harsh and rasping in character, and unaccompanied

by expectoration. It continued until the fifth day of the disease, and showed no tendency to subside until the patient had been given mild doses of codein. By the tenth day the cough had practically disappeared, although it occasionally followed exertion.

On the third and fourth days the child gave the appearance of being extremely ill, and mild delirium was present during the night. The headache disappeared during the second day, and did not return during the entire course of the disease.

Following the chill the temperature rose abruptly to from 102° to 103° F., and remained near this point for a period of about twelve hours. During the second day of the disease the temperature showed an appreciable decline, but did not reach the normal, and upon the fourth day, with the appearance of the eruption, the temperature again rose to 104° F., after which it fell gradually, reaching the normal between the fifth and the seventh days

Physical Examination.—*General.*—When first seen, the child rested quietly in bed, since movement excited both headache and cough. The head was held in a somewhat fixed position. There was an expression of pain on swallowing, and the child was unusually irritable and displayed an intolerance to light.

Local Examination.—Inspection.—The face was flushed; the mucous membrane of the tonsils and pharynx was congested, and by the second day of the disease Koplik's spots were present on the soft palate. The nostrils played freely, and the respirations numbered 40 a minute. The conjunctivæ were reddened. On the third day of the illness a peculiar erythematous reddening of the skin appeared, which was most marked on the inner surface of the arms and about the neck; this continued to extend until, by the end of the fifth day, the entire body was covered with the typical eruption.

Palpation.—The pulse was full and strong at first, the beats numbering 110 a minute; during the fourth day, however, and at a time when the fever was highest, the pulse increased to 120 a minute.

Auscultation.—Numerous fine and coarse râles were heard over both lungs, and at the apices of the lungs the respiratory murmur was harsh.

Laboratory Findings.—During the height of the fever the urine contained a trace of albumin, but microscopic analysis was negative.

Diagnosis by Induction from Clinical Data.—The child had probably been associated with other children who were developing the disease. The diagnosis remained in doubt in spite of the fact that the onset was characteristic, the harsh, rasping cough, photophobia, and pain in the back and limbs suggesting strongly the existence of measles. A positive diagnosis was not possible until the development of the eruption (Koplik's spots). The temperature was in itself quite characteristic (see Thermic Features, p. 873), but in no way aided in the early recognition of the disease.

Course of the Disease.—After the first twenty-four hours there was a distinct amelioration in all the symptoms except the cough, but during the third day the child's condition became less favorable, and he remained extremely ill until the eruption was well developed. (See Thermic Features, p. 873.) Headache and muscular pains, which were present during the first day, were greatly relieved by the administration of proper medicaments, and the cough also improved following treatment. By the beginning of the third week the child was able to leave his room, and from this period recovery was uninterrupted.

Summary of Diagnosis.—The diagnosis is, as a rule, doubtful until the characteristic eruption appears. During the preëruptive stage a provisional diagnosis, based upon the following symptoms, may be made: Headache, malaise, chilly sensations, anorexia, pain in the eyeballs, intolerance of light, and the presence of Koplik's spots upon the mucous membrane of the mouth. With the appearance of the characteristic eruption the diagnosis is confirmed (see Eruptive Stage, p. 873); this eruption fades from the fifth to the sixth day, and is followed by a branny desquamation.

Differential Diagnosis.—Measles is to be distinguished, first, from **scarlet fever,** and the following table sets forth the distinctive clinical features of these two diseases:

Measles.	Scarlet Fever.
1. History of exposure to measles.	1. History of exposure or of an epidemic of scarlet fever.
2. Prodromal symptoms continue for three days.	2. Prodromal period short.

Measles.—(*Continued.*)	Scarlet Fever.—(*Continued.*)
3. Symptoms become more and more intense until the development of the eruption by the end of the third or fourth day.	3. Symptoms violent both before and after the appearance of the rash, which is seen by the end of the first twenty-four to thirty-six hours.
4. Fever falls just before the rash appears and after the eruption is fully developed.	4. Fever remains high.
5. Eruption disappears from the fifth to the sixth day.	5. Eruption disappears by the second to the third day.
6. Eruption maculopapular.	6. Eruption erythematous and punctate.
7. Cough and catarrhal conjunctivitis.	7. Cough less prominent.
8. Photophobia is an annoying feature.	8. Photophobia absent.
9. There may be slight albuminuria.	9. Albuminuria is common, especially during convalescence.
10. Tongue is heavily coated, and often swollen, with Koplik's spots upon the buccal mucous membrane.	10. Characteristic strawberry tongue.
11. Tendency toward the development of bronchopneumonia and eye complications.	11. Renal and cardiac complications quite common.
12. Pulse full and of fair tension, numbering 100 to 110 beats a minute.	12. Pulse full and bounding and of high tension, 120 to 140 beats a minute.
13. Bran-like desquamation by the end of the sixth or beginning of the seventh day, and continuing for approximately one week.	13. Desquamation is scale-like, and may continue for from three to six or more weeks. Entire casts of the hands, fingers, or foot may be given off, and the palms of the hands and the soles of the feet are the last to be concerned in this process.
14. The leukocytes are normal or subnormal in number.	14. Leukocytosis is present.

The absence of leukocytosis also distinguishes measles from **variola, varicella,** and **rubella.**

The accompanying table, taken from Rotch, shows the differential points between measles and other acute infections for which it is likely to be mistaken:

	Measles.	Variola.	Varicella.	Scarlet Fever.	Rubella.
Incubation....	10 days.	12 days.	17 days.	4 days.	21 days.
Prodromata...	3 "	3 "	A few hours.	2 "	A few hours.
Efflorescence..	Papules.	Macules. Papules. Vesicles. Pustules.	Vesicles.	Erythema.	Papules.
Desquamation	Furfuraceous.	Large crusts.	Small crusts.	Lamellar	
Complications and sequelæ.	Eye and lung.	Larynx. Lungs.		Kidney, ear and heart.	

Clinical Course and Duration.—The clinical course is divided into three stages: (1) The preëruptive stage, previously described; (2) the eruptive stage (see p. 873); and (3) the stage of defervescence. The time required for convalescence to be well established varies between ten and sixteen days. In mild cases of the disease in children desquamation may be nearly completed by the tenth or twelfth day, but in adults, in whom the clinical expression of the disease is more severe, a longer period is required.

Complications.—These consist chiefly of pulmonary affections, among which **bronchopneumonia** is prominent; **lobar pneumonia,**

purulent bronchitis, and **chronic bronchitis** are less frequent. **Otitis media** and **simple catarrh of the middle ear** are exceedingly common in measles. **Chronic conjunctivitis, blepharitis,** and **ulceration of the cornea** are by no means unusual. **Nephritis** is occasionally observed when patients are subjected to exposure (cold and wet) during the preëruptive stage or before convalescence is completed. **Gastro-intestinal catarrh** may continue for weeks or even months after an attack of measles, and is usually manifested by exacerbations of diarrhea.

Sequelæ.—Following an attack of measles the hair may fall, and in many instances a luxurious growth is never restored. Permanent impairment or even destruction of the auditory sense is occasionally encountered, as are also chronic conjunctivitis and nephritis.

RUBELLA

(RÖTHELN; GERMAN MEASLES).

Pathologic Definition.—An acute infectious disease, characterized by enlargement of the postcervical glands and the presence of a cutaneous eruption.

Predisposing Factors.—Rubella may occur either epidemically or sporadically. It is generally conceded to be of probable microbic origin, although the specific organism capable of exciting the disease has not as yet been determined. Exposure to the disease appears to be a prominent factor in the majority of instances. House and local epidemics in schools and homes are common. The disease spreads more rapidly in those places in which unhygienic surroundings prevail.

The channel through which the disease is conveyed from sick to healthy individuals is not positively known, although it is believed to take place through cutaneous emanation, exhalation of the breath, and by means of clothing and other articles. The *infective period* is believed to begin some hours, or possibly days, prior to the development of the eruption, and continues until convalescence is well established.

Immunity.—One attack usually establishes immunity, although, in 719 cases studied by us, second attacks occurred in 2.5 per cent.

Incubation Period.—In the vast majority of instances the stage of incubation continues for from ten to sixteen days, but there are exceptional cases in which typical clinical pictures of the disease are seen for three or four days after exposure; on the other hand, cases have developed as late as from the twentieth to the twenty-fifth days following exposure.

Principal Complaint.—Stage of Invasion.—This stage is fairly distinct, and usually persists for from one to three days. The patient complains of feeling indisposed for a period of a few days before the appearance of the rash, and *mild chills*, vague *pains* in different portions of the body, lassitude, feverishness, moderate soreness of the throat, a mild constriction or band-like sensation about the chest, and the symptoms of acute bronchitis occur. After the *eruption* has developed, itching may become intolerable. In mild forms of the disease the initial symptoms may be indefinite or absent, the first indication of ill health being the appearance of the eruption.

Thermic Features.—The eruption is, as a rule, preceded by the onset of moderate fever, the temperature varying between 99° and 100° F.; in exceptionally severe forms, however, it may reach 102° to 103° F. The temperature does not subside with the appearance of the eruption, but remains at or near 100° to 102° F. until the eruptive stage has run its course.

Physical Signs.—**Inspection.**—*The Eruption.*—In typical cases there is an appreciable enlargement of the *postcervical lymph-nodes*, and the characteristic *eruption* consists of papules and is multiform, confluent, and pale rose-red in color. These patches seldom assume any special form, the skin between them being hyperemic. The rash is completely developed on different parts of the body in successive crops, and while it may be fading in

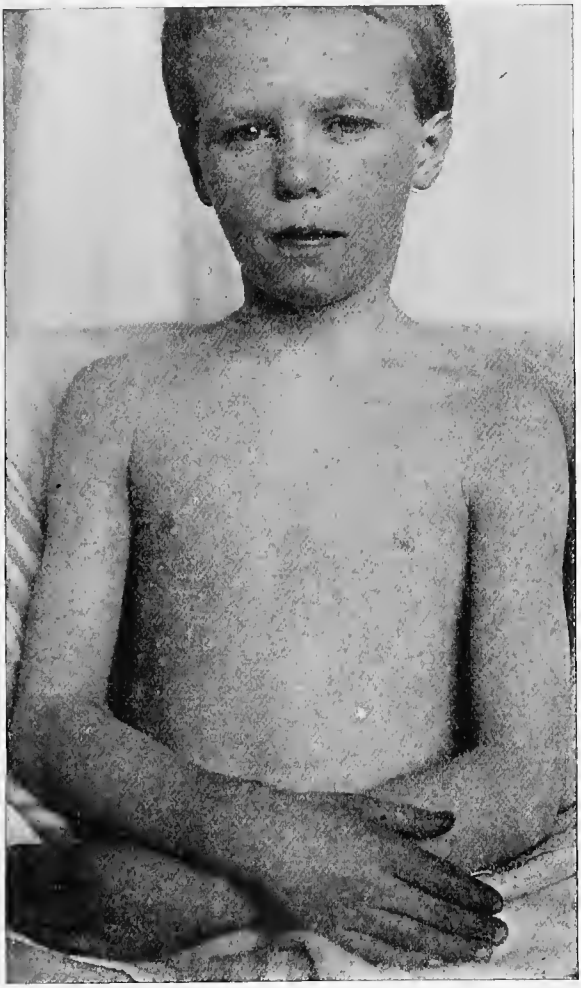


Fig. 324.—Rubella—Characteristic Eruption upon Trunk (Welch and Schamberg).

one portion of the body, it may be appearing in another part. The *duration* of the eruption varies in different cases, two to five or more days being the usual period. *Desquamation* occurs in the majority of cases, and there may be slight evidence of pigmentation after the eruption has disappeared, which usually occurs in from three to seven days. The throat is congested, the tonsils are swollen, and their covering mucous membrane is reddened.

Palpation confirms the findings of inspection with reference to the enlargement of the cervical lymph-nodes and the moderate roughening of the skin. The *pulse* is increased in frequency, of moderate tension, and the respiratory movements are also slightly more frequent than normal.

Summary of Diagnosis.—The diagnosis is based largely upon the absence of severe constitutional symptoms, the character of the eruption, glandular enlargement, and the establishment of convalescence by the end of the first week.

Differential Diagnosis.—Rubella is to be distinguished from **measles** by the extreme mildness of its course and the absence of marked catarrhal symptoms referable to the respiratory tract and conjunctivæ. The eruption of rubella is also a distinctive feature, appearing, as it does, in successive crops. The presence of epidemics should be taken into consideration in differentiating rubella from measles. In rubella the fever does not show a decided fall—a condition that is characteristic of measles. (See Fig. 320.)

Scarlet fever is differentiated from rubella by the fact that in the latter the symptoms are exceedingly mild, whereas in the former disease graver symptoms are manifested at some period during its course. The eruption of scarlet fever is erythematous, whereas that of rubella appears in successive crops. The absence of albuminuria and the character of the desquamation will also serve to distinguish this disease from scarlet fever. In general, the short course, the mildness of the attack, and the absence of complications serve to differentiate rubella from scarlet fever, measles, and other more virulent infections. The accompanying table, modified from Anders, shows the points of differentiation between *rubella, erythema,* and *urticaria:*

Rubella.	Erythema.	Urticaria.
1. The rash occurs first on the face.	1. The eruption is first seen on the hands and feet.	1. The eruption occurs in the form of wheals on the arms and leg.
2. Enlargement of cervical lymph-nodes.	2. No enlargement of cervical lymph-nodes.	2. No enlargement present.
3. At first there is no itching.	3. Burning pain present.	3. Intense itching is a prominent feature.
4. The disease is contagious.	4. The disease is not contagious.	4. The condition is not contagious.
5. The affection has a probable microbic origin.	5. This condition is of reflex origin.	5. Origin is gastro-intestinal.

Clinical Course.—The average case proceeds to convalescence by the end of the first week.

Complications.—These are less common than in measles and scarlet fever, although the following complications may be encountered: severe **bronchitis, bronchopneumonia, gastro-intestinal catarrh,** and other **acute infectious fevers. Relapses** are quite common, and each attack may be as severe as the initial seizure.

MUMPS

(Epidemic Parotitis).

Pathologic Definition.—An acute infectious disease, characterized anatomically by enlargement of one or of both parotid glands.

Predisposing Factors.—Mumps is spread by contagion, and the disease is transmitted by close contact with a patient so afflicted. There are doubtful cases in which epidemic parotitis may have been conveyed

from the sick by one healthy person to another. Mumps is a highly contagious affection, although by no means all persons exposed to it contract the disease.

Age is not without influence, the majority of cases occurring between the fourth and the tenth year; cases under one year are very rare. A child may transmit mumps to several other children before displaying any positive symptoms himself, and he may continue to carry the infection to others for from three to ten days after all swelling of the parotid glands has subsided.

Period of Incubation.—This lasts, as a rule, between ten and fourteen days, the majority of cases developing upon the seventeenth day.

Principal Complaint.—The stage of invasion is mild, and such prodromata as lassitude, headache, vague pains in the muscles, and slight stiffness of the jaw muscles are experienced. By the second day there may be considerable pain upon swallowing, more particularly when opening the mouth or upon taking acids (vinegar).

Thermic Features.—The temperature ranges between 99° and 101° F., but in severe cases the fever may reach 103° or even 104° F. Ringing in the ears and earache may develop on the second or third day, and the child may manifest a variable degree of deafness. Nausea and epigastric distress may be annoying.

Dryness of the Mouth and Salivation.—One of the chief annoyances of epidemic parotitis is the dryness of the mouth, which is frequently persistent from the onset of the disease to the fourth or sixth days, at which time the secretion of saliva may be normal or even increased. A few authentic cases have been reported in which salivation accompanied epidemic parotitis throughout the entire course of the disease.

Clinical Eccentricities.—It may be well to emphazise here that although mumps is a mild type of infection, in adults it may be accompanied by severe constitutional symptoms. Deglutition is difficult, and occasionally impossible, for an indefinite period. Pressure by the enlarged glands upon the veins of the neck may cause venous congestion of the brain, which is followed by cerebral symptoms, the most marked of which is delirium. Gastro-intestinal symptoms suggestive of the typhoid state may be present, but even in these severe cases the symptoms subside by the seventh day.

Physical Signs.—Inspection.—There is a pyriform swelling in front of the ear, extending down into the neck and forward onto the jaw. Both parotid glands are commonly affected, but in more than 50 per cent. of cases in which unilateral involvement is seen the left parotid is first attacked, and from one to two weeks later the opposite side is similarly affected. As a rule, the second attack of parotitis is milder than the first. Between the third and the seventh day of the disease extensive swelling of the other glands of the neck may take place. Tenderness in the epigastrium is occasionally present and is claimed to be due to associated pancreatitis.

Summary of Diagnosis.—The prominent features on which to base a diagnosis of epidemic parotitis are swelling of the parotid glands extending to the front of the ear, stiffness of the jaws, with slight pain, diminished secretion or absence of saliva, acute pain on introducing acids into the mouth, and the presence of mild constitutional symptoms.

Clinical Course and Duration.—Uncomplicated cases of epidemic parotitis tend to go on to spontaneous recovery, which is completed by the end of the second week. In complicated cases recovery is delayed for from one to three weeks, depending upon the nature of the complications.

Complications.—Orchitis is the most frequent complication of

epidemic parotitis, and this condition seldom, if ever, develops in patients under twelve years of age. After puberty, and in young men, orchitis is particularly common; it may be either unilateral or bilateral, and is accompanied by more or less swelling, extreme pain, and marked constitutional symptoms. Only the substance of the testicles is involved, whereas the epididymis is seldom attacked. Orchitis is usually followed by enlargement of the involved gland for a period of weeks, months, or even years, and there may be atrophy of the glandular structure.

Otitis media, although by no means a frequent complication, may occur and result in impairment of the auditory function. Piece collected 40 instances from the literature, in which deafness followed epidemic parotitis.

Mastitis, ovaritis, and vulvitis may complicate epidemic parotitis in adults, but are practically unknown in children. Mastitis is characterized by swelling, pain, and tenderness of one or both breasts. The first symptoms are observed during the second or third week of the disease, and ordinarily last for from two to four days. Ovaritis also develops late, and is characterized by intense abdominal pain, which may be cramp-like in character. Vulvitis and vulvovaginitis are somewhat more common than the two preceding conditions, and are characterized by intense inflammation and swelling of the vulvovaginal mucous membrane.

Nephritis is extremely uncommon, although a few authentic cases have been recorded. In these cases it is probable that the patient suffered from nephritis prior to the attack of mumps.

Nervous complications are unusual in epidemic parotitis, and, together with arthritis, merely deserve mention in this connection.

WHOOPING-COUGH (PERTUSSIS).

Pathologic Definition.—An acute infectious disease, characterized by the sudden onset of a catarrhal inflammation of the respiratory mucous membrane, and later by a similar inflammatory process involving the conjunctivæ, lacrimal duct, and, less often, the Eustachian tube and middle ear. The inflammatory process may extend to the smaller bronchi and air-cells, and gives rise to isolated areas of pulmonary consolidation or pulmonary collapse. In severe types of the disease pulmonary congestion and edema are present, and bronchopneumonia may develop as a complication. The pathologic changes in the bronchial mucosa are determined largely by physical examination, since uncomplicated cases seldom, if ever, come to autopsy. The mucous surface of both the respiratory and the digestive tract may be covered with a viscid mucous exudate. Experiments of Mallory, Hornor, and Henderson point strongly to the Bordet-Gengou bacillus as the exciting factor in pertussis.

Incubation Period.—This is usually from seven to ten days.

Exciting and Predisposing Factors.—Bacteriology and Parasitology.—Alinnæus attributes the disease to the presence of the larvæ of certain insects in the nasal cavity, and Kuoloff regards the specific microörganism as a protozoön, having found it to be present in the sputum of those suffering from the disease. Afanassiff has described a large bacillus (bacillus tussis convulsivæ) which he obtained in pure cultures from cases of whooping-cough. Camplewski and Hensel described a bacillus with rounded ends, often occurring in pairs, which they cultivated from the mucous secretions; a peculiarity of this organism is that it is found free in the sputum and also within the pus-cells. Jochmann and Krause, in 1891, described an organism which they isolated in whooping-cough, and which they named the bacillus

pertussis eppendorfii. None of these organisms is generally accepted as the cause of the disease. The majority of cases are seen in conjunction with *epidemic outbreaks*, although a small number of sporadic cases have been studied. Singularly, in rural districts the disease occurs in epidemic form every two years, whereas in large cities it is present at all times.

Mode of Infection.—**Contact** with a patient suffering from the disease is the usual mode of infection, and there can be little doubt but that the disease is propagated in schools, although it is probably less contagious than either measles or scarlet fever.

Season.—This appears to exercise but little influence. The disease is, however, more likely to prevail in epidemic form during the school months.

Unhygienic surroundings and **environment** exercise but a limited influence, and healthy and delicate children alike develop the disease. A more severe clinical type is, however, encountered in infants and in those previously in ill health.

Age is an important factor, most cases being seen before the tenth year, although the disease may occur both during and after middle life. In three cases seen by us—two males and one female—the patients were over sixty years of age.

Sex is believed by certain writers to serve as a predisposing factor, females being said to be affected more often than males; this observation, however, has not been borne out by general experience.

Immunity.—One attack usually bestows permanent immunity, yet second attacks, although uncommon, may occur.

Infective Period.—The disease is communicated by infected patients to others during the period of the paroxysmal cough. Cases of pertussis in the new-born have been reported.

Principal Complaint.—Pertussis is characterized by the following clinical stages:

(1) **Catarrhal Stage.**—The symptoms in this stage are similar to those of an ordinary cold, the patient complaining of coryza, lacrimation, and cough, all of which increase in intensity until about the tenth day.

(2) **Paroxysmal Stage.**—This stage is marked by the appearance of the characteristic *whoop*, the cough becoming paroxysmal. The child is conscious of an approaching seizure just previous to its occurrence, and will invariably make a strenuous effort to restrain the cough. In severe cases he will seize firm hold of some object and then *cough* violently until he is quite exhausted; following the exacerbation there is a pronounced whooping sound, which usually terminates the paroxysm; rarely two or more whoops may accompany a single seizure. After the *paroxysm* is over a variable quantity of thick, tenacious mucus is expectorated and *vomiting* is also common. Both urine and feces may be passed involuntarily. Paroxysms of coughing are excited by undue exercise, talking, laughing, sneezing, etc. When the seizure is violent, it may be accompanied by epistaxis, and at times blood may gush from the mouth. Lacrimation is a prominent symptom. The face is flushed at first, and later may become cyanosed.

The number of paroxysms occurring during the twenty-four hours will be found to vary greatly in different cases. In mild forms there may be but from four to ten paroxysms a day, whereas in the more severe types the number may exceed 50 during the twenty-four hours.

Physical Signs.—**Inspection.**—During the catarrhal stage the conjunctivæ are congested, and there is edema beneath the eyes. The nasal mucosa is also congested, and the general expression of the child is dull. During the paroxysm of coughing the face becomes cyanosed, the veins of the

neck stand out prominently, the conjunctivæ are markedly congested, and there is a free mucous discharge from both the conjunctivæ and the nose. In young children, and after the disease has continued for several weeks, the chest becomes barrel-shaped, the result of pulmonary emphysema. The *face* presents a characteristic appearance, being swollen and dusky in hue; the eyelids are also edematous and pinkish in color, and there is decided reddening of the conjunctivæ; conjunctival hemorrhages may also be seen.

Palpation, percussion, and auscultation reveal the physical signs characteristic of acute bronchitis with emphysema.

Clinical Course.—The *duration* of the paroxysmal stage varies between three and four weeks. In cases of average severity the patient coughs for approximately six weeks, although not uncommonly the cough may persist for three or four months. Having had one attack of whooping-cough the child is likely for some months to suffer a mild relapse whenever he contracts an acute "cold."

Complications and Sequelæ.—In those cases in which the paroxysms are violent, **epistaxis** may occur; rarely, indeed, is the hemorrhage sufficiently severe to cause alarm. **Hemoptysis** and vomiting of blood are unusual, and intestinal hemorrhage is extremely rare. Extensive extravasations of blood beneath the conjunctivæ may take place.

Among the most serious of the complications are bronchopneumonia and pulmonary collapse. These conditions are, however, extremely uncommon, except in children during the first and second years of life. Lobar pneumonia is occasionally seen, and is due to intense straining and rupture of the lung, when either interstitial emphysema or pneumothorax may follow. Pleurisy is also a serious complication, and may be accompanied by pronounced enlargement of the bronchial lymph-nodes. Inguinal and femoral hernia may result from sudden strain.

Among the **nervous complications** should be mentioned convulsions, hemiplegia, monoplegia, and subdural hemorrhage. Acute nephritis is occasionally seen.

Cardiac complications are unusual, and consist in dilatation of the right ventricle and endocarditis. Chronic catarrh of the bronchial mucosa frequently follows whooping-cough, and when the disease develops at or near the age of puberty, pulmonary tuberculosis may follow. In selected cases **gastro-intestinal irritation** may appear during the later stages of the disease, and continue for an indefinite period after the characteristic whoop has disappeared.

DENGUE (Break-bone Fever).

Pathologic Definition.—An acute, infectious disease, probably transmitted by the bites of infected mosquitos. It is characterized by the occurrence of definite alterations in the blood—*e. g.*, leukopenia, with a decrease in the polymorphonuclear leukocytes and a marked increase in the small lymphocytes and the presence of a maculopapular eruption.

Varieties.—Debrun recognizes the following clinical types: (1) Dengue with high fever and well-marked associated symptoms, including an eruption; (2) afebrile dengue, in which all the symptoms are exceedingly mild, but here, too, the eruption is present; and (3) a type in which the eruption is not only the most conspicuous, but the only, diagnostic feature.

Predisposing and Exciting Factors.—**Season** appears to influence the development of attacks, the disease occurring, as a rule, in warm weather.

Age, race, sex, and **environment** appear to be without effect. A single attack ordinarily confers immunity to subsequent attacks. No evidence has been adduced to support the fact that dengue is contagious.

The report* of P. M. Ashburn and C. F. Craig, who conducted extensive researches and clinical investigations in the Philippine Islands in 1907, shows that dengue is transmitted from man to man through the bites of a mosquito, Culex fatigans.

Period of Incubation.—Clinically, this varies between two and one-half and seven days, the average period being three to four days. Ashburn and Craig have shown, as the result of inoculation of non-immunes with unfiltered blood, filtered blood, and with infected mosquitos, that the incubation period varies between two and one-half and seven days, with an average of three days and fourteen hours, as shown by the accompanying table:

Method of Inoculation.	Incubation Period.
Inoculation of unfiltered blood	3 days, 18 hours.
Inoculation of unfiltered blood	2 " 19 "
Inoculation of unfiltered blood	2 " 18 "
Inoculation of unfiltered blood	2 " 12 "
Inoculation of unfiltered blood	4 " 4 "
Inoculation of unfiltered blood	7 "
Inoculation of filtered blood	3 " 11 "
Inoculation of filtered blood	2 " 12 "
With infected mosquito	About 3 days, 16 hours.

Principal Complaint.—Invasion.—The onset is *abrupt*, beginning with a mild *chill* or *chilly sensations*. By the end of the first or the beginning of the second day the patient complains of *headache* and *muscular* and *joint pains*, and his suffering now becomes intense. He describes the pains as of bone-breaking character—hence the name, "break-bone fever." There is complete anorexia, and nausea and vomiting may occur at different times during the day. Epistaxis and hemorrhage from other mucous surfaces are occasional occurrences. (See Laboratory Diagnosis, p. 886.) Diarrhea may be present. The symptoms of catarrh of the respiratory tract, *e. g.*, coryza, slight bronchial cough, and soreness of the throat, are prominent.

In a fair proportion of all cases there is a variable degree of discomfort in the region of the precordium, and distinct precordial pain is occasionally experienced, followed by a sense of suffocation and threatening syncope. A feeling of faintess follows slight exertion during the febrile period.

Nervous Symptoms.—The severe pains previously referred to constitute the chief annoyance in this particular group of symptoms. Delirium is uncommon, and when present, is usually of a low type. Hysteric seizures and hallucinations have also been observed. Insomnia is frequently an annoying symptom during the febrile period, especially in those cases in which hyperpyrexia exists.

Thermic Features.—Following the invasion, the temperature rises quite abruptly, but continuously, to a maximum of from 103° to 106° F. by the end of the first twenty-four hours. The fever continues at or near its maximum point for from one to three days, when it drops by crisis with diuresis, diaphoresis, diarrhea, or epistaxis, to normal. With the first fall of temperature the erythema disappears, and the symptoms are much improved. This improvement may last twenty-four hours, when the temperature rises grad-

* Philippine Jour. Sci., May, 1907, p. 93.

ually and the terminal eruption appears. The second febrile period may be overlooked, but it usually lasts one or two days and declines by crisis, which is likely to be accompanied by a critical discharge.

Physical Signs.—Inspection.—The joints are red and swollen, and an erythematous rash, the so-called initial rash, is present. The face is deeply flushed, and the conjunctivæ are congested. As a rule, this eruption is most profuse over the exposed parts—*e. g.*, face, neck, and hands. Ashburn and Craig assert that this rash is not the true eruption, but a general capillary dilatation, resembling in appearance a mild sunburn or flushing the result of a hot bath. The characteristic eruption of dengue usually appears on the fourth day, although it may be delayed in some cases until the sixth or the seventh day. It first appears on the backs or on the palms of the hands, extends up the forearms, and then invades the back, the chest, the arms, and the thighs. The lesions are round, dusky red, slightly elevated, and about the size of a small pea. They are surrounded by healthy skin at first, but they have a tendency to spread, forming irregular patches, sometimes as large as three inches in diameter, and separated by normal skin. The eruption disappears in a few days, and is followed by a branny desquamation.

Jaundice may be present throughout the greater part of the febrile period, and may even continue during the convalescence.

The *tongue* is at first covered by a light, creamy coat, which thickens rapidly and becomes darkened in the center, the edges showing an appreciable fading. Late during the course of the disease the tongue displays a heavy, yellowish central coat, whereas the edges and tips are bright red; the tongue remains moist throughout the entire illness.

Palpation elicits tenderness over the large joints, and firm pressure excites pain. With the beginning of the disease the pulse is accelerated, and follows the temperature—a characteristic difference between this disease and yellow fever. Ashburn and Craig, in their study of a somewhat large series of cases of dengue, found the pulse to be moderately accelerated and to follow the course of the fever fairly closely.

Laboratory Diagnosis.—Vomiting occurs in a small percentage of all cases, and the vomitus may give off a foul odor; in such cases the breath is offensive. The vomiting of blood has been reported. Diarrhea is also an occasional symptom, and some writers believe that the stool may contain blood and mucus.

The **urine** was found to contain a trace of serum-albumin in 41 per cent. of cases studied by Guitéras and Cartaya, but other observers believe that pathologic albuminuria does not occur in uncomplicated cases. The question of the occurrence of albuminuria is one that will be largely influenced by the findings of certain epidemics, and, therefore, statistics gathered from a single epidemic would have but limited clinical value. The urine may be bile stained.

Up to the present time no microörganisms of any kind have been detected in the blood of dengue. The number of red blood-cells in a cubic millimeter approximates that of the normal. A fairly well-marked leukopenia is present, the polymorphonuclear leukocytes displaying a decided decrease, the proportionate number of small lymphocytes showing a corresponding increase (30 to 60 per cent.). The blood-platelets are normal in number.

Differential Diagnosis.—Yellow fever has often been mistaken for dengue, and the two affections may be present simultaneously. A

differential diagnosis is made only with great difficulty, as the two diseases present many points of similarity. The following table, modified from Anders, shows the differential features:

Dengue.	Yellow Fever.
1. Affects all races.	1. Caucasians more especially affected.
2. Facies characteristic; face flushed.	2. Mucous membranes injected.
3. Irregular rise of fever, followed by remission, and then a second moderate rise. Duration, five to seven days.	3. The temperature rises regularly. Duration of fever, about seventy-two hours.
4. The pulse keeps pace with the fever.	4. The pulse falls while the fever is rising.
5. Maculopapular eruption present.	5. Eruption unusual.
6. Vomiting rare.	6. Vomiting frequent.
7. Urine seldom contains albumin in uncomplicated cases.	7. Albuminuria common; reaction for bile present.
8. Jaundice unusual.	8. Jaundice present early.
9 Hemorrhage from mucous membranes, generally slight, and black vomit rare.	9. Hemorrhages common and severe. Black vomit an alarming symptom.
10. Nervous symptoms absent or mild.	10. Nervous symptoms of a grave nature present

ERYSIPELAS (St. Anthony's Fire).

Pathologic Definition.—An acute infectious disease, engendered by the streptococcus, and characterized by the presence of congestion, inflammation, and edema of the skin and subcutaneous cellular tissue, with distention of the cutaneous lymph-channels. Suppurative inflammation may attack the subcutaneous tissue. Blisters, blebs, and bullæ appear upon the cutaneous surfaces.

Varieties.—(1) The **ordinary type,** which will be described at length.

(2) **Phlegmonous (cellulocutaneous) erysipelas,** which is characterized by the appearance of a severe inflammation of the subcutaneous connective tissue, with a tendency to go on to suppuration.

(3) **Migratory erysipelas,** a condition in which the erysipelatous process is very acute at first, but tends, as the disease advances, to assume a subacute form and to spread over all portions of the body. We have seen cases of this type in which the erysipelatous process extended from the face to the feet, covering practically the entire body surface, from twelve to twenty weeks being consumed in completing the process.

(4) **Relapsing erysipelas,** a condition in which the inflammatory process is of unusually low grade and tends to recur at longer or shorter intervals.

(5) **Erysipelas neonatorum,** or the erysipelas of infants, follows infection of the umbilical cord. The erysipelatous process spreads rapidly from the umbilicus over the lower portion of the abdomen, and frequently extends to the face, chest, and less often to the back. This is an exceedingly grave variety of erysipelas, and usually terminates fatally within the course of from two to five days.

(6) **Pneumo-erysipelas,** a form in which the specific infection may set up a bronchopneumonia as a complication.

(7) **Nephro-erysipelas.**—Nephritis may develop as a complication, and the nephritic tissue be infiltrated with cocci. The symptoms of acute nephritis are also present.

Exciting and Predisposing Factors.—Bacteriology.—It is generally agreed that the specific cause of erysipelas is the streptococcus erysipelatis of Fehleisen, which is probably identical with the pus-producing organism commonly encountered. An erysipelatous process may, however,

be produced by inoculating the skin with the streptococcus, and, indeed, inoculation with other bacteria will produce an inflammation that is indistinguishable from that of true erysipelas. In 1900 Pfahler cultivated a diplococcus from the blebs in 8 cases of erysipelas at the Philadelphia Hospital, and we produced an erysipelatous process in rabbits by injecting an emulsion containing Pfahler's diplococcus beneath the animal's skin; similar inoculations with other pus-producing organisms in laboratory animals gave rise to an erysipelatous inflammation. In our own experience, covering a bacteriologic study of approximately 40 cases of erysipelas, streptococci were found present in every instance in which cultures were made from several different blebs. Staphylococci and diplococci were also present in many of the cultures, at least two pathogenic bacteria being found in each culture. A detailed study of the bacteria recovered from erysipelas, however, showed these organisms to be practically indistinguishable from those that may be cultivated from the surface of the human skin.

Season.—Anders,* in a statistical analysis of 2010 cases of erysipelas, showed that 19.5 per cent. of all cases develop during the month of April, and one-half of all cases during the months of February, March, April, and May. Boston and Blackburn, in a report of 546 cases of erysipelas seen in the wards of the Philadelphia Hospital, found that 20.3 per cent. of all cases were admitted during the month of April, and that 423 of the whole number, or 77.8 per cent., developed during the months of January, February, March, April, and May. The accompanying table, by Boston and Blackburn, sets forth the influence of season upon the development of erysipelas, and gives an estimate of the severity of the type of infection encountered at different seasons:

INFLUENCE OF SEASON ON ERYSIPELAS.

Month.	Total Cases.	Total Deaths.	Percentage of Mortality.
January	66	12	18.0
February	77	8	14.0
March	104	11	10.6
April	111	15	13.3
May	65	9	13.8
June	11	0	0.0
July	4	0	0.0
August	6	0	0.0
September	10	1	10.0
October	16	2	8.0
November	30	6	20.0
December	46	5	11.0
Total	546	69	

Age.—In an analysis of 1894 cases, Boston and Blackburn found that 25.8 per cent. developed during the third decade, and that the disease was far less common after the age of fifty, whereas 15 per cent. of all the cases developed before the age of twenty.

Sex.—An analysis of the records of 1767 cases showed that males are attacked more often than females, in a ratio of 3 to 2; Boston and Blackburn's analysis of 539 cases gave 342 males and 197 females.

Race.—The African negro rarely suffers from erysipelas, as is shown by the previously named writers' analysis of 545 cases, in which only 5.3 per

* Proc. Amer. Climatolog. Assoc., 1893.

cent. were negroes. **Nationality** appeared to be a marked predisposing factor, 42.2 per cent. of cases developing in Americans and 20 per cent. of those afflicted being of Irish birth.

Previous Attacks.—One attack predisposes to others, and a second, third, and even a fourth attack is not unusual. We have seen a number of cases in which erysipelas developed during the winter months for two or more successive years. In an analysis of 450 cases, we obtained a history of previous attacks in 8.6 per cent.

Coryza.—Acute coryza markedly predisposes to the development of erysipelas of the nose and face, as is shown by M. B. Miller's statistical analysis of 301 cases, in which coryza occurred as an antecedent in 13 instances (4.3 per cent.).

Chronic maladies, after they have appreciably impoverished the system, favor the development of erysipelas. The occurrence of the disease is to be feared late during the course of nephritis, hepatic cirrhosis, valvular heart disease, chronic tuberculosis, diabetes, arteriosclerosis, and in those addicted to the use of alcohol.

Injuries.—An abrasion of the cutaneous surface favors the development of the erysipelatous process, and is said to be essential to the invasion of the specific bacterium. Those whose occupations subject them to frequent abrasions of the skin of the hands, nose, and face, as well as to slight injuries, are especially prone to acquire the disease, although it is often impossible to obtain a definite history substantiating this fact. In an analysis of 643 cases, but 13 gave a history of injuries to the cutaneous surface. Erysipelas is especially likely to develop after surgical treatment, particularly after operations in which it has been impossible thoroughly to cleanse the parts incised.

Puerperium.—Women are especially likely to become infected with erysipelas after delivery, particularly when either the nurse or the physician is also attending patients suffering from the disease. The epidemic outbreaks of erysipelas occurring in hospitals and institutes are possibly explained by the fact that certain of the attendants convey the disease from one patient to another, although it may be transmitted by clothing, towels, napkins, and other objects.

Period of Incubation.—In cutaneous erysipelas this usually varies between one and two weeks, but when erysipelas develops after surgical interference, a much shorter incubation period (three to seven days) may occur. Experimentally, we have found it possible to produce an erysipelatous process in from twenty-four to forty-eight hours by inoculating the skin of rabbits' ears with bacteria cultivated from blebs.

Prodromal Symptoms.—These are often indefinite, and consist in headache, restlessness, slight soreness of the throat, mild cough, fever, and anorexia. The *duration* of the prodromal symptoms will be found to vary from a few hours to several days.

Principal Complaint.—Following the prodromata, the attack sets in quite abruptly, with a distinct *chill* or a sensation of chilliness. The evidence of constitutional depression may not be well marked in those who have previously enjoyed health, but in the debilitated, and particularly in alcoholics, prostration comes on early. The patient complains of a localized sense of tension over the part affected, and later a distinct burning sensation is felt throughout the erysipelatous area; pain is, however, unusual, unless the subcutaneous cellular tissue is involved.

Nervous Symptoms.—In uncomplicated cases the nervous symptoms

are, as a rule, mild, and consist of headache, restlessness, and aching in the back and loins. When complications arise, and, indeed, in the more severe types of erysipelatous infection, delirium occurs during the night. When nephritis and bronchopneumonia develop as complications, maniacal delirium and coma may follow.

Thermic Features.—Following the chill the temperature rises somewhat abruptly, and usually reaches 102° to 104° F. during the first twenty-four hours. The fever remains high, with moderate remissions, for a period of from five to seven days, when, in uncomplicated cases, it falls rapidly to the normal. As each new area becomes involved in the erysipelatous process (*relapse*) the temperature again rises one, two, or three degrees, but the period of pyrexia is comparatively shorter in each succeeding relapse.

Physical Signs (Local).—The area most often affected is the face, as shown by Boston and Blackburn's statistical analysis of 545 cases, in which the face served as the initial site of infection in 485 instances. The affected part at first is reddened, and later becomes intensely congested, swollen, and edematous, and the features may be distorted. Immediately beyond the congested area the swelling ends abruptly—the so-called "line of demarcation." The inflammation may extend from one side of the face across the median line, a feature that was observed in 78.8 per cent. of the series of cases just mentioned. The reddened surface of the skin is often studded with small blebs or blisters, which are filled with serum. When these blisters rupture, an angry, suppurating surface may remain. In the series of cases previously referred to, the spread of the erysipelatous inflammation appeared to be limited by the following: the median line of the body, in 21.2 per cent.; the folds of the skin, and the hair (beard); it is also very unusual for the process to extend beyond the junction of skin and mucous membrane. **Cutaneous edema** may be so pronounced as to distort the features, making recognition of the individual often impossible; the eyes are often closed.

The congested portion of the skin is hot to the touch, and firmer than the adjacent surface. On drawing the finger over the healthy skin to the involved area, a distinct hardening is felt at the junction of the healthy and diseased epidermis, resulting from infiltration of the lymphatics; this is the so-called "line of demarcation." Pressure over the affected area seldom elicits pain unless the deeper structures are infiltrated with pus.

The **tongue** is heavily coated; the mouth is dry and parched; the throat is often congested, and the patient finds it difficult to open the mouth.

The **pulse** is rapid,—100 to 120 beats a minute,—and in severe complicated cases its tension becomes diminished with the progress of the disease, whereas later it is weak, dicrotic, and intermittent.

Constipation obtains in the majority of cases, although diarrhea with the passage of serous stools is occasionally observed.

Laboratory Diagnosis.—The **urine** is of high color, increased in specific gravity, and in uncomplicated cases may contain a trace of albumin. Nephritis is a somewhat common complication, occurring, as it did, in 29 per cent. of 548 cases analyzed by Boston and Blackburn; when present, the urinary findings are those of acute nephritis. The diazo-reaction is present during high fever.

A **leukocytosis** in which the white cells number from 15,000 to 30,000 per c.mm. is to be expected during the initial attack of erysipelas. The increase in the number of leukocytes affects chiefly the polymorphonuclear elements. In cases in which repeated relapses have occurred, the hemo-

globin and red cells become markedly reduced, and the general blood-picture is that of secondary anemia plus moderate leukocytosis.

Cultures from the serum obtained from the cutaneous blisters, as well as those made from the blood-serum that exudes from the skin after incision over an erysipelatous area, will be found to contain streptococci, staphylococci, and diplococci. In the majority of instances, however, streptococci alone are present.

Summary of Diagnosis.—This is based first upon the history of a chill and the characteristic temperature, which are soon followed by swelling and redness of the part, and, later, the formation of blebs upon the surface of the skin and the appearance of a distinct line of demarcation. When the deeper cellular tissue is not involved, a sense of burning and the absence of pain are valuable diagnostic points.

The *duration* of the febrile period—four to seven days—and its characteristic decline by rapid lysis are of great diagnostic importance, as is also the comparatively short febrile period of each successive relapse.

Differential Diagnosis.—**Erythema** produces superficial redness, differing from erysipelas in that it is not attended with heat, swelling, or fever.

The eruption of **urticaria** assumes the form of pale-red, circular wheals, which cause marked itching and appear in successive crops, often disappearing in the course of a few hours. In this condition the initial chill, blebs, and rise in temperature are absent.

Acute eczema of the face, when severe, may somewhat resemble erysipelas, but lacks the peculiar line of demarcation and mode of progression characteristic of erysipelas. Again, eczema is accompanied by troublesome itching, the swelling is less pronounced than in erysipelas, and fever is also absent in uncomplicated cases.

Eczema nodosum is characterized by the presence of nodosities situated near the articular surfaces, and is devoid of the constitutional symptoms seen in erysipelas.

Clinical Course and Duration.—The average duration of a case of erysipelas, including the prodromal stage in early adult life, is fourteen days (Anders). In each relapse the condition is prolonged for from five to seven days, consequently in those cases in which frequent relapses have occurred from four to six and even eight weeks may elapse before convalescence is established. The clinical course of erysipelas is appreciably longer in the aged and the debilitated than it is during the second and third decades. Complications materially retard convalescence.

Complications.—These are numerous, and often unusually serious—*e. g.*, abscess formation, lobar pneumonia, phlebitis, catarrhal pneumonia, nephritis, otitis media, acute bronchitis, laryngitis, and pleurisy may develop.

Sequelæ.—In those cases in which the erysipelatous inflammation involves the scalp the hair falls out, but a rich growth usually returns. When erysipelas attacks persons who have previously suffered from some chronic disease of the skin, a cure of the latter may be effected. As an example, may be mentioned the case of a male, aged sixty-six years, who had been under treatment for twelve years for lupus involving both sides of the face. He suffered a severe attack of erysipelas, which extended only over the right half of the face, and after recovery it was found that the half of the face attacked by the erysipelatous process was free from lupus, whereas the other side remained unchanged. Chronic eczema may also be similarly

affected by erysipelas, and some writers maintain that this disease is antagonistic to the development of malignant disease.

ACUTE ARTICULAR RHEUMATISM.

Pathologic Definition.—An acute infectious disease, characterized by the presence of an acute inflammation of the synovial membranes of various joints, with the accumulation of fluid. The surface of the synovial membrane of the affected joints is injected and swollen, and may be more or less completely covered with a fibrinous exudate. Later the effusion contains fibrin and leukocytes. The tendinous sheaths in relation with the diseased articulation may be attacked, and in severe cases erosion of the cartilages may occur. A similar inflammatory process generally affects the other serous surfaces, particularly the endocardium and pericardium, and less often the meninges, pleura, and peritoneum are involved.

Exciting and Predisposing Factors.—**Bacteriology.**—A bacteriologic study of the exudate obtained from the synovial sacs may reveal the presence of bacteria, staphylococci, streptococci, and diplococci. Singer, in a study of 92 cases, asserted that, in the majority of them, either streptococci or staphylococci were present. Pierre Achalme has described a bacillus that he recovered from the blood of those suffering from acute articular rheumatism. In certain cases anaërobic diplococci are present in the synovial fluid, and many observers believe that this is the probable cause of the disease, although streptococci are more commonly encountered in this situation.

Those suffering from other acute infectious conditions appear to be more susceptible to the disease than healthy individuals; indeed, acute articular rheumatism often follows an attack of acute tonsillitis.

Season.—The greatest number of cases are seen during the months of February, March, and April, although the disease is also quite common during October, November, December, and January. At times the disease is so prevalent as to suggest the possible existence of an epidemic. It occasionally prevails during the summer months.

Exposure to **cold** and **wet** is a prominent predisposing factor, males being consequently attacked more often than females.

Age.—The majority of cases occur during early adult life, or between the years of fifteen and thirty-five, although the disease may be encountered in the young and in those over fifty years of age.

Heredity is believed by many writers to play an important rôle.

Epidemic Influence.—Distinct epidemics may arise in certain localities at any time during the year, although they occur most commonly during cold weather.

One attack does not establish *immunity*, but appears to predispose to subsequent attacks.

Incubation Period.—The occurrence of an incubation period is doubtful, although certain prodromal symptoms are occasionally experienced, and usually consist in the following: malaise, a slight degree of fever, soreness of the throat, laryngitis, and the like. These may continue for a day or two, and then be followed rapidly by definite local symptoms.

Principal Complaint.—**Onset.**—This is quite abrupt, often beginning with a *chill*, followed by other constitutional symptoms. (See Thermic Features.)

Nervous Symptoms.—*Pain* is a prominent feature, and is localized to one

or more joints. The medium-sized and larger joints (ankle, knee, wrist) are most often attacked at the time of onset. The initial site of synovitis may also be at the shoulder, hip, or elbow, and still less frequently the joints of the fingers and toes are attacked primarily. In severe types of the disease the articular surfaces of the vertebræ may manifest tenderness. The pain increases on moving the joint, and the patient usually places the affected part in a certain position, which he claims appreciably lessens the pain. A characteristic feature of articular rheumastim is that from time to time during the course of each attack different joints become involved, and in each the pain is equally fugacious and out of proportion to the anatomic distortion of the part. In some cases in which cardiac complications exist precordial pain is present.

In those cases in which hyperpyrexia is present, other nervous symptoms are prominent; but certain nervous manifestations may also be observed even when high temperatures are absent. The patient is usually restless and unable to sleep. Delirium is rarely present, except in complicated cases and when the temperature exceeds 104° F. In the adynamic type of the disease, which is rare, delirium, stupor, and coma may develop. The presence of endocarditis is likely to intensify the nervous symptoms, and outbreaks of maniacal excitement are prone to occur as the result of cerebral embolism. Involvement of the pericardium is also accompanied by more marked nervous symptoms, which may lead to stupor. When alcoholics are attacked by acute articular rheumatism maniacal delirium is apt to develop. Convulsive seizures, although rare, may precede the onset of coma. The writers have recently encountered the case of a young female in whom melancholia developed during convalescence from acute articular rheumatism. Meningitis and chorea are nervous conditions rarely seen to accompany or complicate this disease.

Symptoms referable to the pulmonary system are by no means uncommon, pleuritic pain being a frequent symptom. The general clinical features of acute bronchitis frequently coexist with articular rheumatism. Pulmonary complications occurring during the course of acute articular rheumatism are always of severe type.

Thermic Features.—Following the chill the temperature rises abruptly to from 100° to 104° F.; the fever is remittent in character. A hectic type of fever is never seen except in those cases in which suppuration exists. Hyperpyrexia may develop somewhat suddenly at any time during the course of the disease, but occurs most often during the second week (seventh to tenth days); it is generally accompanied by other severe constitutional symptoms, such as delirium and stupor. In severe and complicated forms the temperature may even reach 106° to 108° F.

Physical Signs.—Inspection.—The involved joints are swollen and reddened at first, and the patient holds the affected limb in one position, usually a partially flexed one. When he is asked to move the part, he does so guardedly, an expression of pain accompanying each movement. After the disease has persisted for some days or weeks, extreme enlargement of the affected joint may take place, especially if one of the larger articular surfaces, such as the knee, elbow, wrist, or ankle, is affected. During convalescence, and after an attack of acute articular rheumatism, small subcutaneous nodosities may appear along the course of the tendinous insertions and within the fasciæ. The skin overlying these nodules is merely elevated, but does not display the usual evidences of inflammation.

The face is flushed, but the expression remains unaltered except when an

effort is made to move the affected part. The entire body is bathed in perspiration several times during the twenty-four hours, the excretion having an acid reaction, although during the later stages of the disease it may become alkaline. Sudamina appear in successive crops during the febrile period. Occasionally a distinct erythema may develop, and erythema nodosum may also occur.

Urticaria occasionally develops during the febrile period, and in severe types of the disease there may be purpura hæmorrhagica (the so-called hemorrhagic polyarthritis, also considered under the heading Peliosis Rheumatica). Cutaneous hemorrhages may become extensive, and coalesce to form ecchymotic areas in both the skin and mucous membranes.

Palpation.—The affected joints are tender to the touch, and firm pressure elicits pain. The muscles of the region are spastic, and prevent movement of the joint. Within the course of a few days an exudate into the synovial sac takes place, and fluctuation may then be easily elicited. During convalescence the joints become less sensitive, and within the course of a few weeks there may be impairment of movement, the result of an apparent ankylosis. In the region of the affected joint nodular enlargements may be

Fig. 325.—Fingers in Acute Articular Rheumatism.

detected along the sheaths of the tendons, and usually remain for an indefinite period after the patient has apparently recovered. In exceptional cases infiltration into the sheaths of certain tendons occurs; a well-marked case representing this type of the condition recently came under our personal observation.

The skin is hot to the touch, bathed in acid perspiration, and nodules of urticaria may be felt. In selected cases the spleen is enlarged.

The *pulse* is increased in frequency, even in mild types of the disease, and is soft and full, numbering 100 beats a minute. In certain cases it becomes unusually rapid (120 to 140 beats a minute), and when hyperpyrexia and other grave constitutional manifestations are displayed, the pulse becomes feeble, decidedly irregular, and often compressible. The apex-beat becomes forcible and quickened, this feature becoming especially prominent when endocarditis develops as a complication. According to Anders, endocarditis is seen in from 25 to 30 per cent. of all cases. If pericardial involvement has taken place, a friction fremitus synchronous with the heart's action may be detected.

Percussion is of but limited value, except in those instances in which effusion accumulates in the pericardial or pleural sacs.

Auscultation.—During convalescence, and in those cases in which deformities or loss of mobility of the joint have become permanent, a peculiar friction crepitus may be present for a long period upon movement of the joint. This sign is elicited by placing the stethoscope over the articular surface, and then forcibly manipulating the part.

The characteristic signs of *endocarditis* are commonly elicited, and even before true cardiac murmurs can be detected there is an appreciable irritability of the heart's action and a slight prolongation or loss of distinctness of the first sound. The physical signs of bronchitis may be distinct over the entire surface of both lungs, although in mild cases these signs are absent. (See Acute Bronchitis, p. 88.)

Laboratory Diagnosis.—**Secondary anemia** develops early and is progressive in character, and a moderate **leukocytosis** generally occurs.

The **saliva** may display an acid reaction during the acute symptoms, and in selected cases the sulphocyanids are in excess.

The **fluid obtained from the synovial sacs** is usually clear, and may be free from bacteria, although pathogenic bacteria may be cultivated from such exudates. (See Bacteriology, p. 892.)

The **urine** is diminished in quantity during the febrile period, and is high in color and unusually acid. On standing, it often deposits a heavy sediment, made up for the most part of amorphous urates. A trace of albumin may be present, and the reaction for chlorids is, as a rule, present.

Illustrative Case of Acute Articular Rheumatism.—Charles M., aged fourteen years.

Family History.—Parents and one brother living and in good health; no history of rheumatism, tuberculosis, or carcinoma in the family.

Previous History.—The patient had the diseases of childhood, but does not remember whether any complications occurred. He has been subjected to repeated and somewhat periodic attacks of tonsillitis, which usually develop during the fall, and upon one occasion his tonsils were lanced. Four years ago he suffered from a mild attack of what his physician regarded as rheumatism; at that time the right knee was swollen and painful, and he was compelled to remain indoors for at least one week.

Social History.—Family environment good. The patient has always been properly nourished. He is a messenger boy by occupation, and is therefore exposed to cold and wet.

Present Illness.—On March 14th he complained of slightly chilly sensations, malaise, and anorexia, and on the following morning, on attempting to put his weight upon the right foot, he found that the right ankle was extremely painful. The ankle continued to swell, and when seen on the second day of his illness the swelling was marked and the slightest movement caused extreme pain. Anorexia, obstinate constipation, and mild headache were present. Upon the third day after the appearance of joint symptoms a similar inflammatory process attacked the left ankle, the right ankle showing marked improvement simultaneously. The wrist and left knee then became similarly affected. Profuse sweating occurred during the night, making it necessary to change the garments and bed linen in the morning.

Pain was increased upon moving either of the affected joints, this painful condition continuing for a period of about three weeks, when the sensitiveness gradually lessened. During the third week of the illness he complained of a sense of weight in the region of the heart, and although this was never referred to as distinctly painful, yet he was conscious of some distress in the precordium for a period of about one week.

When seen upon the second day of the illness, the temperature was 101° F.; it continued to fluctuate between 99° and 101.4° F. until involvement of the left knee occurred, when the temperature rose to 102° F.; it soon fell to its usual level, however, and continued of irregular type for a period of about two weeks, when it fell to the normal.

Physical Examination.—*General.*—The skin was bathed with perspiration, the lower extremities could not be moved without eliciting an expression of pain. The body

appeared well nourished, the skin and mucous membranes and the tone of the muscles normal. The face was flushed, and the expression one of pain; the tongue was heavily coated. The right ankle was swollen during the first week, when the left ankle became involved, and later also the wrists and left knee became markedly swollen. The skin over the affected joints was red, and pressure caused paling of the skin.

Local Examination.—Palpation.—The involved joints were extremely tender, and even moderate palpation, as well as movement of the joint, produced great pain. Late during the third week of the disease, and at the time when precordial distress was present, there was a distinct irritability of the cardiac impulse, and the pulse was full and strong, numbering 100 to 120 beats a minute. From the third to the fifth week of the disease there was distinct fluctuation at the left knee, the fluid present being gradually absorbed and fluctuation disappearing by the seventh week.

Percussion.—The area of cardiac dullness was found to extend slightly to the left and downward, showing dilatation of the left ventricle.

Auscultation.—From the third to the fifth week of the illness the heart-sounds were rapid, the first sound evinced alteration (muffling), and a distinct murmur, soft and blowing in quality, was audible. The murmur became clearer and clearer from the fifth to the tenth week, and during this time dyspnea, cyanosis, coldness of the extremities, and evidence of cardiac weakening were present.

Laboratory Findings.—The quantity of urine voided during the day varied between 25 and 35 ounces; it was high colored, with a specific gravity of 1.025, and at times it showed a trace of albumin

Diagnosis by Induction from Clinical Data.—The age of the patient, the history of repeated attacks of tonsillitis and of an attack of acute articular rheumatism four years ago, go far to suggest at least the nature of the condition. This evidence, together with the onset and the characteristic appearance of the ankle on the second day of the illness, was quite sufficient to warrant the belief that acute articular rheumatism was present. A similar involvement of a joint of another limb rendered the diagnosis positive. Pain was an important symptom, and the fact that pressure upon the involved joints and movement of the affected limbs increased the pain were not without significance. Precordial distress, even though it never became severe, was highly suggestive of the existence of cardiac involvement. The temperature was that of acute articular rheumatism, rather than of other acute infections known to attack the serous surface of the joints.

Differential Diagnosis.—The fact that the larger joints were attacked made a gonorrheal origin possible, but the condition was differentiated from gonorrhea by the absence of a history of urethritis.

In certain respects the condition simulates tuberculosis of the joint, but the acuteness of the onset, the involvement of other joints, and the clinical course all distinguish it from a tuberculous process.

Course of the Disease.—This appeared to be modified as the result of treatment, the pain being greatly lessened as the result of the judicious administration of salicylates. The quantity of urine voided during the twenty-four hours soon increased from 35 to 50 ounces, this being probably attributable to the fact that the patient drank a large quantity of water. He was able to leave his bed during the sixth week of the disease, but owing to the fact that both lower extremities were involved in the process, he was unable to walk about the room until the tenth week, after which time he improved rapidly. When seen one year later, a systolic murmur was heard at the apex of the heart, and a slight degree of hypertrophy was present.

Summary of Diagnosis.—The history of previous attacks and of recurring attacks of tonsillitis is of considerable importance in formulating a diagnosis. The characteristic features of the disease are few—chill followed by a rapid rise in temperature, the fever running an irregularly remittent course, pain, swelling, redness, and tenderness of the affected joints, and the influence of motion upon the degree of pain. The occurrence of intercurrent acute endocarditis is also strongly confirmatory.

Differential Diagnosis.—Tuberculous arthritis in children may be confounded with acute rheumatic arthritis. The former is less indurating, the swelling is less symmetric, and it runs a far less acute course than the latter condition. In acute articular rheumatism the pain is definitely localized to the joint and the general clinical course is acute, whereas tuberculous arthritis runs a more chronic course.

Gonorrheal arthritis may closely resemble acute articular rheumatism; here, however, a history of an attack of gonorrhea, the character of the pain, the less marked constitutional disturbances, and the tendency toward chronicity strongly favor a diagnosis of gonorrheal infection and discourage the possibility of the existence of acute articular rheumatism. In gonorrheal arthritis aspiration of the affected joint will result in the recovery of fluid in which gonococci will be found.

Scurvy.—During the course of scurvy and the allied conditions, purpura and hemophilia, an effusion into the synovial sacs takes place, but in these conditions the effusion is likely to consist of blood, which will differentiate if from the serous effusion that collects in acute articular rheumatism. The tendency toward hemorrhage from the mucous surfaces is a marked feature of scurvy, but uncommon in acute rheumatism. In the so-called "*peliosis rheumatica*," petechial hemorrhages may occur in both the skin and the mucous membranes, but even in this disease hemorrhage into the serous sacs is uncommon. A history of the patient's life during the past year, the character of food he has eaten, as well as a general knowledge of his environment during that time will often be of great value in differentiating acute articular rheumatism from scurvy.

Pyemia.—Here the general condition is more grave; fever of the irregular intermittent type precedes the local manifestations. Rigors also occur at varying intervals, accompanied by a marked elevation of temperature—symptoms that are absent in rheumatism. In pyemia suppurative processes occur in the various viscera and skin, and slight jaundice is present. Rheumatic symptoms fluctuate greatly, whereas those of pyemia do not.

The multiple swelling of the joints which develops after child-birth is to be regarded as septic in nature. In these cases arthritis leads rapidly to suppuration, with more or less destruction of the joints.

Clinical Course.—In an average case of acute articular rheumatism in which a single joint is involved the febrile period lasts from seven to fourteen days, and is followed by convalescence, the patient being able to leave his room by the end of the third or during the fourth week. When several of the articulations are attacked in succession, the illness may be prolonged to from six weeks to several months. Cardiac complications practically always retard convalescence, and may leave a permanently damaged heart in their wake. Judicious treatment materially shortens the course of the disease, whereas retention of the serous fluid in the synovial sacs may in turn retard convalescence for weeks, and even necessitate aspiration for the removal of such fluid.

Complications.—Acute endocarditis is the most frequent complication. Pericarditis and pleurisy are occasionally encountered. Bronchitis, bronchopneumonia, and lobar pneumonia are rare complications.

Gonorrheal Arthritis.

Pathologic Definition.—An acute septic inflammation involving both the synovial membranes and the periarticular tissue of the larger joints. The inflammation may extend along the tendon-sheaths. Effusion into the synovial sac usually follows, and may, in some cases, be purulent in character. The joint may become edematous and swollen in proportion to the virulence of the inflammatory process, and ankylosis may follow.

Exciting and Predisposing Factors.—**Bacteriology.**—The exciting factor is the gonococcus, although other bacteria may also be present

in the purulent exudate recovered from the synovial sacs. If the blood-current becomes contaminated by the gonococcus, endocarditis results, and positive blood cultures are then obtained. An attack of gonorrhea is an essential factor in the development of the disease, although the urethral mucosa may not be involved; Lucas collected 23 cases in which gonorrheal arthritis followed gonorrheal ophthalmia.

Sex.—The disease appears in both sexes, and, according to Gather, it occurs in about 22 per cent. of all cases of gonorrheal urethritis. Invasion of the synovial sacs by the gonococcus is less common in females, yet such cases have come under our observation.

Principal Complaint.—A history of gonorrheal infection is usually obtained, but when such history is not elicited, careful clinical research is necessary. Two distinct subclasses of gonorrheal arthritis have been described:

(*a*) A type in which the inflammatory changes are mild and in which the patient suffers a moderate amount of pain in one or probably in two or three joints, but in which the affected joints show but little, if any, evidence of inflammation.

(*b*) **Typical Form.**—In this variety the *pain* is more pronounced, and a single joint becomes markedly incapacitated in the course of a few days. Soreness radiates from the joints and runs along the tendons, and despite treatment the patient's condition goes from bad to worse. *Polyarthritis* is present in a small proportion of all cases, the more pronounced symptoms being confined to one or two of the large joints, *e. g.*, the knee, wrist, elbow.

Transitory arthritis may be seen during childhood.

Physical Signs.—Inspection.—The affected joint is swollen, reddened, and partly flexed. If arthritis persists for some weeks, extreme pallor of the skin and mucous membranes and emaciation take place.

Palpation.—The affected joint or joints are found to be greatly enlarged and tender upon even mild pressure. Following the accumulation of an exudate in the synovial sacs fluctuation is present, and aspiration results in the recovery of fluid. In long-standing cases a fibrinous ankylosis may develop and complete motion of the joint becomes impossible. In those cases in which the endocardium becomes involved the pulse is increased in frequency.

Laboratory Diagnosis.—If a urethral discharge is present, a specimen must be examined for the presence of the gonococcus. Cultures on blood-serum or glucose-agar should be made from the fluid obtained by aspirating the synovial sacs. The gonococcus will be obtained in a large proportion of cases. Other bacteria may also be present in the synovial fluid, *e. g.*, staphylococci, pneumococci, and streptococci.

If the type of gonorrheal infection has been unusually severe and has continued for several weeks or even months, the *blood-picture* is that of chloranemia. Following the accumulation of pus in the synovial sacs a well-marked *leukocytosis* may be present, although this is not a constant finding. If the condition is complicated by endocarditis, the gonococcus may be cultivated from the circulating blood.

Summary and Differential Diagnosis.—(*a*) History of gonorrheal infection, urethritis, leukorrhea, or ophthalmia; (*b*) involvement of the articular surface some weeks or months after the initial symptoms of gonorrhea; (*c*) detection of the gonococcus in the synovial fluid.

The foregoing diagnostic features serve to distinguish gonorrheal arthritis from other forms of joint inflammations. The following table sets forth the differential features between gonorrheal and tuberculous arthritis:

Gonorrheal Arthritis.	Tuberculous Arthritis.
1. Histroy of gonorrheal infection from a few months to a year prior to the development of articular symptoms.	1. May be a history of tuberculosis prior to the development of articular symptoms.
2. Fluid in synovial sacs, purulent in most cases.	2. Fluid in synovial sacs serous in character.
3. Synovial fluid contains the gonococcus, and possibly other bacteria.	3. Synovial fluid may contain tubercle bacilli.
4. Tuberculin reaction negative.	4. Tuberculin test positive.

Clinical Course.—Despite the apparent virulence of the articular inflammation, the process tends to become subacute or chronic in nature. Surgical treatment is usually necessary, and materially shortens the course of the disease.

VARIOLA (Smallpox).

Pathologic Definition.—An acute infectious disease characterized by the appearance of an eruption that presents four distinct stages: (1) The macule; (2) the papule; (3) the vesicle; and (4) the pustule. During the healing process the lesion is covered with a scab, which, when removed, leaves a scar.

The mucous membrane of the mouth, pharynx, and esophagus may also display a characteristic eruption. In the severe type of the infection, known as hemorrhagic smallpox, extensive cutaneous hemorrhages and hemorrhages into the muscular tissue may occur; hemorrhagic infarction of the lung and of other viscera is also occasionally seen.

Varieties.—(1) **Discrete smallpox,** which may be mild or moderately severe. (See Fig. 326.)

(2) The **confluent form,** which appears to follow a severe type of infection, and in which the disease is ushered in by grave constitutional symptoms. The eruption appears early and is profuse, and the pustules may coalesce, with extensive destruction of tissue and resulting deformity. The thermic features and the nervous manifestations of this type of the disease are pronounced, the patient soon entering into the typhoid state. The lymph-nodes become markedly swollen, the features are distorted, and salivation is frequently an annoying symptom. The cardiovascular disturbances are marked, the pulse being frequent, weak, and irregular; and there may be unusually severe gastro-intestinal symptoms, such as nausea, vomiting, and diarrhea. In favorable cases convalescence is retarded.

(3) The **hemorrhagic form** (black smallpox), another extremely malignant type of the disease, in which, owing to certain hemic changes or to changes in the other tissues, hemorrhagic extravasations into the skin (Fig. 327), mucous membrane, and viscera take place. This type of smallpox is divided clinically into the following subvarieties: (*a*) A form in which there is an effusion of blood into the pustules, brought about probably by permitting the patient to leave his bed too soon or as the result of undue excitement during convalescence. In this subvariety of hemorrhagic smallpox the lower extremities are involved in the majority of instances. (*b*) During the eruptive stage of the ordinary type of variola, to be subsequently described, a moderate amount of hemorrhage may take place into certain of the pustules. (*c*) The patient may manifest a hemorrhagic tendency during any portion of the eruptive stage of the disease, and there may be bleeding from the mucous surfaces (mouth, lungs, kidneys, uterus, bladder).

In this grave form of the disease the initial symptoms are intense, the eruption is profuse, and collapse may follow the hemorrhages. In this subvariety complications are unusually common; among these are pneumonia and nephritis. (*d*) Rarely do we encounter cases of variola in which the hemorrhagic tendency is displayed during the period of invasion, with ecchymoses into the skin and mucous surfaces as early as the second day of the disease; these hemorrhagic areas develop rapidly, and may involve the greater portion of the body. In these cases the typical eruption of variola is not present, and the thermic manifestations are also unusual, the fever being but moderately elevated. A fatal termination usually occurs early.

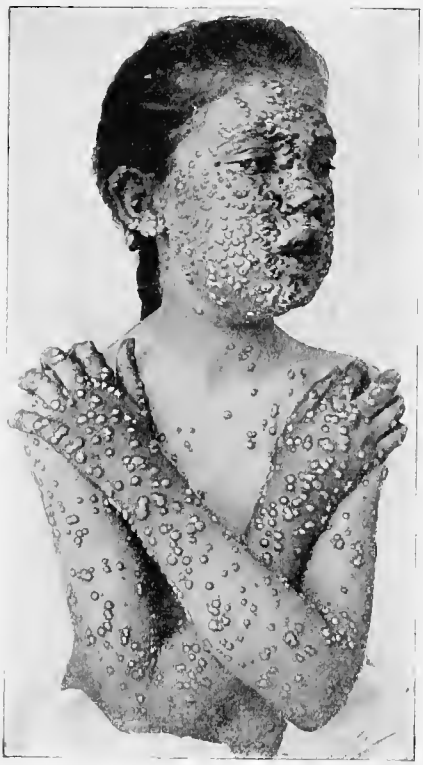


Fig. 326.—Discrete Smallpox in an Unvaccinated Girl.
Eighth day of eruption (Welch and Schamberg).

(4) **Varioloid** is a mild form of smallpox developing in an individual who has been protected by one or more successful vaccinations. This mild form of smallpox may, however, occur in those who possess a variable degree of natural immunity, and who have not been vaccinated. The initial symptoms of varioloid are practically identical with those of variola, but the general clinical course of the disease is usually somewhat milder, the eruption displaying certain irregularities.

Immunity and Susceptibility.—One attack establishes permanent immunity, and successful vaccinations produce the same effect in a vast majority of cases.

Practically every case of smallpox must have had its origin in some previous case, the specific virus being conveyed from one patient to another through various channels and by various methods. (See Modes of Infection.) The disease usually spreads with great rapidity among Caucasians who have not been vaccinated, whereas among American negroes and American Indians it is disseminated with still greater rapidity.

Exciting and Predisposing Factors.—**Parasitology.**—The more recent investigations regarding the etiology of smallpox, made by Councilman and his associates, have resulted in the finding of a body believed to be a protozoan parasite in the epithelial cells and in the fluid of the vesicles and the pustules. Councilman's findings have been confirmed by other investigators. Funk has described a protozoön found in this disease, and Pfeiffer discovered a protozoön in the pustules of vaccinia. Among other observers who have detected the presence of protozoa in vaccinia are Iskigami, Rosenberger, Haushalter, and Etienne.

Bacteriology.—Streptococci and the other bacteria commonly present on the cutaneous surface may be recovered from the pustules of smallpox, but they have no etiologic significance.

Age.—Smallpox occurs during all periods of life. It is especially common in children, and may affect the fetus *in utero*. The disease may develop during the puerperal state in those exposed to the infection.

Period of Incubation.—This varies; six or seven days usually constitute the period of incubation when the disease is directly inoculated from man to man; when, however, it develops as the result of exposure, the incubation period ranges from ten to fourteen days. Ill-defined prodromal symptoms are present at times.

Principal Complaint.—Following the history of exposure to a case of smallpox, there develop, within the course of approximately twelve days, the following symptoms: A rigor, accompanied by intense headache, and followed by a sense of chilliness that persists for from twelve to twenty-four

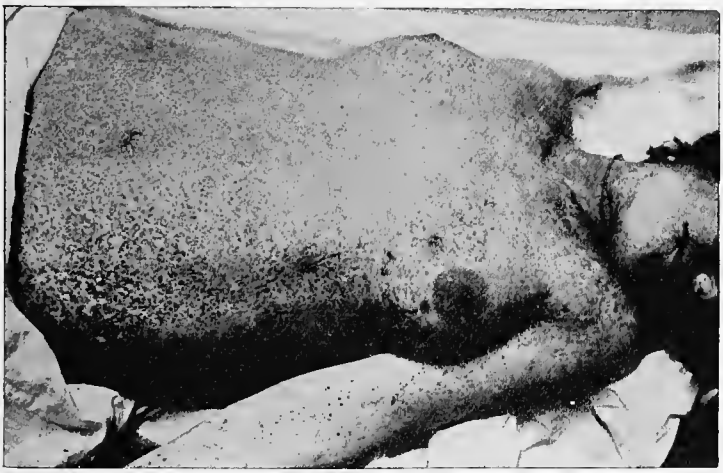


Fig. 327.—Hemorrhagic Smallpox in a Puerperal Woman; Fatal.
Cutaneous surface covered with petechiæ and ecchymoses. A few ill-formed papules were present (Welch and Schamberg).

hours, lumbar pains, and aching muscles. The patient's complaint at the onset of the disease will be found to vary greatly in different epidemics; we have observed epidemics in which these symptoms were unusually mild. (See Thermic Features, p. 902.) Following the chill there generally occur anorexia, constipation, nausea, and, in severe types of infection, vomiting. The constipation may disappear later and be replaced by diarrhea. Sore throat is common, and the patient may complain of a shooting pain, extending from the throat into one or both ears; suppurative otitis media occasionally occurs during convalescence, and should probably be regarded as a complication. Coryza develops early and persists for several days, and severe lacrimation is also present.

Nervous Symptoms.—The nervous manifestations are usually in direct relation to the severity of the type of the disease in question. Restlessness, and frequently mild delirium, are always present, whereas in severe cases maniacal outbreaks, low muttering delirium, and even coma are observed.

In children convulsions not uncommonly occur. Paraplegia and multiple neuritis and myelitis may appear during convalescence, and, indeed, these grave nervous conditions may develop as a sequel rather than as a symptom of the disease. Insanity, hemiplegia, aphasia, and epilepsy may also follow an attack of variola.

Cutaneous Features.—After the development of the pustule there is intense itching of the skin. (See Inspection, p. 903.)

Respiratory Symptoms.—There is always an associated *pharyngitis* and a *laryngitis*, both of which result from eruption upon the mucous surfaces. The laryngeal condition may be so severe as to result in the development of a perichondritis, which is likely to be followed by edema of the glottis. In variola, as in most acute febrile conditions in which acute bronchitis accompanies the disease, pulmonary congestion and bronchopneumonia are to be dreaded as complications. As in certain other acute infections, variola shows a predilection to attack the serous surfaces, consequently pleurisy may develop during the acute stage of the disease and during convalescence;

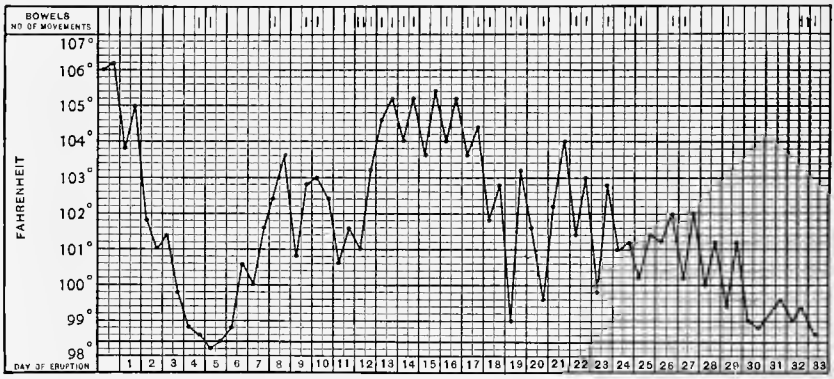


FIG. 328.—TEMPERATURE-CHART OF A CASE OF VARIOLA, FROM A PATIENT IN THE MUNICIPAL HOSPITAL, PHILADELPHIA (J. M. Anders).
A. F——, aged three years; not vaccinated.

its onset is indicated by the presence of intense lancinating pain in the chest. *Cough* is an early symptom, and may continue until convalescence is established; in those cases in which pulmonary complications develop, the cough persists for some time.

Thermic Features.—At the onset of the disease the temperature will be found to rise rapidly, reaching 103° to 105° F. by the end of the first twenty-four hours following the chill. The fever is of the continued type, remaining high until the papular eruption appears,—about the third day,—when there is a decided fall in the temperature. Following the appearance of the papular eruption the temperature continues slightly elevated until the development of suppuration, when it again rises—the so-called secondary fever. (See Fig. 328.) This secondary exacerbation of temperature is decidedly irregular or septic in character, displaying exaggerated points of elevation and of marked remission. In mild cases of variola the secondary febrile expression may be feeble or even absent. In cases of average virulence the secondary fever continues for three or four days, and declines gradually with the improvement of the general symptoms.

A third febrile exacerbation should be regarded as due to the presence of some complication.

Physical Signs.—Inspection.—With the beginning of the fever a diffuse erythema is frequently seen on the arms, legs, and trunk. This is sometimes called the initial eruption, but it has nothing to do with the characteristic rash of the disease, being caused by the capillary dilatation, the result of the influence of toxins on the vessels, which is seen in the early stages of nearly all acute febrile diseases. This erythema in some cases resembles the exanthem of scarlet fever, when it is called *scarlatiniform*, and in other cases it resembles the eruption of measles, when it is said to be *morbilliform*.

The true eruption develops upon both the skin and the visible mucous surfaces, appearing first upon the face, forehead, and scalp, and extending in a downward direction to the thighs, and finally to the legs. The femoral region is more likely to escape than are other portions of the cutaneous surface. Each pock passes through the following stages:

Fig. 329.—Smallpox—Dried Pocks Embedded in the Horny Layer of the Palms (Welch and Schamberg).

(1) *Macule*, in which the mark is reddish in color, resembling the bite of an insect; it increases in size for twenty-four hours, at the end of which period each macule is developed into a distinct papule.

(2) The *papule* continues as such for a period of three days, up to the sixth day of the disease, when the conical apices of the papules become filled with liquid, and vesicles are formed.

(3) The *vesicles* increase in size until the entire lesion becomes filled with exudate, its apex being depressed—the so-called umbilicated vesicle. At this stage puncturing of the vesicle does not cause it to collapse, but is followed by the escape of but a small portion of the liquid contents—a clinical fact that indicates that the vesicular lesion of smallpox is divided into several compartments.

As the vesicle increases in size its contained fluid becomes opaque, and three days later—the ninth day of the disease and the sixth day of the eruption—it is converted into a *pustule*. Lesions are seen upon the soles of the feet and upon the palms of the hands (Fig. 329).

With the development of the pustular stage the umbilicated appearance

of the lesion is lost and the pustule is surrounded by an inflammatory areola. In those cases in which the pustules are in close proximity, generally upon the wrist, face, and fingers (Figs. 326 and 329), the skin connecting the pustules becomes edematous. In confluent smallpox the pustules coalesce and there is marked swelling of the skin, which is often so severe as to distort the features. The pustules usually rupture soon after they appear.

(4) Following the escape of the purulent exudate from the pustule a scab is formed, which remains until about the twelfth day of the eruption. After the scabs have been shed, a permanent whitening of the spot, with a depression of the skin, remains.

Extensive cutaneous gangrene, bed-sores, abscesses, and the development of erysipelatous processes are among the annoying features, and should be classed as *cutaneous complications*. The face is swollen, and in many instances the eyes are completely closed; the lips and mucous membrane of the mouth and throat, the buccal and pharyngeal mucosæ, and the tongue are swollen and coated, and a false membrane may be detected upon the pharynx and tonsils.

Ocular Phenomena.—The conjunctivæ are congested, and pustules and ulcerations may be present upon them; one of the most serious of the ocular phenomena that may occur is ulceration of the cornea. Keratitis, choroiditis, and panophthalmitis are among the less common ocular disturbances.

The *joints* are often swollen. Owing to pain, the patient may persist in holding the arm or limb partially flexed. As the result of an associated peritonitis the thighs may also be flexed upon the abdomen.

Palpation.—Immediately after the appearance of the eruption the skin is dry and hot, and later it is slightly roughened along those areas in which the initial eruption appears. During the papular stage the papules upon the forehead and about the wrists have a distinct, shot-like feel; after pus has accumulated within the lesion, the intervening skin pits upon pressure, the result of edema.

Following the onset of the disease, the *pulse* is accelerated, reaching 100 to 120 beats a minute; it is of good volume and of moderate tension. During the stage of remission the pulse is diminished in frequency, and may be but slightly above that of the normal; with the development of the secondary fever, however, the pulse is again markedly accelerated, the number of beats varying in frequency between 100 and 130 a minute. If the disease occurs in a patient who has previously suffered from cardiac disease, or if cardiac complications, such as endocarditis, pericarditis, or myocarditis, are present, the pulse may become weak, dicrotic, irregular, intermittent, and easily compressible. The liver and the spleen are often palpable, and the apex-beat of the heart is forcible.

Auscultation.—The signs of acute bronchitis are present (p. 88). The heart action is rapid, and in severe and in complicated cases the muscular element of the first sound is deficient.

Laboratory Diagnosis.—Fluid obtained from the vesicles may be sterile, although some observers assert that they have found it to contain bodies that appear to be parasites. (See Parasitology, p. 900.) After the serum has become infected with pathogenic bacteria, the pus will be found to contain a variety of pus-producing organisms. (See Parasitology, p. 900.)

According to the reports of Welch and Schamberg, *albuminuria* is present in 65 per cent. of all cases of smallpox, whereas casts were found in 45 per cent. of a series of 128 cases. *Hematuria*, although rare, may occur in those

cases having a hemorrhagic diathesis. *Constipation* is present, as a rule, but it may be replaced by diarrhea, and rarely there is profuse hemorrhage from the bowel. R. G. Curtin has reported a case where intestinal hemorrhage caused a fatal termination before the eruption had fully developed. Vomiting of blood is also an unusual complication.

The *sputum* may be blood-streaked, although such blood may have its origin in the buccal cavity or pharynx. If otitis media develops, a purulent bloody discharge will be recovered from the external auditory canal.

Summary of Diagnosis.—The history of an epidemic or of exposure to a case is of great importance, as is also the evidence of a previous vaccination. Sudden onset with a chill, followed by a rapid rise in temperature, which continues for a period of about three days, when first a macular, then a papular, later a vesicular, and finally a pustular eruption develops, accompanied by secondary fever, forms a highly characteristic grouping of features. Prior to the development of the eruption, pain in the back and loins, cough, acute bronchitis, and sore throat are to be considered, although these symptoms may be present in the other acute infections; hence in the preëruptive stage the diagnosis is made not only with difficulty, but is often impossible.

Differential Diagnosis.—**Scarlet fever** is to be distinguished early from the erythematous (scarlatinous) rash that is often a precursor of the variolous eruption; this is, as a rule, neither so intense nor so uniformly distributed over the surface of the body as in true scarlatina. Hemorrhagic scarlatina, which is extremely uncommon, closely resembles "black" smallpox.

Measles.—During the first three days of the development of the smallpox eruption, while the rash is still in the macular stage, the disease may be mistaken for measles. The latter disease, however, presents more marked evidences of respiratory disturbance than does smallpox. In measles the conjunctivitis, photophobia, and coryza are more marked than in smallpox. In the papular stage of the variola eruption the shot-like feel of the lesion will distinguish it from the papule of measles.

Typhus Fever.—The onset of typhus fever resembles closely that of smallpox. The former disease is usually imported, and is not prevalent in America. The appearance of the eruption first upon the trunk (chest and abdomen), in the form of macules, later becoming petechial, is characteristic of typhus fever. Moreover, in typhus the temperature does not remit with the appearance of the eruption. The temperature chart of typhus (see p. 760), when compared with that of variola (p. 902), will be found to display distinctive characteristics.

It is at times extremely difficult to distinguish between hemorrhagic smallpox and virulent types of typhus fever. The nodular or shot-like feel of the papules, so characteristic of smallpox, is, however, absent in typhus.

Cerebrospinal fever may be mistaken for hemorrhagic smallpox, but the history of the case, the prominence of nervous symptoms (see Meningitis, p. 838), together with the evidence obtained from lumbar puncture and an analysis of the cerebrospinal fluid, will serve to differentiate these maladies from each other.

Syphilis is marked by a milder initial stage, by the indurated base of the pustule, and by the appearance in crops of the skin lesions and their polymorphous character. The pitting characteristic of smallpox does not follow syphilis, although a copper-like tint of the skin, the result of pigmentation, is seen.

Impetigo contagiosa does not present an initial stage, but begins as

vesico-pustules that appear on the normal skin and enlarge by peripheral extension. In impetigo the characteristic febrile expression is absent.

The accompanying table, modified from Anders, sets forth the distinctive features between variola (smallpox) and varicella (chicken-pox):

Variola.	Varicella.
History.	
1. Previous or existing case in the vicinity.	1. Traceable to previous or present case of varicella.
2. Not successfully vaccinated.	2. Negative.
3. Occurs at any age.	3. Is more common in childhood.
4. Characteristic preëruptive stage, rash on the third day.	4. Eruption not preceded by prodromes.
5. Sacral pain, high fever, and vomiting common.	5. Pain, high fever, and vomiting uncommon.
Eruption.	
6. Appears first upon the forehead, extending downward.	6. Appears first over the parts covered by clothing. No regular distribution over the body.
7. Vesicles uniform in size, umbilicated, and deep seated.	7. Vesicles vary much in size, are rarely umbilicated, and feel soft and velvety.
8. Contains serum and later pus.	8. Contains only serum, giving it a pearly translucency.
9. Most abundant on face and fingers.	9. Most abundant upon back and lower extremities.
10. Appears in a single crop.	10. Various lesions present side by side.
11. Pin-prick does not cause collapse of vesicles, they being multilocular.	11. Pin-prick causes collapse, vesicles being unilocular.
12. Secondary fever usually present.	12. Secondary fever absent.

Clinical Course and Duration.—This will be found to vary greatly, depending upon the severity of the type of infection and upon the presence or absence of complications. In uncomplicated cases the eruption will have advanced to the pustular stage by the ninth day, and if it is but moderately extensive, the formation of scabs will be observed after the twelfth day, when, in favorable cases, the secondary fever will gradually decline, reaching the normal during the third week of the disease. The disease runs a somewhat shorter course in the aged than in young subjects.

Complications.—Among the more serious complications are bronchopneumonia, lobar pneumonia, hemorrhage from the bowel, acute nephritis with hematuria, grave nervous manifestations, ulceration of the cornea, purulent conjunctivitis, and otitis media. Almost all these complications have been considered in the general description of the disease.

VACCINIA (Cowpox).

Pathologic Definition.—An attenuated form of smallpox resulting from vaccination with serum collected from bovines that have previously been inoculated with the disease.

Clinical Characteristics.—Within the course of three to five days following vaccination a distinct papule appears at the site of the lesion, which is surrounded by a red areola. The congested area extends, and by the sixth day a well-marked umbilicated vesicle or a crop of vesicles are present. These often show distinct umbilication, and by the tenth day they contain purulent fluid. On or about the twelfth day following vaccination the lesion tends to disappear, and within the course of three or four weeks the scab is shed and distinct pitting remains at the site of each papule.

Thermic Features.—In from four to six days following vaccination

mild fever and constitutional disturbances appear, and may continue until the ninth day.

The lymph-nodes in the corresponding axilla are often enlarged, and may be tender or even painful.

Atypical Forms.—If the patient is especially susceptible to the virus, or if the virus is unusually active, extensive erythema and a papular eruption may occur, and go on to suppuration, leaving distinct pock-marks on certain portions of the body.

Complications.—Those resulting from external infection of the wound are erysipelas, impetigo, extensive ulceration, and tetanus. Tetanus has been found to follow vaccination in 33 out of 863 cases in which the disease occurred after operation, injury, and the like (Anders and Morgan). The transmission of syphilis through vaccination has been reported.

VARICELLA (CHICKEN-POX).

Pathologic Definition.—An acute infectious disease characterized by the presence of a cutaneous eruption of vesicles distributed over the body.

Exciting and Predisposing Factors.—Bacteriology.—Various investigators have isolated different bacteria from the vesicles, but the etiologic factor is as yet unknown.

The disease is seldom, if ever, conveyed by clothing, etc., personal contact being necessary to produce the disease.

Incubation Period.—The eruption develops in from fourteen to sixteen days after exposure.

Principal Complaint.—Slight prodromal symptoms may be experienced, but these, as a rule, are so mild as to be unappreciated by the patient.

Thermic Features.—Upon the second and third days of the disease the temperature will be found to range between 100° and 102° F.; occasionally a higher temperature is observed. The fever declines by lysis.

Physical Signs.—Inspection.—*Eruptive Stage.*—In many instances the patient appears to be in perfect health until the eruption appears. The latter is characteristic, appearing in the form of small reddish *puncta* that later develop into rose-colored *macules.* As the disease progresses the macules may become converted into *papules,* and later into *vesicles,* the lesions becoming distended to approximately the size of a pea.

The distribution of the eruption is somewhat characteristic, appearing first upon the upper portion of the body—*i. e.,* the chest, back, neck, and scalp. The face is, as a rule, but sparingly covered, whereas the scalp contains many lesions. Vesicles may also appear upon the lips, within the buccal cavity, and on the palate. These are transparent at first, but later become translucent, and the vesicular contents may become seropurulent. A narrow areola the result of congestion surrounds each vesicle. Later *crusts* form, which drop off in from the sixth to the twentieth day after the appearance of the eruption. Pitting is not common, and is rarely seen on those portions of the body covered by clothing. Some of the lesions may go on to form well-marked pustules, although this manifestation is by no means characteristic of the disease. When the eruption begins to fade, intense itching of the scalp occurs, and in those cases in which the eruption is profuse, itching of the entire body may be an annoying feature. The eruption of chicken-pox appears in successive crops, so that macules, papules, and vesicles may be seen side by side in a given cutaneous area. This is a distinctive feature between varicella and variola. In the latter disease the

eruption involves the entire body at one time, and the lesions in various situations are of the same degree of evolution.

Complications.—These are unusual, although the disease is occasionally complicated by **erysipelas**, which may extend from certain of the infected areas, and is most likely to affect those sections in which there has been distinct ulceration. **Isolated abscesses** and **adenitis** are occasionally seen. **Acute nephritis** may develop when the surroundings are unhygienic, and when the patient has been unduly exposed to cold and wet.

HYDROPHOBIA (RABIES).

Pathologic Definition.—An acute infectious disease, characterized, according to Van Gehuchten and Nelis, by lesions in the ganglia, on the posterior roots of the spinal nerves and of the sympathetic system. These lesions consist of atrophy and invasion and destruction of the nerve-cells by newly formed cells derived from the endothelial cells of the capsule of the ganglion. The cerebral vessels may contain soft thrombi, and hemorrhagic extravasations into the perivascular spaces may take place, as has been stated by Fitz.

General Remarks.—The specific infection is conveyed to man by the bite of an infected animal.

Rabies is constantly present in certain parts of the country. In 1906 there was hardly a county in Pennsylvania in which the disease had not been reported. In Chester County, Pennsylvania, during the summer of 1907, the destruction of 154 dogs, 25 cows, and 10 horses was necessitated by reason of the fact that they had been exposed to the bites of rabid dogs. The increasing prevalence of hydrophobia is further supported by the statistics of the State of Connecticut for 1906, when, in the city of Waterbury, several persons were bitten by rabid animals and 175 dogs were destroyed. At Torrington, Conn., seven cows died of hydrophobia. In 1905–06 the disease prevailed extensively in Florida, and Hill reports the necessary destruction of 1200 dogs. Twelve persons were bitten, and of these three died of hydrophobia. In Norfolk, Va., nine persons have been bitten by rabid dogs during the past five years, and a large number of domestic animals have been destroyed. At Charleston, W. Va., 12 cows and 40 dogs are reported as having died from the disease during the past few years. This brief statistical résumé is in itself sufficient to convince the most skeptical of the increasing prevalence of hydrophobia in the United States. The reader is referred for further information to the detailed report issued by the Bureau of Animal Industry, January, 1908.

Exciting and Predisposing Factors.—No specific organism has as yet been detected. In 1903 bodies were found in the large ganglion-cells of the brain; particularly in the hippocampus major; these were described by Negri, and are known as Negri bodies. Some authors believe them to be protozoan parasites, and the cause of the disease. The bodies are round, oval, or triangular in shape, and vary in size from 1 to 23 microns in diameter. They are composed of a homogeneous, non-granular substance, which is strongly eosinophilic, and which resembles coagulated albumin. The bites of infected dogs are the usual cause in man.

Clinical Stages.—The **prodromal stage** lasts from two weeks to four months, the average case developing symptoms in from six to eight weeks after exposure. George H. Heart, V.M.D., cites the case of a dog that received a bite from a rabid animal and developed hydrophobia just

one year later. The diagnosis in both animals was made from a pathologic study of their tissues and by the inoculation of rabbits.

In experimental hydrophobia, when the virus is introduced directly into the nervous system (meninges), definite symptoms develop in from fourteen to twenty-one days. An unusually prolonged incubation period, extending over months, is occasionally seen, and is explained in the following way: If a rabid animal bites a human subject through the clothing or inflicts but a slight wound, the virus is not introduced directly into the circulation, but is taken up by the lymphatics and held within the lymphatic system for an indefinite period (weeks, months); when, however, the virus passes beyond the barriers of the lymphatics and reaches the nervous system, the characteristic symptoms of the disease follow in from fifteen to twenty-one days.

Among the **initial symptoms** are depression of spirits, malaise, headache, impaired appetite, insomnia, slight fever, photophobia, intolerance of sound, and alterations in the voice, such as hoarseness and dysphagia.

Second Stage.—At this time the patient becomes extremely excitable, and there is hyperesthesia of the special senses and of the skin. The muscles of the throat become more or less fixed, and attempts at swallowing are followed by *violent spasms* that involve the muscles of the pharynx, mouth, and upper portion of the chest. During the spasm the patient becomes cyanosed and presents a picture of great distress. Owing to the hypersensitiveness of the nerves, the paroxysms may be excited by drafts, the sight of water or of food, an attempt to swallow, startling noises, or even by an attempt to move the patient in bed. Intense thirst is present. Consciousness may be retained during the attacks, although in certain cases delirium occurs.

Thermic Features.—Mild fever is usually present during this stage of the disease, the temperature ranging between 99° and 103° F. This stage of the disease continues for from thirty-six to seventy-two hours.

Third Stage.—This is often referred to as the paralytic stage, and is characterized by the absence of spasms and the development of stupor, followed by coma, which terminates in death in from six to eighteen hours.

Summary of Diagnosis.—A history of being bitten by an animal believed to be suffering from the disease at the time, together with the characteristic symptoms, is strong evidence of the existence of hydrophobia. The animal that inflicted the bite should in no case be immediately killed. Instead, he should be placed in a safe cage, and given both food and water. If the dog has rabies, he will die within two or three days, and an autopsy will determine the cause of death. It is unfortunate that so many animals are shot immediately after inflicting a bite upon either man or domestic animals, for if he were immediately placed under the care of a veterinarian, definite knowledge could be ascertained as to the presence or absence of rabies, and proper treatment of the bitten individual accordingly instituted.

Lyssophobia (Pseudo-hydrophobia) is a condition affecting persons of neurotic or hysteric temperament some months after being bitten by a dog. They then develop symptoms simulating those of hydrophobia. Among the characteristics that differentiate this condition from true hydrophobia are the following: Irritability, despondency, emotional seizures, absence of fever, and the fact that the disease does not progress through the successive clinical stages.

Clinical Course.—Hydrophobia usually terminates fatally on about the third day. Dogs generally die on the third day, rabbits on the ninth day, and monkeys on the fourteenth day.

TETANUS (TRISMUS; LOCK-JAW).

Pathologic Definition.—An acute infectious disease caused by the bacillus tetani. The toxins act upon the nerve-cells of the medulla and the spinal cord, resulting in congestion, edema, inflammation, and softening of the gray matter. An ascending neuritis extends from the initial wound, and is characterized by reddening and swelling of the neurilemma.

Clinical Types.—The disease, as a rule, follows the infliction of punctured or lacerated wounds. In the new-born it results from infection of the umbilical cord.

Varieties.—(1) **Acute tetanus**; (2) **chronic tetanus**; and (3) **cephalic tetanus.**

Exciting and Predisposing Factors.—Bacteriology.—The bacillus of tetanus was first recovered from the tissues of man by Rosenbach in 1886, although it had been described in 1884 or 1885 by Nicolaier. It is a long, slender rod, clubbed at one extremity, and is anaërobic. Animals inoculated with cultures of this organism develop typical attacks of tetanus.

The tetanus bacillus is a normal inhabitant of the soil of certain localities, and is also present in the intestine of the horse.

Modes of Infection.—Anders and Morgan's analysis of the records of 1201 cases of tetanus shows conclusively that the introduction of the tetanus bacillus is usually effected through a lesion of the skin, and that the so-called idiopathic or rheumatic type of the disease does not exist.

Season.—The accompanying tables, taken from the paper of Anders, in collaboration with A. C. Morgan, the result of an analysis of 687 cases, is of special value as showing the influence of season upon tetanus:

SEASONAL OCCURRENCE OF TETANUS.

	Number of Cases.		Number of Cases.
January	35	August	59
February	36	September	68
March	41	October	75
April	42	November	47
May	57	December	37
June	61		
July	129	Total	687

SEASONAL OCCURRENCE OF TETANUS NEONATORUM.

January	12	August	8
February	10	September	14
March	24	October	9
April	5	November	9
May	6	December	7
June	11		
July	18	Total	133

Immunity.—Animals may be rendered immune by injecting them with cultures of the tetanus bacillus after such bacteria have been treated with iodin trichlorid.

Incubation Period.—The duration of the period of incubation is dependent entirely upon whether the case pursues an acute or a chronic course. In acute tetanus the incubation period lasts from one to two weeks, whereas in the chronic type the first symptoms are manifest after the second week. The accompanying table, taken from the paper just cited, shows the average incubation periods in infants and in adults developing the disease:

TABLE OF AGES OF PATIENTS IN TETANUS CASES.

	Number of Patients.		Number of Patients.
3 to 15 days	13	Over 50	14
1 to 5 years	24		
5 to 10 years	99	Total	583
10 to 15 years	130	Unclassified	618
15 to 20 years	70		
20 to 25 years	75	Total	1201
25 to 30 years	44		
30 to 35 years	42	Males	778
35 to 40 years	37	Females	203
40 to 45 years	22		
45 to 50 years	13	Total	981

Sex.—Males, as seen from the foregoing table, are far more likely to develop the disease than are females, a clinical fact that is probably explained, in part, at least, by the exposure to which males are subjected in the various trades. The disease appears to attack most frequently those who have previously enjoyed good health.

"**Age.**—An analysis of 593 cases gave 229, or 39.3 per cent., of them from the fifteenth to the twenty-fifth years; while there were 86 cases, or 14.8 per cent., between twenty-five and thirty-five years. After the fiftieth year only 14 cases occurred" (Anders).

Acute Tetanus.—Clinical Picture.—The patient usually experiences mild *prodromal symptoms*, such as languor, headache, mental hebetude, and anorexia. The characteristic symptoms develop somewhat insidiously, the patient's first complaint being of stiffness of the muscles of mastication and of those at the back of the neck. *Tonic spasms* soon follow, the muscles of the face become spastic, and there is locking of the jaws.

Spasm.—Severe convulsive seizures are often excited by slight movement of the patient, by sounds, or by currents of air; the attacks are accompanied by excruciating pain.

Rigidity of the cervical muscles becomes marked, and the patient is unable to bring the chin forward upon the chest; retraction of the head soon follows. The *facies* is characteristic, the forehead being wrinkled and the corners of the mouth drawn down, giving to the face the expression of a peculiar sardonic smile. Further examination shows that the reflexes are increased. The skin is beaded with perspiration.

The *pulse* is increased in frequency even in mild cases, and in the more severe types it varies from 140 to 160 beats a minute; it is small, irregular, dicrotic, later becoming compressible.

The muscles of the body are next attacked, in the following order: First, those of the trunk and spine are affected, causing the body to assume a bowed or arched attitude (opisthotonos), and there may be lateral arching (pleurothotonos); the abdominal muscles are next affected, becoming unduly rigid, and their spasmodic contraction may incline the body forward (emprosthotonos). Later the legs become involved, the arms, however, being in most cases capable of some movement. The patient complains of thoracic oppression and agonizing pain at the base of the chest.

Thermic Features.—Moderate fever is present, as a rule, although in selected cases the temperature may suddenly rise to 105° or even 110° F. In other instances fever may be absent throughout the attack.

Laboratory Diagnosis.—*Constipation* continues throughout the attack. The *urine* is usually voided with each spasmodic seizure. Scrapings from

the initial wound may show the presence of the bacillus tetani. Moderate leukocytosis—12,000 to 14,000—has been observed.

Chronic Tetanus.—Clinical Picture.—Here practically all the symptoms previously outlined under the acute form of the disease are present, but they develop less rapidly. The painful spasms may disappear, making the administration of liquid food possible. A feature that is characteristic of the chronic form of the disease is that the patient experiences partial freedom from painful seizures, the intervals becoming longer and longer as convalescence proceeds. *Relapses* are prone to occur.

Cephalic Tetanus.—Clinical Picture.—Rose described this type of tetanus, which follows injuries to the face. Among the most characteristic features are spasms of the masseter muscles and the pharyngeal muscles, causing dysphagia and rarely contraction of the muscles of the neck and abdomen. Paralysis of the facial nerve may take place. Approximately 25 per cent. of all cases of cephalic tetanus go on to recovery.

Summary and Differential Diagnosis.—Given the history of a punctured wound or of an abrasion of the skin, the diagnosis is based upon the following clinical features: The presence of rigidity of the muscles of the jaw and of the neck, with retraction of the head, spasm of the muscles of the trunk, and later of the lower extremities and of the arms. The detection of the specific microörganism in scrapings from the wound makes the diagnosis positive, even in those cases in which the symptoms are atypical.

Tetany differs from tetanus first by its clinical history, and second by the fact that the extremities (hands) and larynx are involved intermittently. Tetany is a disease of the young, and the attitude of the patient is unlike that seen in tetanus.

Hydrophobia.—Here there is a history of being bitten by an animal in practically all cases, and the spasmodic seizures are limited more especially to the respiratory system. In hydrophobia the jaws are free and opisthotonos is practically unknown.

Strychnin Poisoning.—The distinctive features between strychnin poisoning and tetanus are set forth in the accompanying table taken from Anders:

Tetanus.	Strychnin Poisoning.
1. The receipt of a wound, generally followed by a period of incubation.	1. Ingestion of strychnin, immediately followed by the symptoms.
2. Begins with lock-jaw; later spreads downward, the arms and hands escaping.	2. Begins with gastric disturbance or a tetanic contraction of the extremities. Hyperesthesia of the retina occurs and objects appear green.
3. Reflex spasms not present at the onset.	3. Violent convulsions present from the onset.
4. Rigidity is persistent, except in the chronic form.	4. Intervals of complete relaxation occur.
5. The course is prolonged into days or weeks.	5. The course is brief, terminating in death or recovery.
6. Cultures made from the discharges of the wound show the presence of the bacillus tetani.	6. Examination of the gastric contents shows the presence of strychnin.

Clinical Course.—In cases in which the symptoms develop within one or two weeks following an injury the disease usually runs a rapid course, terminating fatally in a few days. In the chronic type the disease may become much protracted. Tetanus neonatorum is probably the most fatal form of the disease, and pursues a rapid clinical course. Of 870 cases analyzed by Anders and Morgan, 338 terminated fatally before the fifth day;

275 died between the fifth and the tenth day, and 211 cases lived for more than fifteen days.

GLANDERS (FARCY).

Pathologic Definition.—An acute infectious disease caused by the presence of the Bacillus mallei. It is characterized by the development of granulomata. These growths are nodular, and when situated upon the nasal mucous membrane, become soft and eventually ulcerate. On the other hand, when they are situated on the skin, multiple abscesses result. Microscopically, sections from the new-growth are seen to contain numerous bacilli.

Clinical Varieties.—(1) **Acute glanders.**

(2) **Chronic Glanders.**—This is a mild and unusual form of the disease. The symptoms are vague, nasal catarrh being the most significant indication.

(3) **Acute farcy** is a form of cutaneous glanders in which the nasal manifestations may be mild or absent. In this type of the disease the *local symptoms* at the seat of the primary lesion may be acute, and numerous cutaneous abscesses which are distributed along the lines of the lymphatics develop later. The *constitutional symptoms* of pyemia are manifested early.

(4) **Chronic Farcy.**—This form of the disease is characterized by the formation of granulomatous tumors of the skin and subcutaneous tissue, which eventually result in abscesses. The lesions are most commonly seen in the neighborhood of the large joints. In chronic farcy the abscesses usually discharge their contents externally, leaving behind an offensive ulcer. Owing to suppuration, the temperature takes a hectic curve.

Exciting and Predisposing Factors.—Bacteriology.—The disease is due to infection with the bacillus mallei, an organism that is readily cultivated, and is found present in sections of the granulomatous mass, and in the purulent discharge from the nasal mucous membrane, as well as from abscesses and ulcers.

Sex.—This disease is usually transmitted directly from domestic animals to man, being generally contracted from horses; hence males are most often affected.

Modes of Infection.—The medium of conveyance from the equine family to man is usually through the purulent nasal secretion, which is expelled from the animal's nostrils and alights upon open wounds, upon abrasions of the skin, or upon the mucous surface.

Incubation Period.—This varies, lasting usually from three to five days.

Immunity.—Man possesses an almost complete natural immunity to this disease, and Singer asserts that he has produced immunity by making intravenous injections of sterilized cultures of the bacillus mallei.

Acute Glanders.—**Clinical Picture.**—The first evidence of the disease is the presence of inflammation at and surrounding the point of infection. *Lymphangitis* follows within the course of a few days, and all the adjacent lymph-nodes become involved. Later a distinct *eruption* involving the face and trunk is seen, the extremities, particularly in the region of the joints, becoming finally affected. The papules rapidly become converted into pustules, discharging a seropurulent fluid that contains the specific organism of the disease. Following the accumulation of pus in the lesions extensive swelling of the nose and of other portions of the body occurs. In practically all cases the conjunctivæ are attacked, and lesions may extend to the mucous surfaces of the mouth and pharynx, and, rarely, the respiratory

and gastro-intestinal tracts may become involved. Necrosis of the bones is occasionally seen.

Summary of Diagnosis.—The diagnosis of glanders is made with difficulty unless a clear history of exposure to an infected animal is obtained. In doubtful cases the pus from the lesions should be injected into the peritoneal cavity of a male guinea-pig. The early development of edema and the formation of pus within the tunica vaginalis testis of the animal will give an opportunity for the cultivation of the bacillus mallei. The detection of the specific organism in the exudate recovered from the pustules and nasal mucous membrane confirms the diagnosis.

ANTHRAX

(MALIGNANT PUSTULE; SPLENIC FEVER; WOOL-SORTER'S DISEASE, ETC.)

Pathologic Definition.—An acute infectious disease due to the presence of the bacillus anthracis, and characterized by the formation of a rapidly extending pustule, by a bacteriemia, or by lesions of the gastro-intestinal tract or the lungs. The bacillus or its spores may be readily demonstrated in the lesions. The local manifestations of the disease are inflammation, ulceration, gangrene, and edematous infiltration, together with degenerative changes in the heart muscle and in the kidneys. Hemorrhagic and gangrenous infiltration of the intestinal tract and of the retroperitoneal lymph-nodes may also be present. Enlargement of the spleen may be a conspicuous symptom.

Clinical Varieties.—External anthrax (malignant pustule); internal anthrax (intestinal mycosis).

Exciting and Predisposing Factors.—**Bacteriology.**—The exciting cause of the disease is the bacillus anthracis.

Incubation Period.—This lasts, as a rule, between one and three days.

Immunity.—Pasteur has prepared an attenuated virus that has been used extensively in localities in which anthrax is common, and good results are said to have followed its use. Other investigators have obtained less satisfactory results, the majority agreeing that temporary immunity can be effected by its use.

Modes of Infection.—It is highly probable that the bacillus gains entrance into the human body through slight wounds, abrasions, and scratches of the cutaneous surface. The intestinal form of the disease probably follows the ingestion of food containing the specific organism. Primary lesions of the lung occur, but they are rare, and the channel through which the exciting bacterium gains extrance to the pulmonary tissue remains in question. It is asserted that the bite of certain insects, particularly the fly and the mosquito, may convey the disease to man.

Occupation is a prominent predisposing factor, the disease being more prevalent among those brought in direct contact with infected animals or with their hides or wool. It follows, therefore, that those employed in woolen mills and those engaged in handling cattle and sheep are especially prone to contract the disease. Persons engaged in the manufacture of mattresses, rugs, and hair goods are also frequent sufferers from the disease.

Sex.—The disease is more commonly seen in males.

Malignant Pustule.—Clinical Picture.—Three days after infection has taken place an appreciable reddening is seen at the wound of entrance, and at this site a papule forms, rapidly developing into a vesicle containing bloody fluid. During the papular and vesicular stages the patient may

complain of intense burning in the region of the lesion. The *vesicle* soon ruptures, leaving behind a blackish *scab*, surrounded by a brawny area of edematous induration. Radiating from the initial lesion red lines, corresponding to the lymphatic vessels, are to be seen, and within the course of from twenty-four to thirty-six hours vesicles form at variable distances from the initial lesion.

During the second day of the disease the patient may display *constitutional symptoms*, such as high fever, extreme prostration, nausea, vomiting, profuse sweats, appreciable enlargement of the spleen, and, in severe cases, delirium, followed by coma and death.

Internal Anthrax.—Clinical Picture.—For convenience of description and clinical study this type of the disease is divided into two sub-classes:

(*a*) *Wool-sorter's Disease.*—This is marked by sudden *onset* with a severe chill, followed by a rapid rise in temperature, which may reach 103° F. or even higher. *Prostration* is apparent early, and there are severe *pains* in the back and in the muscles of the legs, and intense headache.

As the disease progresses the *heart* becomes rapid, the *pulse* weak and irregular, and severe gastro-intestinal symptoms, *e. g.*, nausea, vomiting, and diarrhea, are apt to occur. *Nervous symptoms* may also be prominent, delirium being followed by coma.

(*b*) *Intestinal Form* (*Intestinal Mycosis*).—In this variety the disease develops abruptly with a *chill*, followed by well-marked constitutional symptoms and by *pain* in the head, back, and legs. Nausea and vomiting develop early, and are usually followed by intestinal cramp and diarrhea. *Hemorrhages* from the mucous surfaces, bowel, stomach, and pharynx may occur, and petechial hemorrhage is also occasionally observed. Muscular spasms are not infrequent, and in all cases extreme restlessness in a conspicuous feature. Moderate fever is, as a rule, present.

Laboratory Diagnosis.—The bacillus anthracis will be found in blood obtained from the initial lesion, both by cultural methods and by the staining of smears. The existence of the bacillus may also be demonstrated by inoculating animals with the serum.

The **urine** becomes scanty, high colored, and of high specific gravity. Hemorrhagic fluid from the mucous surfaces may also contain the anthrax bacillus.

Blood.—Royer and Holmes,* in a report of a clinical study of 15 cases treated at the Municipal Hospital, Philadelphia, gave out the following data: Anthrax bacilli were frequently discovered in the circulating blood both by smears and by cultural methods. In 13 cases a study was made with reference to alteration in the leukocytes, and in these leukocytosis was the rule, the highest count obtained being 25,000 in a cubic millimeter, whereas the average count for the 13 cases was 13,900. In two fatal cases the leukocytes numbered 12,600 and 9600 respectively. In 11 cases a differential leukocyte count gave the following as an average: Polymorphonuclear leukocytes, 77.6 per cent.; large lymphocytes, 17.7 per cent.; small lymphocytes, 5.3 per cent.; eosinophiles, 3 per cent.; basophiles, 0.1 per cent., and myelocytes, 0.4 per cent.

Clinical Course.—In malignant pustule the clinical course is greatly modified by surgical treatment, but in those cases in which treatment is administered late, the disease usually progresses from bad to worse for a period of from five to eight days. Internal anthrax pursues a rapid course, and may soon terminate in coma and death.

*Therapeutic Gazette, January 15, 1908.

SEPTICEMIA.

Pathologic Definition.—A systemic disease due to invasion of the blood and the tissues by pathogenic microörganisms. Among the manifestations likely to be present are endocarditis and an acute catarrhal inflammation of the gastro-intestinal mucosa, with punctate mucous hemorrhages. The spleen is enlarged; there is cloudy swelling of the liver, and acute inflammatory changes take place in the parenchyma of the kidneys; minute hemorrhages into the various serous surfaces (pleuræ, pericardium, and peritoneum) also often occur.

General Remarks.—Toxemia results from the absorption of the poisonous products of local bacterial growth. Septicemia, with the presence of bacteria in the circulating blood, and a toxemia from the absorption of toxins may be present in the same patient.

Exciting and Predisposing Factors.—**Bacteriology.**—A septicemia may be caused by any pathogenic organism, but the majority of cases are due to the staphylococcus aureus or streptococcus pyogenes.

Modes of Entrance of Pathogenic Microorganisms into the System.—(*a*) The pathogenic organisms may enter the circulation through wounds made at surgical operations or through those inflicted accidentally. (*b*) Through wounds in the mucous membrane of the uterus following parturition, abortion, and curetment. (*c*) Through minute cracks and fissures in various parts of the body. (*d*) Through sloughing ulcerative surfaces, as in carcinomata, leg ulcers, bed-sores, gangrene, and the like. (*e*) Through lesions of the mucous membranes, in any infectious disease, as, for example, in typhoid fever, dysentery, gonorrhea, and suppurating processes involving the tonsils. (*f*) The so-called "*sepsis intestinalis*" is due to the ingestion of decomposed meats, ice-cream, and other articles of diet. Strictly speaking, in this connection one would be dealing with a true toxemia, or particularly with symptoms resulting from the toxins of bacteria that have developed upon dead tissue. Vaughan calls the ptomains of these substances tyrotoxicon, and believes that this chemical substance is concerned in the production of true toxemia.

Symptoms of Toxemia.—The fact that this form of poisoning may occur without bacterial infection, either local or general, having taken place, must be emphasized, but more frequently either local infection or putrefactive changes with the production of a grave general condition, due to the absorption of poisonous chemical products, will have occurred. In other acute infections (diphtheria, tetanus, typhoid fever, erysipelas) the general symptoms are similar. Perhaps the most typical examples of toxemia are those due to the formation of tyrotoxicon and to the unaccustomed inhalation of foul odors.

A *chill* may be present. In "sepsis intestinalis" marked local symptoms, such as nausea, vomiting, colic, and diarrhea, may usher in the attack; in all forms the *temperature* rises rapidly to 101° to 105° F. *Prostration* and *anemia* are prominent symptoms. The toxemia following childbirth is a subvariety, and, apart from the special history, the symptoms are usually similar to those outlined above. This is the form most amenable to treatment, the removal of the cause being followed by a rapid disappearance of all alarming symptoms.

Symptoms of True Septicemia.—The *incubation period* is indefinite, usually, however, averaging several days. The *onset* is more gradual than in toxemia, although it is often marked by a chill. When it follows

surgical procedures, there are *fever*, with headache, anorexia, prostration, sometimes vomiting and diarrhea, and mental dullness, occasionally amounting to mild stupor. As the disease progresses the symptoms become intensified, and the patient may enter into the so-called typhoid state.

Thermic Features.—The fever rises abruptly to from 101° to 103° F. or higher, and is often of the continued type. The duration of the febrile period varies with the character of the infection.

Physical Signs.—**Inspection.**—The face may be flushed at first, but extreme pallor is often present. Punctate hemorrhages into the skin are quite common, and these may coalesce to form ecchymoses; also less commonly there is a scarlatinal eruption. Herpes labialis, cutaneous edema, and jaundice are among the rarer features of the disease.

Palpation.—The skin is hot, and may be beaded with perspiration. The pulse is, as a rule, rapid from the onset, the beats numbering 100 to 120 a minute. As the disease progresses the pulse becomes more rapid, irregular, dicrotic, and compressible. After a few days the spleen may be felt below the costal margin, and is often tender upon firm pressure.

Laboratory Diagnosis.—The number of red corpuscles is moderately reduced, and there is a corresponding reduction in the hemoglobin. Leukocytosis is generally present, and a differential count shows an increase in the number of the polymorphonuclear cells. Cultures from the blood may show the presence of pathogenic bacteria.

Diarrhea is not unusual, and may be severe. Vomiting is also frequent.

Nephritis may develop at any time during the course of the attack, casts, albumin, and at times pus being found in the urine. The quantity of urine excreted is diminished, and the fluid is of high specific gravity and has a high color.

Summary of Diagnosis.—The clinical history, especially with reference to the existence of local septic processes or of exposure to or ingestion of toxic substances, is of great importance. The brief incubation period, together with the onset of a chill or of chilly sensations, with a rapid elevation in temperature to from 101° to 103° F., should always suggest the possibility of septicemia. Later, the continuous type of the fever, the rapidity of the pulse, and the degree of prostration are almost constant features. Leukocytosis and albuminuria are often present.

PYEMIA.

Pathologic Definition.—Pyemia is a form of septicemia in which the pathogenic organisms form thrombi or emboli in the small blood-vessels, with the development of multiple abscesses. These abscesses are most frequently found in the skin, the spleen, the liver, the kidney, and the brain. Hemorrhagic extravasations into both the skin and mucous membranes occur, and these ecchymoses may later contain purulent fluid. Ulcerative lesions of the endocardium are by no means uncommon, and in rare cases myocardial abscess is observed.

Exciting and Predisposing Factors.—**Bacteriology.**—Pyemia is believed to result, in the majority of instances, at least, from the invasion of the blood by streptococci and staphylococci. Other pus-producing organisms capable of exciting this condition are pneumococci, the pneumobacillus (Friedländer's), bacillus coli communis, bacillus anthracis, gonococcus, micrococcus tetragenus, and bacillus pyocyaneus.

Paths of Invasion.—The bacteria enter the circulation through the

blood-vessels, by the same routes described under septicemia. The organisms excite either phlebitis and thrombosis, or they form emboli in the smallest arteries.

The bacteria may enter the system through the lymphatics, but here the dissemination of the organisms is slow and consequently pyemia of lymphatic origin is rare.

Pyemia may also follow purulent appendicitis and other concealed abscess formations, and such cases, especially when the site of the lesion is unknown, are said to be suffering from spontaneous pyemia, although in reality they do not differ either etiologically or clinically from the types previously described.

Age and Sex.—The greater number of cases of pyemia develop during middle life, at a time when the greatest number of wounds of the body are inflicted. From the same cause, probably, males are more frequently affected than females.

Season appears to exercise a mild influence, the greater number of cases being seen during cold weather—in this climate, during February and March. Epidemic outbreaks of pyemia are recorded, and local epidemics, limited to institutions, are occasionally encountered.

Period of Incubation.—The characteristic symptoms usually develop first about one week after the patient has received an injury to the skin or after a surgical operation has been performed.

Principal Complaint.—A history of injury from which the patient has not fully recovered is usually obtained. The onset is marked, as a rule, by a distinct chill, although the patient may have felt feverish for one or more days prior to the occurrence of this characteristic symptom. The chill may have been so slight as to escape the patient's notice, yet when questioned carefully, he will usually recall having experienced a series of chilly sensations. *Prostration* is a conspicuous feature early during the disease, and drenching *sweats* occur throughout the twenty-four hours. The seat of the wound may or may not be painful. When there is pleural involvement, *pain* is present over the lungs, and there is also pain over the region of the spleen and liver, due to the septic process. *Abscesses* of the skin and extensive areas of cutaneous and subcutaneous inflammation are also characterized by pain. Metastatic purulent processes localized in or about the larger joints are occasionally seen and are productive of severe pain. Acute osteomyelitis may also develop, and is characterized by the presence of intense pain along the course of one of the long bones.

Vague *gastro-intestinal* symptoms are present, although anorexia usually obtains, and there may be septic diarrhea.

Nervous Phenomena.—The mind is clear until the later stages of the disease, and delirium is a late symptom, usually terminating in coma. Hyperesthesia of the skin may be present early. Purulent meningitis due to metastasis may develop at any time during the course of pyemia, and in such cases the characteristic features of this condition are present. (See p. 1124.)

Thermic Features.—During the chill the temperature rises rapidly, reaching from 101° to 105° F. The course of the fever is remittent, the temperature fluctuating greatly on different days of the disease. In exceptional cases the temperature is intermittent, falling to or below the normal line for a time each day.

Physical Signs.—Inspection may disclose the initial injury or wound of operation, which is seldom completely healed. The expression is

anxious, the face is drawn, and the skin is pale and beaded with perspiration. The nostrils dilate, particularly in those cases in which pulmonary complications exist. Late during the course of the disease there is a variable degree of jaundice, and general cutaneous eruptions—*e. g.*, purpura, localized general erythema, and numerous pustules—are not uncommon.

Palpation.—Pustules and areas of inflammation involving the skin are detectable. If hepatic abscesses or abscess formation in other viscera are present, pressure over the diseased organ will elicit extreme tenderness. The spleen is decidedly tender, and pressure over any portion of the body may excite pain. When the fever is high the skin is unusually hot, but following sleep it is frequently cold and clammy.

The *pulse* is accelerated early during the disease, but is, as a rule, moderately full and regular. As the disease progresses the pulse becomes very rapid,—numbering 120 to 160 beats a minute,—weak, and compressible, and in advanced cases it may be impossible to count the beats.

Auscultation.—In those cases in which pleurisy, pneumonia, endocarditis, or pericarditis develops the auscultatory and other signs of these conditions will be present.

Laboratory Diagnosis.—The *urine* is scanty, of high specific gravity, rich in albumin, and often contains casts, pus, and blood. Cultures from the urine show the presence of colonies of pus-producing bacteria. The diazo-reaction is very common.

Destructive changes in the *blood* soon begin, and there is progressive reduction in the percentage of hemoglobin and red cells. Leukocytosis develops early, the number of white cells varying between 15,000 and 60,000 in a cubic millimeter. Microscopically, the red cells are found to be greatly distorted, and nucleated erythrocytes are by no means uncommon.

Bacteriologic Study of the Blood.—In making a diagnosis of septicemia and pyemia blood-cultures will show the presence of the organism producing the symptoms. The method of making the blood-culture has been described on p. 342. The bacteriologic study of the blood should be undertaken in every case of general infection, in order to determine the nature of the organism producing the symptoms. If the culture remains sterile, the condition is more likely to be a toxemia than a septicemia.

Summary of Diagnosis.—In formulating a diagnosis it is especially important to consider carefully the prominent etiologic factors. The history of a chill, followed within a few hours by a distinct rise in temperature, and the character of the fever-curve are also important. The rapidity of the pulse, with its tendency to become weak, rapid, and dicrotic late during the disease is a marked feature of all pyemic processes. Tenderness over the spleen, and at times over the liver, with enlargement of both these viscera are valuable diagnostic signs. Prostration and profuse sweats are practically always present, and the occurrence of purpura, with the presence of cutaneous pustules, renders the diagnosis almost positive.

Clinical Course.—The course of the disease will be found to vary within certain limits, depending upon the character of the exciting factor; generally speaking, however, pyemia runs a rapid course, progressing from bad to worse.

ACTINOMYCOSIS ("BIG-JAW"; "LUMPY-JAW").

Pathologic Definition.—An infectious disease of cattle, transmissible to man. It is caused by the actinomyces bovis, or ray fungus, and is characterized by the formation of granulomatous tumors in various tissues of the body.

Exciting and Predisposing Factors.—The exciting cause of actinomycosis is the *ray fungus* (actinomyces bovis), which gains entrance to the buccal cavity through slight wounds of the mucous membrane or through cavities in the teeth. Grass and grain are the normal habitats of the ray fungus, and in the majority of cases of human infection the habit of chewing hay or straw explains the occurrence of the disease.

Age.—The disease is more common in adults than in children. Farmers and those working about hay and grain are more liable to become infected than those residing in cities. The American negro is believed to be highly susceptible to actinomycosis.

Principal Complaint.—A hard, tumor-like mass appears in the region of the mouth and progressively increases in size; it may ulcerate or form a sinus, and discharge its pus either externally or into the oral cavity.

Physical Signs.—Inspection.—Neither the location of the tumor nor its general appearance is characteristic of the disease, but an associated involvement of the submaxillary gland and the lymph-nodes of the neck points strongly toward the presence of actinomycosis. (See Actinomycosis of the Lung, below.)

Laboratory Diagnosis.—A microscopic examination of the pus or of scrapings from the tumor is the only positive guide to a diagnosis. The pus will be found to contain leukocytes, pus-cells, epithelial cells, granular débris, and at times much necrotic tissue. When smeared thinly upon a slide and studied with a high-power objective, the pus of actinomycosis will be found to contain the ray fungus (Fig. 33, p 82), which may appear in its characteristic branching forms, although this finding is by no means constant. The morphology of the fungus varies directly with the character of the soil upon which it develops, and with the resistance offered to such development by the patient. Not infrequently actinomycotic pus has been seen in which no characteristic ray fungi were found, but in which mycelial threads were present, which, when placed on suitable soil, rapidly produced a characteristic lesion from which the branching forms of the fungus were recovered. In the pus from thirteen sinuses of the mouth studied by us the ray fungus was found seven times, both on microscopic examination of the pus and in cultures. Wright describes a most interesting case of infection of the tonsil with actinomycosis.*

Summary of Diagnosis.—The detection of the ray fungus in the exudate from the lesions is necessary in order to establish a diagnosis of actinomycosis. When a positive diagnosis is reached, the case becomes one of surgical, rather than of medical, interest.

Pulmonary Actinomycosis.

Pathologic Definition.—A chronic infectious disease of the lung caused by the ray fungus (actinomyces bovis), which develops in the pulmonary tissues, giving rise to consolidation and ulceration.

History.—In America actinomycosis is, comparatively speaking, rare. In 1885 Murphy gave an accurate clinical picture of human actinomycosis, and seven years later Bellinger, Harz, and Israël published original articles in Germany, but even before the appearance of Murphy's paper cases had been reported from various sections of the United States. In 1899 Ruhräh collected 58 cases, many of which were then recorded for the first

*Amer. Jour. Med. Sci., July, 1904, p. 74.

time. In 1900 Ruhräh* reported 4 additional cases, and December 1, 1901, 25 more cases were added to the list; in 1902 W. G. Erving reported 10 cases of human actinomycosis. The possibility of its occurrence must be borne in mind, since cases are occasionally encountered; it is only within the past few years that any effort has been made to detect the ray fungus in the sputum, consequently cases must have been overlooked. Pulmonary actinomycosis can hardly be said to be increasing in North America, the increase in the number of cases reported during the past ten years being due probably to the fact that a more careful study of the sputum and of the clinical course of the disease is being made.

Mode of Infection.—The actinomyces is believed to be found normally on both growing and harvested rye, wheat, barley, and oats—grains that are employed as food. According to Wright, however, this generally accepted idea of the normal habitat of actinomyces is erroneous. He believes it to be a normal inhabitant of the intestinal tract of man and the lower animals. In man the disease has been known to follow exposure to the discharges of actinomycotic cattle. Certain clinicians believe that pulmonary actinomycosis is due to the inhalation of the dust of grain upon which the actinomyces is developing, but there is little conclusive evidence to show that this is the mode of infection. In view of the frequency of actinomycosis of the pleuræ, other sources of infection are not improbable. Moreover, since, as is shown by the history, human actinomycosis is fairly common, secondary pulmonary actinomycosis is not improbable. The actinomyces finds a suitable soil for its development in and about decaying teeth, and it is highly probable that the fungus is more often carried into the stomach from this site than into the trachea and lung. (See Actinomycosis of the Pleuræ.)

Symptomatology.—The clinical picture is similar to that of pulmonary tuberculosis, although there are certain important distinctive differences:

(1) In the bronchopneumonic form the history is unlike that of tuberculosis, and during the early stage there is but slight cough, and the sputum is scanty, mucoid, and rarely purulent, but the fungus is absent. The degree of anemia and prostration is, as a rule, out of proportion to the lung involvement, and, indeed, the physical signs are not only indefinite, but, except for the presence of numerous râles and exaggerated breath-sounds, may be absent. In this type of pulmonary actinomycosis several months must elapse before the disease is sufficiently developed to give definite physical signs upon palpation and percussion. Canalis has reported a case in which actinomyces were present in the sputum of a boy for a period of several years without doing marked damage to the lung.

(2) The sputum does not resemble closely that of either incipient or advanced tuberculosis, and no tubercle bacilli are present.

(3) Cough is less likely to be paroxysmal, and throat symptoms (hoarseness, laryngitis, etc.) are less frequent than in tuberculosis.

(4) The feces do not contain tubercle bacilli, whereas Rosenberger has shown that this is an almost constant finding in all forms of tuberculosis.

Physical Signs.—**Inspection.**—Not infrequently there is an external tumor or a sinus from which the actinomyces may be recovered. Deformity of the chest is, as a rule, absent, and when present, there is but moderate retraction of the affected side.

Palpation.—Tactile fremitus is increased most often over the pectoral region, but the disease may be well advanced without disclosing any alteration in the fremitus. In the bronchopneumonic type of actinomycosis the

*Ruhräh, Annals of Surgery, vol. xxxi, 1900.

fremitus is often diminished, and, indeed, owing to the presence of isolated areas of compensatory emphysema, it may be absent.

Percussion.—In direct relation to the degree of consolidation of the lung or of involvement of the pleura the percussion-note is found to be impaired. This impairment may be elicited over the pectoral region, but extensive areas of dullness are usually located at one or the other base; apical dullness has, however, been reported.

Auscultation.—In those cases in which appreciable impairment of the percussion-note is present the breath-sounds are intensified, the respiratory murmur over such areas being bronchial or bronchovesicular. In advanced cases râles are audible not only over the affected areas, but over the greater portion of the lungs, and, indeed, the signs generally regarded as characteristic of pulmonary cavity may be present late during the course of actinomycosis.

Laboratory Diagnosis.—The sputum may contain granules resembling grains of sulphur. These are composed of the mycelial threads of the actinomyces. The detection of these threads in the sputum and in the discharge from a thoracic sinus points to the presence of pulmonary actinomycocis. We have seen a number of specimens of sputum in which the actinomyces was present, but the clinical course could in no case be followed.

Summary of Diagnosis.—The following features will serve, in a measure, to differentiate actinomycosis from pulmonary tuberculosis: (*a*) The tendency to anemia and the degree of prostration in actinomycosis are out of proportion to the severity of the chest involvement; (*b*) actinomycosis displays a special tendency to attack the bases of the lungs, although apical involvement is possible; (*c*) in actinomycosis the pleura is frequently attacked, and in such instances a sinus is often present; (*d*) tumor and abscess of the chest-wall are frequent accompaniments; (*e*) the detection of the actinomyces in the sputum or in the exudate from the pleural sinus is an essential finding in formulating a diagnosis of actinomycosis.

Differential Diagnosis.—(1) **Pulmonary Tuberculosis.**—The differential points between these diseases have just been detailed.

(2) **Carcinoma.**—A history of previous or coexisting malignant growth elsewhere is of great diagnostic importance; particularly is this true after removal of the breast for malignant disease. The absence of fever, the result of secondary infection, also goes far to support a diagnosis of malignant growth. Again, in malignant disease of the lung a microscopic study of the sputum will reveal nothing of clinical value.

(3) **Syphilis.**—It must be borne in mind that syphilis, like actinomycosis, frequently attacks the central or lower portions of the lung. In syphilis an examination of the sputum is negative, and there are, as a rule, a history of the disease and the other visceral changes common to this condition. In luetic disease there is but little tendency toward the formation of tumors and abscesses of the chest-wall, features that are common to actinomycosis. The recovery of the treponema pallidum in the secretion obtained by puncture of the diseased area is valuable evidence of the existence of syphilis.

(4) **Echinococcus Cyst of the Lung.**—Here the diagnosis rests entirely upon the detection of either hooklets or scolices of the echinococcus in the sputum or in fluid obtained by puncture (Fig. 361), and upon the absence of the actinomyces.

Clinical Course.—The disease usually runs a course of from one to two years after the actinomyces can be detected in the sputum. The disease displays a tendency to extend slowly and progressively. Surgical intervention may in rare cases give relief, thereby prolonging the clinical course

until the pleura is involved. In the bronchopneumonic type the progress of the disease is more rapid than in cases in which a single isolated area of consolidation exists.

PULMONARY BLASTOMYCOSIS.

Pathologic Definition.—A chronic disease of the lung caused by some form of blastomyces, and characterized by the presence of pulmonary consolidation.

Remarks.—During recent years a number of brilliant papers on the subject of cutaneous blastomycosis has appeared, but we know of many instances in which the lung has been primarily involved. Walker and Montgomery * have reported a case of secondary blastomycosis, which is sufficient to show that the blastomyces may involve the lung secondarily and excite mycotic changes there.

Symptomatology.—After the patient has suffered for some time from cutaneous blastomycotic disease, he is particularly likely to develop a generalized type of this disease; in which event the lungs become involved early, the chief complaint being of extreme prostration and cough.

Expectoration.—The sputum may be copious, and is, as a rule, mucopurulent in character, although at times it may be blood-streaked. It has been claimed that the associated bronchitis is responsible for the amount of sputum, since there is little or no tendency toward cavity formation. Microscopically, the sputum is always rich in bacteria, containing, as it does, various pus-producing organisms. The yeast fungus is often present.

Thermic Features.—Fever is a somewhat constant finding in blastomycosis, and probably depends upon infection with the streptococcus.

Chill.—Late during the disease, and when the patient has become markedly exhausted, he may suffer daily from a severe rigor, followed by profuse sweating.

Summary of Diagnosis.—The diagnosis is based on the detection of the blastomyces in the sputum.

Clinical Course.—Cutaneous blastomycosis may persist for a period of years, but the majority of cases run a slightly more rapid course after the lung becomes involved.

STREPTOTHRICOSIS.

Pathologic Definition.—An infectious disease caused by the presence of a streptothrix, and characterized by pulmonary consolidation, caseation, and cavity formation, with a tendency toward metastasis to other viscera and to the lymph-nodes.

Remarks.—The streptothrix is a vegetable organism, occupying a position intermediate between molds and bacteria, making its classification extremely difficult. Generally speaking, it forms an ill-defined genus of fungi, at the same time resembling more or less closely the bacteria, possessing features similar to both these types of vegetable organisms. Flexner has found that the streptothrix develops from spores into branching threads.

Varieties.—(*a*) **Acute streptothricosis,** (*b*) **subacute or chronic streptothricosis.**

Exciting and Predisposing Factors.—Infection with the streptothrix is essential to the development of the disease. Nothing is known concerning the conditions that promote infection of the respiratory tract with this microörganism.

* Jour. Amer. Med. Assoc., April 5, 1902.

Sex.—Males appear to be attacked about twice as often as females.

Age.—Streptothrix infection has been encountered in persons between the ages of twelve and seventy years, and most cases have been seen during the third and fourth decades.

Exposure to dust is believed to act as a predisposing factor; in Australia, it is said, streptothricosis often follows in the wake of sand-storms.

Principal Complaint.—The general clinical picture of pulmonary streptothricosis closely resembles that of pulmonary tuberculosis. The acute variety of the disease appears to be the more common form, the symptoms developing abruptly, and consisting of severe cough, free, and often bloody, expectoration, and pain in the chest. Weakness and emaciation soon set in, and if there is ulceration of the lung tissue, the symptoms of secondary infection appear, the patient's condition now being identical with that seen in tuberculosis. Anorexia is experienced early, and as the disease progresses profuse night-sweats occur.

Thermic Features.—After the lung tissue is broken down the morning temperature will be found to vary between 99° and 101° F., with an evening rise to from 102° to 104° F. This febrile expression of streptothricosis resembles closely that displayed by pulmonary tuberculosis with cavity formation, and is possibly dependent upon infection of the lung tissue with pus-producing organisms.

Physical Sign.—Inspection.—The respirations are seen to increase in frequency from day to day, and within the course of a few weeks expansion is diminished at one or at both apices. Emaciation becomes evident, the nostrils dilate, and cyanosis of the lips and finger-tips occurs, the last two symptoms being dependent on the degree of pulmonary involvement. In those cases in which the disease does not run a rapid course clubbing of the fingers is seen. The patient inclines toward the affected side, or may hold the chest firmly in one position, owing to an associated pleuritis.

Palpation.—The findings by this method will be found to vary in accordance with the pulmonary and pleuritic condition present. For example, in those cases showing an apical lesion without involvement of the pleura tactile fremitus will be increased over the lesion; if, however, pleurisy with serous or serofibrinous exudate into the pleural sacs should be present, fremitus is absent over those portions of the chest occupied by pleural fluid. (See Pleurisy, p. 145.) Rarely, both lungs may be affected, in which case the physical signs of pulmonary consolidation are bilateral.

Auscultation.—When there is pulmonary consolidation, the breath-sounds over the apices are intensified (bronchovesicular breathing), and may approach true bronchial breathing. Moist râles of a mucous character are audible over the upper portion of the affected lung, and may often be detected on the opposite side of the chest. When the pleura is first attacked by the inflammatory process, a friction murmur may be present, but after the fluid has accumulated in the pleura, the breath-sounds are absent over the area occupied by the exudate, and egophony is obtained at the point of junction of the fluid with the compressed lung. Distinct signs of cavity formation, as shown by auscultation, are seldom, if ever, obtained, yet, from a theoretic point of view they should be present whenever there is extensive excavation and the cavity communicates with a bronchus.

Laboratory Diagnosis.—At first the expectoration is moderate in quantity and serous in character, but later it becomes profuse, mucopurulent and purulent, and is of a yellow or greenish-yellow color. The sputum may contain elastic tissue and alveolar epithelial cells. In those cases in which

the onset is abrupt the sputum is bloody, and, indeed, hemoptysis may be one of the alarming symptoms of streptothricosis.

The streptothrix will be stained fairly well by the Gram method. A peculiarity of this organism is that the staining is irregular, and, therefore, some of the organs resemble cocci. The streptothrix is seldom, if ever, the only organism present in the sputum, the rule being to find streptococci, diplococci, and various other bacteria.

Blood.—We have but few records of complete blood examinations, and these, although not sufficient in number to permit of definite deductions being drawn, show the existence of a secondary anemia in which the leukocytes have been found to vary between 7400 and 13,600 in a cubic millimeter. The polymorphonuclear cells have been found to be normal or increased in number.

Summary of Diagnosis.—A diagnosis is possible only by detecting the streptothrix in the sputum.

THRUSH

(Mycotic Stomatitis; Fungous Stomatitis).

Pathologic Definition.—A parasitic disease of the mouth, due to the presence of the oïdium albicans. It is characterized by extensive ulceration, with the formation of whitish, soft, and lightly adherent flakes, and the presence of mycelial threads of the fungus in scrapings from the ulcerated surface.

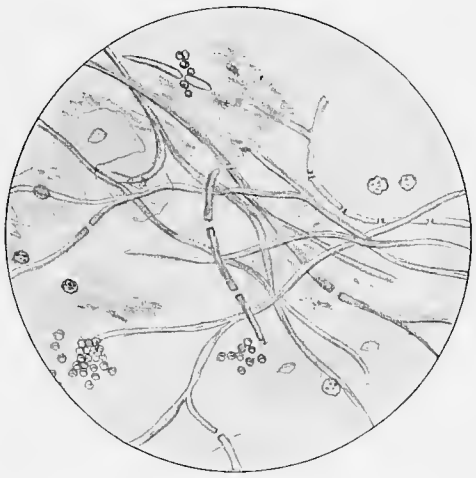


Fig. 330.—Thrush Fungus, Epithelial Cells, and Leukocytes from a Child Suffering from Ulcerative Stomatitis (Boston).

Exciting and Predisposing Factors.—The exciting factor is the presence, in the mouth, of the oïdium albicans, or thrush fungus. Children are more susceptible to the disease than are adults, although we have seen the disease in those at the extremes of life. Lowered vitality, such as is seen in marasmus, congenital syphilis, and poorly nourished children, is a predisposing factor, and the condition may also arise during the course of such chronic afebrile conditions as diabetes, nephritis, valvular heart disease, and the anemias. Thrush may also complicate febrile maladies, as, for example, pulmonary tuberculosis, chronic suppuration, and similar conditions.

Environment plays an important part as a predisposing factor, thrush being common in homes, asylums, and similar institutions. It is seen to develop in infants when the nursing-bottle and nipple are not properly cleansed after each feeding. Thrush is especially common in children having deformities of the mouth, *e. g.*, harelip and cleft-palate. It may be transmitted to the mouth from other portions of the body, as the foot or the vagina. We have seen two instances in which thrush developed a few days after the patient had been treating a domestic animal known to have an affection of the foot.

Principal Complaint.—The essential complaint is of a sore mouth, the tongue or the inner surface of the cheek being first affected. The extent of involvement of the mouth varies greatly, depending upon the stage of the disease and the resistance offered by the patient. Tenderness and pain develop early, and are excited by hot, cold, or solid foods. At first the patient's mouth is dry, and he experiences great difficulty in swallowing. When thrush complicates either chronic or acute conditions, it causes a variable degree of exaggeration of the symptoms belonging to the primary disease.

Physical Signs.—Inspection.—The oïdium albicans displays a special predilection to attack the flat epithelial cells, and seldom invades other epithelial tissues, although the deeper structures may be involved. The initial lesions appear upon the mucous membrane of the mouth or upon the tongue or the cheek, and consist of small, milk-white flakes dotted over the mucous surface. These flake-like particles can be removed from the mucous surface with ease, leaving an intact mucosa, or, if the process extends deeply and they are torn forcibly away, a bleeding, slightly ulcerated surface is presented. The number of milk-white patches disseminated over the buccal mucous membrane may vary from one-half dozen to several hundreds.

The mouth appears parched and dry, and the tongue heavily coated. In two cases under our personal observation the primary site of infection was the hard palate. Other observers have found thrush to involve the lips, tonsils, pharynx, and pillars of the fauces. Infection of the stomach will be considered in another chapter. (See p. 482.) Holt states that upon two occasions he has seen the ulceration of thrush extend to the esophagus, and in European countries involvement of the back of the throat and larynx appears to be more common and is seen far more often than in America.

Laboratory Diagnosis.—The saliva is, as a rule, highly acid in reaction, and it is to be remembered that the thrush fungus develops best in a faintly acid medium, although the oïdium albicans may grow in an alkaline medium. In a few cases of thrush infecting the mouth the saliva has been alkaline in reaction.

Microscopic Study.—This is best done by removing a portion of one of the white mycotic sloughs from the mucous surface, placing it upon the center of a microscopic slide, and then rubbing it out to a thin smear with a needle or with a match-stick. The unstained specimen will be found to contain leukocytes, an occasional red blood-cell, many pavement epithelial cells, spore-like bodies, and numerous filamentous and segmented mycelial threads. The mycelium may show branching at different portions; segmentation is quite distinct, and the entire mass is more or less completely embedded in granular débris. The shred and spore of the thrush fungus stain readily with ordinary aqueous basic dyes, *e. g.*, methylene-blue.

Summary of Diagnosis.—Although the disease presents apparent characteristic features, such as ulceration of the mucous membrane, a microscopic examination, with the detection of the thrush fungus, is necessary in order to form the diagnosis. The age of the patient, the early involvement of the lips and tongue, the presence of glandular involvement, and the dryness of the mouth are suggestive of mycotic stomatitis.

Differential Diagnosis.—Thrush must be distinguished carefully from aphthæ, and the following table (modified from Anders) shows the chief points of differentiation:

Thrush.	Aphthous Stomatitis.
1. Dryness of the mouth.	1. Salivation.
2. Whitish, raised spots or patches having no red areola; these are easily removed, causing no bleeding.	2. An ulcer with a yellowish-white, depressed base, surrounded by a red areola. The base is removed with difficulty by forceps, and bleeding results.
3. Spots are numerous.	3. Spots are usually few in number and discrete.
4. Begins in the form of minute spots.	4. Does not begin in the form of small spots, but ulcers appear, preceded by the formation of herpetic vesicles.
5. Ulcers are not painful. Discomfort depends upon the associated stomatitis.	5. Ulcers are exquisitely tender.
6. The characteristic thrush-fungus is always detectable with the microscope.	6. No specific microörganism has been found, though one is probably present.

Clinical Course.—Mycotic stomatitis cannot be regarded as a self-limited disease. In marantic children and in the aged the mycotic process has grave significance; it may extend to the throat and cause a variable degree of dyspnea, in which case the clinical course is quite unfavorable. The general symptoms accompanying thrush are indirectly caused by the interference with the taking of sufficient food. In favorable cases thrush yields to treatment in from a few days to two weeks, and it is unusual for the mycotic condition to continue for a longer period.

ASPERGILLOSIS OF THE LUNGS.

Pathologic Definition.—A chronic disease of the lung, caused by the aspergillus fumigatus, and characterized pathologically by consolidation with cavity-formation.

Remarks.—This form of mycotic destruction of lung tissue has been studied by both French and German clinicians. Virchow and his followers believe that the infection is a secondary one. The researches of Renon and of numerous other observers go far to support the belief that pulmonary aspergillosis may be a primary disease. Rothwell,* of Manchester, England, in 1899, wrote an elaborate monograph upon this subject, giving a complete history of the disease. Attention must here be called to the fact that this disease may affect other portions of the body, particularly the external auditory canal, the majority of such cases being due to the presence of the aspergillus. Mycotic disease of the cornea, although a rare condition, deserves mention in this connection. It may be caused by the aspergillus nigra, a fungus that is ordinarily non-pathogenic for man. Other types of aspergillus may also develop upon the cornea; on injecting an emulsion containing the aspergillus into the eye of an animal, corneal abscess ensues.

During the course of experimental aspergillosis Renon detected threads of this fungus in the urine of animals inoculated with the disease, and it was further found that within from twenty-four to forty-eight hours after inoculation the aspergillus fumigatus could be cultivated from the animal's urine, and, indeed, within this short time mycelia were often found. As the disease progressed, the aspergillus was almost constantly found in the urine of inoculated animals, and at postmortem the bladder or the kidneys of such animals were always found to contain fungoid lesions. As the result of his experiments Renon suggests that infection of the urinary tract probably

* Dissertation on Aspergillosis, Victoria University, 1899.

takes place by way of the venous blood-supply. Acute nephritis commonly develops during experimental aspergillosis.

Ravenel studied aspergillosis in cattle both clinically and pathologically, and we made a study of cultures of the aspergillus fumigatus obtained from this source, confirming many of the findings of Renon. Pigeons not infrequently show ulceration of the buccal cavity that is mycotic in nature, and although in the majority of cases the condition remains localized, mycotic pseudotuberculosis often follows.

Aspergillosis has been known to occur in connection with pulmonary tuberculosis.

Exciting and Predisposing Factors.—The disease occurs as the result of infection with the aspergillus fumigatus (Fig. 331), a fungus commonly developing upon grain. Its spores may resist exposure for an indefinite period.

It has not been definitely established whether pulmonary aspergillosis results from the inhalation of the fungus, or whether the parasite must be ingested in order to produce infection. In animals ulceration of the buccal cavity has been found to antedate pulmonary involvement, a feature that strongly suggests that the lung involvement is a secondary condition.

FIG. 331.—ASPERGILLUS FUMIGATUS, FROM THE LUNG OF A PARROT (Plaut).

Occupation is an important predisposing factor, men who handle grain being often affected, probably as the result of being brought into direct contact with the fungus. In France aspergillosis is chiefly seen to occur in feeders of pigeons, haircombers, and millers. This is due to the fact that the pigeon-feeder first takes the grain into his own mouth, masticates it thoroughly, and then forces it into the bird's throat. The hair-comber and miller become infected in a somewhat similar manner.

Symptomatology.—The symptomatology of aspergillosis at first resembles closely that of chronic pulmonary tuberculosis and also of actinomycosis (p. 920). After the consolidated areas break down, secondary infection is likely to occur, the fever that occurs being the result of infection with pathogenic bacteria.

Physical Signs.—The physical signs are those of pulmonary consolidation, with, later, destruction of the lung tissue. (See Actinomycosis, p. 920.) The signs of definite cavity-formation may at times be present.

Laboratory Diagnosis.—The sputum is negative while the consolidated portions of the lung have not broken down, but after ulceration takes place the sputum often becomes greenish or grayish brown in color, and when examined microscopically, will be found to contain mycelia and the various forms of aspergillus (Fig. 331). Specimens of sputum fixed and stained with a weak solution of safranin or a solution of thionin will clearly show the presence of the mycelial threads, the aspergillus staining readily with these dyes.

Cultural studies of the fungus must be made for purposes of identification. This fungus will be found to grow well upon potato and other solid and semisolid culture-media. The surface of the culture-medium is covered with a greenish mold that, when placed under the microscope, will be found to be composed of mycelial threads, some of which are expanded and contain many spores.

Summary of Diagnosis.—Practically speaking, the diagnosis rests upon two clinical points: (1) The physical signs of consolidation of one or of both lungs; and (2) the detection of the aspergillus in the sputum.

Clinical Course.—The disease is chronic in nature, and, as a rule, goes from bad to worse, pronounced cachexia, extreme prostration, and emaciation developing progressively. In rare cases the more acute symptoms may be arrested, the cough and expectoration become less marked, and the patient gradually improves, showing an increase in weight.

MYCETOMA.

Pathologic Definition.—An inflammatory process usually involving the foot, excited by different species of discomyces and aspergillus. The foot and leg are most often involved and the disease invariably follows

FIG. 332.—CONDITION OF DORSUM OF FOOT IN A CASE OF AMERICAN MYCETOMA (Dr. R. L. Sutton).

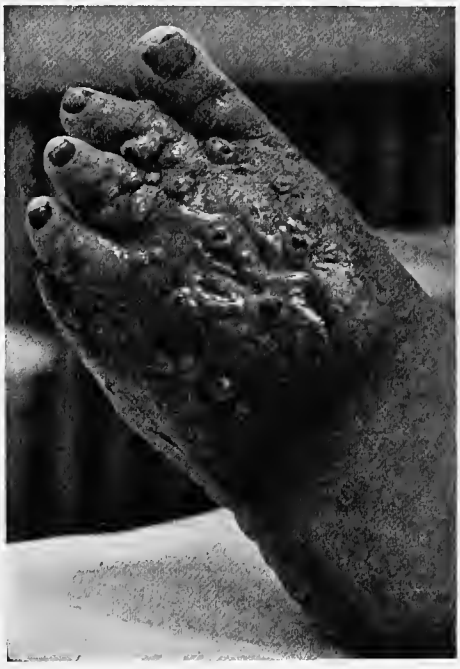


FIG. 333.—SOLE OF FOOT SHOWING SINUSES, SOME OF WHICH ARE COVERED BY HARD BROWNISH CRUSTS (Dr. R. L. Sutton).

traumatism. In rare instances mycetomatous ulceration may be found in other parts of the body, *e. g.*, shoulder, axilla, and scrotum. The foot is swollen, presents numerous small nodules (Fig. 332), which later develop small sinuses (Fig. 333), through which a peculiar viscid, yellowish, blackish, or reddish pus exudes. The discharge from mycetomatous lesions always

contains small granular bodies, which, when studied microscopically, display fungi.

Geographic Distribution.—Until recently madura-foot disease was decidedly rare in North America, and was generally conceded to be a disease of Asia. Within the past few years there have appeared in American literature many reports upon this disease, and certain of the patients so afflicted have been born in North America. Sutton,* in addition to an interesting report upon this subject, has given a complete bibliography of American mycetoma.

Clinical Course.—There is usually a history of a punctured wound, following which there are evidences of a subacute inflammatory process which continues for an indefinite period. There are seen small nodular areas, varying from the size of a pin's head to that of a pea, located over the inflamed area (Fig. 332). When the foot is involved, these nodules later develop sinuses that are seen on both the superior surfaces and the sole of the foot (Fig. 333). The viscid, syrupy, and slightly purulent discharge may at times be blood-streaked.

The small granules suspended in the discharge is a feature characteristic of madura-foot. After the disease has continued for a long period there is appreciable enlargement of the infected part, and should the foot be involved, there follows a well-marked atrophy of the muscle of the leg and hip. Mycetoma is to be distinguished from thrush and actinomycosis, and the most reliable distinctive features are obtained through a microscopic examination of the exudate

PROBABLE INFECTIOUS DISEASES.

MUSCULAR RHEUMATISM (MYALGIA).

General Remarks.—A painful disease of the muscles and of the structures to which they are attached, *e. g.*, the fasciæ and the periosteum. Muscular rheumatism is a common disease, probably of a general nature, but exhibiting local symptoms. The latter may be deep-seated, affect various parts of the body, and in this way give rise to a number of subvarieties. The condition may accompany either acute or chronic rheumatism, or may occur independently. It often follows joint rheumatism, and some authors believe that the affection is a neuralgia of the sensory nerves of the muscles.

The fibers of the affected muscles are swollen and undergo a more or less granular change. In long-standing cases, owing to trophic disturbance, the muscles become atrophied. Strauss found well-circumscribed nodules in the muscles.

The changes are essentially those of myositis, and in the acute form extensive round-cell infiltration of the connective tissue, with swelling and partial degeneration of the muscle-fibers, and the formation in them of vacuoles, often occur. In the chronic form there is a proliferation of the interfascicular tissue.

Clinical Varieties.—Lumbago (Myalgia Lumbalis).—This is by far the most common form of muscular rheumatism, and is believed to be a special type of myalgia. The onset is sudden, at times instantaneous, and the stitch-like pain in the back is excited by the slightest movement. The condition is exceedingly painful, and at times the muscles of the lower por-

* Jour. Amer. Med. Assoc., May 3, 1913, p. 1339.

tion of the back may be sensitive. Exacerbations of pain are likely to follow any change in the patient's position. One attack markedly predisposes to others.

The disease most often attacks men, and is unusually common in those subjected to heavy strain. Erben believes that the symptoms are caused by disease of the lumbar vertebræ, or that the condition is a neuralgia of the cutaneous nerves.

Torticollis (Myalgia Cervicalis; Wry-neck).—In this variety the muscles of one side of the neck are involved, and occasionally the muscles of the throat are implicated. Among the characteristic clinical features are the following: (*a*) The head is held in a fixed position, and the chin is directed toward the unaffected side; (*b*) the muscles of the affected side are contracted; (*c*) because of pain the patient makes little or no effort to turn the head, but, instead, turns his entire body.

This form of myalgia occurs more commonly in the young than after middle life, and is of short duration. One attack seems to predispose to subsequent ones.

Cephalodynia.—Under this head are included all types of rheumatism or myalgia of the muscles of the scalp and face. Cephalodynia is often localized to the frontal, temporal, or occipital muscles, or attacks a single group of muscles, although it may at times involve the entire scalp and one side of the face. Movement of the affected muscles gives rise to extreme pain.

Pleurodynia is a form of myalgia involving the intercostal muscles, and less frequently the pectoralis and serratus magnus muscles. Like torticollis, this is a unilateral affection, the left side being affected more often than the right. Movements of the chest and trunk give rise to some pain, whereas the respiratory movements, and particularly deep inspiration, are attended with agonizing pain. Heavy lifting, stretching, reaching, and turning of the trunk from side to side may also be painful. The acts of laughing, sneezing, and coughing are followed by lancinating pain involving the base of the affected side. Pleurodynia must be distinguished from pleurisy and from that type of myalgia following strain and traumatism to the chest muscles.

Myalgia of Special Groups of Muscles.—Under this head should be mentioned *omodynia*, or myalgia of the deltoid; *abdominal rheumatism*, a type involving the abdominal muscles; *rheumatic myositis*, a condition in which the muscles of the extremities are attacked; *myotonia* and *paramyoclonus multiplex* (see section on Nervous Diseases, p. 1169); *dorsodynia*, involvement of the muscles of the upper part of the back.

Predisposing and Exciting Factors.—Among the most important factors in the causation of the affection are: (1) The rheumatic diathesis; (2) heredity; (3) exposure to cold, damp, and strong air-currents, especially after heavy exercise or during free perspiration; (4) *sex*, the more frequent exposure of men while following their occupations making them more susceptible; (5) *age*—although it occurs at all ages, the acute and subacute varieties most frequently affect children and young adults, whereas the chronic form frequently attacks those beyond middle life; (6) previous attacks increase the susceptibility to subsequent seizures.

Principal Complaint.—There is usually a history of previous attacks and of an inherited tendency. (See Predisposing and Exciting Factors, above.) Obstinate constipation often precedes the attack. The classification of clinical types previously outlined shows the degree of limita-

tion to which the various forms of myalgia are subject. Pain of a sharp, lancinating, and more or less paroxysmal nature follows movement of the affected muscles. In certain cases the pain may be deep-seated, dull, more or less boring in character, and practically continuous. It is also influenced by climatic changes. Contraction of the affected muscles may prove an annoying symptom. Pain is at times relieved by making firm pressure over the involved muscle.

Thermic Features.—Leube, in a study of 200 cases, found fever to be a conspicuous feature in about 33.33 per cent. of cases, the temperature rarely exceeding 102° F. The fever is of short duration in typical cases, lasting one or two days.

Physical Examination.—This may reveal no positive findings, although in certain chronic cases distinct nodular hardenings of the affected muscles may be felt. Of Leube's cases, 16.66 per cent. displayed distinct cardiac murmurs.

Diagnosis and Differential Diagnosis.—Diagnosis is based on the etiologic influences and on the presence of pain, which is greatly increased by muscular contraction.

Muscular rheumatism differs from **neuralgia** in that there are no painful points, and in that firm pressure with the palm of the hand often affords relief.

Dermatomyositis must be distinguished from muscular rheumatism. Unverricht first differentiated between the two conditions by the presence of pain and swelling of the muscles in muscular rheumatism, dermatomyositis showing, in addition, redness (erythema) and hyperesthesia of the skin overlying the affected structures. Chief among the general symptoms of dermatomyositis are fever and physical prostration. The spleen is enlarged, and angina and hemorrhages have been observed. Unlike muscular rheumatism, dermatomyositis is more frequently seen in women.

Clinical Course and Duration.—This condition lasts from a few hours to several days, the attack usually terminating by the end of the first week. Muscular rheumatism may become chronic.

CHRONIC ARTICULAR RHEUMATISM.

Pathologic Definition.—A chronic disease of the joints characterized by the presence of inflammatory changes in the synovial membranes and inflammatory thickening of the articular and peri-articular structures (capsule, ligaments, tendon-sheaths, etc.). In some joints there may be erosions and adhesions, with loss of function and atrophic changes in the muscles.

Predisposing and Exciting Factors.—The probable causes of these atrophic changes have been pointed out in connection with muscular rheumatism. When the shoulder-joint is the seat of a chronic inflammation the muscular atrophy, affecting chiefly the deltoid, reaches its highest degree of development.

Age predisposes to this affection, the greatest number of cases being seen to occur during the fourth and fifth decades.

Sex exerts a slight predisposing influence, the disease being observed more frequently among females. Poverty, occupations that entail exposure to cold and dampness, and hereditary tendencies predispose to the disease.

Clinical Picture.—The involved joints may or may not show evidence of disease. The patient complains of pain, which eventually becomes

more severe at night, and is appreciably increased during cold and damp weather. It does not affect any particular joint or set of joints, but both large and small articular surfaces may be attacked, although the former, as a rule, suffer most. Occasionally acute or subacute attacks occur, one or more joints becoming swollen and slightly reddened. The affected joints are tender upon pressure, and their movements are appreciably restricted. A somewhat characteristic feature is that after a night's rest the affected joints are less painful and move more freely than they do after prolonged use. Chronic rheumatism is characterized by exacerbations that are followed by remissions. On placing the stethoscope over the affected joint, a distinct crepitation may be audible during movements. Partial dislocation and ankylosis occur late during the disease. In uncomplicated cases fever is absent, and prostration and emaciation do not appear until the patient has suffered severely for months or even years.

Clinical Course.—The disease rarely shortens life and tends to progress from bad to worse for a period of several years. A late feature may be lack of assimilation and exhaustion from pain.

Complications.—Chronic articular rheumatism is likely to be complicated by endocarditis, and, as a consequence, cardiac murmurs are often present.

MOUNTAIN FEVER (MOUNTAIN SICKNESS).

General Remarks.—The term "mountain fever" should be applied only to a condition produced by the action of rarefied air upon the organic functions. It has no definite pathology. Aron has shown that the intake of oxygen is diminished at high altitudes.

The *symptoms* are a markedly quickened pulse, urgent dyspnea, headache, vertigo, and, at times, nausea and vomiting. The temperature may reach 100° to 101° F. Malaise, inordinate thirst, and anorexia are present. Hemoptysis has occasionally been observed. The effect of high altitudes upon the human economy varies with the amount of reserve nerve-force the individual possesses.

The "mountain sickness" of the older writers is now almost universally conceded to be typhoid fever modified by the effects of extreme altitudes.

Laboratory Diagnosis.—The number of red and white blood-cells as well as the percentage of hemoglobin are influenced by altitude.

ROCKY MOUNTAIN FEVER.

Pathologic Definition.—An acute infectious disease prevailing during the summer months in certain portions of the Rocky Mountain district and the Bitter Root section of Montana. The disease also occurs in the mountainous districts of Nevada, Idaho, and Wyoming.

Exciting Factors.—The exciting cause of the disease is as yet unknown. Anderson and other observers regard it as due to infection with piroplasma hominis, a microörganism believed to be transmitted to man by the bite of ticks that are native to the particular district in which mountain spotted fever prevails. Later observations have failed to corroborate the assertions of Anderson concerning the existence of *piroplasma hominis*, but the work of Ricketts has definitely shown that the tick, *dermacentor occidentalis*, is the intermediate host of the parasite, whatever this may chance to be.

Predisposing Factors.—Climate figures prominently, mountain spotted fever not being observed south of 40 degrees nor north of 47 degrees

N. latitude. The disease prevails in epidemic form only at high altitudes (3000 to 4000 feet). Heineman and Moore have found that the horse is susceptible to the injected virus of this disease.

Season exercises a decided influence, the disease being prevalent only during the spring and early summer months.

Occupation, Sex, and Age.—Lumbermen, farmers, ranchers, and those following an outdoor life are especially likely to develop mountain spotted fever. Anderson, in an analysis of 121 reported cases, found 76 of them to have developed in males while 45 were in females. The disease is usually limited to early adult and middle life, most cases appearing between the ages of fifteen and fifty.

Principal Complaint.—There is a history of the bite from a tick, and one or more of these insects are found embedded in the skin of the patient. The disease is ushered in by a decided chill or series of chills, following which the temperature rises steadily, with slight morning remissions, for two or three days, when there is an intermission for about two days. A second febrile period then occurs. As many as seven febrile paroxysms occur with afebrile intervals before the disease terminates. In severe types of the affection the patient may complain of severe pain in the back and loins, intense soreness over the large muscles, and difficulty in moving the limbs. Toward the end of the first and during the second week of the disease epistaxis may be an annoying symptom. Nausea and vomiting are not uncommon, and persistent constipation is usually seen. The temperature in the febrile periods rises to 103° or 104° F., but the first paroxysm is usually more severe than the subsequent ones. In the fatal cases the temperature-curve shows no intermissions, but continues with fairly well-marked remissions until the end.

Physical Signs.—Inspection.—The tongue is heavily coated over the center and at the base, the edges and tip being intensely reddened. The conjunctivæ are markedly congested, and late in the disease may show a yellowish tinge. The eruption is characteristic, appearing on the third or fourth day following the chill, and seen first on the wrists and ankles, spreading to the arms and legs, and then over the greater portion of the body, involving the abdomen last. The spots first appear as a bright red macule, varying in size from that of the point of a pin to that of a pea. If hemorrhage into the skin occurs, the hemorrhagic areas may assume a bluish tinge. The petechial eruption begins to fade at about the sixth day of the disease, and in favorable cases has almost disappeared by the fourteenth day. The respiratory movements are greatly accelerated, and number between 25 and 60 a minute, becoming more frequent when an associated bronchitis develops.

Palpation.—The pulse is weak and rapid. The liver is not usually enlarged, but the spleen is always increased in size and is frequently tender.

Laboratory Diagnosis.—There are appreciable destructive changes in the red blood-cells, which are always reduced in number. The reduction in hemoglobin follows the red blood-corpuscle loss closely, and may fall to 70 or even to 50 per cent. There is no marked leukocytosis, but there is a slight increase in the number of large mononuclear cells.

Albuminuria develops during the height of the disease, and the evidence of acute nephritis may be present.

Differential Diagnosis.—Rocky Mountain spotted fever must be distinguished from those diseases in which there is a purpuric eruption. It is differentiated from **pyemia** (with purpura) by the fact that in Rocky

Mountain fever cultures from the venous blood do not show the presence of pathogenic bacteria. Rocky Mountain fever is differentiated from epidemic meningitis by the fact that in the former condition an examination of the cerebrospinal fluid gives negative results.

Clinical Course.—In approximately 10 per cent. of all cases the disease reaches its height by from the eighth to the tenth day, after which the fever declines and there is an improvement in all the general symptoms. Convalescence is well established in from the fourteenth to the eighteenth day. In the majority of cases, however, the disease progresses from bad to worse until the fourteenth day, when complications, such as nephritis, bronchopneumonia, and cardiac failure, ensue. In Montana the death-rate is as high as 30 per cent., but in Wyoming and Idaho it is only 2 or 3 per cent.

MILK-SICKNESS

("TREMBLES").

Pathologic Definition.—A disease occurring in man and in the lower animals. When it occurs in the latter, it is known as "trembles." The disease is unknown east of the Alleghany Mountains, but formerly prevailed in many of the western and southwestern States as the result of denudation of the forests and of advancing cultivation.

Predisposing Factors.—It has been proved that many cases of milk-sickness were communicated to man by the use of milk and its products, or by the ingestion of meat obtained from cattle suffering from "trembles." Jones, among other writers, believes the disease to have been transmitted to man by the use of vegetables and of drinking-water. Cattle have become infected as the result of drinking water flowing from a certain spring.

Season.—The disease generally appears during the spring and autumn months, although it may occur at any season. A single attack does not bestow immunity, but, on the contrary, predisposes to subsequent attacks. A. C. Crawford* has compiled valuable data concerning the disease, and discussed at length the relation believed to exist between it and white snakeroot.

Incubation Period.—This varies greatly, but in the majority of instances it is said to be between two and ten days.

Symptoms.—Such prodromata as anorexia, languor, headache, and fatigue may occur. Later nausea and violent vomiting develop, and vomiting of blood has been noted in certain cases. Obstinate constipation is the rule. There is pain in the abdomen, and as the disease progresses the pain may be referred to various portions of the body. A peculiar form of dyspnea may be present, and hiccough and difficulty in swallowing are occasionally seen. Inordinate thirst is the rule, and the patient's breath gives off a fetid odor that is said to be highly characteristic of the malady.

Nervous Features.—When the patient is directed to protrude his tongue, this organ is seen to be unusually large, and affected by decided tremor. Restlessness, followed by apathy and mental dullness, is the rule, and in severe cases the condition may go on to stupor and even to coma. The typhoid state and convulsive seizures are occasionally observed.

Thermic Features.—The temperature is, as a rule, normal or subnormal, and, according to Kimmell, rarely exceeds 99° F.

Physical Signs.—In mild cases the pulse remains nearly normal, but in severe cases it becomes accelerated and fluctuates in direct proportion to

* U. S. Department of Agriculture, Bulletin No. 121, part I.

the degree of prostration present. The abdomen is at first scaphoid, and in severe types of the disease tympanites occurs later.

Laboratory Diagnosis.—The vomitus shows nothing characteristic, and is occasionally found to contain blood. The urinary secretion is decidedly diminished in quantity.

Course.—In mild cases the patient recovers within a few days, but in the more severe types the symptoms may continue for from twelve to twenty days. Yandell and Kimmell have observed cases in which the disease ran a chronic course.

MILIARY FEVER

(SWEATING DISEASE).

Pathologic Definition.—An infectious disease, characterized by the occurrence of copious sweats and the presence of a vesicular (miliary) eruption. It has prevailed epidemically in England, Italy, Germany, and France in 1887, and in Austria in 1893. The severity of the disease is shown from a statistical report of Schaffer, in which, of a total population of 5097 persons, 158 were shown to have developed miliary fever, the disease attacking 31 adults, 17 of whom were men, and 128 children.

Predisposing Factors.—Ordinarily, women between twenty and forty years of age are attacked more often than are men, and, according to Schaffer, childhood is a marked predisposing factor.

Clinical Picture.—The disease is marked by fever, accompanied by epigastric oppression and sweating, which symptoms continue until the third or fourth day, when an eruption occurs, first appearing in the form of a mild irritation of the skin, but later developing into vesicles that soon rupture; within the course of forty-eight hours following their appearance there is a decidedly scaly desquamation. Hemorrhages may occur and may precede general collapse. Relapses are not uncommon.

Nervous Manifestations.—In severe cases there are restlessness and delirium.

Diagnosis.—This is based largely upon the following characteristics: Fever, and the typical cutaneous manifestations that appear on the third or fourth day, together with the tendency toward the occurrence of relapses. The fact that the disease is almost always limited to a comparatively small area is also worthy of consideration.

FOOT-AND-MOUTH DISEASE

(EPIDEMIC STOMATITIS; APHTHOUS FEVER).

Pathologic Definition.—An acute infection attacking cattle, sheep, swine, and goats, and rarely transmitted to man. Klein has described a special micrococcus that he recovered from the lesions.

Predisposing Factors.—The prevalence of an epidemic among the lower animals appears to be the leading predisposing factor when the disease is transmitted to man, and it is then known as epidemic stomatitis. In the fall of 1908 several cases of foot-and-mouth disease developed in cattle, sheep, and swine in the States of New York and Pennsylvania, and so widespread was this epidemic, that hundreds of animals had to be destroyed. There is no record of the disease being transmitted to man during this epidemic.

Incubation Period.—This is from three to five days.

Principal Complaint.—A chill or a series of chills usually marks the onset, following which the patient complains of malaise and of symptoms referable to the mouth, resembling those of aphthous stomatitis. (See p. 395.) The vesicles first appear upon the tongue and mucous surface of the mouth, extending to the lips. The temperature of the mouth is increased, the mucous membrane is greatly reddened and edematous, and salivation occurs. The oral vesicles may go on to pustule formation, and may extend to the face, and occasionally to other portions of the body. A tendency toward hemorrhage has been observed in certain epidemics.

Summary of Diagnosis.—A history of the disease in lower animals, together with the characteristic eruption of the mouth, which is also transmitted to the extremities (fingers), are the leading features of the disease.

Clinical Course.—This is usually mild, extending over a period of approximately one week.

GLANDULAR FEVER.

Pathologic Definition.—An acute infectious disease of children, characterized by an adenitis affecting the lymph-nodes of the neck.

Etiology.—This disease was first described by Filatow, of Moscow, Donkin, Fischer, and Dawson Williams, of England. J. Park West has also given an excellent description of the disease. It usually occurs in the form of house epidemics. West, of Ohio, described a wide-spread epidemic in which 96 cases occurred in 43 families.

Age.—The disease usually occurs during childhood, although cases occurring in adults have been reported.

Season.—Most of the cases are seen between the months of October and May.

Incubation Period.—This is from five to eight days.

Clinical Picture.—The onset is sudden. Because of pain on movement the child holds its neck in one position. There are anorexia, nausea, occasional vomiting, and often abdominal pain. The tonsils are enlarged, and in some cases there is injection of the pharyngeal mucosa. Mental hebetude and delirium are occasionally seen. Glandular enlargement appears on the second or third day, and generally attacks the left side first, the opposite side of the neck becoming enlarged a few days later. The glands vary in size from that of a bean to that of a hen's egg, and are painful. The axillary and inguinal glands may also be enlarged. Cough and dyspnea may occur, as the result of involvement of the bronchial and tracheal glands. Splenic enlargement occurs in 50 per cent. of the cases, and in almost all the liver is increased in size.

Thermic Features.—The temperature ranges between 101° and 103° F.

Clinical Course and Complications.—The average duration is sixteen days. Among the common complications are hemorrhagic nephritis, bronchitis, and otitis media.

INFECTIOUS JAUNDICE

(ACUTE FEBRILE JAUNDICE; FIEDLER'S DISEASE; WEIL'S DISEASE; EPIDEMIC CATARRHAL JAUNDICE).

Pathologic Definition.—An acute infectious disease, characterized by jaundice, wasting, moderate enlargement and cloudy swelling of the liver and spleen, the former occasionally showing small foci of fatty

degeneration. The kidneys are also the seat of a diffuse tubular nephritis, and hemorrhages into the serous sacs and the spleen have been observed. Infectious jaundice comprises a group of conditions intermediate between catarrhal jaundice and grave destructive jaundice (acute yellow atrophy), all grades of transition toward these extremes being observed.

Remarks.—There are several more or less distinct types of acute infectious jaundice, all of which are characterized by sudden onset with fever, gastro-intestinal symptoms, and jaundice. Some of the cases exhibit enlargement and tenderness of the liver and spleen, as well as the clinical manifestations of nephritis. *Nervous symptoms* are prominent in some epidemics, whereas in others in which the disease is of milder type, the nervous features are not pronounced. As early as 1866 French observers described both isolated cases and epidemics, but it was not until 1886 that Weil gave a detailed report of epidemic jaundice. "Many English and French authors, however, demur to the designation of Weil's disease as separate from other types of benign infectious jaundice" (Osler).

Exciting and Predisposing Factors.—The exciting cause of epidemic jaundice remains in doubt, in spite of many laboratory attempts to isolate a specific microörganism. Anders believes that the condition may be an acute febrile jaundice of varied etiology. Infectious jaundice occurs most often in those between twenty and forty years of age. **Butchers** have been found to suffer from the disease more often than those engaged in other occupations, and, judging from the recorded cases, men are more often attacked than women. **Season** is believed to exert a slight influence, the majority of cases developing during the summer months.

General Complaint and Characteristic Signs.—The onset is usually sudden, and may be fulminating, although such prodromal symptoms as lassitude, headache, and anorexia are occasionally encountered. The disease is generally ushered in with a chill, followed by fever, which rises quickly to 103° or 104° F., and is of the remittent type, continuing from ten to fourteen days, and terminating by lysis. Headache, vertigo, nausea, vomiting, and at times diarrhea are present. Jaundice usually appears in from the second to the fourth day, and may be slight or intense. If the disease is due to obstruction, the stools may be clay-colored, showing the absence of bile. The liver and spleen are often enlarged, and the latter may be tender on pressure. In grave cases cerebral symptoms, such as delirium, convulsions, and coma, may rarely occur. Herpes, diffuse or macular erythema, and urticaria are occasionally seen.

In certain cases hemorrhages may occur—*e. g.*, epistaxis, hemoptysis, petechial eruptions, and bleeding into the serous cavities and from the intestine.

Laboratory Diagnosis.—The urine is high-colored, bile-stained, and shows the presence of albumin, casts, and sometimes of blood.

Diagnosis.—This is based on the acute onset, fever, pains in the muscles, joints, and epigastrium, nephritis, icterus, a tendency toward hemorrhages, and the frequent occurrence of relapses.

Clinical Course.—The prognosis, both as to life and recovery, is good. W. E. Hughes, notwithstanding, records two cases that proved fatal within forty-eight hours of the onset.

In cases of average severity the temperature shows a tendency to decline in from the fourth to the ninth days, and reaches the normal about five days later. Muscle pains, however, disappear slowly, and may be present even when convalescence is apparently well established. The nervous symptoms,

the enlargement of the liver and spleen, and the evidences of nephritis gradually subside. There is usually a marked loss in weight, and convalescence is somewhat protracted. Relapses occur in approximately 40 per cent. of all cases, and are prone to appear in from three to eight days after the temperature has reached the normal. The period of each relapse is ordinarily shorter than that of the initial attack.

AUTUMNAL CATARRH

(HAY-FEVER; HAY ASTHMA).

Pathologic Definition.—A disease of doubtful origin, in which many predisposing factors are concerned, and which is characterized pathologically by congestion of the nasal mucosa, with overactivity of the glands. Congestion may extend from the nasal mucous membrane to the conjunctivæ, the pharynx, and the larynx, and in some instances to the bronchi. The inflammatory process frequently extends along the Eustachian tube, and hyperemia of the middle ear may follow.

Predisposing and Exciting Factors.—Age and Sex.—Age is a prominent predisposing factor, since practically one-third of all cases develop before the twentieth year. Males are affected more often than females. Nasal polypi, spurs, defective septa, and other abnormalities of the nasal mucous surface may serve as predisposing factors in some cases. **Reflex irritation** is also known to figure in the production of this disease, and **heredity** is a potent factor.

Season acts as the most important predisposing factor, since in the United States the majority of cases are seen during the months of August and September and in the early part of October. A limited number of cases are also encountered during the months of May and June—the so-called "rose fever."

Exposure to the pollen of certain plants may excite the disease in those who have previously been free from it. But dust of any kind will bring on a paroxysm. Attacks of sneezing are more likely to develop during the middle of the day and when the sun is hot, than during the early morning and evening hours. The **application of local treatment** to the nasal mucous membrane is, in many instances, sufficient to precipitate an attack of hay-fever, and such an attack may continue for weeks. **Walking against a strong wind** and riding upon both steam and electric cars intensify the symptoms in those suffering from the disease, and are often sufficient to precipitate an attack in persons previously healthy. There are certain cases in which the patient suffers more or less from hay-fever during the entire year, but the vast majority of all cases are free from the disease from the first appearance of frost (October or November) until May or June of the following year. The patient frequently states that he is positive there has been a frost in the immediate vicinity, although he has not been out of the house, and may have no other evidence except that his breathing is improved.

Change of location of patients who are already suffering from an attack may, in certain instances, intensify the symptoms, whereas in others they may become ameliorated. Patients seldom suffer from the disease when on the high seas, whereas the symptoms usually develop promptly when such individuals reach the land; the disease is extremely uncommon at an elevation of from 4000 to 6000 feet.

Odors.—The odor of certain plants, ammonia, and other gases may not

only predispose to, but appears to excite, an attack, and hay-fever may also follow great excitement and the inhalation of inorganic and organic dusts.

Prodromal Symptoms.—These are common to the majority of all patients, and in typical cases consist of the following: For several days prior to the initial attack of paroxysmal sneezing a variable degree of constipation occurs, which may be obstinate in some cases; undue itching of the skin, and particularly of the scalp, may also be present, and at the same time there is likely to be itching of the eyelids and of the nose; vague pains and soreness of the muscles upon movement are by no means unusual, although these are of a mild nature. Upon rising after a night's rest certain of the joints feel stiff, a condition that more commonly involves the ankles and feet. The mental condition is somewhat sluggish, and victims of the disease find it impossible to concentrate their mental faculties both immediately before and during an attack of hay-fever. Drowsiness is often present during the day, and in severe cases the patient may sleep for hours during the afternoon, and still secure restful sleep at night.

Varieties.—(1) At times pathologic lesions of the upper air-passages are to be found, such as a deflected nasal septum, disease of the turbinated bones, disease of the sinuses that communicate with the nasal fossæ, and the like.

(2) A somewhat larger class includes those cases in which no disease of the respiratory tract is detectable, and still, for some unknown reason, these patients develop typical attacks of hay-fever during the autumn months, and some have two or more attacks (one in May or June—rose fever) during each year.

(3) There is a special class of sufferers from this disease in whom there is a hypersensitiveness of the nasal mucous membrane for certain odors or for dust. Such patients may develop an attack of hay-fever whenever they are exposed to the particular excitant for which they possess an idiosyncrasy.

Principal Complaint.—A history of previous attacks is usually obtainable in all but that small proportion of cases in which the patient consults his physician during the primary attack. Most patients assert that they have had similar mild attacks at intervals during the past few years, but that they always regarded such attacks as acute "colds." These early mild attacks of hay-fever may last for but from twenty-four to forty-eight hours, during which time the patient sneezes frequently and suffers from increased lacrimation and a copious discharge from the nose.

The symptoms of hay-fever may be divided into two great clinical classes: (1) *Local;* (2) *general.*

(1) **Local Symptoms.**—Following the prodromal stage, the onset of the disease is abrupt, and the attacks tend to return annually at approximately the same day; the invasion is ushered in by a paroxysmal attack of sneezing, which is accompanied by the other symptoms of severe coryza, such as temporary obstruction of the nasal passages and profuse rhinorrhea, the discharge being thin and of the consistence of water, although in some cases the nasal secretion may be mucopurulent. The conjunctivæ are greatly congested, and the patient experiences intense itching of the eyelids, conjunctivæ, and tip of the nose. In typical cases of hay-fever the patient will be seen to be continually rubbing the tip of the nose; this he does unconsciously, while at the same time he may irritate his scalp by scratching; with the onset of the disease there is more or less itching over the entire body. As the disease progresses paroxysmal attacks of sneezing and coryza become more and more frequent, and occur at intervals of from a few hours to a few minutes. In severe cases, during a paroxysm the patient will continue

to sneeze every few seconds for from two minutes to one-half hour, and after he has sneezed from ten to forty times, his body becomes immediately covered with beads of perspiration, and, indeed, this sweating may be so profuse as to saturate his linen. The more profuse the perspiration at these paroxysms, the more profound is the prostration following each of them. In nearly every case of hay-fever a peculiar scaly exudate develops upon the scalp during the attack. In many patients, however, the scalp remains unusually clean.

Paroxysm.—As a precursor of each paroxysm, both nostrils appear to be more or less completely closed as the result of edema or of swelling of the nasal mucosa. Following this difficulty in breathing the following symptoms occur:

(*a*) The patient experiences a peculiar tingling sensation of the palate and the tip of the nose, and within the course of a few seconds this sensation is communicated to the nasal cavity, and especially to the region of the turbinated bones.

(*b*) The patient sneezes violently several times, following which there are profuse discharges from the conjunctivæ and the nose. Attacks are commonly provoked by irritation of the nose or by rubbing of the eyes. Cleansing of the nose serves as one of the commonest causes for a precipitation of these attacks, and we have found that in the majority of cases local treatment renders the attacks more frequent except in those instances in which cocain or adrenalin chlorid is employed, and even here violent paroxysms commonly occur between the treatments.

Without apparent cause the local symptoms become appreciably ameliorated for periods of one or more days, but following such amelioration there is likely to be a temporary exacerbation in both the local and the general symptoms. Exacerbations are frequently attributed to exposure to the air, to the inhalation of dust, and to climatic changes, paroxysms being more common during those hours of the day when the sun is brightest, and appreciably more frequent when there is a strong breeze. In other words, the pleasanter the day, the more does the victim of hay-fever suffer.

(2) **General Symptoms.**—These are, as a rule, mild, and consist of chilly sensations, alternating with slight flushings of the face and a sense of feverishness; there are also lassitude and a moderate degree of anorexia; insomnia may be present, and results directly from interference with respiration during sleep.

Late during the course of the disease the catarrhal process may invade the larynx, and even the bronchi, as previously stated (see Pathologic Definition, p. 939), and in consequence of congestion of the mucous surface of these organs, the patient is annoyed by cough, and attacks of asthma (see Bronchial Asthma, p. 96) may be experienced.

Laboratory Diagnosis.—During the prodromal stage, in special cases, the quantity of urine voided during the twenty-four hours is appreciably diminished, but at or near the time of the initial paroxysm the patient may void an unusually large quantity of pale urine of low specific gravity. Throughout the course of hay-fever the quantity of urine voided during the twenty-four hours bears a more or less close relation to the severity of the paroxysmal attacks and the degree of sweating.

The nasal secretion contains a few epithelial cells, leukocytes, some bacteria, and occasionally red blood-cells.

Summary of Diagnosis.—This depends upon the following: (*a*) A history of previous attacks of either autumnal or rose fever. (*b*) The

characteristic paroxysms, accompanied by profuse sweating and prostration. (*c*) Congestion and itching of the conjunctivæ and nasal mucosa. (*d*) Cutaneous itching, particularly of the scalp; (*e*) chilliness and feverish sensations, together with the characteristic sensation at the palate and nose.

Clinical Course.—In typical cases of autumnal catarrh this lasts for from four to six or even eight weeks, but those who develop an attack during the spring months (rose fever) are seldom annoyed for more than two weeks. In certain years sufferers from attacks of autumnal catarrh experience immediate relief after the first frost, but occasionally such relief is felt through portions of the country in which frost has not yet been seen. Many cases end abruptly as soon as the patient is at sea. Change of climate may also be followed by an almost immediate disappearance of the annoying symptoms. A change of location from the city or country to a mountainous section containing dense forests is commonly followed by relief. A sea-voyage may give similar relief. Not infrequently a patient going from America to Europe will not suffer from the disease while in Europe, even though he reside in a climate in which hay-fever is common.

HEMORRHAGIC DISEASES OF THE NEW-BORN.

EPIDEMIC HEMOGLOBINURIA

(WINCKEL'S DISEASE).

Pathologic Definition.—An affection, probably septic in nature, occasionally seen in lying-in hospitals, and occurring in infants in from one to ten days after birth.

General Features.—The infants refuse the breast and exhibit hematogenous jaundice. Gastro-enteric catarrh is present, and hemorrhages occur into the viscera and into the mucous membranes. Mild fever, rapid emaciation, and convulsive seizures are present.

Laboratory Diagnosis.—The stools contain meconium; the urine is scanty, dark colored (from methemoglobin), often albuminous, and may contain casts. Kilham and Mercelis, of New York, isolated a diplococcus from 10 cases, but, in all probability, this organism is not the true cause of the disease.

ACUTE FATTY DEGENERATION OF THE NEW-BORN

(BUHL'S DISEASE).

General Remarks.—This disease is probably similar to Winckel's in nature. It was first described by Hecker and Buhl as an infectious disease of the new-born.

Clinical Characteristics.—There are cyanosis, jaundice, and profuse visceral hemorrhages. The chief pathologic change is an acute fatty degeneration of the viscera.

MORBUS MACULOSUS NEONATORUM.

General Remarks.—In this affection there is hemorrhage from the gastro-intestinal mucosa of the new-born (melæna neonatorum), due probably to intracranial lesions the result of pressure received during birth, although the condition may take place independently of the latter. Preuschen

collected the reports of 37 cases, in 5 of which the brain was examined, and in all of these cerebral hemorrhage was found. Townsend found 459 cases among the records of 6700 births. Gärtner believes the disease to be of infectious origin, and asserts that in two cases he was able to identify a bacillus. Hemorrhage may also take place from the mouth, nose, umbilicus, etc. General rather than local bleeding is the rule.

Clinical Course.—Hemorrhage is usually marked during the first week of life, and continues for from a day to a week, at the end of which time practically 50 per cent. of cases enter upon a stage of convalescence and go on to recovery.

HISTOPLASMOSIS.

Remarks.—A fatal disease somewhat resembling kala-azar, due apparently to an organism similar to Leishmania donovani, which has been observed in persons in the canal zone, Panama.

Among the characteristic features are splenomegaly, irregular remittent fever, leukopenia, and emaciation. Glandular enlargement, tenderness over the spleen and liver, and absence of the patellar reflexes were also observed. Thus far only three cases have been reported.* The parasite recovered from the splenic tissue resembles in some respects the Leishman-Donovan body, but is regarded by Darling and others as being distinguishable from the last-named parasite. Darling suggests for it the name histoplasma capsulatum.

Laboratory Diagnosis.—According to Darling's observations, oval and round bodies were found free in the blood-plasma at autopsy, and were also present in smears made from the marrow of the ribs and from the spleen.

SUNSTROKE.

Pathologic Definition.—Following undue exposure to heat, the blood is fluid, dark in color, and there is degeneration of the red cells and an absence of Rouleaux formation. Parenchymatous degeneration of the kidneys and liver and of the whole neural axis may be present. The cerebrospinal fluid is albuminous, occasionally blood-stained and under increased tension.

Predisposing and Exciting Factors.—Practically, anything that lessens bodily resistance to external high heat predisposes to heat-stroke. Thus, privation, unsanitary surroundings, fatigue of body or mind, emotional excitement, worry, overeating, indulgence in alcoholics, and previous attacks of sunstroke are conducive to heat-stroke on exposure to high temperature.

Heat-stroke and thermic fever are terms more appropriately applied to those similarly affected in midsummer while working in places not exposed to the sun, but yet close, confined, and excessively hot.

Heat-exhaustion (prostratio thermica) is caused under similar conditions as the preceding, but manifests dissimilar effects. The majority of the cases of sunstroke occur between 2 and 5 P. M., although heat-stroke and heat-exhaustion may occur at night as late as 10 or 11 P. M.

Clinical Varieties.—Two forms of heat- or sunstroke are common: (1) The asphyxial or apoplectic form; (2) the hyperpyrexial form. The majority of the cases of sunstroke are possibly combinations of apoplexy and exhaustion. "Valin puts all cases of insolation into two classes: the first,

* Samuel T. Darling, M. D., Archives of Internal Medicine, September, 1908.

sthenic or asphyxial, corresponding to our hyperpyrexial or congestive variety; the second, asthenic or syncopal, corresponding to our heat-exhaustion. Mixed forms may occur quite frequently, the most prominent symptoms being referable to the organs suffering the most, as the cerebro-spinal system, heart, lungs" (Anders).

General Complaint.—There may be sudden premonitions or dizziness, chromatopsia, throbbing headache, cessation of sweating, or dyspnea. The patient while working in the sun may suddenly fall unconscious, convulsions may occur, and in this state he may die from cardiac failure. More often unconsciousness is not so profound, but there is much restless-

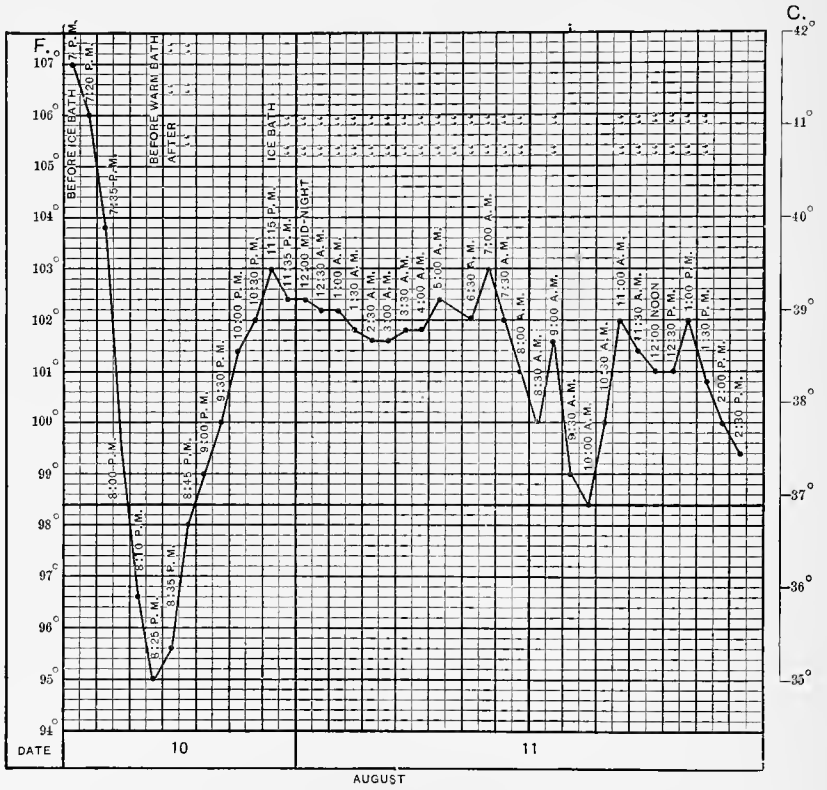


FIG. 334.—CHART OF A CASE OF SUNSTROKE. (C. B., aged twenty-nine years. Recovery.) (J. M. Anders.)

ness, and epigastric "cramp" may be present. Also a sense of thoracic oppression, and occasionally there are nausea and vomiting.

There are often prodromes as cramp-like pains on the abdomen, blurring of vision, mental hebetude, anorexia, intense headache, irritability of the bladder, general nervousness, and progressive weakness.

Thermic Features.—These fluctuate within wide limits, for example, the temperature may be subnormal in certain cases, while in others it may reach 101° to 102° F. Again, the mouth or rectal temperature may be found to register 104° to 106° F. In the hyperpyrexial variety high temperature is practically always observed, and may fluctuate between 106°

to 112° F., and there are on record many instances where a much higher temperature has been observed. (See Fig. 334.)

Physical Signs.—General.—In ordinarily severe types of heat-exhaustion the face is flushed, the vessels of the neck are seen to pulsate, respirations are labored, and the skin is hot, dry, and may display minute hemorrhages. At times the skin is clammy, and there is also present extreme cyanosis. In the more severe type the general appearance of the patient is that seen in profound exhaustion and in coma. The patient may be restless, and occasionally delirious. The movements of the chest are increased in frequency, varying between 25 and 50 per minute.

Local Examination.—Inspection.—The tongue is usually coated with a heavy whitish fur. The eyes are suffused, the pupils pin-point, and there may be a rather fixed stare. In the nervous types there is picking at the bed-clothes.

Palpation.—The skin is at first hot and dry, or may later be cold and clammy. The pulse may vary between 90 and 160 beats per minute. Its force, frequency, and general characteristics correspond more or less closely to the degree of temperature, becoming weak, dicrotic, and irregular in severe cases.

Laboratory Diagnosis.—The blood is decidedly fluid and shows no tendency to Rouleaux formation; and in cases displaying cyanosis it is dark in color. In mild cases there is a tendency to frequent urination, with the passing of the urine of normal color. In the more severe forms of heat-exhaustion the urine is scanty and albuminous. The cerebrospinal fluid is found upon aspiration of the spinal canal to be under unusually high pressure. The spinal fluid is often blood tinged or slightly turbid, and contains an abnormal number of cellular elements.

ANIMAL PARASITIC DISEASES.

Man is subject to diseases that are caused by animal parasites that belong to the following subclasses: Protozoa, Vermes, and Arthropoda. Among the parasites belonging to the class Protozoa we find members of the orders Rhizopoda, Sporozoa, Flagellata, and Ciliata. Among the parasites belonging to the class Vermes we find members of the orders Nematoda, Cestoda, and Trematoda. Of the parasites belonging to the class Arthropoda we find members of the orders Diptera, Hemiptera, and Acarina. Some of these parasites live on the surface of the body, and are therefore called *ectoparasites;* others develop within the tissues, and are therefore called *endoparasites.* In order to follow the zoölogic classification the diseases produced by these parasites should be considered in order from lowest to highest position of the causative factor in the zoölogic scale. There are certain considerations that make such a method of discussion inadvisable, and for this reason the diseases produced by animal parasites are considered in order of their frequency and importance in human pathology.

PROTOZOAN DISEASE.

MALARIA.

Pathologic Definition.—An acute infectious disease caused by a member of the genus *plasmodium,* transmitted to man by the bites of infected mosquitos, and characterized by destruction of the red blood-cells and the deposition of pigment in the organs, notably the spleen, the liver, and the bone-marrow. In some varieties of the infection the blood-vessels of the brain and of other organs may become plugged with sporulating parasites.

Remarks.—Clinically, the important features of the symptom-complex of malaria are the periodic occurrence of paroxysms of chill, fever, and sweating, which are followed by prostration, headache, drowsiness, and sleep. The presence of one of the species of plasmodium in the peripheral blood or in blood obtained by splenic puncture is requisite for the establishment of a diagnosis. Extreme prostration, emaciation, pallor, secondary anemia, nervous symptoms, hemoglobinuria, hematuria, hemorrhages from the mucous membranes, and purpura may supervene.

Exciting and Predisposing Factors.—(1) The exciting factor of malarial fever is a species of the plasmodium that is transmitted to man by the bites of infected female mosquitos of the family Anophelinæ. The predisposing factors to this infection are, therefore, conditions favorable to the development of the variety of mosquito known to convey the disease. A warm climate with a high degree of humidity and heavy rainfall, so that pools of stagnant or slowly flowing water are numerous, with thick vegetation,

favor the propagation of mosquitos. It has been repeatedly shown that highly malarial districts may be rendered non-malarial by draining pools of stagnant water, and quickening the flow of streams by clearing and deepening their channels.

Race appears to exercise little influence, although in the United States the full-blooded Negro is slightly less susceptible to malarial infection than other races.

Sex exerts no influence when men and women are equally exposed, although males who follow certain outdoor occupations are especially prone to become infected. Children are likely to develop the disease when exposed. A natural immunity is occasionally observed, but persons who cannot be infected with malaria are rarely seen.

Age.—Malaria may occur at any age, but young men are affected oftener than persons at the extremes of life.

Incubation.—The period of incubation varies slightly in different types of the malarial infection, and at times there is a slight variation in the same type of parasite. Bignami and Bastianelli give the incubation period of benign tertian malaria as fifteen days; of estivo-autumnal tertian, as five days. Celli reports cases in which the period of incubation of the tertian parasite was twenty-two days, and of the estivo-autumnal parasite, seventeen days.

Clinical Classification.—Clinically, four varieties of malaria are recognized: (1) Benign tertian malaria, characterized by the occurrence of a paroxysm every other day. (2) Quartan malaria, in which a paroxysm occurs every third day. (3) Estivo-autumnal malaria, characterized by paroxysms that occur at irregular intervals. (4) Chronic malarial cachexia. Other classifications of malaria have been made, based on the type of the temperature-curve or other clinical manifestations, as intermittent fever, remittent fever, pernicious malaria, malarial cachexia, malarial hematuria, malarial hemoglobinuria, latent malaria, and recurrent malaria.

Explanation.—The benign *tertian parasite* (*plasmodium vivax*) requires forty-eight hours for its endogenous cycle of development; as a consequence, when there is a single infection or an infection with but one crop of this parasite, the malarial paroxysms recur every other day. If the patient is infected with two crops of this parasite, he will have a paroxysm every day—the so-called double tertian fever (quotidian malaria). This double tertian type of malarial fever is common in the northern portion of the United States.

The *quartan parasite* (*plasmodium malariæ*) requires seventy-two hours for its endogenous cycle of development, consequently when there is infection with but one crop of the quartan parasites, the paroxysms occur every third day. In cases of infection with two crops of this parasite, the paroxysms occur two days in succession, after which there is a day on which no paroxysm occurs. Should the patient be infected with three crops of the quartan parasite, he would have a paroxysm every day.

The estivo-autumnal parasite (*plasmodium falciparum*) has a developmental period that appears to vary from twenty-four to forty-eight hours. On this account some writers declare that there are two forms of estivo-autumnal parasite—the plasmodium falciparum quotidianum, and the plasmodium falciparum tertianum (Craig). In case of infection with the former variety of parasite, the patient has a paroxysm every day; in infection with the latter variety, the paroxysm appears every other day. The paroxysms of estivo-autumnal infection are more severe and last longer than

those of benign tertian malaria, so that, particularly in case of infection with plasmodium falciparum quotidianum, one paroxysm is likely to extend into a second paroxysm, producing a continued type of fever.

In malarial infection the fever may be one of three types: (1) The intermittent type; (2) the remittent type; (3) the continued type. The intermittent type of fever generally occurs in infection with the benign tertian parasite and with the quartan parasite. In some cases of infection with the estivo-autumnal parasite an intermittent fever is seen (pernicious intermittent), but the remittent and continued fevers are the types most commonly met.

Intermittent Fever.

In intermittent fever the patient experiences certain prodromal symptoms, among which are mental apathy, dull headache, pain or stiffness of the neck muscles, and an expression of imperfect oxidation of the blood, shown by frequent yawning. It must be remembered, however, that the paroxysm not infrequently develops abruptly. In intermittent fever there is a decided rigor, and the patient shivers incessantly and the teeth chatter. The chill may occur at any time, but is most likely to take place between midnight and midday. It usually lasts from one to two hours, but may continue for several hours.

Physical Signs and Description of Paroxysm.—The Cold Stage.—*Inspection.*—The patient is seen to be resting under a blanket, or is well wrapped and sitting near the fire or in the sunlight. The skin is often pale, and cyanosis of the lips is not uncommon; later the face becomes flushed. If the patient has suffered from malaria for several weeks, a decided yellowish-brown tinging of the skin is apparent, said to be due to a deposition of blood-pigment in the cutaneous tissues. Jaundice may be present. Herpes involving the lips and nose is quite common during the course of intermittent fever, and such other cutaneous manifestations as purpura and urticaria have been observed.

Palpation.—During the chill the skin is cold, the muscles of the arms and legs are tender and at times painful, and the pulse increases in frequency, becoming rapid, bounding, and of high tension. Before the chill has subsided the internal, and at times the external, temperature begins to rise.

The Hot Stage.—The patient passes from the cold to the hot stage in quite rapid succession.

Inspection.—The face is flushed, and the skin becomes hotter and hotter. The muscle soreness is now less decided than it was during the chill. The spleen is enlarged and slightly tender.

Palpation.—The pulse remains rapid and full, although in cases of virulent infection it may become weak, rapid, and even irregular, due probably to acute dilatation of the heart following extreme toxemia.

Percussion.—The splenic dullness will be found to increase slightly after each succeeding paroxysm. *Palpation* and *percussion* reveal the fact that the liver is moderately enlarged.

Auscultation.—The respirations are at times hurried, and hemic murmurs are heard over the base of the heart.

The *temperature*, which has begun to rise before the close of the first stage, continues to mount rapidly until it reaches 104° to 106° F. (See Fig. 335.) It may remain near the maximum point for one or more hours, or it may fall suddenly by crisis; in either case there may be two moderate remissions

before there is a decided lowering of the temperature, but it must be remembered that, as a rule, there is a rapid decline at the close of the febrile stage which lasts from three to six hours. (See Fig. 335.)

Sweating.—Profuse sweating is followed by an amelioration of all the symptoms of the hot stage. The temperature falls by crisis to the normal, as is shown in the accompanying chart (Fig. 335). After treatment has been instituted the temperature may fall to the normal and remain at or

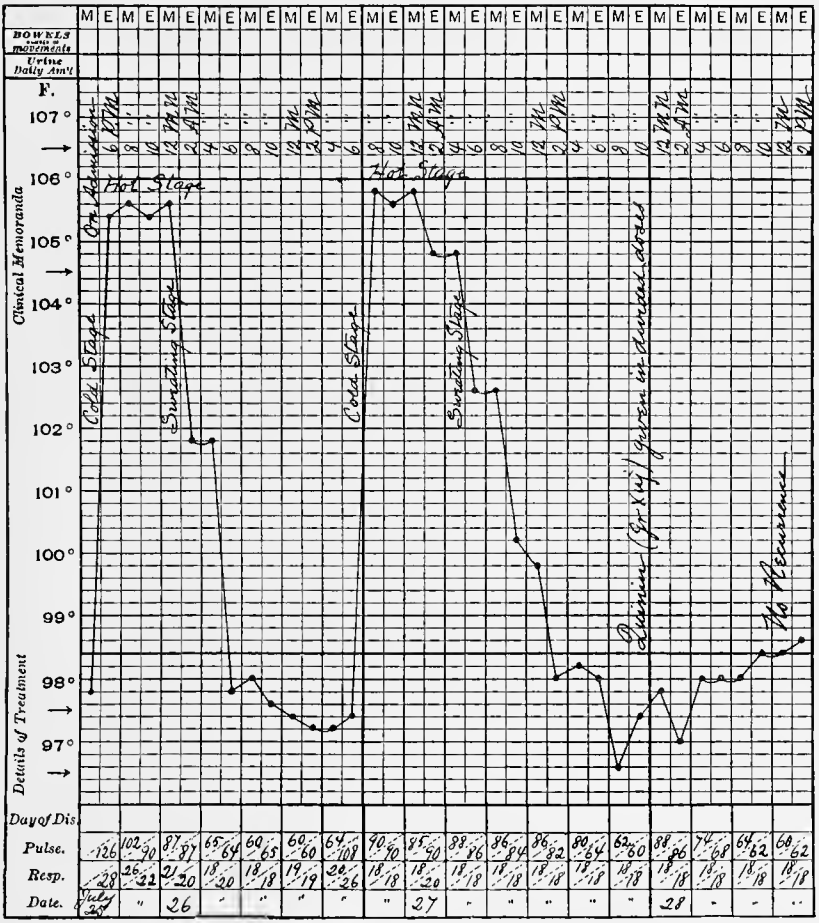


Fig. 335.—Temperature-curve in a Case of Double Tertian Fever. (C. F. C., aged forty-one years.) (J. M. Anders.)

near that point. If the treatment has not been successful there will be recurrent elevation of temperature, with the associated phenomena just described. Following these recurrent elevations of temperature the fever-curve is occasionally seen to fall in a step-like manner, dropping one or two degrees and then remaining at this point for an indefinite period, but even in this step-like defervescence only a few hours are required to reach the normal.

Laboratory Diagnosis.—During the chill and even a few hours prior to the rise in temperature blood obtained from the peripheral circulation, when studied in the fresh state under a $\frac{1}{12}$-inch oil-immersion objective, shows the presence of living malarial parasites. The number of parasites present in the fresh blood varies greatly in different persons, but the morphologic characteristics of the parasite are constant. (See Plate XV.) When the disease has progressed for some time, the red blood-cells show marked evidences of degeneration, as, for example, imperfect distribution of hemoglobin, cracks and fissures, streaks, irregular areas of decoloration, and poikilocytosis. The number of red cells in a cubic millimeter is reduced in direct proportion to the length of time the disease has existed and the severity of the type of infection. The hemoglobin also undergoes destruction. When intermittent fever has continued for a long time pigment particles are seen in the blood-plasma. In uncomplicated cases of malaria leukopenia is a characteristic feature.

The *stained blood* shows all the changes more clearly than the fresh films. The change in size and shape of the erythrocytes, the various degenerations of the erythrocyte, the morphologic characters and the stage of development of the parasite, the characters of the leukocytes, and the presence or absence of malarial pigment are much better appreciated in stained smears. A *differential leukocyte count* will show an increase in the percentage of the large mononuclears.

The **urine** not infrequently contains traces of albumin, and in severe types of the infection one occasionally finds the evidences of acute nephritis—*e. g.*, albumin, casts, red and white blood-cells. This condition may be regarded as a complication, and is said by Jones to occur quite commonly in the American negro. Jaccoud asserts that the amount of urine excreted is increased for from three to six hours prior to the development of the chill. There are certain symptoms that point strongly to the existence of gastro-intestinal catarrh, but a clinical analysis of the stomach-contents does not disclose anything of special importance.

Summary of Diagnosis.—Prodromata are of little value in making the diagnosis of malaria. The occurrence of exacerbations, consisting of a chill, a hot and a sweating stage, which occur periodically and are accompanied by prostration and blood destruction, with the occasional presence of herpes, point strongly to malarial infection. The diagnosis is confirmed by the finding of the plasmodium in either the living or the stained peripheral blood.

Differential Diagnosis.—Malaria is to be distinguished from certain other infectious maladies, among which infection with pyogenic organisms occupies a prominent place. In the presence of **abscess,** whether deep or superficial, there are likely to be periodic paroxysms of chill, fever, and sweating, which simulate those seen in malaria. The presence of a leukocytosis and the absence of the malarial parasite from the blood serve to distinguish such a suppurative process from malaria.

Pulmonary Tuberculosis.—After the stage of cavity formation periodic exacerbations of fever commonly occur, which are followed by profuse sweats. Here the distinguishing features are: the history of pulmonary tuberculosis, with the presence of a cavity, leukocytosis, and the absence of the malarial parasite from the blood. An intermittent fever, accompanied by chill and nocturnal sweats, is occasionally observed in the earlier stages of pulmonary tuberculosis. A physical examination may make the

diagnosis clear, but if it does not, the blood should be examined microscopically.

Hepatic colic not infrequently gives rise to a temperature that may be mistaken for that of malaria—the so-called Charcot's intermittent fever. Tenderness over the liver, with pain in the region of the gall-bladder radiating to the right scapula, the presence of jaundice, and the findings of a blood examination will exclude malarial fever.

Renal calculi may at times excite a temperature that resembles in many respects that of malaria, but the abdominal pain and the area of distribution of the pain, with the passing of bloody urine, precludes the diagnosis of malaria.

Urethral fever frequently closely resembles malarial fever.

Clinical Course.—In the ordinary type of intermittent malarial fever the prognosis is favorable, and in cases in which treatment is instituted early complete recovery ensues. If, however, the infection is permitted to exist for a long period before treatment is instituted, recovery is delayed, and the condition tends to assume a chronic form.

Estivo-autumnal (Malarial) Fever.

Pathologic Definition.—Estivo-autumnal malarial fever is a type of malarial infection due to the presence of the plasmodium falciparum in the blood. Two varieties of this organism may be present: plasmodium falciparum tertianum, in which the cycle of development is forty-eight hours, and plasmodium falciparum quotidianum, in which the cycle of development is twenty-four hours.

Remarks.—Plasmodium falciparum produces the most severe types of malaria clinically. The paroxysms are of longer duration than are those produced by infection with plasmodium vivax and plasmodium malariæ. The paroxysms are likely to anticipate one another, so that one has not ended before another seizure begins. The sporulation of the parasites takes place almost exclusively in the internal organs, the rosets often blocking the blood-vessels of the brain, the heart, the liver, the spleen, or the kidneys, producing the various types of pernicious fever, depending upon the organ involved.

Varieties.—Algid Form.—The algid form of estivo-autumnal malaria is characterized by the occurrence of purging, vomiting, intestinal pain, and collapse. The condition closely resembles dysentery or cholera. Jaundice and severe nervous symptoms are present. The fever may be intermittent, and reach a maximum of 106° or 107° F. (pernicious intermittent), or it may be remittent. This form is due to the sporulation of the parasites in the blood-vessels of the intestine.

Comatose Form.—This variety is characterized by the sudden development of coma, with cyanosis, contracted pupils, stertorous breathing, rapidly failing pulse, and death. In some cases the coma appears gradually after the development of restlessness, delirium, or mental depression. This form of the disease is due to the sporulation of the parasites in the blood-vessels of the brain.

Cardialgic Form.—In this type of estivo-autumnal malaria the parasites sporulate in the heart muscle. There are precordial or epigastric pain, vomiting of blood, hiccough, dyspnea, weak, rapid pulse, collapse, and death.

A hemorrhagic form, a choleraic form, a dysenteric form, and a

pneumonic form have also been described. The most common variety of estivo-autumnal infection, however, is the so-called **bilious remittent fever.**

Period of Incubation.—Celli gives the period of incubation of the estivo-autumnal parasite as five days.

General Complaint.—Such prodromal symptoms as headache, epigastric oppression, uneasiness, and anorexia occur, and a distinct chill may or may not take place.

Sweating Stage.—Following the remission in the temperature profuse sweating occurs, and during this stage the headache and epigastric symptoms disappear.

Nervous Symptoms.—The patient may be extremely nervous, and delirium is occasionally seen.

Thermic Features.—One or even two elevations in the temperature may occur during the twenty-four hours, and these resemble closely the fever of intermittent malaria, except that in estivo-autumnal fever the paroxysms are considerably longer, and may last from twelve to twenty hours. The temperature rises more gradually in estivo-autumnal fever than in the ordinary intermittent type of malaria, and the decline of the fever likewise requires a longer period; consequently the temperature may not reach the normal after one exacerbation until a second elevation has set in. The peculiarities of estivo-autumnal fever just described have caused it to be regarded as a form of continued malarial fever, in which case the periods of intermission become progressively shorter with the progress of the disease.

The patient may give a history of but one initial chill, which was soon followed by fever, the temperature rising gradually to from 104° to 105° F. within twelve hours.

Physical Signs.—Inspection.—During the attack the patient may become slightly cyanosed, and if the paroxysm is a severe one, there is a peculiar pallor of the cheeks that may be followed by a hectic flush. The tongue is parched and coated, the conjunctivæ are congested, the skin is slightly jaundiced, and herpes labialis is commonly present.

Palpation and **percussion** show the spleen to be moderately enlarged, and there is also slight increase in the area of liver dullness.

Laboratory Diagnosis.—Microscopic examination of the blood discloses the presence of the estivo-autumnal parasite. (See Plate XVI.) Early during the course of remittent malarial fever there is a moderate reduction in the percentage of hemoglobin and in the number of red cells, but in neglected cases, and when repeated infections have occurred, the red cells become fewer and the hemoglobin shows a decided reduction in percentage. Leukopenia is an important feature of uncomplicated remittent fever.

In **stained blood** the red cells show the usual evidences of degeneration, the presence of the small ring forms of the parasites. and the characteristic crescentic gametocytes. There is an increase in the percentage of large mononuclear leukocytes.

Urine.—During the course of remittent fever the urine is diminished in quantity, its specific gravity is higher, and albumin is present. In about 5 per cent. of the cases true nephritis develops, in which case the color becomes high and casts are present. Mild types of remittent fever are seen in which the recurring paroxysms grow shorter day by day.

Differential Diagnosis.—The following tables show the distinctive features between remittent malarial fever and **typhoid fever,** and remittent malarial fever and **puerperal sepsis.**

DESCRIPTION OF PLATES XV AND XVI.[1]

The drawings were made with the assistance of the camera lucida from specimens of fresh blood. A Winckel microscope, objective ¼ (oil immersion), ocular 4, was used. Figures 4, 13, 23, 24, and 42 of Plate XV were drawn from fresh blood, without the camera lucida.

PLATE XV.

THE PARASITE OF TERTIAN FEVER.

1.—Normal red corpuscle.
2, 3, 4.—Young hyaline forms. In 4, a corpuscle contains three distinct parasites.
5, 21.—Beginning of pigmentation. The parasite was observed to form a true ring by the confluence of two pseudopodia. During observation the body burst from the corpuscle, which became decolorized and disappeared from view. The parasite became, almost immediately, deformed and motionless, as shown in Fig. 21.
6, 7, 8.—Partly developed pigmented forms.
9.—Full-grown body.
10–14.—Segmenting bodies.
15.—Form simulating a segmenting body. The significance of these forms, several of which have been observed, was not clear to Drs. Thayer and Hewetson, who had never met with similar bodies in stained specimens so as to be able to study the structure of the individual segments.
16, 17.—Precocious segmentation.
18, 19, 20.—Large swollen and fragmenting extracellular bodies.
22.—Flagellate body.
23, 24.—Vacuolization.

THE PARASITE OF QUARTAN FEVER.

25.—Normal red corpuscle.
26.—Young hyaline form.
27–34.—Gradual development of the intracorpuscular bodies.
35.—Full-grown body. The substance of the red corpuscle is no more visible in the fresh specimen.
36–39.—Segmenting bodies.
40.—Large swollen extracellular form.
41.—Flagellate body.
42.—Vacuolization.

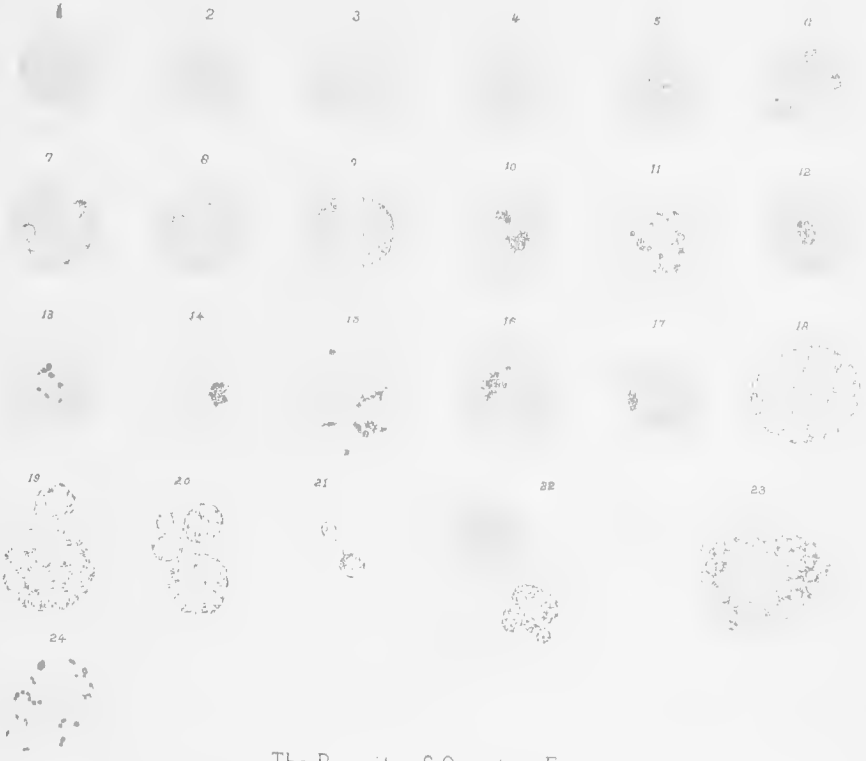
PLATE XVI.

THE PARASITE OF ESTIVO-AUTUMNAL FEVER.

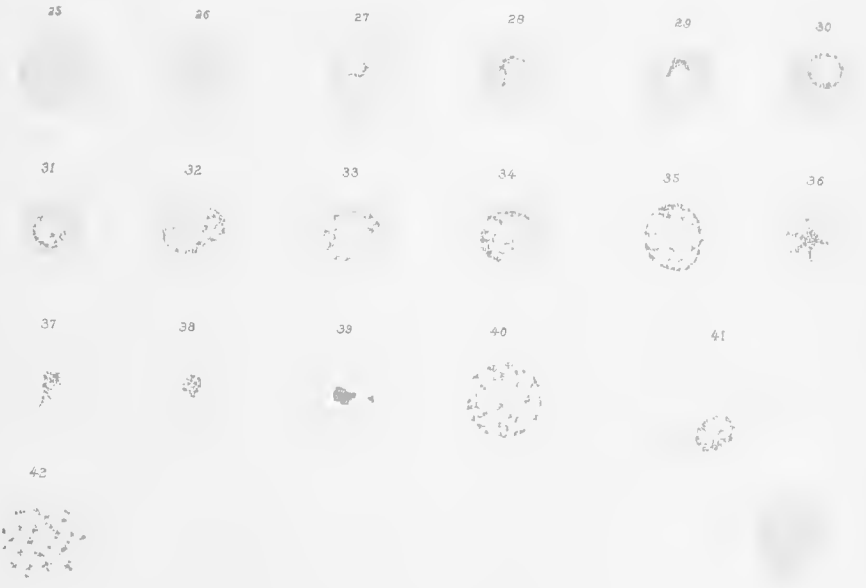
1, 2.—Small refractive ring-like bodies.
3–6.—Larger disk-like and ameboid forms.
7.—Ring-like body with a few pigment-granules in a brassy, shrunken corpuscle.
8, 9, 10, 12.—Similar pigmented bodies.
11.—Ameboid body with pigment.
13.—Body with a central clump of pigment in a corpuscle, showing a retraction of the hemoglobin-containing substance about the parasite.
14–20.—Larger bodies with central pigment clumps or blocks.
21–24.—Segmenting bodies from the spleen. Figs. 21–23 represent one body where the entire process of segmentation was observed. The segments, eighteen in number, were accurately counted before separation, as in Fig. 23. The sudden separation of the segments, occurring as though some retaining membrane were ruptured, was observed.
25–33.—Crescents and ovoid bodies. Figs. 30 and 31 represent one body, which was seen to extrude slowly, and later to withdraw, two rounded protrusions.
34, 35.—Round bodies.
36.—"Gemmation," fragmentation.
37.—Vacuolization of a crescent.
38–40.—Flagellation. The figures represent one organism. The blood was taken from the ear at 4.15 p. m.; at 4.17 the body was as represented in Fig. 38. At 4.27 the flagella appeared; at 4.33 two of the flagella had already broken away from the mother body.
41–45.—Phagocytosis. Traced with the camera lucida.

[1] These illustrations are reproduced by permission from the article by Drs. Thayer and Hewetson in *The Johns Hopkins Hospital Reports*, vol. v., 1895.

The Parasite of Tertian Fever.



The Parasite of Quartan Fever.



The Parasite of Estivo Autumnal Fever.

DIFFERENTIAL DIAGNOSIS BETWEEN REMITTENT MALARIAL FEVER AND TYPHOID FEVER

Remittent Malarial Fever.	Typhoid Fever.
1. There may or may not be a history of exposure to the bites of mosquitos, or of having had malaria during the past few weeks or months.	1. The history of an epidemic is often obtained.
2. Begins abruptly with a chill or a series of chills, which are followed by headache, and the characteristic fever and sweating stage.	2. A distinctive rigor is extremely uncommon, although chilly sensations may be experienced, but are not followed by high fever.
3. An examination of the fresh blood reveals the presence of the plasmodium. Pigment granules may be seen free in the plasma.	3 A blood examination is negative.
4. The Widal reaction is negative.	4. The Widal reaction is positive in dilution of 1: 50 or higher.
5. The temperature rises abruptly and may display decided remissions for one, two, or even more days, when another elevation takes place. These elevations and remissions display a variable degree of periodicity. When treatment is not instituted, the fever continues over a period of several weeks.	5. There is but slight fever at first (99° to 100° F.); the temperature then rises gradually for a period of from five to seven days, and reaches its height in from ten to fourteen days; it continues high until between the seventeenth and twenty-first days, after which it gradually declines.
6. The skin may show periodic flushings.	6. A characteristic eruption appears on the abdomen in from the seventh to the ninth days, and continues to appear in two or more successive crops.
7. Constipation the rule.	7. At first there is constipation, but diarrhea develops toward the end of the first week, and there are usually from 3 to 12 pea-soup-like stools daily by the end of the second week.
8. Intestinal hemorrhage is rare.	8. Intestinal hemorrhage is a frequent complication.
9. The abdomen is normal or scaphoid.	9. Tympanites is a prominent symptom after the second week.
10. Nervous symptoms slight, and delirium unusual.	10. Nervous symptoms pronounced; delirium common.
11. Hemoglobinuria and hematuria with pronounced albuminuria quite common.	11. A mild grade of albuminuria in uncomplicated cases. Urine contains typhoid bacilli in from 6 to 20 per cent. of cases.

THE DISTINCTIVE FEATURES BETWEEN REMITTENT MALARIAL FEVER AND PUERPERAL SEPSIS.

Remittent Malarial Fever.	Puerperal Sepsis.
1. Chill may develop at any time during the puerperium, and recurs with a variable degree of periodicity.	1. Chill from the third to the ninth day after delivery.
2. A study of the blood shows leukopenia.	2. Leukocytosis 10,000 to 30,000 in a cubic millimeter.
3. Plasmodia present in the blood.	3. Blood examination negative.
4. Differential leukocyte count shows an increase in the percentage of large mononuclear leukocytes.	4. Increase in the polymorphonuclear cells, 85 to 95 per cent.
5. The lochia remains normal.	5. Lochia diminished prior to the development of the chill and fever, but may become profuse and of an offensive odor later.

Remittent Malarial Fever.—(*Continued.*)	Puerperal Sepsis.—(*Continued.*)
6. No tenderness either in the uterine or in the pelvic regions.	6. Pelvic and uterine tenderness common, though by no means a constant feature.
7. Involution of the uterus normal.	7. Subinvolution the rule.
8. Temperature affected by the administration of quinin.	8. Quinin exercises but little influence, but temperature declines following intrauterine douches and curetment.

Malarial Cachexia.

Pathologic Definition.—Malarial cachexia is the resulting anemia and wasting, with splenomegaly and enlargement of the liver, which follow repeated attacks of malarial infection. There may be hemorrhages from the various mucous surfaces, hemorrhages into the skin, and joint and muscle tenderness. Those affected with chronic malarial cachexia often display chronic nephritis, myocarditis, etc., and are especially likely to develop tuberculosis. (See Latent Malaria, p. 956.)

Recurrent Malaria.

Pathologic Definition.—As its name implies, in this form of malarial infection there is a reappearance of the general symptoms, due to the same group of parasites that caused the original infection, the symptoms recurring after an initial attack without reinfection by another group of plasmodia having taken place.

Remarks.—The exact time that has elapsed between the initial attack of malaria and the recurrence should be ascertained. This clinical problem is solved most satisfactorily by reference to the accompanying tables by Craig:

TIME OF RECURRENCES IN 18 CASES OF TERTIAN INFECTION.

Case No.	Date of Initial Attack.	First Recurrence.	Second Recurrence.	Third Recurrence.	Fourth Recurrence.	Fifth Recurrence.
1	Nov. 2	20 days	21 days	.....	.....	.....
2	Aug. 4	18 "	20 "	.....	.....	.....
3	Aug. 28	19 "	30 "	26 days	46 days	.....
4	Nov. 6	20 "	24 "	.....	.....	.....
5	Jan. 17	20 "	32 "	30 days	24 days	.....
6	Nov. 23	21 "	20 "	26 "	.....	.....
7	Oct. 6	21 "	30 "	.....	.....	.....
8	Sept. 17	21 "	22 "	27 days	.....	.....
9	Aug. 27	22 "	36 "	.....	.....	.....
10	Feb. 12	22 "	18 "	16 days	27 days	30 days
11	Jan. 17	27 "	.....	.....	.....	.....
12	July 20	30 "	.....	.....	.....	.....
13	May 3	30 "	.....	.....	.....	.....
14	Nov. 1	30 "	.....	.....	.....	.....
15	Sept. 22	33 "	.....	.....	.....	.....
16	Sept. 1	37 "	.....	.....	.....	.....
17	Dec. 13	38 "	.....	.....	.....	.....
18	Sept. 22	41 "	.....	.....	.....	.....

ESTIVO-AUTUMNAL TERTIAN RECURRENCES. TIME OF THE VARIOUS RECURRENCES IN 55 CASES OF TERTIAN ESTIVO-AUTUMNAL INFECTION.

Case No.	Date of Initial Attack.	First Recurrence.	Second Recurrence.	Third Recurrence.	Fourth Recurrence.	Fifth Recurrence.
1	Oct. 12	10 days	30 days	36 days	.....	.....
2	Nov. 19	12 "	.....	.....	.....	.....
3	Feb. 27	15 "	20 days	30 days	.....	.....
4	Nov. 2	18 "	30 "	30 "	.....	.....
5	Mar. 30	19 "	20 "	.....	.....	.....
6	Dec. 8	19 "	.....	.....	.....	.....
7	Jan. 24	20 "	.....	.....	.....	.....
8	Feb. 12	20 "	60 days	.....	.....	.....
9	Dec. 24	20 "	.....	.....	.....	.....
10	Feb. 6	20 "	20 days	.....	.....	.....
11	Feb. 6	20 "	48 "	.....	.....	.....
12	Dec. 25	21 "	33 "	.....	.....	.....
13	Mar. 1	22 "	.....	.....	.....	.....
14	Nov. 29	22 "	.....	.....	.....	.....
15	Nov. 14	24 "	.....	.....	.....	.....
16	Feb. 4	24 "	20 days	38 days	30 days	.....
*16½	Oct. 30	24 "	16 "	.....	.....	.....
17	Aug. 29	24 "	26 "	.....	.....	.....
18	Mar. 17	24 "	.....	.....	.....	.....
19	Feb. 4	25 "	16 days	20 days	.....	.....
20	Dec. 30	26 "	36 "	30 "	90 days	30 days
21	Jan. 26	26 "	48 "	90 "	.....	.....
22	Jan. 11	26 "	22 "	.....	.....	.....
23	Oct. 2	27 "	.....	.....	.....	.....
24	Nov. 2	27 "	.....	.....	.....	.....
25	Mar. 2	27 "	52 days	.....	.....	.....
26	Feb. 5	28 "	21 "	20 days	21 days	.....
27	Dec. 12	28 "	28 "	.....	.....	.....
28	Oct. 29	29 "	48 "	15 days	.....	.....
29	Jan. 17	30 "	.....	.....	.....	.....
30	Jan. 1	30 "	30 days	.....	.....	.....
31	Jan. 19	30 "	.....	.....	.....	.....
32	Jan. 20	32 "	.....	.....	.....	.....
33	Oct. 19	33 "	26 days	90 days	.....	.....
34	Jan. 19	34 "	40 "	.....	.....	.....
35	Oct. 18	34 "	50 "	.....	.....	.....
36	Jan. 25	34 "	26 "	17 days	.....	.....
37	Oct. 21	36 "	56 "	.....	.....	.....
38	Feb. 30	36 "	66 "	.....	.....	.....
39	Aug. 13	36 "	35 "	.....	.....	.....
40	Nov. 27	36 "	.....	.....	.....	.....
41	Sept. 1	37 "	49 days	.....	.....	.....
42	Oct. 18	38 "	.....	.....	.....	.....
43	Oct. 17	38 "	.....	.....	.....	.....
44	Aug. 13	38 "	.....	.....	.....	.....
45	Sept. 6	41 "	.....	.....	.....	.....
46	Oct. 31	42 "	20 days	.....	.....	.....
47	Jan. 1	45 "	30 "	.....	.....	.....
48	Nov. 3	46 "	21 "	.....	.....	.....
49	Dec. 7	49 "	.....	.....	.....	.....
50	Feb. 24	50 "	24 days	41 days	.....	.....
51	Oct. 24	51 "	39 "	.....	.....	.....
52	Jan. 18	61 "	156 "	.....	.....	.....
53	June 14	64 "	66 "	14 days	20 days	20 days
54	Mar. 3	80 "	120 "	96 "	.....	.....

* The case numbered 16½ brings the total of the table to 55 cases.

It is frequently impossible to estimate with any degree of accuracy the interval between the initial attack of malaria and that of the recurrence, and while it shall not be our purpose to outline such difficulties, we are inclined to believe that in many instances the time cannot be determined. The time will also be found to differ somewhat depending upon the type of organism represented by the case in question. The exact method as to how these recurrences are produced has baffled even the most competent students of the age, and at present various theories are offered.

Hemoglobinuria and Hematuria.

In malarial infection the detection of red corpuscles (hematuria) or of hemoglobin (hemoglobinuria) in the urine constitutes the most important finding. The number of red cells in the peripheral blood is generally reduced, and may fall below 2,000,000 in a cubic millimeter; in fact, cases have been reported in which the number of red cells was below 1,000,000.

The peripheral blood shows great numbers of pigmented malarial parasites, and many of the leukocytes show pigmentation.

The general history obtained is that of a mild cold stage, following which the temperature becomes subnormal and remains so for an indefinite period, when hemoglobinuria and hematuria develop. These paroxysms occur with decided periodicity, although in some patients bloody urine may be voided daily or even hourly. Hemoglobinuria is occasionally observed to occur at irregular intervals. Chemically, the urine is found to contain a considerable amount of albumin, and in a small percentage of cases casts are found. In the hemoglobinuric variety red blood-cells are also present, but in the hematuric type it is unusual to find many erythrocytes.

Latent Malaria.

Pathologic Definition.—By the term latent malaria is meant a condition in which plasmodia may be demonstrated in the blood of an individual in whom no definite clinical symptoms of the disease are observed. "The term should not be confined to those instances in which no symptoms of malaria have ever been present, for if the parasites be present in the blood in recurrent cases, between the attacks the disease is as truly latent as it may be before the initial one" (Craig).

Remarks.—In a statistical analysis of 1653 cases, Craig* found 424 (25 per cent.) to be latent infections, and of these, 307 occurred in American soldiers or civilians, whereas 115 were in Filipinos.

Variety of Organism.—Among these 424 cases, the tertian parasite was present in 110; the quartan parasite, in 8; the tertian estivo-autumnal parasite, in 272; the quotidian estivo-autumnal parasite, in 25; combined tertian and tertian estivo-autumnal parasites, in 7; combined tertian and quotidian estivo-autumnal parasites, in 2. In 307 cases studied in Americans the tertian organism was found 81 times; the estivo-autumnal tertian, 199; the quotidian estivo-autumnal, 21; and combined infections, 6.

Latent Infection in Children Natives of the Philippine Islands.—Craig examined the blood of 180 cases, and found that 87 (48.3 per cent.) showed the presence of plasmodia. The plasmodium vivax was present in 34; the plasmodium malariæ, in 6; plasmodium falciparum tertianum, in 44; the plasmodium falciparum quotidianum, in 4; combined infections were found in 3 cases.

* Jour. of Infectious Diseases, vol. iv, No. 1, January 1, 1907, p. 100.

"The infections in children diminished in number with advancing age; thus, between the ages of one month and five years, among 40 children, 79 per cent. were infected; between five and ten years, 37 per cent.; and between ten and fifteen years, 24.5 per cent" (Craig). The researches of Craig confirm the observations of Koch, Stephens, Christopher, James and other observers, all of whom found that the younger the child, the more susceptible is it to malarial infection.

Family Infection.—Several members of the same family are commonly found to be infected, a feature that further supports the fact that the transmission of malarial infection is likely to be limited, a finding that is borne out by the accompanying table by Craig:

Family.	Number of Members.	Number Infected.	Variety.
1.....	4	2	1 estivo-autumnal; 1 tertian.
2.....	3	2	2 estivo-autumnal.
3.....	4	2	1 estivo-autumnal; 1 tertian.
4.....	5	4	2 estivo-autumnal; 1 tertian; 1 quartan.
5.....	4	2	2 estivo-autumnal.
6.....	3	2	2 estivo-autumnal.
7.....	4	3	2 estivo-autumnal; 1 tertian.
8.....	3	2	1 estivo-autumnal; 1 tertian.
9.....	3	2	2 tertian.
10.....	6	4	2 estivo-autumnal; 2 tertian.

TRYPANOSOMIASIS (KALA-AZAR).

Pathologic Definition.—An acute infectious disease caused by the Trypanosoma gambiense. In this connection a variety of severe anemia occurring in Assam, associated with pyrexia and enlargement of the spleen and liver, has been shown to be due to a variety of trypanosomiasis in which only immature forms of the parasite (Leishman-Donovan bodies) have been found in the fluid obtained by splenic puncture. The trypanosomes are found to invade the blood-stream, connective structures of all organs, the reticular tissue of the lymph-nodes and spleen, and the substance of the brain.

Clinical Remarks.—Trypanosomiasis begins as a febrile affection, with enlargement of the superficial lymph-nodes and the presence of a diffuse erythema. The fever is of the continued type, and varies in degree in different cases. After a week or more the temperature falls, and there is a period of apyrexia of indefinite duration. The periods of pyrexia and apyrexia alternate irregularly, and in time the patients become wasted, anemic, and mentally deficient. During the first febrile paroxysm the skin presents irregular areas of erythema, associated with edema of the underlying connective tissue. The enlarged lymph-nodes are tender, but they seldom go on to pus formation. Headache, neuralgic pains, rapid pulse, cardiac weakness, painful local swellings, enlargement of the spleen and liver, and orchitis are among the symptoms that have been observed. After a period of several years, during which these alternating attacks of fever and apyretic intervals have been observed, the patient gradually becomes lethargic, and the terminal stage of the infection, known as the *sleeping sickness*, begins. The weakness, wasting, and anemia now increase, the patient becomes indifferent to his surroundings, and is incapable of exertion. His gait is shuffling. His mental processes become very sluggish, and localized edemas appear. He presents fibrillary twitchings of the muscles of the face and tongue, and tremors of the hands and legs

develop. He takes to bed or sleeps on the ground. At first he can be aroused to take his meals, but this soon becomes impossible, and death occurs either in convulsions or in coma, or is dependent on some intercurrent disease, such as dysentery and pneumonia.

Exciting Factor.—Trypanosoma gambiense is transmitted from man to man by the tsetse fly, *glossina palpalis*, and possibly also transmitted by other insects.

Protozoology.—Trypanosoma gambiense is an animal parasite, belonging to the genus protozoa; order, flagellata. It has a spindle-shaped cytoplasmic body, having a nucleus (macronucleus) and a centrosome (micronucleus). The latter is situated at the posterior end of the body of the parasite. From the centrosome a flagellum arches over the dorsum of the cytoplasmic body, to project beyond the anterior end of the parasite as a free flagellum. The cytoplasm of the organism is prolonged from the dorsum of the body to the flagellum to form the *undulating membrane.*

The **cutaneous manifestations** of trypanosomiasis may consist mainly of localized areas of erythema, or there may be a peculiar blotching over the extremities and upon the face and trunk.

Nervous Symptoms.—Restlessness and mental dullness are present, and when the parasite infects the meningeal fluid, the patient becomes dull and sleepy. Cheyne-Stokes respiration develops late.

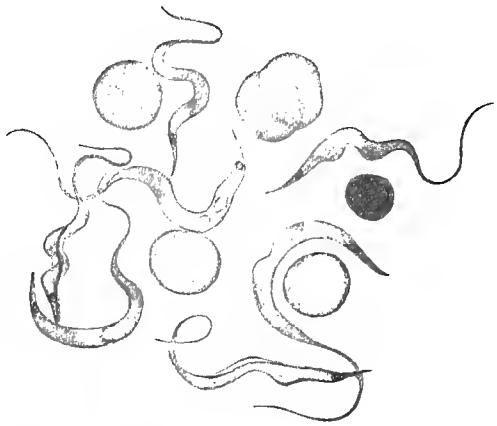


FIG. 336.—TRYPANOSOMA LEWISI STAINED WITH A 2 PER CENT. AQUEOUS SOLUTION OF METHYLENE-BLUE (Boston).

Circulatory Phenomena.—The chief circulatory manifestation of the disease consists of thrombosis of the vessels of the extremities. The **ocular manifestations** consist of pallor and mottling of the fundus.

Laboratory Diagnosis.—The trypanosoma gambiense is detected in the peripheral blood of the infected person only with difficulty, even during the height of the paroxysms. Puncture of the enlarged lymph-nodes or injection of the blood of the suspected patient into monkeys or white rats will, however, clear up the diagnosis. The lymph-node puncture is performed as follows: The skin over the enlarged organ is washed with soap and water, followed by sterile water, and a 1 : 1000 mercury bichlorid dressing is put on the skin for an hour. Then a sterile hypodermic needle attached to a syringe is plunged into the organ, and after being moved backward and forward a few times, so as to loosen the contents, the piston is pulled out and a

few drops of the contained fluid are withdrawn. This fluid is then examined —as fresh specimens and as stained smears.

Inoculations of the blood are made by puncturing a vein—usually the median cephalic—of the suspected patient, after the usual aseptic preparation. Next 10 c.c. of blood are withdrawn, and 5 c.c. are injected into each of two monkeys, or white rats may be employed for the inoculation. Examination of the peripheral blood of the inoculated animals will show the presence of trypanosoma gambiense within about one week after the inoculation.

Blood studies in cases of human trypanosomiasis show a marked chloranemia and a leukopenia. The differential leukocyte count shows an increase of the large mononuclear leukocytes.

NEMATODES.

FILARIASIS.

Pathologic Definition.—A condition due to the presence of the embryos of filaria bancrofti (filaria nocturna) or of filaria loa (filaria diurna) in the circulating blood, which is believed to result finally in obstruction to the lymph-channels, with the development of elephantiasis, obstruction to the lymphatics of the kidney and rectum, and, rarely, cutaneous abscesses are formed.

Exciting and Contributing Causes.—The exciting cause of filariasis is the filaria bancrofti or other species of the genus Filaria. Man becomes infected by the bites of infected mosquitos, usually of the genus Culex. Filariasis is a disease of the tropics and of subtropical regions. In the United States several cases of filariasis have occurred in a small section of country in North Carolina. A few cases have also been reported from Illinois, and we have studied a case of filariasis in a patient who had never lived south of New Jersey.

Principal Complaint.—The patient may harbor filaria embryos in his blood for a long time and yet be in perfect health. The development of symptoms is thought by many writers to be dependent upon injury to the adult worm and the consequent blocking of the lymphatic vessels with improperly developed embryos. Upon injury to the adult worm the first symptoms to appear are attacks of fever that somewhat resemble malaria, and are known as *filarial fever*. The patient may complain of mental depression, anemia, weakness, and fatigue upon slight exertion. In some instances there is intense itching of the skin, as was seen in a case studied in Philadelphia. After several attacks of filarial fever separated by intervals of apyrexia, elephantiasis (Figs. 337, 338) begins to develop. The patient usually complains of inability to move the affected parts or of discomfort caused by scrotal and labial hypertrophy. Roughness and scaling of the skin of the lower extremities appear early.

Physical Signs.—Inspection.—There may be general pallor, due to secondary anemia. When elephantiasis develops, the skin of the affected part presents a peculiar rough, scaling, and wrinkled appearance, not unlike the skin of the elephant, hence its name. As the result of this enlargement movements of the limbs are but slightly restricted. One or both limbs, the scrotum, or the vulvæ may be involved. (See Figs. 337 and 338.)

Palpation confirms inspection as regards those portions of the body showing elephantiasis.

Laboratory Diagnosis.—There are three types of filaria embryos: (1) Those found in the peripheral blood during the night (filaria nocturna); (2) those found during the day (filaria diurna); and (3) those continually present. In four cases studied in Philadelphia the filaria could be detected at practically any time during the twenty-four hours, but in one of these cases filariæ were abundant in the blood in but two days of each week. The method for detecting filaria embryos in the living blood is practically identical with the method described for the detection of the malarial parasites. (See p. 950.) The embryos are easily discerned under a $\frac{2}{3}$-inch objective, and when the parasites are very active, this degree of magnification is most desirable. In studying organisms that are but slightly motile, a $\frac{1}{5}$- or a $\frac{1}{6}$-inch objective is entirely satisfactory (Fig. 339).

FIG. 337.—LATERAL VIEW OF CASE OF ELEPHANTIASIS OF THE LABIA MAJORA.

Light area corresponds to opening of the vagina. (Patient studied and photographed by Dr. B. B. Ussher, Jamaica, W. I.)

FIG. 338.—ANTERIOR VIEW OF CASE OF ELEPHANTIASIS OF THE LABIA MAJORA.

Patient studied and photographed by Dr. B. B. Ussher, Jamaica, W. I.

The hemoglobin and the red blood-corpuscles do not undergo marked changes unless some other cause for the secondary anemia is present. This last statement may not hold true, however, for advanced cases of filariasis. In slides smeared thickly with blood containing the filaria embryos the hemoglobin should be dissolved with distilled water, and then stained for twenty minutes with warm Delafield's hematoxylin.

The **urine** often contains red blood-cells, hemoglobin, and filaria embryos in cases of chyluria. The urine is milky in appearance, and contains numerous particles of fat (chyle). When the quantity of chyle entering the urinary tract is extremely large, the urine may give off the odor of the food

taken. The filaria embryos found in the urine are non-motile. In 132 cases of chylous ascites collected from the literature by one of us, there were but three instances in which this condition was dependent upon the filaria.

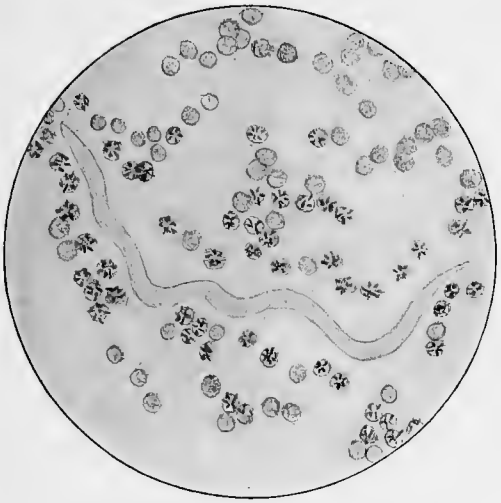


FIG. 339.—FILARIA SANGUINIS HOMINIS (Boston). Living filaria in blood from case at Pennsylvania Hospital. Sketch forty-eight hours after blood was taken.

Summary of Diagnosis.—A history of having resided in the tropics or in districts in which the filaria are known to infest man is of great value in formulating a diagnosis. The presence of elephantiasis and the detection of the parasite (Fig. 339) in the circulating blood or in the urine furnish conclusive evidence of the existence of filariasis. In all cases of hematuria and chyluria the question of possible infection with the filaria should be entertained.

Differential Diagnosis.—Filariasis is to be distinguished from **obstruction to the lymphatic channels** following surgical operations upon the abdomen, the inguinal region, and the thighs; but here the history usually serves to distinguish traumatic elephantiasis. **Chyluria** is occasionally seen after surgical operations upon either the bladder or the pelvic viscera. In one instance we have seen chyluria follow puerperal sepsis. Intermittent attacks of fever may make a microscopic examination of the blood necessary in order to distinguish between trypanosomiasis and filariasis.

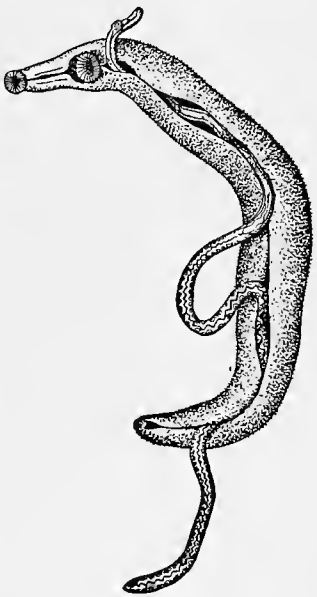


FIG. 340.—MALE AND FEMALE SPECIMENS OF THE HUMAN BLOOD-FLUKE (SCHISTOSOMUM HÆMATOBIUM) ENLARGED. × 12 (after LOOSS).

SCHISTOSOMUM HAEMATOBIUM

(BILHARZIA HAEMATOBIA; BLOOD-FLUKE).

This is a trematode furnished with two sucking disks. The male is shorter and thicker than the female, the former being 4 to 15 mm. ($\frac{1}{6}$ to $\frac{3}{5}$ in.) long, and the latter, about 20 mm. ($\frac{4}{5}$ in.) in length. It is found in Egypt, Cape Colony, and other parts of Africa.

Its presence in the blood gives rise to the following *symptoms:* hematuria, with stinging and burning of the urethra and pain during micturition. The ova of the parasites are found in the urine.

The adult parasites (Fig. 340) inhabit the veins of the portal system. Their ova, however, seldom enter the general circulation, but are found in the veins of the rectum and of the bladder. When the ova escape from the veins ulceration of the mucous surfaces ensues, and the organisms are

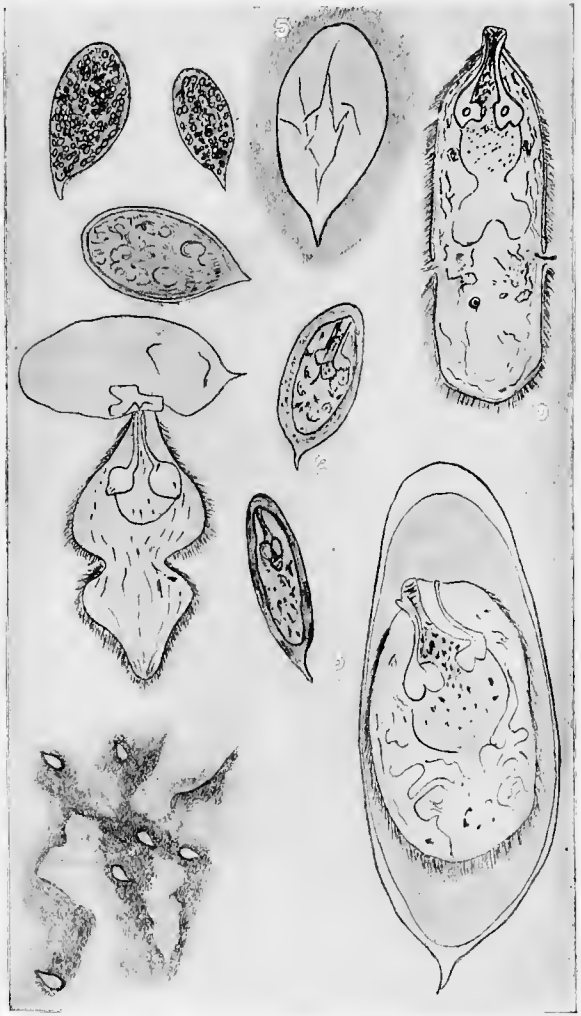


Fig. 341.—Schistosomum Hæmatobium (Bilharz).
1–4, Various stages in development of embryo; 5, empty shell; 6, surviving embryo (after Brock); 7, ova in urinary sediment (Boston).

found enmeshed in blood-clots in the urine (Fig. 341) or the feces. (See Hematuria, p. 956.)

Mode of Detection of the Parasite.—Transfer a portion of the thick urinary sediment to a glass slide, apply a cover-glass, and examine under a two-thirds inch objective. If large blood-clots are present, these

should be broken up or compressed gently between the slide and cover-glass to release the ova from the clotted material. The ova cannot be detected unless the smear of the sediment be comparatively thin, so that each individual cell will stand out prominently in the field. A high-power $\frac{1}{6}$-inch objective may be employed for the study of individual ova, but ordinarily there is no advantage in employing this degree of magnification.

Anemia may follow hemorrhage from the bladder, which is generally persistent, and may continue for a period of several years. Eosinophilia has been detected soon after infection.

INTESTINAL ANIMAL PARASITES AND THEIR OVA.

AMEBIC DYSENTERY.

Pathologic Definition.—An acute infectious disease caused by entamœba histolytica, and characterized by the presence of multiple ulcers in the colon, which show a tendency to coalesce and to produce communicating sinuses in the submucous tissue and amebic abscess of the liver.

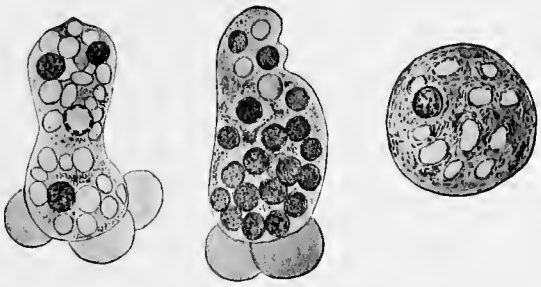


Fig. 342.—Entamœba Histolytica (Kruse and Pasquale).

a and *b*, Amebæ as seen in the fresh stools, showing blunt ameboid processes of ectoplasm. The endoplasm of *a* shows a nucleus, three red corpuscles, and numerous vacuoles; that of *b*, numerous red corpuscles and a few vacuoles; *c*, an ameba as seen in a fixed film preparation, showing a small rounded nucleus; × 600.

Exciting and Predisposing Factors.—The exciting cause is the entamœba histolytica (Stiles). Among the predisposing factors are **climate,** the disease being seen more commonly in the tropics than in temperate and more northern districts.

Age and Sex.—Adults seem to be afflicted oftener than children, and males more frequently than females. Amebic dysentery has long been regarded throughout India, China, Formosa, and the Philippine Islands as extremely fatal, and the disease is also encountered along the Mediterranean Sea and Nile River. One of us has studied a case where the patient had resided in Philadelphia and Atlantic City for ten years before his illness. The feces showed many amebæ.

Feeding Experiments.—Amebic dysentery may be induced in the lower animals (cats) by injecting the living entamœba histolytica into the rectum. Craig* produced typical dysentery in 66 per cent. of cases in which kittens were fed 5 c.c. of feces that contained motile entamebæ mixed with

* Jour. of Infectious Diseases, June 4, 1908, p. 324.

milk. Amebæ were found present in the feces in each instance, and the autopsy findings were those characteristic of this type of dysentery.

Incubation Period.—In Craig's experiments upon kittens the shortest period of incubation was seven days, the longest eleven days, and the majority of the animals developed diarrhea with blood-stained stools by the eighth day. The period of incubation is somewhat longer when living entamebæ are injected into the rectum.

Principal Complaint.—The disease develops insidiously, with a mild diarrhea that continues for a period of from four to ten days, following which a variable degree of constipation occurs, lasting a somewhat longer time. There is always a succession of attacks of diarrhea alternating with periods of constipation. Clinically speaking, the disease assumes a chronic nature from the time the patient observes the first symptoms.

With the progress of the disease there is gradual loss of strength and of flesh, and the skin assumes a yellow hue. In *advanced cases* the patient may be unable to leave his bed, and in the majority of instances he complains of the general symptoms of anemia, *e. g.*, dyspnea, palpitation, vertigo, ringing in the ears, anorexia, and occasional attacks of nausea. The mind is, as a rule, clear, and when marked nervous symptoms occur, they are to be regarded as of serious import.

Thermic Features.—In the acute stage of the disease there is usually a marked febrile reaction, but as the condition becomes chronic the temperature is likely to fall to the normal or below.

Physical Signs.—**Inspection.**—The skin is yellow, the cheeks are sunken, the expression is anxious, the tongue is dry and pale, and there are evidences of emaciation.

Palpation.—The muscles are soft and flabby, and the skin is loose and dry. As the disease progresses the skin becomes cool and clammy. In advanced cases there may be edema of the ankles. The pulse is at first of about normal frequency, but later becomes more rapid, weak, and dicrotic.

Auscultation.—The heart-sounds are weak and fetal in character (late), a condition that is probably dependent upon an associated myocarditis.

Laboratory Diagnosis.—During the attacks of diarrhea the stools contain shreds of mucus, blood, pus, and occasionally sloughs from the mucous surface of the colon. When studied under the microscope,—$\frac{1}{12}$-inch oil immersion objective,—the gelatinous mucoid substance present in the stool will show the presence of many amebæ (Fig. 342).

The *hematologic findings* are those of profound anemia, the hemoglobin and red blood-cells showing great loss. In the stained blood the red cells are found to be markedly degenerated, and to resemble in many respects the changes known to pernicious anemia. Eosinophilia has occasionally been observed.

Illustrative Case of Amebic Dysentery.—C. H., aged twenty-one years; height, 5 feet 10¼ inches; usual weight, 165 pounds; present weight, 128 pounds.

Family History.—Father living at the age of forty-six; mother in apparent health at forty-two; three younger brothers and a sister all in good health.

Previous History.—The patient had the diseases of childhood, including diphtheria at the age of nine years, which condition was complicated by otitis media. He was treated six years ago for influenza, and two years later had an attack of tonsillitis that went on to peritonsillar abscess. Six months ago he had malaria, and two months ago an acute attack of dysentery.

Social History.—The patient is single, a bookkeeper by occupation. At the outbreak of the Spanish-American War he enlisted in the United States Army, and was sent to the Philippine Islands, where he remained for seven months. While there he

suffered from malaria and from dysentery, and upon returning to the United States he found he was unable to resume his usual vocation.

Present Illness.—Following a ten days' stay in the hospital in Manila for what he described as an acute attack of dysentery, he had two similar, though much milder, attacks during the voyage from Manila to San Francisco. An interval of approximately seven to fourteen days occurred between the attacks of dysentery, during which time constipation was present. After returning to the United States he suffered from constipation, which was obstinate at times. When first seen he was so weak as to be scarcely able to walk about the room. The appetite was poor, and he complained of indigestion and of extreme shortness of breath following exertion. He was annoyed by ringing in the ears and by occasional attacks of cardiac palpitation. He slept well and did not display any well-marked nervous symptoms.

It was not until three months after he had reached this country that a dull, boring pain developed in the region of the liver, which later became intense. Upon one occasion he suffered from paroxysmal pain that was followed by the general symptoms of shock, from which he rallied a few hours later.

Upon reaching America he had a more or less chronic cough, which became gradually worse until the rupture of an amebic abscess into the lung took place, following which the cough became persistent, being frequently accompanied by bloody expectoration.

The temperature ranged between 97.3° and 99.6° F., and at no time was there high fever.

Physical Examination.—*General.*—The skin presented a cachectic appearance, and hung in folds, as the result of emaciation. Upon careful questioning it was learned that the patient had lost 40 pounds within the past six months.

Local Examination.—*Inspection.*—The conjunctivæ appeared to be slightly jaundiced, the hair poorly nourished, and the abdomen scaphoid. For a few days preceding the attack of paroxysmal pain in the region of the liver there appeared to be limited expansion at the base of the right chest. The apex impulse was rapid, and, following exertion, there was some pulsation over the greater portion of the precordial region. The tongue was heavily coated, and the mucous membrane of the mouth appeared to be unusually dry.

Palpation.—Firm pressure over the region of the liver produced a dull pain that radiated along the right border of the sternum. The pulse was weak, the beats numbering 100 to 110 a minute.

Percussion.—The inferior area of liver dullness was limited to the right costal margin, whereas the superior boundary of hepatic dullness was at the lower border of the fourth rib, in the nipple-line.

Auscultation.—The heart-sounds were clear, although not strong, and when the stethoscope was placed over the base of the organ, a soft systolic murmur was audible, but this murmur was not transmitted along the route where true organic murmurs are usually heard. Over the base of the right lung anteriorly numerous, fine, crackling râles were heard, and after the attack of paroxysmal pain, many coarse bubbling râles were audible in this region.

Laboratory Findings.—During his stay in the hospital at Manila the feces contained considerable blood, and amebæ were also present. No examination of the feces was made during the attacks, which occurred while traveling from the Philippines to the United States, and which were separated by an interval of approximately two weeks. After reaching this country the feces were examined repeatedly, but at no time was it possible to demonstrate the presence of amebæ. Following the paroxysmal pain at the base of the right chest he expectorated several ounces of blood-streaked, mucopurulent material, a microscopic analysis of which showed it to contain active amebæ, red blood-cells, leukocytes, and shreds of partially necrotic tissue.

The urine was high colored, had a specific gravity of 1.016, and gave a decided reaction for indican. The patient's sputum continued to show amebæ for a period of about seven months, since which time it has not been examined.

When first seen the blood showed: hemoglobin, 64 per cent.; red blood-cells, 2,110,000. The leukocytes were not estimated before rupture of the abscess, but while studying smeared blood a differential leukocyte count showed 6.2 per cent: of eosinophile cells. Four months following the rupture of the abscess into the lung the red cells numbered 4,200,000; hemoglobin, 82 per cent.; leukocytes, 8700.

Course of the Disease.—The condition doubtless began with the original attack of dysentery while in the Philippine Islands, and the fact that amebæ were not found in the stools ten weeks later suggests that the intestinal process had possibly subsided by that time. The patient presented the further evidence that while in the hospital he was given repeated rectal injections of a quart of water which, the attendant said, contained

quinin. The profound anemia from which he was suffering disappeared gradually after the rupture of the amebic abscess of the liver with the escape of its contents through the lung, although hematinics and highly nutritious foods were administered at the same time. The patient was restored to apparent health within the course of approximately two years.

Summary of Diagnosis.—Given a patient who has resided in the tropics and who presents the typical clinical history, *e. g.*, repeated attacks of dysentery alternating with constipation, progressive anemia, and marked emaciation, and the diagnosis becomes quite clear. It may be substantiated, however, by the detection of amebæ in the feces. The presence of anemia and of eosinophilia is of some value in formulating a diagnosis.

Differential Diagnosis.—Amebic dysentery is to be distinguished from **chronic dysentery.** In the latter the degree of emaciation and of prostration is but slight, the quantity of material dejected at each stool is large, and the ameba is not found in the feces.

Duration and Clinical Course.—Uncomplicated cases that tend toward a favorable termination go on to recovery in about ten weeks. The course of the disease may be greatly curtailed by treatment. It must be remembered that the tendency is always toward repeated relapses, and that a relapse may occur when the patient is apparently cured. In relapsing cases convalescence is protracted over a long period, and the anemia and emaciation continue.

The *mortality rate* in certain epidemics has been found to range between 70 and 80 per cent., whereas in others it may fall to from 10 to 15 per cent. "In sporadic cases the mortality rate in temperate climates is not over 5 or 6 per cent" (Anders).

Complications.—Among the complications that occur in amebic dysentery are peritonitis, pyemia, malaria, hepatic abscess, bronchopneumonia, and hepatopulmonary abscess. Hemorrhage from the bowel has been reported.

Hepatic Abscess.—This complication may develop at any stage of the disease. In subacute cases it is prone to occur in from the fourth to the twelfth week. The abscess often occupies the convex surface of the liver, near the coronary ligament. In these cases the lung is also likely to become involved. Abscess of the liver is dealt with in the section devoted to Hepatic Diseases, but it is important to note here that hepatic symptoms may occur in cases in which, on account of the mildness of the attack, the local intestinal disturbance may have escaped observation.

Hepatopulmonary Abscess.—The character of the expectoration points conclusively to the nature of the lung complication. A history of a dry, hacking cough, with the sudden expectoration of a large quantity of gelatinous or mucoid and bloody material, is suggestive of hepatopulmonary abscess. In typical cases the expectorated material is of a brown or light chocolate color.

The sputum may be bile-stained, and when studied microscopically under a $\frac{1}{6}$-inch objective, it often shows liver-cells, bile-pigment, and crystals. The amebæ are readily seen with the $\frac{2}{3}$-inch objective, but the $\frac{1}{6}$ gives better magnification. The first expectoration from an amebic abscess does not contain bacteria unless the abscess has become infected secondarily. After the patient has been expectorating the contents of the liver abscess cavity for a time, secondary infection occurs, and then the expectorated material will also be rich in pyogenic bacteria. With the increase in the number of bacteria in the sputum the number of amebæ present is reduced. In a patient seen at the Medico-Chirurgical clinic (Philadelphia) amebæ were pres-

ent in the sputum for a period of nearly nine months. This patient is at present in perfect health.

FLAGELLATA.

"Members of the group flagellata or mastigophora are characterized by the fact that each organism displays from one to eight flagella. These flagella, by their active movements, render the animal capable of locomotion" (Fig. 343). Infection with these worms is common in temperate climates, as well as in the tropics.

BALANTIDIUM COLI.

This is an oval organism that measures about 1 mm. in its greatest diameter. It differs from the flagellata in that its entire body is covered with

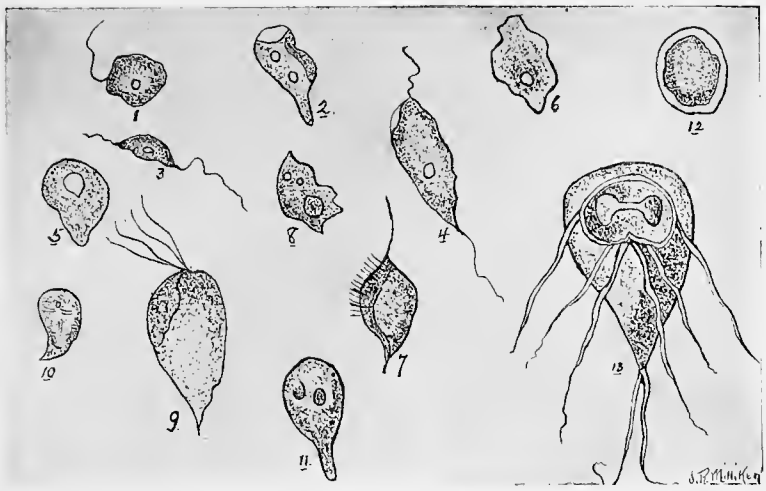


FIG. 343.

1, 2, 3, 4, 5, 10, and 11, Various forms of Cercomonas intestinalis (after Leuckart and Lamb); 6, 7, 8, and 9, various forms of trichomonas (after Scanzoni and Kölliker and Dock); 12, encysted form, and 13, adult form, of Megastoma entericum (Cercomonas) (after Grassi and Schewiakoff).

fine cilia, which are thickest about the mouth of the parasite, and thinly distributed over the remainder of the body. The balantidium coli has a pale nucleus, and from two to four distinct vacuoles (Fig. 345).

Within the body of the parasite small particles of starch may be seen, and at times droplets of fat are detected.

Clinical Significance.—Infection of man with the balantidium coli is believed to have its origin in the dejecta of swine. In all, 89 cases of persistent diarrhea due to this parasite were reported in 1904. The balantidium coli has been recovered from the feces in persons suffering from this infection, and at a time when other intestinal parasites were also present.

CESTODES.

TAPE-WORMS.

These parasites inhabit the small intestine, where they give rise to considerable irritation, which results in the development of a variable amount

of intestinal catarrh. As the result of this condition, body depletion, toxemia, nervous manifestations, and progressive anemia may follow. Parasitic tape-worms in man include species belonging to the family Tæniidæ and to the family Bothriocephaloidæ. There are eight species that are known to be parasitic for man.

The segments (proglottides) of the tape-worm vary greatly in size (Fig. 347). The longer the tape-worm, —that is, the further it is from the head,—the larger is the segment. Passing from the largest segment, it will be found that each segment is smaller until the neighborhood of the head of the parasite is reached, where the segments appear to the naked eye as a slightly flattened thread. The segments of the tape-worm are of a yellowish-white or bluish-white color.

Fig. 344.—Eggs of Tænia Saginata (Mosler and Peiper).

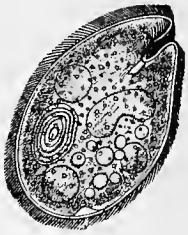


Fig. 345.—Balantidium (Paramœcium) coli (Eichhorst).

TAENIA SAGINATA (BEEF TAPE-WORM).

This worm is four or five meters long. The head is pigmented (Fig. 346), and the segments are long and fat (Fig. 347). The head is supplied with four powerful sucking cups, but has no rostellum or crown of hooklets. The uterus in the ripe segment is finely branched, and these segments are capable of *independent movement.* The eggs are characteristic (Fig. 344).

TAENIA SOLIUM (PORK TAPE-WORM).

The parasite consists of a number of segments and a single head (Fig. 350). When the segments are introduced into the stomach of an animal they undergo partial digestion by the animal's juices, the ova liberating their young in the intestinal canal; these young immediately find their way through the intestinal wall into the body-tissues of the animal, which now becomes their intermediary host. In the animal's tissues the young tape-worm develops only as far as the head, becomes encysted, and remains quiescent here until the flesh of the animal containing such cyst is ingested by a second animal, possibly man. In the stomach or the duodenum of the second animal the cyst is digested and the head of the tape-worm liberated. The liberated head then fastens itself to the mucous membrane of the intestine, and here rapidly develops its characteristic segments (Fig. 349).

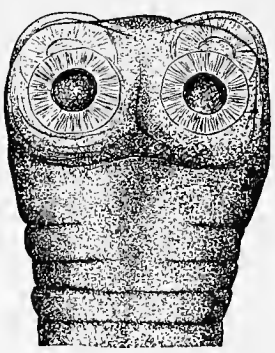


Fig. 346.—Head of Tænia Saginata (Mosler and Peiper).

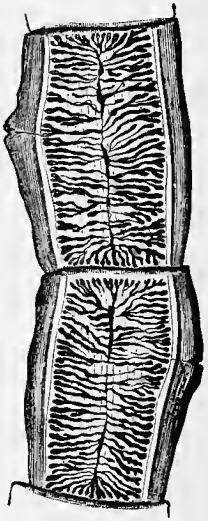


Fig. 347.—Mature Segments of Tænia Saginata.

Life Cycle.—When the segments of the mature tape-worm are introduced into the human stomach, cystic formation in the body-tissues follows, and the parasite is then known as the cysticercus. A fact to be borne in mind is that persons having matured segments of any form of tape-worm in their intestinal canal may, as the result of violent retching or vomiting, regurgitate some of the segments into the stomach, where, suspended in an acid medium, the ova are liberated and develop into scolices and then go on to form cysticerci. Autoinfection with the beef tape-worm is by no means uncommon, and a knowledge of the symptoms to which it gives rise is of great clinical value.

FIG. 348.—EGGS OF TÆNIA SOLIUM.

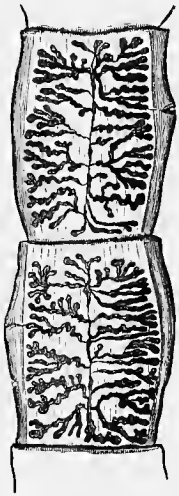


FIG. 349.—MATURE SEGMENTS OF TÆNIA SOLIUM.

Principal Complaint.—As a result of infection with any form of tape-worm there is an abnormal appetite, and a peculiar parched condition of the throat and mouth is present. A variable degree of mental hebetude, constipation alternating with diarrhea, and the passing of segments of the parasites (Fig. 349) from the rectum are observed.

If the parasite has been harbored for months or even years, a high-grade anemia is present, and this anemia may, in some instances, closely resemble one of the essential blood diseases.

Persons infected with cestodes become extremely nervous and irritable.

Physical Signs.—Inspection.—The skin is pale, often of a lemon-yellow or greenish hue, and the conjunctivæ generally show small, milk-white areas, due to deposits of fat. The abdomen is, as a rule, scaphoid in shape.

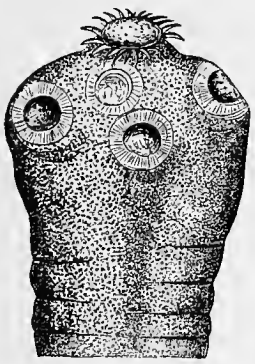


FIG. 350.—HEAD OF TÆNIA SOLIUM (Mosler and Peiper).

Laboratory Diagnosis.—The detection of segments (proglottides, Fig. 349) or of ova in the feces is the only positive evidence of infection with a cestode.

Ova.—The ova of the tænia saginata (Fig. 348) closely resemble those of the tænia solium. The slight difference in size is no guide to the diagnosis unless measurements are taken. The ova escape with the feces.

DIBOTHRIOCEPHALUS LATUS (FISH WORM).

Description.—A form of worm common in all countries bordering upon the Baltic Sea, in the vicinity of Lake Geneva, and in Holland. This parasite may attain a length of from one to five meters, the average size found being two meters, or about seven feet, in length.

Proglottides.—The mature proglottides do not escape from the rectum singly, but the segments are, as a rule, passed in large numbers, one foot or more of the worm being passed at a time. The individual segment is very thin at a point near the head. The segments gradually increase

in size, the largest being those situated farthest from the head. The small segments appear to be greater in length than in breadth, whereas the medium-sized ones are almost square. In the center of each segment is a dark or slightly bluish spot, indicating the position of the genital pore.

Head.—The head is 2 or 3 mm. long by about 1 mm. broad. It is perfectly ovoid in contour, and closely resembles the expanded portion and handle of a spoon. It has two suckers, resembling slits, on the lateral margin of the head.

To the naked eye the head of this worm corresponds in size to that of a small pin. It may be of a light-gray or pearl-white color, and is sometimes opalescent.

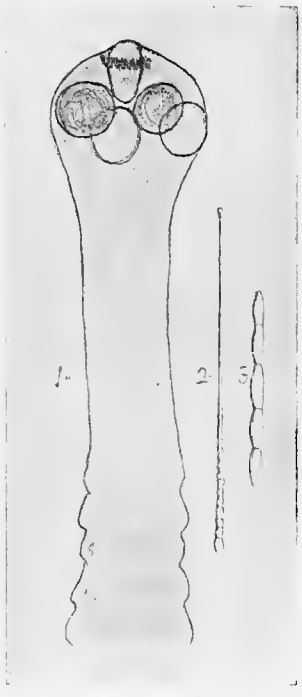


FIG. 351.—HYMENOLEPIS NANA FROM INTESTINE OF A CAT (Boston)

1, Head and neck (obj. B and L. two-thirds); 2, head and neck (natural size); 3, largest segments (natural size).

Ova.—In fully matured segments the body of the uterus is seen to be so packed with ova that the center of the segment protrudes slightly. The detection of ova in the stool of man is of great diagnostic value. These ova are elliptic or ovoid in contour, and, as a rule, are of a muddy-white, brownish-white, or brown color. They vary in length from 0.06 to 0.07 mm., their width being usually equivalent to about one-half their length. With a one-sixth or a one-eighth inch objective a faint hyaline band may be seen at one end of the ovum, outlining an apparent lid.

HYMENOLEPIS NANA.

This is a parasite occasionally encountered in the intestinal canal of man, but for more commonly seen in the intestine of the lower animals. It measures from 10 to 15 mm. in length.

Geographic Distribution.—The parasite is common in Italy, Egypt, and along the shores of the Mediterranean Sea. Deaderick found it to be quite common in the State of Arkansas, he having detected the ova of this parasite 8 times in 40 specimens of feces examined.

Description.—Proglottides and Ova.—The proglottides of the hymenolepis nana may be clearly seen in the accompanying illustration (Fig. 351). The ova are numerous and slightly opalescent oval bodies, enveloped in a distinct membrane.

Head.—The head of the parasite differs markedly from the heads of other tape-worms previously described, being more or less pear-shaped, and displaying four suckers and a club-shaped rostellum (Fig. 351). It contains from 24 to 30 hooklets, which are arranged in a single row to form a crown at the anterior portion of the head, instead of being inverted, as is shown in the illustration (Fig. 351).

HYMENOLEPIS DIMINUTA.

This parasite was first described by Leidy, and in 1900 Packard reported the ninth case of infection in man.

The parasite varies in length between 25 and 60 mm. The head is provided with four quite well-marked sucking cups. The ova resemble those of the tænia solium (Fig. 348).

TAENIA MARGINATA.

This parasite resembles in certain respects both the beef and the pork tape-worms, and has occasionally been known to infest the intestinal canal of man.

DIPYLIDIUM CANINUM.

Description.—The dipylidium caninum, or dog tape-worm (Figs. 353 and 354), belongs to a family of intestinal parasites rarely encountered in man, but commonly attacking the cat and the dog. Its segments, which are elliptic, elongated, tape-like bodies, are not easily confused with those of other intestinal parasites. The larval stage of this worm develops in

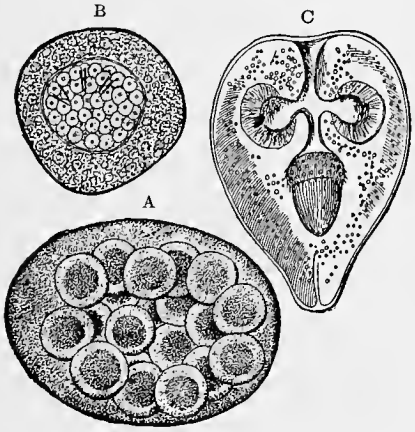


Fig. 352.

A, Egg packet of Dipylidium caninum; B, egg of same—six-hooked embryo (after Stiles); C, Cryptocystis tricodectis, as found in the flea (after Leuckart).

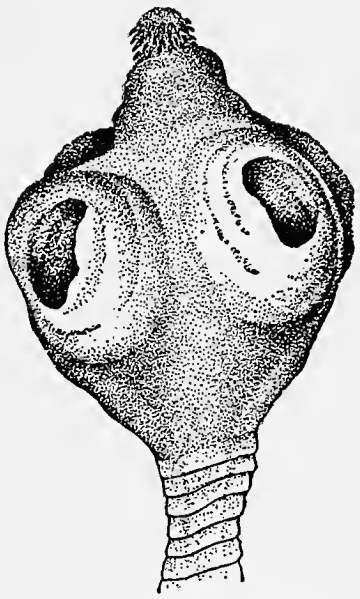


Fig. 353.—Head of Dipylidium Caninum (Stiles).

Showing four rows of rose-thorn hooks on the rostellum and four unarmed suckers.

lice and in fleas (Fig. 352). Stiles states that the dipylidium caninum is one of the smaller tape-worms, but should be looked upon as a pathogenic parasite, as it sometimes burrows into the intestinal mucosa.

The **head** of the parasite shows four sucking cups and a rostellum, surrounded by four rows of hooklets.

Ova.—The ovum of dipylidium caninum differs markedly from that of any form of tenia known to infest man. "In the genus Tænia we find a thick, striated inner shell (embryophore), while in dipylidium the inner shell is thin" (Stiles). But few ova are found in the feces of persons infected with this parasite.

TAENIA MADAGASCARIENSIS (Grenet).

Description.—A form of tape-worm found to infest persons residing on the eastern coast of Africa. It may attain a considerable length, its segments reaching a maximum number of 600.

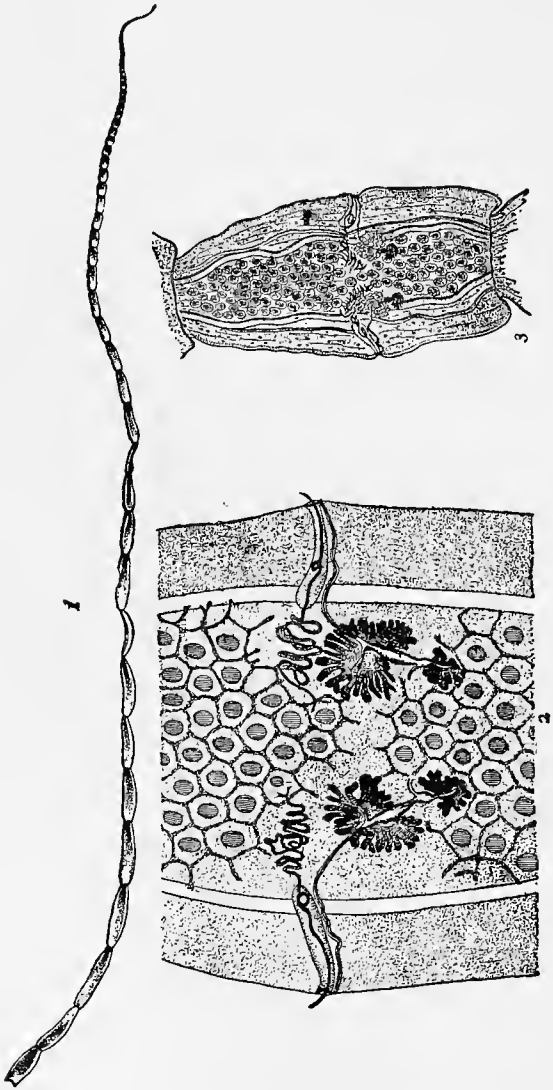


FIG. 354.—DIPYLIDIUM CANINUM.
1, Adult strobila (natural size); 2, segments (Stiles) (highly magnified); 3, ground segment (after Diamari).

A distinguishing feature of tænia Madagascariensis is that its segments are trapezoid, its rostellum being surrounded with a double row of hooklets, and the sucking-cups being well defined.

Under this name another type of tape-worm has recently been reported from eastern Africa. A single case has been reported.

Fig. 355.—Tænia Echinococcus, Enlarged (after Heller).

Above, at the right, echinococcus of natural size.

TAENIA ECHINOCOCCUS.

This parasite is another species of dog tape-worm, commonly found in members of the canine family, and rarely in the intestine of man.

In man the larval stage of the tænia echinococcus appears in the form of hydatid cysts. (See also Animal Parasites of Liver, p. 980.)

Description.—The tænia echinococcus is about one-fourth inch in length, and is composed of four segments. The cephalic extremity, which is prolonged to form a well-marked net, is capped by a pointed rostellum. In the center of the head are four well-marked sucking cups (Fig. 355). The rostellum is surrounded by a double row of hooks, numbering between 30 and 40. The last segment, when sexually matured, is as long as the three anterior segments; it is provided with papillæ at the margin of the proglottis, below the central line. The uterus is packed with ova.

General Remarks.—A hydatid cyst is an embryo tape-worm. It consists of a vesicle in which there is a scolex with four sucking discs and six hooklets, circularly arranged. After a time the scolex degenerates, the hooklets are shed, the cyst increases in size, and contains a clear fluid in which the hooklets and some cells may be found. The wall of the cyst is composed of fibrous tissue that gradually becomes dense.

Clinically, echinococcus disease is of great importance because it is a type of infection found wherever man is brought in contact with the dog or the wolf. Infection with this parasite is common in Russia, Finland, Iceland, and Australia.

Sites of Development.—The echinococcus cyst develops in any part of the human tissues, the favorite site, however, being the liver. We have seen echinococcus cysts in the liver and in the lung at post-mortem nine times, and in one case 19 of these cysts were found in the brain.

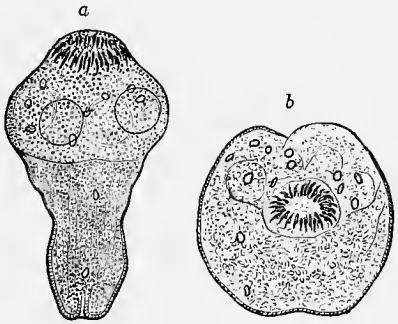


Fig. 356.—Head of Echinococcus (Mosler and Peiper).

a, Head protruded; *b*, head drawn in.

Summary of Diagnosis.—The diagnosis is based entirely on the recovery of fluid from the tumor and the detection therein of hooklets of the echinococcus.

Differential Diagnosis.—Among the conditions that may be confused with hydatid of the liver are: **distention of the gall-bladder, hydronephrosis, syphilis, carcinoma,** and **amebic abscess of the liver.**

The following tables, modified from Anders, outline the distinctive differences between hydatid disease of the liver and disease of the gall-bladder and of the kidney:

Hydatid Cyst.	Dilatation of the Gall-bladder.
1. Previous history negative, except for the companionship of dogs.	1. A history of having passed biliary calculi is often obtained.
2. Pain and jaundice absent.	2. Attacks of biliary colic followed by jaundice either are present or enter into the previous history.
3. Enlargement in any direction is dependent upon the location of the cysts.	3. Enlargement is always in one direction—downward and posteriorly.
4. Tumor is firmly fixed to the liver or other viscera.	4. The tumor is somewhat movable.
5. Aspiration recovers fluid containing Charcot-Leyden crystals and hooklets of echinococcus.	5. Aspiration recovers bile-stained fluid.

Hydatid Cyst.	Hydronephrosis.
1. The history is negative.	1. There is a history of renal calculi or of vesical inflammation.
2. There is no pain.	2. There may be severe pain.
3. The tumor is most prominent over the hepatic area, and is associated with enlargement of the liver.	3. The tumor is most prominent in the flanks and iliac fossæ. If it extends to the right hypochondriac region, it *does not* move with the liver.
4. The duration is indefinite, and the tumor is permanent.	4. The duration is short; a large amount of urine may be passed and the tumor disappear; termination in uremia is common.
5. Aspiration recovers cyst fluid, etc.	5. Aspiration recovers urine.

TREMATODES OR FLUKES.

Trematodes are parasitic for man in nearly all parts of the tropics and in many of the subtropical regions. *Fasciola hepatica* is a parasite of sheep which is occasionally found in man if he has been closely associated with these animals.

Paragonimus Westermanii (see p. 988), *Schistosomum hæmatobium* (see p. 961), *Fasciolopsis Buski*, and *Opisthorchis sinensis* are frequently found in man in certain parts of the world.

These parasites, when present in the intestinal tract or the liver, produce diarrheal attacks, the feces being found to contain blood, mucus, and pus. Abdominal pain and distention, anemia, and cirrhosis of the liver with ascites are important symptoms of infection with these parasites.

The *ova* of the trematodes vary in size, but are characterized by the presence of a lid or operculum, with the exception of those of schistosomum hæmatobium, which are furnished with a spine.

INTESTINAL NEMATODES.

ROUND-WORMS.

Ascaris Lumbricoides.—The common round-worm is from four to twelve inches in length, the females being somewhat longer than the males. They are of a reddish-white or milk-white color and bear a more or less close resemblance to common earth-worms. They inhabit chiefly the small

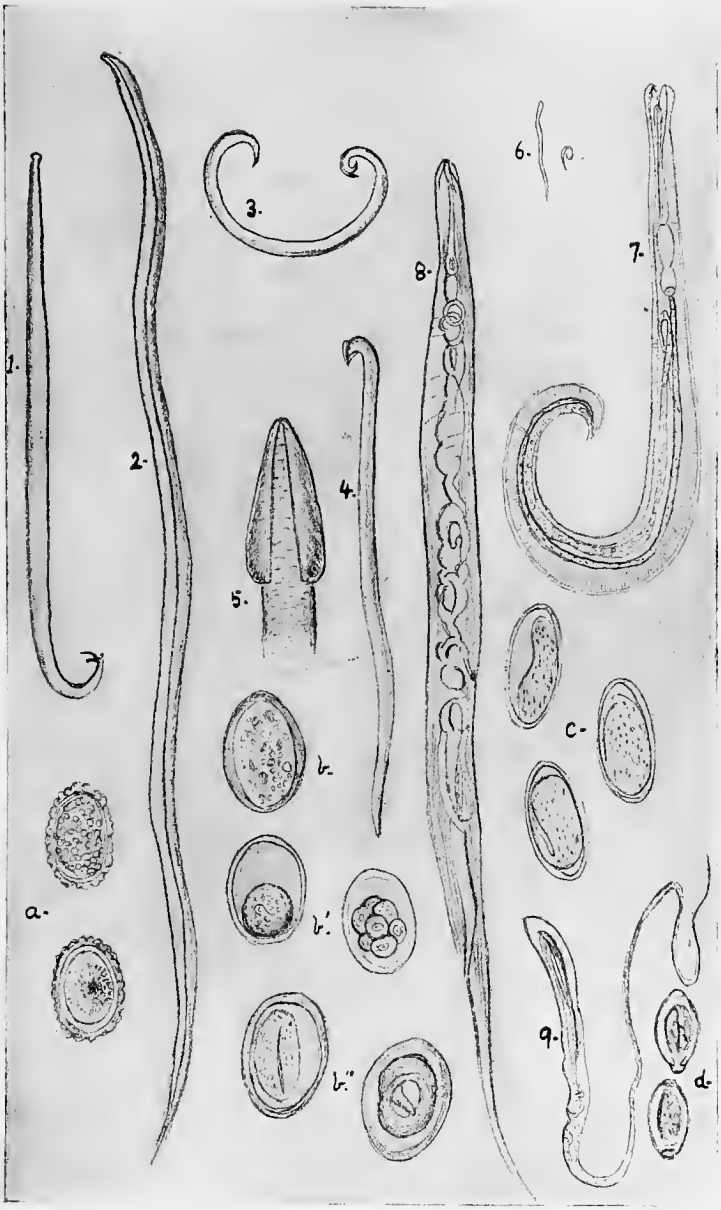


Fig. 357.—Ascaris, Oxyuris, and Trichocephalus

1, 2, and *a*, Ascaris lumbricoides: 1, Male; 2, female; *a*, ova.

3, 4, 5, *b*, *b'*, *b''*, Ascaris canis: 3, Male; 4, female; 5, head of female (magnified); *b*, ovum; *b'*, ova, showing segmentation; *b''*, ova showing embryo (Kobbold).

6, 7, 8, and *c*, Oxyuris vermicularis: 6, Male and female (natural size); 7, male; 8, female (magnified); *c*, ova (Boston) (obj. B. and L. one-sixth).

9 and *d*, Trichocephalus dispar: 9, Female (magnified); *d*, ova (obj. Queen one-sixth) (Boston).

intestines, but frequently migrate to the stomach, large intestine, or even to the gall-bladder.

Several parasites may be present in the intestine at one time, and in exceptional instances many hundreds may be found. They gain entrance into the system by means of their ova, which are swallowed with the food; the envelopes surrounding the ova are dissolved by the gastric juice, and the embryos are thus set free.

Principal Complaint.—The symptoms produced by the presence of round-worms cannot be clearly distinguished from those due to gastro-intestinal catarrh, with which the ascarides are so commonly associated. The first sign to draw attention to the disorder is the passage of a round-worm by the rectum.

The presence of one or two worms rarely gives rise to any symptoms unless they pass into the stomach or bile-duct. When, however, *large numbers* exist, they may give rise to colicky pains, coming on especially at night; diarrhea, vomiting, and symptoms of intestinal obstruction have also been observed.

Nervous Symptoms.—There are general nervousness, picking of the clothing and of the face, etc., and in children convulsions are by no means uncommon. In rare instances worms have found their way into the peritoneal cavity, and have been discharged through the abdominal wall, together with the contents of an abscess.

Anemia develops early and is of the secondary type. A differential leukocyte count is of service in making a diagnosis of infection of the intestine with the various parasites, such infection showing, as a rule, an abnormally high percentage of eosinophiles.

Laboratory Diagnosis.—The ascaris lumbricoides is readily detected in the feces (Fig. 357).

Ova.—The ova of the ascaris lumbricoides are easily found in the feces. They are of a yellowish-brown color (Fig. 357), and vary in size, being from 0.06 to 0.07 mm. in diameter.

Oxyuris Vermicularis.—(**Thread-worm.**)—This parasite inhabits the lower bowel—the cecum, colon, sigmoid flexure, and rectum—and the vagina. A diseased condition of the mucous membrane and sluggish bowels favor their development. To the naked eye they appear as short bits of white thread. Under a low magnification the females, which are the most numerous, will be seen to taper at each end; their uterine ducts are filled with numerous ova (Fig. 357), some of which contain embryos.

Predisposing and Exciting Factors.—These parasites gain entrance into the system by means of their ova, which are ingested with the food, or perhaps more frequently by the ova adhering to the fingers of those already affected; they are thus conveyed directly or indirectly to others.

Principal Complaint.—The symptoms are vague, and the diagnosis is usually made by finding the parasites in the stools of the child. The most common symptom is an irritation and itching at the anus or at the vulva. In girls, when vaginal infection exists, there is a discharge of mucus and blood from the vagina.

ANKYLOSTOMIASIS (UNCINARIASIS; HOOK-WORM DISEASE).

Pathologic Definition.—A disease caused by the presence of a nematode worm in the intestinal canal, and characterized by a severe secondary anemia, catarrhal enteritis, and secondary fatty degeneration of the viscera.

Predisposing and Exciting Factors.—This disease was known before the Christian era. Of recent years, and particularly since 1898 in the United States, the disease has assumed great importance, because of its presence in Porto Rico and in the southern States. Two parasites are known to be responsible for the disease: that form seen in Europe, Asia, and Africa is caused by *Ankylostoma duodenale;* that seen in the western hemisphere is caused by a worm, *necator americanus* (Fig. 358).

Age.—Children are chiefly affected, but adults may also harbor the parasite.

Occupation is an important predisposing factor. The infection is known to be transmitted by soil pollution. The ova of the parasite are deposited on the ground with human feces, where they develop into larvæ, particularly in moist, sandy soil. The larvæ gain entrance to the tissues of the human host by burrowing through the skin, upon which they have been deposited with the mud containing them. Consequently, any occupation in which a person is likely to get mud containing larvæ on his hands or feet is one that predisposes to the infection. Hence it is seen most often in miners, brick-makers, farmers, civil engineers engaged in operations in an infected region, laborers, etc. In Porto Rico females afected with the hookworm were found to have contracted the infection by working in their rose-gardens. The larvæ, passing through the skin, produce a pustular dermatitis known in Porto Rico as *mazamorra,* and in the southern United States as *ground itch.*

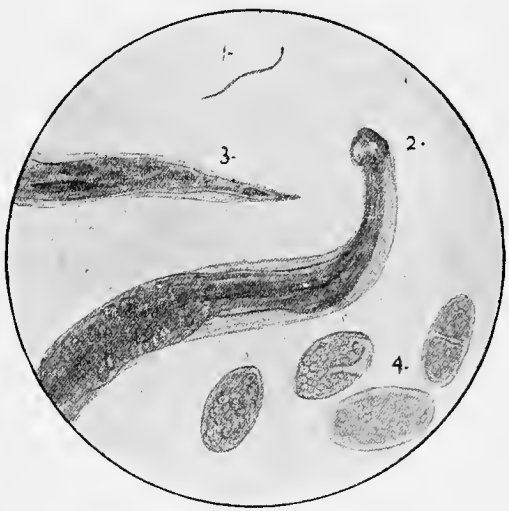


Fig. 358.—Uncinaria Americana (Boston). 1, Female, natural size; 2, head; 3, tail; 4, ova.

Principal Complaint.—The chief symptom of uncinariasis is a marked anemia, the characteristics of which will be described under Laboratory Diagnosis. There is a history of progressive weakness, nervousness, loss of weight, anorexia, perverted appetite, etc. In children infected before puberty physical development is retarded and mental evolution is delayed; the pubic hair fails to appear, the genitals remain infantile, and in girls menstruation is delayed. The symptoms of anemia are prominent: pallor of the skin and mucous membranes, headache, palpitation of the heart, dyspnea on exertion, vertigo, drowsiness, and localized edemas. Constipation, alternating with attacks of diarrhea, is a common symptom. Digestive disturbances are not marked or common; catarrhal stomatitis, salivation, flatulence, heartburn, nausea without vomiting, pain and tenderness in the epigastrium are among the symptoms more frequently observed.

Physical Signs.—Inspection.—The skin is ashy gray and often pigmented; the conjunctivæ and lips are extremely pale; the tongue is flabby and heavily coated. The body shows evidences of emaciation, although

the face is usually full, and in advanced cases there is edema beneath the eyes. According to Ashford, this edema may so distort the face as to make recognition impossible. Edema of the hands and feet and abdominal distention due to ascites are seen late in the disease. The impulse of the apex-beat is indistinct.

Palpation reveals the presence of edema and fluid in the peritoneal cavity. The pulse is weak, irregular, and compressible.

Percussion is negative, unless there is effusion into the serous sacs, *e. g.*, peritoneum, pericardium, and pleuræ. The liver and spleen are not usually enlarged.

Auscultation.—The heart-sounds are rapid and weak, and soft (hemic) murmurs are heard over the base of the organ.

Laboratory Diagnosis.—The total length of the female worm, necator americanus, varies between 9 and 11 mm., whereas the male worm is from 7 to 9 mm. in length. The tapering form of the neck and head, which is slightly turned, is shown in Fig. 358. The female worm tapers gradually, terminating posteriorly in a slightly rounded point (Fig. 358). The tail of the male parasite displays a bursa from which two spicules project.

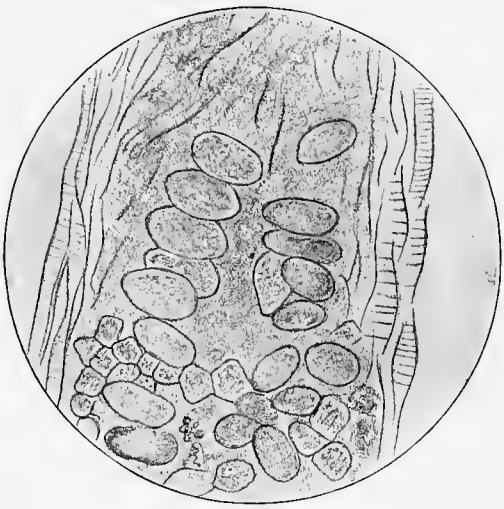


Fig. 359.—Section of Adult Uncinaria (Ankylostoma), Uterus Containing Ova (Boston).

Detection of the Ova in the Feces.—The ova of necator americanus are found in the feces. They measure between 0.064 and 0.075 mm. in length by 0.036 to 0.04 mm. in breadth. They are always deposited with segmentation begun, so that they are filled with two, four, or more segmentation spheres. They have a thin, transparent, clear white shell. The embryo may at times be seen in the shell. The detection of the ova in the feces is positive evidence of infection with the hookworm.

The Blood.—The anemia of ankylostomiasis is a secondary anemia of high grade. The percentage of hemoglobin is markedly reduced—often from 30 to 50 per cent. The red blood-cells are also greatly diminished in number, and show the changes of degeneration, *e. g.*, poikilocytosis, variation in size, unequal distribution of the hemoglobin, granular degeneration and polychromatophilia, and the presence of normoblasts and megaloblasts.

In uncomplicated cases of uncinariasis the leukocytes are usually normal in number, eosinophilia is common, and myelocytes are sometimes present.

Summary of Diagnosis.—The clinical history is in no way typical of infection with the necator nor is the additional fact that the patient has resided in the tropics of special importance. A positive diagnosis is attained only by finding the ova or the adult parasite in the feces.

The presence of eosinophilia is of great value in making a diagnosis of

intestinal parasites, but is not pathognomonic of hook-worm infection. The high grade of anemia and the edema of the face and ankles are suggestive of uncinariasis.

Differential Diagnosis.—Uncinariasis may be mistaken for **leukemia, pernicious anemia,** and **chronic plumbism.** In each of these, however, the clinical history will be found to be vastly different, and the detection of the parasite in the feces will furnish positive differential evidence. The hematologic findings are of service, since leukocytosis is characteristic of leukemia and fairly constant in chronic plumbism. In chronic lead intoxication the urine gives a reaction for lead.

Clinical Course and Duration.—When judicious treatment is instituted early, the disease runs a favorable course, practically all cases going on to recovery within a period of from two to six months. In cases of long-standing infection, and in the presence of profound anemia, the prognosis is guardedly favorable. In those in whom general edema is present there is a great tendency for complications to develop—*e. g.*, bronchopneumonia. It has been estimated that infection by the hook-worm is accountable for more than 20 per cent. of all deaths occurring in Porto Rico.

Fig. 360.—Trichocephalus Dispar (Heller).

a, Female; *b*, male (natural size). (Also known as trichuris trichiuria.)

TRICHURIS TRICHIURIA.

Description.—This parasite is a member of a family of Trichotrachelidæ, which inhabit the cecum in man. It will be found to vary between 40 and 50 mm. in length (Fig. 360).

Ova.—The ovum is of about the same size as the egg of the uncinaria, and measures about 50 to 54 microns in length by 21 to 23 microns in breadth. These ova are distinctly barrel-shaped, and present a light-colored plug at each pole. They are red brown or yellowish in color (Fig. 357).

Clinical Significance.—Trichuris trichiuria is a common intestinal parasite. It is usually productive of no symptoms.

STRONGYLOIDES INTESTINALIS.

This parasite is commonly found in the feces of persons residing in tropical and subtropical countries. In recent years it has been found by A. J. Smith, Claude A. Smith, Daland, Woldert, and Thayer in the States bordering on the Gulf of Mexico.

TRICHINIASIS.

Remarks.—Embryos of the trichina spiralis may appear in the intestine and escape with the feces during infection with that parasite. The adult parasite may also be found during the initial diarrhea. (See Trichiniasis, p. 982.)

ANIMAL PARASITES OF THE LIVER.

ECHINOCOCCIC DISEASE.

Pathologic Definition.—An affection of the liver characterized by the formation of a multilocular cyst containing a limpid fluid in which hooklets of the encysted scolex of tænia echinococcus are found.

Principal Complaint.—The majority of cases of echinococcus disease of the liver are discovered at autopsy, the symptoms being vague except in those cases in which the cysts reach a large size. Generally speaking, the only symptoms of echinococcus cysts are caused by pressure upon the bile-ducts or upon adjacent structures. Marked pressure upon the portal vein and the bile-duct may be followed by the development of ascites and jaundice. Occasionally there is a distinct history of the tumor having disappeared suddenly, the patient having passed by the rectum a large quantity of peculiar looking material at the time of its disappearance. It is doubtless this form of cyst that ruptures into the colon, nature in this way effecting a cure. When an echinococcus cyst becomes infected with pathogenic bacteria the symptoms described under Hepatic Abscess appear. (See p. 587.)

When the cyst occupies the superior surface of the liver and forces the diaphragm well up against the lungs, coughing results. Spontaneous rupture through the diaphragm may occur, and in such cases the patients expectorate the contents of the cyst and a cure is effected.

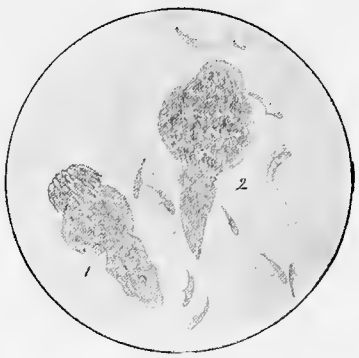


FIG. 361.

1, Scolex of Tænia echinococcus, showing crown of hooklets; 2, scolex and detached hooklets (obj. B. and L. one-sixth) (Boston).

Physical Signs.—Beyond revealing the fact that the cyst is unusually large, **inspection** is negative.

Palpation may be of great value when the cyst is large and exhibits fluctuation. By making deep palpation over one portion of the cyst and percussing over another the palpating hand occasionally detects a peculiar thrill (hydatid thrill), which many writers regard as pathognomonic of the disease. Splenic enlargement is quite commonly associated with hydatid disease.

Percussion, as previously stated, will elicit the "hydatid thrill," or fremitus, and also confirms the findings of palpation as to the size of the spleen and the liver. Movable dullness shows the presence of ascites.

Auscultation.—Upon placing the stethoscope over one portion of the cyst and percussing over a distant area, a peculiar short, sharply defined, booming sound will be obtained (Santoni).

Laboratory Diagnosis.—**Aspiration** of the cyst usually results in the recovery of a fluid that contains the hooklets and scolices of the tænia echinococcus (Fig. 361). In those cases rupturing through the diaphragm and communicating with a bronchus, hooklets are found in the sputum. We have detected both scolices and hooklets of the tænia echinococcus in the dejecta of two patients in the Philadelphia Hospital after a cyst had probably ruptured into the colon. In a third case hooklets of the tænia echinococcus were found in the urine, and within forty-eight hours the degree of hepatic enlargement was decidedly reduced. In the latter case it was believed that the hepatic cyst had probably ruptured into the pelvis of the right kidney. These cases went on to recovery, and the laboratory findings were the only positive evidences displayed.

Summary of Diagnosis.—The diagnosis is based largely upon the presence of hepatic enlargement without tenderness, pain, or fever. The

detection of the so-called hydatid fremitus is also of value. Aspiration of the cyst and the finding of hooklets in the cyst fluid constitute the only conclusive evidence unless either hooklets or scolices of the tænia echinococcus are found in the sputum, urine, and feces. The cysts may rupture externally, when the discharge will be found to contain cyst products. The presence of shreds of cyst membrane also furnishes conclusive evidence as to the nature of the disease.

Differential Diagnosis.—Hydatid disease of the liver must be distinguished from **dilatation of the gall-bladder,** from which condition it is readily differentiated by the fact that jaundice is more common in gall-bladder disease. The history is also of great importance in differentiating these two conditions, since previous hepatic disease is common in enlargement of the gall-bladder. Again, dilatation of the gall-bladder is always discovered at one anatomic point, and extends below the surface of the liver, whereas a hydatid cyst is more nearly oval in contour, and usually develops at a point near the center of the right or the left hepatic lobe. Cysts are more common upon the superior surface of the liver.

The distinctive features between hydatid cysts of the liver and **hydronephrosis** are shown in the accompanying table:

Hydatid Cyst.	Hydronephrosis.
1. The history is negative.	1. There is a history of renal calculus or of vesical disease.
2. Pain is absent.	2. Pain is common when the tumor is large.
3. The tumor is more prominent over the hepatic area, and is associated with enlargement of the liver.	3. The tumor is most prominent in the flank and the iliac fossa. If it extends to the right hypochondriac region, it *does not* move with the liver.
4. There is no history of the patient having voided a large quantity of urine with disappearance of the tumor.	4. There is a history of the voiding of a large quantity of urine followed by disappearance of the tumor.
5. Auscultatory percussion shows the tumor to be connected with the liver.	5. The tumor is not attached to the liver.
6. The duration is indefinite, and uremia is absent.	6. Uremia is a common termination.

In rare cases an echinococcus cyst may resemble clinically a **unilateral pleural effusion of long standing,** but the distinction is made clear by examination of the aspirated fluid. (See Laboratory Diagnosis, p. 980.)

Other Cysts of the Liver.—Other cysts may develop in the liver, but seldom attain sufficient size to be of clinical importance, and there is no means of recognizing them unless they press upon the bile-passages or adjacent vessels. Clinically speaking, cysts of the liver are rare, Lipmann's analyses of the literature showing but 16 cases, 3 of which were retention cysts, 9 cystic adenomata, and 1 a chylous cyst.

LIVER FLUKES.

General Remarks.—The ordinary liver fluke (fasciola hepatica) inhabits the hepatic ducts in man, and is also quite commonly seen in sheep, deer, swine, and the bovines. The ova of the parasite collect in the bile-ducts, and, together with the parasites, may cause a marked dilatation along certain portions of their course. When there is an acute inflammation of the lining membrane of these expanded ducts, the patient is likely to become jaundiced.

Among other types of trematodes (flukes) occasionally encountered in man are dicrocœlium lanceatum, fasciolopsis Buski, and opisthorchis sinensis.

Diagnosis.—This is based on the detection of the ova of the particular liver fluke in question in the feces.

NEMATODES (ROUND-WORMS).

Round-worms may enter the common bile-duct and cause obstruction, giving rise to acute obstructive jaundice. Such cases are rare.

AMEBIC ABSCESS OF THE LIVER.

Pathologic Definition.—A condition excited by the entamœba histolytica, and secondary to amebic dysentery. It is characterized by extensive destruction of the hepatic tissue, with the formation of pus.

General Remarks.—Amebic abscess may develop at any time during the course of amebic dysentery, but is most likely to appear after the disease has become chronic. Residence in the tropics and a previous attack of dysentery are among the predisposing factors.

There is progressive emaciation, together with a high grade of anemia, although the blood-findings are not characteristic. The recovery of amebæ from the stools points strongly toward the existence of hepatic abscess, when there is questionable enlargement of the liver.

In those abscesses developing on the superior surface of the liver there is likely to be cough, and the abscess may rupture into a bronchus.

ANIMAL PARASITES OF THE MUSCLES.

TRICHINIASIS.

Pathologic Definition.—A disease caused by infection of the intestinal tract with the trichinella spiralis. It is characterized by the deposit of embryos of the trichinella in the muscles and by eosinophilia.

Remarks.—Trichiniasis is primarily a disease of the rat.

Sources of the Trichinella.—The parasite was first found in pork—the usual source of transmission to man. The swine become infected by eating diseased rats, trichinous meats, or human dejecta containing embryos of the trichinæ. About 2 per cent. of hogs are found to be trichinous. Man is infected by eating raw or partially cooked meat containing the encysted larvæ of the trichinæ (Fig. 364).

Principal Complaint.—There is usually a history of having eaten raw or partially cooked pork or other meats, followed in from two to five days by anorexia, nausea, vomiting, and cramp-like pains in the abdomen. The patient always suffers from headache and insomnia. If the amount eaten has been large, vomiting and diarrhea are severe. Following the acute intestinal symptoms the patient appears to improve slightly for a period of from ten to fifteen days, when the embryos begin to migrate, and a class of symptoms appear that are in many instances misleading. There may be a series of chills, chilly sensations, or a distinct rigor, followed within a few hours by fever ranging between 100° and 104° F. Within two or three days after the chill, distressing polymyositis appears. Practically all the muscles are stiff. There is a variable degree of muscular spasm, and the muscles of the calf and of the frontal region are extremely tender. There is intense soreness at the base of the chest, and agonizing pain upon deep inspiration—a

symptom due probably to the presence of great numbers of larval trichinæ in the fibers of the diaphragm. Dyspnea is at times extreme.

The muscles of mastication are sore, and the patient is unable to open his mouth, and even swallowing may give rise to pain.

Thermic Features.—The temperature usually falls to near the normal

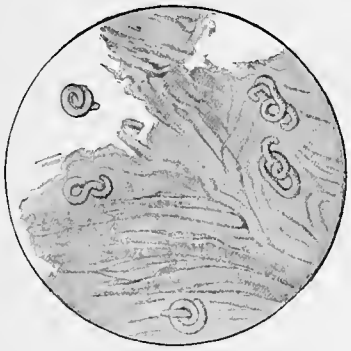


Fig. 362.—Trichinella Spiralis (Larvæ) from Head of Right Gastrocnemius Muscle; Seventh Week of Disease (Boston).

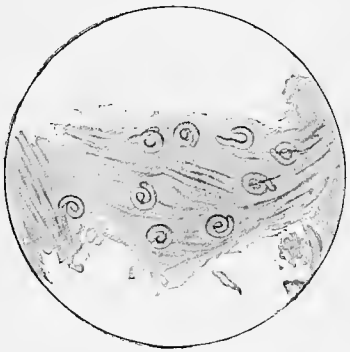


Fig. 363.—Trichinella Spiralis (Larvæ) from Outer Head of Left Gastrocnemius Muscle; Twenty-first Day after Symptoms (Boston).

within from six to fourteen days. The pulse, as a rule, is increased in proportion to the degree of fever.

Physical Signs.—**Inspection.**—The occipitofrontalis and the jaw muscles are swollen, and the calf muscles and those of the arms are similarly affected, the greatest degree of swelling taking place near the tendinous insertion of the muscles. There is swelling of the frontal region and of the eyelid and face. The conjunctivæ are greatly congested, and minute hemorrhages beneath the conjunctiva are not unusual. The tendon reflexes are diminished or absent. Urticaria, herpes, and pruritus are present. The pupils are dilated.

Palpation.—Firm pressure exerted over any group of muscles gives rise to pain. The tendon-reflexes are lessened or abolished.

Fig. 364.—Encapsulated Trichina from Muscle One Year after Infection (Boston).

Laboratory Diagnosis.—There are but three points of special interest in the laboratory diagnosis—(1) The detection of the adult or embryo trichinæ in the dejecta during the initial attack of diarrhea; (2) the discovery of the embryos in the patient's muscle tissue in from twelve to twenty days after the initial diarrhea (Figs. 362 and 363); and (3) blood findings (p. 984).

Methods for Detection.—The skin should be cleansed thoroughly near the tendinous insertion of one of the calf muscles, and the skin and fascia

then divided down to the muscle-sheath; the sheath is next incised, and a small portion of the muscle removed. This removed tissue should be placed in water and a portion of it teased thoroughly, placed upon a slide, and studied under a ⅔ inch objective 'when, if larval trichinæ are present, they will be detected among the muscle-fibers. Several months after the initial symptoms the larvæ are found to be encysted (Fig. 364).

Persons infected with trichinella spiralis display a high degree of eosinophilia—from 20 to 40 per cent.; the total number of leukocytes may, however, remain near the normal. The blood findings are: (*a*) eosinophilia, and (*b*) the detection of the embryo-trichinelli, in the peripheral blood.

Herrick and Janeway first demonstrated embryos of the trichinella spiralis in the circulating blood, and their observations have been confirmed by a number of writers, including A. H. Lamb. Embryos are present in the circulating blood a few days after the onset of symptoms. Experimentally, embryos are found to enter the blood in from eight to twenty-five days after infection. The embryo is cylindric, refractile, and curved, with both ends rounded, one extremity being slightly tapered and surrounded by a hyaline capsule.

Detection.—Dilute the blood with ten times its volume of three (3) per cent. solution of acetic acid. Shake well, and after the sediment forms lift a portion of it into a pipet for examination. Place on a slide, cover with a thin glass, and examine under a one-sixth (⅙) objective. At times it may be well to close down the diaphragm, since these highly refractile bodies are often best seen under a rather feeble illumination.

Summary of Diagnosis.—A history of having eaten raw or partially cooked meat should always be regarded as of importance when coupled with the general symptoms of muscular rheumatism. Edema over the muscles, and particularly over the frontal region, is of great diagnostic value. A girdle pain following an attack of gastro-intestinal catarrh, accompanied by diarrhea and vomiting, should give rise to the suspicion of infection with the trichinella spiralis.

The diagnosis is substantiated by the detection of the larval trichinæ in the muscle tissue (Fig. 363) and blood.

Differential Diagnosis.—The accompanying table shows the distinctive features between acute trichiniasis, acute articular rheumatism, and acute muscular rheumatism:

TABLE SHOWING THE POINTS OF DIFFERENTIATION BETWEEN ACUTE TRICHINIASIS, ACUTE ARTICULAR RHEUMATISM, AND ACUTE POLYMYOSITIS.

Acute Trichiniasis.	Acute Articular Rheumatism.	Acute Polymyositis.
1. There is a history of having eaten raw or partially cooked meats (pork).	1. History of previous attacks.	1. History of exposure to cold and wet.
2. Preceded eight to fourteen days by gastro-intestinal disturbances, diarrhea, nausea, vomiting, and cramps.	2. Gastro-intestinal symptoms absent.	2. Gastro-intestinal symptoms absent.
3. Edema of the forehead and face seen early.	3. Edema absent.	3. Edema absent.
4. Swelling of muscles near tendinous insertions occurs late.	4. Muscles not swollen.	4. Swelling of body of muscles appears early.

Acute Trichiniasis	Acute Articular Rheumatism.	Acute Polymyositis.
5. Tenderness over the body of the muscles and near their tendinous insertions.	5. Tenderness over the articular surface of the long bones, and may involve the small joints.	5. Tenderness over the body of the muscles.
6. Joints not swollen.	6. Joints swollen, red, and tender.	6. Joints not swollen.
7. Effusion into the serous sacs of the larger joints absent.	7. Effusion into the serous sacs of the larger joints and fluctuation a common sign.	7. Effusion absent.
8. Dyspnea pronounced, and girdle pain upon deep inspiration.	8. Dyspnea not marked, and no girdle pain.	8. A sense of soreness over the chest.
9. Eosinophilia develops with the muscular symptoms (4 to 8 per cent. or higher). Embryos found in blood.	9. Eosinophilia not constant and seldom high.	9. Eosinophilia absent, as a rule.
10. Detection of adult trichinella in the dejecta during the initial attack of diarrhea.	10. No trichina in the dejecta. Constipation present early.	10. Feces negative.
11. Larval trichina present in the muscle tissues.	11. Muscle tissue normal.	11. Muscle tissue normal.
12. Conjunctivitis.	12. Rare.	12. Unusual.

Clinical Course and Duration.—Frederick A. Packard, in an analysis of 357 reported cases, found the mortality rate to be 44.07 per cent. Cases terminating favorably go on to recovery in from three to six months. An early diarrhea is said by some writers to be a favorable symptom, whereas others regard it as evidence of serious intestinal irritation, and as a sign that the patient has ingested a large amount of infected meat.

Cysticercus.—The cysticercus represents one developmental stage in the life-cycle of the tape-worm (see Tænia solium, p. 968). It is found in the muscles and viscera.

PARASITES OF THE BLADDER AND KIDNEY.

Among the animal parasites that infect the urinary tract are the **schistosomum hæmatobium,** which may be recognized by the presence of its ova in the urine. Schistosomiasis has been further considered in detail on p. 961. The ova are always found in the urinary sediment and in the small blood-clots. They vary in size from 135 to 160 microns in length, and from 55 to 66 microns in breadth. The extremity of each ovum is provided with a spine (Fig. 341).

Ova of **oxyuris and ascaris** are occasionally seen in the urinary sediment. Ova of the **trichuris trichiuria** and the adult **rhabditis pellio** are rarely seen in human urine, and cases have been reported in which the proglottides of **dibothriocephalus** have been found in the bladder.

Echinococcus Cyst of the Kidney.—Infection of the kidney with the tænia echinococcus is rare in the United States, though quite common in Scandinavia, Greenland, and Iceland. The tumor develops somewhat slowly, but displays all the physical signs characteristic of other growths of the kidney. In one case studied at the Philadelphia Hospital the cyst rup-

tured into the pelvis and the patient voided a large amount of bloody urine that contained hooklets of the tænia echinococcus.

Eustrongylus.—Rarely, indeed, ova of eustrongylus gigas are found in the urine, three such cases having come under our observation.

PARASITIC DISEASES OF THE LUNGS.

ECHINOCOCCUS DISEASE.

General Remarks.—Echinococcus disease of the lung is due to infection with the embryo of an animal parasite, the tænia echinococcus. The condition is frequently encountered in Australia, Iceland, and Russia. Hydatid cyst of the lung may be either primary or secondary, but in the majority of cases hydatid disease of the liver or of other viscera is also present. The statistics collected by authors in different portions of the world vary widely as to the frequency of involvement of the lung in this disease. Thomas, in an analysis of the reports of 1897 cases of echinococcus disease collected from practically all parts of the world, found the lung to be the site of disease in 11.59 per cent., whereas the cases collected in Australia showed pulmonary involvement in 16.56 per cent.

Mosler and Peiper assert that secondary echinococcus disease of the lung frequently results from perforation of the diaphragm by a hydatid cyst of the liver, in consequence of which the hepatic cyst communicates with the pleural sacs or directly with the lung as the result of adhesions existing between the visceral and diaphragmatic pleuræ.

Exciting and Predisposing Factors.—Infection with the echinococcus follows the ingestion of the ova of the adult parasite, which is known to infest the intestinal canal of dogs.

The most common predisposing factor is intimate association with dogs, the disease being common in Iceland and other countries in which these animals occupy the same quarters as their masters.

Principal Complaint.—Frankel distinguishes three clinical stages of the disease:

First Stage.—In this the symptoms are vague, but are suggestive of pulmonary congestion. Emaciation and fever are, as a rule, absent, the only symptom of clinical importance being the repeated attacks of hemoptysis. The quantity of blood expectorated at one time is small, the sputum being merely tinged or blood-streaked. Delgrange declares that hemorrhage from the lung is seldom absent altogether during the initial stage of pulmonary echinococcus disease. Repeated attacks of acute pleurisy are prone to occur, and moderate effusion into the pleura is not uncommon, although such liquid is absorbed within the course of a few weeks. In cases in which the pleura is attacked dyspnea and cough may be annoying.

Second Stage.—This is marked by the appearance of definite physical signs referable to pulmonary disease.

Inspection.—If the cysts are numerous, dyspnea is extreme. If a large cyst is present, there may be local bulging of the chest-wall or displacement of the heart.

Palpation may show that the tactile fremitus is diminished over the affected areas, whereas in those cases in which the pleura is involved or in which the cyst is surrounded by a dense fibrous capsule, the tactile fremitus may be increased.

The *percussion-note* is impaired. As a rule, the breath-sounds over the affected area are feeble, but, on the other hand, they may be exaggerated, depending upon the location of the cyst, the area of congestion surrounding it, and the presence of involvement of the pleura. Impairment may be detected over any portion of the lung, but by outlining the dull area in echinococcus disease this will often be found to be curvilinear, the convexity of the curve being directed upward.

During this stage pleural effusion is by no means uncommon, and when present, the symptoms of this condition (p. 142) will also appear. Pain in the side, cough, dyspnea, together with dullness and crepitant râles, should suggest echinococcus disease, particularly when the patient has previously displayed the symptoms of the first stage of this malady.

Late during this stage the general symptoms suggestive of chronic destructive changes in the lung make their appearance, and, indeed, at this time the general clinical picture may simulate closely that of pulmonary tuberculosis (p. 803).

Third Stage.—This is marked by and succeeds rupture of the cyst or cysts, and the symptoms following such rupture vary directly with the direction in which the contents of the cyst are discharged; *e. g.*, in those cases in which the cyst ruptures into a bronchus, this may be ascribed to paroxysmal coughing, heavy lifting, or violence to the thorax. Cough is, as a rule, present during the stage of rupture, irrespective of whether or not the cyst communicates with a bronchus. Following rupture into the bronchus there is copious expectoration of a clear fluid or semifluid material that is found microscopically to contain hooklets, scolices, and often fragments of cyst membrane (Fig. 361), all of which are characteristic products of the tænia echinococcus. Secondary infection of the cyst may have taken place, when the material expectorated will be purulent in character. Pulmonary hemorrhage may be profuse at the time of rupture.

Rupture into the pleural cavity may be accompanied by a mild expression of shock, following which pneumothorax frequently occurs, although the cyst contents may escape into the pleural cavity without evincing the usual symptoms; on the other hand, the symptoms and signs of pleural effusion may be present.

Dieulafoy has called attention to the development of urticaria prior to rupture of the cyst, and artificial evacuation of the cyst may also be followed by this cutaneous manifestation.

X-Ray Diagnosis.—In 1907 Levy-Dorn and Zadak reported their findings in a case of pulmonary echinococcus disease in which a distinct oval, black shadow was seen in the left lung, and another lighter shadow in the middle portion of the right lung.

Summary of Diagnosis.—The diagnosis is based solely on the detection of the products of the echinococcus cyst in the sputum or in the fluid obtained by aspiration.

Clinical Course.—Months and sometimes years are necessary for the cyst to develop to a size sufficient to produce physical signs. The chronicity of the disease depends largely upon the number of cysts present, and to some extent upon their location. Surgical interference not only modifies the course of the disease, but is said by Luffier to effect a cure in 90 per cent. of all cases. Spontaneous rupture into a bronchus is, as a rule, followed by recovery, although the illness is somewhat protracted. (See Diseases of the Lung, Collection of Sputum, p. 82.)

AMEBIC ABSCESS.

Pathologic Definition.—A disease of the lung secondary to amebic abscess of the liver, caused by the entamœba histolytica, and characterized by destruction of the pulmonary tissue with pus-formation.

Principal Complaint.—There is usually a history of having resided in the tropics, and of having suffered from an attack of dysentery, the clinical course of which corresponded closely to that described under Amebic Dysentery (p. 964). Primary amebic abscess of the lung is extremely rare, the condition usually complicating amebic abscess of the liver (p. 982).

Summary of Diagnosis.—The diagnosis is based entirely upon the detection of the entamœba histolytica in the sputum. In a case under our observation the sputum was frequently tinged with blood, and amebæ were present over a period of several months.

PARAGONIMIASIS

(Endemic Hemoptysis).

Pathologic Definition.—A chronic disease of the lung caused by a trematode worm, paragonimus Westermani, which produces cavities in the lung tissue, in which there is a characteristic exudate containing the ova of the fluke.

Geographic Distribution.—Thus far almost all cases of paragonimiasis have been reported from Japan, Korea, the Philippine Islands, Formosa, and China. The disease has been transported to the United States, although but few cases are on record. A peculiar disease characterized by bloody sputum and pulmonary hemorrhage has been detected in various portions of the United States among cats, dogs, and hogs, and the transmission of this disease to North America is doubtless explained by the great numbers of emigrants from the far east that settle in all sections of this country.

Exciting and Predisposing Factors.—The disease is caused by the presence of the lung fluke, paragonimus Westermani.

Residence in Formosa, Japan, China, Korea, and certain of the East India Islands is the most potent predisposing factor.

Principal Complaint.—Cough is the most annoying symptom, being present during almost the entire course of the disease, and is usually most urgent upon rising after a night's sleep. Paroxysms of coughing are not unusual, and are frequently accompanied by the expectoration of a rusty brown, bloody-looking sputum that resembles anchovy sauce in appearance. When the patient clears his throat he is often able to eject a small quantity of this somewhat characteristic sputum. As a rule, there are repeated attacks of hemoptysis; these are slight at first, but as the disease progresses, profuse hemorrhage may follow. The patient states that upon slight exertion, even that of walking hurriedly, this bloody-looking fluid may be expectorated. Extreme weakness is present, and the general symptoms referable to secondary anemia appear, being dependent for their intensity upon the amount of lung destruction that has taken place.

Laboratory Diagnosis.—The sputum is usually quite profuse, especially after violent coughing; it is dark brown in color, and, as a rule, contains no blood, the color being due to the ova that are present. Free red blood-corpuscles may at times be found.

Filaria.—The literature contains records of the embryo filaria bancrofti having been found in the bloody sputum.

CUTANEOUS PARASITES.

DRACUNCULUS MEDINENSIS
(GUINEA-WORM).

Remarks.—Dracontiasis, or guinea-worm disease, is a tropical affection caused by the dracunculus medinensis. The adult parasite inhabits the connective tissue, and belongs to the class of nematodes (Fig. 365). It is found on the western coast of Africa, India, Brazil, and Arabia. The adult

FIG. 365.
A, Embryo of guinea-worm (Dracunculus medinensis); *B*, adult female guinea-worm (Boston, after Bristow).

female worm is cylindric in form, about 26 inches in length, and $\frac{1}{10}$ inch in diameter; it is of a milky color, and has smooth surfaces, with a tapering tail that is bent abruptly near its tip. The head is provided with a triangular mouth surrounded by six papillæ. The uterus extends nearly from the head to the tail, and is filled with embryos.

Predisposing Factors.—Man is probably infected by drinking water containing a small crustacean known as cyclops, which acts as the intermediary host.

The Embryo.—The embryo (Fig. 365) is nearly one-half inch in length, and its alimentary canal is readily distinguishable. The impregnated female works her way through the intracellular connective tissue for a period of from nine to twelve months, and when fully matured, burrows toward the legs, just above one of the malleoli; and she then migrates toward the surface of the skin, where a small vesicle is produced, which finally ruptures. The head of the worm, near which the uterus is located, is now in a position from which the embryos may be discharged. Symptoms of guinea-worm disease do not develop until the parasite is fully matured, when a vesicle or abscess appears at the site where the parasite comes in relation with the skin. There may be localized swelling, a feeling of tension, sensitiveness, and redness, and in many instances the worm may be felt beneath the skin. Developing from the abscess is a more or less extensive ulcer, from which a portion of the adult parasite may protrude.

Detection.—The milky discharge from the ulcer, when examined microscopically, will be found to contain a number of embryos (Fig. 365).

Summary of Diagnosis.—The finding of the embryo of the parasite in the discharge from cutaneous ulcers is positive evidence of the existence of this disease.

PSOROSPERMIASIS.

Psorosperms belong to that order of protozoa known as sporozoa. A common form occurs in the muscles of swine (sarcocystis Miescheri).

Internal Psorospermiasis.—In man hepatic disease similar to that found in the rabbit is produced by the coccidium oviforme. The tumors formed by the coccidia may be palpable, and the liver may be quite tender. Chilliness, fever, malaise, stupor, and coma have been observed.

In the intestinal variety of internal psorospermiasis nausea, vomiting, diarrhea, and the typhoid state may be seen. Involvement of the kidneys has given rise to hematuria.

External Psorospermiasis.—Cutaneous psorospermiasis, one variety of which was formerly termed *keratosis follicularis*, is characterized by the presence of lesions that at first are hard, crusty, and papular, later becoming confluent, situated on the face and lumbo-abdominal and inguinal regions. These growths contain either parasitic sporozoa or, as suggested by Montgomery, Darier, and others, parasites that belong to the blastomyces.

PARASITES OF THE EYE.

CYSTICERCUS.

This form of the tape-worm has been known to invade the orbit.

FILARIA.

The adult filaria loa has been recovered from the conjunctiva of man, as well as from the eye of the horse.

MYIASIS.

This disease is an infection of the human tissue with the larvæ of certain dipterous insects. If these larvæ are found in the skin or in the mucous membrane of a cavity communicating with the surface of the body, such as the nose, the external auditory canal, or the vagina, it is known as **external**

myiasis. If the larvæ are passed with the feces, the condition is termed **internal myiasis.** In the United States *compsomyia macellaria,* the screw-worm, is the most important of these parasites, although cases of infection with the larvæ of *dermatobia noxialis* are on record, and Swan* reported a case of infection with the larvæ of *lucilia serricata,* and a case of infection with the larvæ of *lucilia cæsar,* which occurred in Philadelphia.

Clinical Features.—The larvæ are known to burrow through the tissues, destroying the mucous membrane, the muscles, and the cartilages. They may invade the serous sacs and the bones, producing extensive lesions. They have been recovered from the eye and the conjunctivæ. George Gray has reported five instances in which the larvæ of the screw-worm were found in human beings. He states that the parasite is common in domestic animals, and that it is widely distributed throughout America.

PARASITIC DISEASE OF THE BRAIN.

Cysticerci are occasionally found within the substance of the brain and the ventricles, a case having been reported by J. Hendrie Lloyd. (See section on Nervous Diseases.)

The **plasmodium of malaria** may be found to plug the smaller blood-vessels of the brain.

*Jour. of Tropical Medicine and Hygiene, January 1, 1910.

CONSTITUTIONAL DISEASES.

DIABETES MELLITUS.

General Remarks.—A disease of metabolism characterized by atrophy of the islands of Langerhans, fatty infiltration of the liver, or organic changes in the brain, with abnormal carbohydrate digestion.

The syndrome necessary to produce typical diabetes is composed of: (1) Intense thirst; (2) polyuria with glycosuria; (3) progressive emaciation and weakness; and (4) an inordinate appetite. When the urinary phenomena constitute the chief symptoms presented by the patient, the condition is known as glycosuria.

Clinical Types.—(1) **Infantile Diabetes.**—Diabetes of the newborn and diabetes occurring during the first decade of life is, as a rule, hereditary, although traumatism and acute infectious maladies have been regarded as potent factors in the production of this affection. Infantile diabetes is a rare condition, but we have studied the urine of 11 cases of diabetes mellitus occurring in children under ten years of age.

(2) **Pancreatic Diabetes.**—In this type of the affection other evidences of pancreatic disease are commonly present. This variety may differ from other forms of diabetes in that, in quite a large percentage of cases, polyuria and intense thirst are absent or but feebly manifest. Pancreatic diabetes runs a more chronic course than infantile diabetes, but the two varieties are equally fatal.

(3) **Phosphatic diabetes** is a condition in which many of the general symptoms of diabetes are present, and, in addition, there is a decided increase in the elimination of phosphates. This increase has been known to reach a maximum of nine grams excreted during the twenty-four hours. When phosphates are present in abundance, glucose is often absent from the urine, but following a decrease in the phosphates, glucose appears in the urine; the alternation of phosphaturia with glycosuria constitutes the cardinal symptom of this malady.

(4) **Alimentary Glycosuria.**—This is a form dependent upon the too liberal imbibition of carbohydrates, together with the inability to digest such substances.

(5) **Transitory Glycosuria.**—This type of glycosuria is oftenest encountered after a breakfast that has been rich in carbohydrates. In certain low-grade conditions transitory glycosuria is not unusual, and for convenience of description these have been classified under the following subheadings: (*a*) Toxic; (*b*) puerperal; (*c*) digestive; (*d*) intermittent glycosuria of arthritis; (*e*) cerebral glycosuria.

Toxic glycosuria is the term applied to the appearance of glucose in the urine after the administration of such toxic substances as hydrochloric acid, sulphuric acid, mercury, strychnin, glycerin, alcohol, nitrobenzol, lead, arsenic, phosphorus, potassium iodid, caffein, thyroid extract, tuberculin, pancreatin, phloridzin, diuretin, carbon monoxid, and morphin. Analgesics

and anesthetics also possess the power of exciting transitory glycosuria, and several instances are recorded in which glycosuria has followed the administration of chloral, chloroform, and amyl nitrite. Several observers found small quantities of glucose in the urine after ether anesthesia, and Andral reports a case of true diabetes developing after ether narcosis.

Pathologic transitory glycosuria (toxic) is best exemplified by the glycosuria of cerebrospinal meningitis; it is also seen, though less commonly, in relapsing fever, typhoid fever, cholera, and diphtheria, in the advanced stages of pulmonary tuberculosis, rickets, and gastritis. During the course of certain acute infections, viz., scarlatina, measles, smallpox, malaria, and whooping-cough, glycosuria is occasionally encountered, and it has been known to follow such chronic conditions as interstitial nephritis, gall-stone, asthma, and syphilis.

Glycosuria developing during the course of another malady is often referred to as *functional glycosuria.*

Digestive glycosuria is recognized by its disappearance after the withdrawal of carbohydrates from the diet and the correction of digestive disorders.

Intermittent glycosuria of arthritis should possibly be included with hereditary glycosuria of the young or with that of gouty and obese adults.

(6) **Cerebral Glycosuria.**—A condition in which the presence of glucose in the urine is dependent upon a pathologic state of the central nervous system. Von Jaksch has found glycosuria in hysteric women and in cases of phosphorus-poisoning in which autopsy disclosed the presence of fatty degeneration of the liver. We have found glucose in the urine during the febrile stage of cerebrospinal meningitis, and glycosuria has been known to develop during the course of neuritis. Transitory glycosuria occurs in disseminated sclerosis, epilepsy, neuralgias, neuroses, psychoses, exophthalmic goiter, myxedema, after prolonged mental strain, sudden emotions, anxiety, and in certain forms of insanity.

(7) **Traumatic glycosuria** not infrequently follows severe injury to the head, chest, abdomen, or extremities; but in this case the presence of glucose in the urine is, as a rule, of short duration or intermittent in nature.

(8) The so-called **puerperal glycosuria** is usually due to the absorption of lactose from the mammary gland. It may make its appearance at any time throughout the course of gestation or during the puerperium. We have studied more than 50 cases of puerperal glycosuria, and in many of these the condition did not exist prior to conception; in others a history of intermittent glycosuria was obtained. In more than 25 per cent. of the cases the patients were Hebrews, but in none did the amount of glucose present exceed 2 per cent.

Predisposing and Exciting Factors.—(*a*) **Age and heredity** figure prominently as etiologic factors of diabetes. Heredity is generally conceded to be the most potent predisposing influence, two, three, and in one instance five members of the same family having suffered from the disease. Heredity is said to figure prominently in the diabetes of children.

The majority of all cases of diabetes occur between the thirty-fifth and sixtieth years; the malady is rarely seen before the tenth year, although it has occasionally been observed in children under one year of age.

(*b*) **Sex.**—Males are more frequently affected than females, except in the diabetes of children, in which sex does not appear to exercise any appreciable influence.

(*c*) **Season.**—The greatest number of cases are seen in March, July, and

November, but since diabetes is usually chronic in nature, but little importance can be attached to season as a predisposing factor.

(*d*) **Race** exerts a decided influence in the production of diabetes, the disease being extremely rare in the negro and particularly common in the Caucasian.

(*e*) **Nationality** also figures in the production of diabetes. Hebrews are more often afflicted than Gentiles, a fact thought to be due not so much to the habits and customs of the Jewish people as to their intermarriages.

(*f*) **Station.**—Diabetes is a disease of the well to-do members of society; in our service at the Philadelphia General Hospital and in various medical clinics in Philadelphia it is rarely encountered, whereas in private practice a large number of cases are seen.

(*g*) **Nervous Influences.**—Sudden shock, such as the loss of friends, or severe financial reverses are not infrequently followed by the onset of diabetes; prolonged mental strain, study, and combined mental and physical overwork are also potent factors in its causation.

(*h*) **Occupation** plays a small part in the etiology of diabetes, although the disease is somewhat more common in clerks than in those who do strenuous muscular work. Obesity, gout, rheumatism, syphilis, and pregnancy (puerperal diabetes) have all been named as etiologic factors.

(*i*) **Incidence.**—Diabetes is said to affect the residents of cities more often than those living in the country. This is possibly due to the fact that little attention is paid to urinalysis in the rural districts. Hare's statistics show that diabetes is increasing in America; and there is statistical evidence to show that in India and in France the percentage of cases of diabetes is progressively increasing.

(*j*) **Contagion** is believed by a few to exert an etiologic influence in diabetes, and according to Senator's analysis of 770 cases, in 9 both husband and wife suffered from this malady. Shram's statistics, covering 5000 cases, further supports the theory of contagion.

Among the **exciting factors** are: (1) Pancreatic disease; (2) hepatic disease; (3) disease of the brain or nervous system (sclerosis, tumors, cysts, lesions of the fourth ventricle or of the spinal cord); and (4) traumatism—*e. g.*, trauma to the spine, loins, and abdomen, injury to the head and extremities.

Special Clinical Types.—For convenience of description and to avoid confusion the disease will be considered under the heads of acute Diabetes Mellitus, Chronic Diabetes Mellitus, and Glycosuria, the three varieties presenting one common leading symptom, namely, the appearance of glucose in the urine.

Acute Diabetes Mellitus.—Instances in which diabetes may be said to run an acute course are extremely uncommon, yet it is not unusual to see cases in which the various symptoms of the disease follow one another in rapid succession. In such cases emaciation, progressive weakness, a high percentage of glucose in the urine, and the presence of acetone and diacetic acid in the fluid all develop within a few months, a fatal termination occurring in less than one year. To be accurate, this group of cases should be considered as subacute, but when compared with the chronic types of the malady, which may continue over a period of from three to thirty years, this variety may be regarded as acute.

Chronic Diabetes Mellitus.—The onset is insidious, and the patient is often unaware of his condition until it is discovered as the result of urinalysis. Among the early symptoms there may be dyspnea or gastritis, or the

patient may complain of being constantly tired, and exhibit some mental hebetude. Following these symptoms the knees may seem to bend too far backward or the clothing lie as a weight upon the shoulders. Later more pronounced and characteristic symptoms of the malady appear, but these, as a rule, follow one another slowly, an interval of from three to six months elapsing between the appearance of the different characteristic features to be described further on.

Diabetes following shock, traumatism, or extreme grief and anxiety may begin abruptly, and pursue a somewhat acute course for several months, when all the manifestations of the disease become chronic in nature, and the patient may live for several years. In this type of diabetes glycosuria may occur at intervals, particularly after a meal rich in carbohydrates. This symptom is almost always intermittent in character for a period of months or even years. Polyuria, ravenous appetite, intense thirst, and gradual progressive weakness, with marked nervous symptoms, constitute the initial symptoms that suggest true diabetes.

Glycosuria.—Under this head are included those cases in which glycosuria is the only symptom, the other characteristic manifestations of diabetes mellitus being absent. In the majority of cases of simple glycosuria, therefore, polyuria, emaciation, progressive weakness, and nervous symptoms are not present. Glycosuria is seldom continuous, but, instead, runs a remittent or an intermittent course. Again, glycosuria is commonly dependent upon dietetic errors, overexertion, either physical or mental, or the presence of some acute or chronic infection. Glycosuria may occur as the result of faulty digestion, either gastric or intestinal, or from faulty metabolism of a questionable nature, which is not essentially associated with appreciable disease of the pancreas, liver, or brain.

In glycosuria the cutaneous symptoms, intense thirst, and ocular manifestations common to diabetes mellitus are lacking. The condition is readily amenable to treatment, in which respect simple glycosuria differs markedly from diabetes mellitus.

Principal Complaint.—The patient may complain of general malaise and weakness, or state that he is tired after a night's rest. His clothing appears to be too heavy for comfort, and he is frequently unable to wear an overcoat. There are soreness and weakness of the calf muscles. A history of having taken on flesh during the past few months or years is often obtained, and the patient attributes his shortness of breath and exhaustion to the increase in weight.

The appetite may at first be unaffected, but as the disease progresses it becomes ravenous, and there is a craving for sugars and carbohydrates. Occasionally, however, the appetite may remain normal throughout the entire course of the disease.

After diabetes has progressed for an indefinite period **thirst** develops, and increases with the progress of the disease. The greater the amount of glucose in the blood, the more intense is the thirst, until, in advanced cases, it is not unusual for the patient to drink several gallons of water during the twenty-four hours. Cases have been observed, however, in which all the other cardinal symptoms of diabetes were present except the intense thirst and polyuria.

Oral Symptoms.—The tongue is large and dry, even in the early stages of diabetes, and as the disease progresses this dryness becomes more and more marked, until, in the later stages, the tongue is parched, fissured, and of a bright-red color. In some cases it is coated, and the lips are dry and fissured,

the gums swollen and edematous, and, rarely, a bloody exudate escapes from the bases of the teeth.

The secretion of the mouth is commonly acid in reaction, and when the saliva is subjected to chemical analysis, it will be found to contain glucose.

Stomatitis develops during the course of advanced diabetes, when the gums recede from the teeth, the teeth decay and become loose, and small ulcers form along the margins of the gums and on the cheeks. Infection of the buccal mucous membrane by the thrush fungus and other extensive ulcerations of the mucous surface occasionally develop.

Digestive Symptoms.—Considering the quantity of food ingested, the digestive function is almost normal early during the course of diabetes, but later, after the patient has become extremely emaciated and weakened, less food is taken, and the digestion becomes impaired.

The bowels may move regularly or there may be constipation or diarrhea; the latter condition lasts for but a short period, and is followed by constipation. In those cases in which the diabetes is due to disease of the pancreas or to obstruction to the escape of bile into the intestines, the stools may contain fat.

Cutaneous Manifestations.—The skin is apparently normal to the touch early during the course of diabetes and before polyuria has developed, but after the disease is well advanced, the skin becomes dry and rough, and sweating is absent, even in the groins and axillæ. In advanced diabetes the patient is extremely pale, and at times a slight lemon tint is observed. Boils and abscesses of the skin are a prominent feature of well-established diabetes. Itching of the skin, particularly at night, is present in about 60 per cent. of all cases. In fact, itching of the scalp and of the skin over the calf muscles may be among the earliest symptoms of diabetes. Eczema of diabetic origin is one of the most annoying manifestations of this affection; it usually develops about the genitalia, but may affect any portion of the body. In a case studied at the Philadelphia Hospital the entire body was involved in the eczematous process.

The hair becomes dry and lusterless, and in nearly all cases tends to fall out after the other symptoms of diabetes become well marked. The nails become thickened and horny, or extremely brittle, and their surfaces are marked by furrows and ridges. In a case seen by us onychia with shedding of the nails occurred. Owing to the extreme emaciation the bony skeleton becomes especially prominent. Gangrene of the feet, particularly of the toes, is a serious cutaneous symptom, and is due to general arteriosclerosis.

Pruritus vulvæ in the female and balanitis in the male constitute troublesome symptoms, and are probably due to the irritating properties of the diabetic urine. Abscesses of the vulva often cause intense suffering.

Ocular Phenomena.—The conjunctivæ are pale and often show peculiar yellowish spots—deposits of fat. The margins of the eyelids are commonly reddened and covered with small scales. Abscesses and boils of the eyelids are distressing, and failing vision, retinal hemorrhages, and cataract all occur during the later stages of diabetes.

Aural Symptoms.—Among these may be mentioned otitis media, otalgia, tinnitus aurium, and, rarely, mastoid disease.

Respiratory Symptoms.—Pulmonary complications do not occupy a prominent place in the general symptomatology of this affection. Pulmonary tuberculosis is a common termination of diabetes, but among the cases studied by us it was not of frequent occurrence. Pulmonary gangrene is an occasional finding. Both lobar and lobular pneumonia have been

known to complicate diabetes. Dyspnea is often present, and is dependent either upon pulmonary disorder or upon general weakness with cardiac failure.

The mucous membrane of the nose and pharynx is dry, and the patient's voice is somewhat husky. Rhinitis is uncommon. Abscesses of the nose are of frequent occurrence, and we have seen several cases in which abscess of the nose preceded the onset of diabetic coma.

Circulatory Peculiarities.—In advanced diabetes the circulatory tension is, as a rule, high, and the arteries are wiry and atheromatous. The frequency of the heart's action is not increased unless the patient is unusually weak, and in fact during the early stages of arteriosclerosis the pulse may be found to vary between 40 and 70 beats a minute.

Sexual Weakness.—Impotence and a premature menopause may be among the earliest symptoms of diabetes, and should always be regarded as suggestive of this malady. Diabetic women rarely conceive. The disease often develops during the period of gestation, and in such instances is likely to continue after delivery. It has been asserted that premature delivery is to be expected, but there is little evidence to show that instrumental interruption of pregnancy materially benefits the sufferer. Intense itching of the vulva, with shooting pains in the clitoris, is a most troublesome symptom. In the male, hypersensitiveness of the glans penis may be present in advanced diabetes.

Muscular cramps occur in a large proportion of all cases of confirmed diabetes, and these are most likely to affect the muscles of the calf of the leg. Cramp in the region of the stomach (gastric crises) may occur at any time during the day, the ingestion of certain foods being held responsible for the gastric pain. In one of our patients intestinal cramp was excited by the eating of an orange or the drinking of orange-juice, and a much less severe pain followed the ingestion of tomatoes.

Nervous Symptoms.—Peripheral neuritis may develop during the course of diabetes, and is accompanied by numbness and tingling of the extremities, and such trophic disturbances as perforating ulcer and thickening or even shedding of the nails. Neuralgia is a prominent symptom, and may affect the lower extremities, loins, back, face, or arms.

Diabetic tabes exhibits many of the features characteristic of ataxia; thus the knee-jerks are diminished or absent, as is shown by Williamson's series of 50 cases of diabetes, among whom 25 showed this sign. Lancinating pains, paralysis of the extensor muscles of the feet, and the ataxic gait are observed, and paraplegia has also been encountered.

Temperament.—The patient is extremely irritable, and is unduly affected by trivial causes. Hysteric outbreaks and hypochondriasis are seen to occur during any stage of the disease. The mentality may be unusually active at times, the power of concentration of mental forces being abnormally increased, but following such period there is a corresponding stage of mental hebetude.

Coma.—Diabetic coma develops only at the terminal stage of this affection in about half the cases. It is almost invariably fatal. We have found coma to occur earlier during the course in young patients and in those in whom emaciation and prostration were rapid and progressive. The precursors of coma are: A fruity odor of the breath and of the urine; a reduction in the amount of glucose excreted; a diminution in the quantity of urine voided during the twenty-four hours; an increased reaction for acetone and for diacetic acid in the urine; the appearance of β-oxybutyric acid in the

urine; the occurrence of a chill or of a series of chilly sensations; intense headache, dimness of vision, and neuralgic pains in various parts of the body.

For convenience of study we have classified diabetic coma under five heads, as modified by Anders:

(1) *Abortive coma,* which tends to run a short course and terminates in recovery; in this class of cases there is a special tendency for repeated attacks of coma to occur, one of which ends fatally.

(2) A group in which diabetic coma follows violent exercise with extreme exhaustion and circulatory collapse. To this group the largest number of cases belong. This form of coma is, as a rule, fatal, lasting for from a few hours to four days.

(3) Cases in which headache and the other signs of severe autointoxication are followed by coma, a fatal issue ensuing within a few hours.

(4) Coma developing abruptly during the course of an acute inflammatory process, such as abscess, tonsillitis, gastritis, etc. In this type of coma the circulatory, respiratory, and febrile symptoms may be prominent, but bear no direct relation to the degree of coma, which continues for from one to five days, ending fatally.

(5) Coma developing in aged persons during the course of such chronic conditions as eczema and gangrene.

Thermic Features.—The temperature may be normal throughout the course of diabetes. Not uncommonly, however, the temperature fluctuates between 99° and 100° or even 101° F. in cases in which there is no positive evidence of the existence of an acute inflammatory process. An elevation of temperature the result of a complicating acute infection resembles more or less closely that peculiar to the existing condition. In many cases of diabetes a subnormal temperature is present, particularly during the morning hours.

Laboratory Diagnosis.—One of the early symptoms of diabetes is the presence of glucose in the urine. (See Tests, p. 653.) Numerous writers have maintained that glucose is found in normal urine. It is our opinion that normal urine does not contain glucose in sufficient quantity to reduce Fehling's solution, and that a urine that will give a positive reaction for glucose with that solution is a pathologic one. Undoubtedly, normal urine contains a carbohydrate substance, but, we believe, no glucose.

The quantity of urine voided during the twenty-four hours is, as a rule, above the normal (50 ounces), and as much as from 200 to 500 ounces may be excreted. The specific gravity is high, varying between 1.025 and 1.050, although we have detected small amounts of glucose in urines of low specific gravity—1.010 and even 1.006. The reaction of diabetic urine is acid; it is of normal color, free from sediment, emits a sweetish odor, and upon shaking displays a heavy white froth that remains for some time.

Chemically, the urine is found to contain glucose in variable amounts, a typical case showing from 1 to 3 per cent. of glucose. Early in the disease, however, there is merely a trace of glucose—too small a quantity to be estimated; in other cases the glucose may exceed 5 per cent. As a rule, the percentage of glucose present in the urine in a given cases of diabetes or of glycosuria will be found to fluctuate in direct proportion to the amount of carbohydrates ingested; thus, we have seen the percentage of glucose fall from 4.5 to 1 per cent. after the withdrawal of carbohydrates from the diet. Occasionally a specimen of urine will be found to contain both albumin and glucose. The amount of colloidal coefficient is high, exceeding 1.79.

The detection of fat in the feces is highly suggestive of pancreatic diabetes.

A microscopic study of the urine should always be made. Evidences of nephritis are uncommon in diabetes, but when the disease has reached an advanced stage, marked nephritis may be present, and even before the terminal renal lesion develops, casts may be found in the urine.

Blood.—There is a secondary anemia, and a differential count shows an increase in the lymphocytes to 40 or even 70 per cent.

In eczema and inflammatory conditions of the skin (boils, abscesses) cocci, bacilli, and fungi may be detected in the secretions from these lesions, but these are probably but another proof of the lowered resistance of the patient.

Illustrative Case of Diabetes Mellitus.—P. R., male, aged thirty-four years; apparent age, forty years. Height, 5 feet 8 inches; weight, 170 pounds. Since the age of thirty he has taken on weight somewhat rapidly, going from 150 to 170 pounds.

Family History.—Father living and healthy at the age of fifty-six years; mother died at forty-five after having suffered from diabetes for several years. One maternal uncle, now fifty years of age, has periodic attacks of glycosuria. A brother of eighteen and one of twenty-six are reported as being in good health.

Previous History.—In addition to the usual diseases of childhood, he had diphtheria at the age of twelve. From this time until after the age of thirty he does not recall having consulted a physician, except, perhaps, for an acute cold.

Social History.—Married four years ago; one child living and in good health. A business man by occupation, and has undergone heavy financial strain during the past two years.

Present Illness.—For some months past he has noticed that his appetite was abnormally increased, and while he has not observed a special fondness for sweets, he admits taking a large amount of starchy food. During the past year there has been inordinate thirst, although no accurate estimate of the quantity of liquid taken during the day could be ascertained. He has been annoyed at night by itching of the skin and of the rectum and genitalia. Six weeks ago an abscess developed on the buttocks, and since then four smaller abscesses have appeared. The patient tires easily on moderate exercise, and states that even the weight of his coat on his shoulders is distressing.

Pain.—There is a slight stiffness and soreness of the joints, particularly of the ankles and knees, most marked on arising after a night's sleep or after sitting for some time.

Nervous Phenomena.—He becomes exhausted after mental strain, and has observed that he has been unable to cope with financial problems that ordinarily caused him no annoyance. He sleeps fairly well during the night, but on certain days the mind is not clear and he becomes drowsy, such mental hebetude not being relieved by sleep.

Physical Examination.—*General.*—The patient is well nourished; the skin and mucous membranes display an unusual color, and the cheeks are slightly flushed. The skin is dry, and there is slight scaling upon the arms and limbs, and eczema of the genitalia and inner surface of the thighs. The hair has lost its luster, and is sparsely distributed over the scalp, although the patient states that his hair was unusually heavy two years ago. At times the mind is remarkably clear, although he tires easily after moderate exertion; mental dullness and irritability are present.

Palpation.—The skin is dry and somewhat rough.

Laboratory Diagnosis.—The quantity of urine voided during the twenty-four hours varied between 90 and 120 ounces, was clear, did not show a sediment upon standing, emited a sweetish odor, and had a specific gravity of 1.035. Chemically, it gave a reaction for glucose, of which 2 per cent. was present.

Course of the Disease.—Following medication and the observance of a restricted diet the quantity of glucose excreted was reduced by about one-half, but despite this reduction the patient continued to lose weight until two years later, when he weighed 128 pounds. Weakness was progressive, and at the age of thirty-eight he was unable to follow his usual vocation. At this time an ophthalmoscopic examination was made and retinal changes were found. His condition progressed from bad to worse and terminated in diabetic coma and death within the course of five years.

Summary of Diagnosis.—The syndrome necessary to the diagnosis of diabetes mellitus includes intense thirst, polyuria with glycosuria, pro-

gressive weakness and emaciation, and polyphagia. If any one of these symptoms is absent in a given case, we are dealing with atypical diabetes or with alimentary glycosuria. For convenience of study diabetes and glycosuria have been considered together, although they may differ widely in their clinical manifestations, etiology, and prognosis.

Course and Duration.—The conditions influencing the prognosis in diabetes are: (*a*) The age of the patient; (*b*) the presence or absence of a history of traumatism to the trunk or cranium; (*c*) a history of heredity; (*d*) the mode of development, *e. g.*, whether it occurred during gestation, after delivery, or following severe shock or mental strain; (*e*) overeating of all kinds of foods, particularly of starches and sugars. Again, the prognosis is influenced by environment, occupation (sedentary or active), and the patient's ability to carry out a proper course of treatment.

Diabetes developing during the first year of life, and even before the tenth year, runs a rapid course, and usually terminates in death within from six months to two years. The younger the patient, as a rule, the more rapid and shorter is the course of diabetes. When the disease appears during the third, fourth, and fifth decade, it runs a more chronic course, and may continue over a period of from ten to thirty years.

Diabetes following traumatism is seldom amenable to treatment and generally runs a rapid course. Hereditary diabetes developing early in life is of short duration, but if it develops later, it may be materially influenced by judicious treatment, although, as a rule, the prognosis in these cases is unfavorable.

Diabetes developing during the course of pregnancy, soon after delivery, or following severe shock and mental strain may be either mild or severe in character. We have seen cases belonging to this last class in which diabetes persisted for more than thirty years. In one instance, that of a woman now under observation, glycosuria developed during a period of gestation thirty-five years ago, and she is at present suffering from diabetes, and has had one attack of coma within the past year.

When diabetes appears as the result of overeating and of insufficient exercise, sedentary habits, and the like, the correction of such habits is followed by a decided amelioration in all the symptoms, and the prognosis is favorable.

DIABETES INSIPIDUS.

Pathologic Definition.—A chronic disease without characteristic organic changes demonstrable at autopsy. In some cases hypertrophy of the kidneys has been seen; in others tumors of the floor of the fourth ventricle have been discovered.

Predisposing and Exciting Causes.—(1) **Heredity** serves as the most potent predisposing factor, a statement that is borne out by the statistics of Weil, who found that in the 91 descendants of a certain family, 23 suffered from polyuria. The records of the descendants of a man living in northern Pennsylvania who was afflicted with diabetes insipidus showed that 18 cases of diabetes insipidus appeared in three generations. In the series of cases just referred to males and females seemed equally prone to transmit the disease, although not a single instance of polyuria in a female was found. It was the rule, however, for each female who married to bear one or more sons who suffered from diabetes insipidus, but in no instance did a son have more than one heir who displayed this malady.

(2) **Sex.**—Males are afflicted more often than females.

(3) **Age.**—It is generally conceded that diabetes insipidus occurs most frequently in children and during early adult life, although we have encountered several cases occurring in men from thirty to sixty years of age.

(4) **Temporary diabetes insipidus** may follow extreme shock, as from fright, nervous strain, traumatism to the head, and, rarely, trauma of the trunk and extremities. It may also occur in acute infectious diseases, *e. g.*, acute nephritis, influenza, etc. Lesions of the fourth ventricle are said to cause diabetes insipidus, and it has also been known to follow paralysis of the sixth nerve. The polyuria following hysteric attacks also belongs to this category.

Principal Complaint.—Diabetes insipidus develops gradually with the growth of the child, so that the mother does not realize that the child is taking an abnormal quantity of liquids until it is two or more years old. If the malady follows traumatism or disease of the brain, it develops abruptly.

The frequent passing of large quantities of urine is a constant symptom and is usually followed by intense thirst and the imbibition of a large quantity of water. The appetite is normal. The skin and mucous surfaces are generally drier than in health. Indigestion is frequently present, although it is by no means a constant finding. In one case seen by us the patient was able to take large quantities of intoxicants, particularly beer, without becoming affected.

Laboratory Diagnosis.—The quantity of urine voided during the twenty-four hours is in direct proportion to the amount of fluid ingested, and will be found to fluctuate between 20 and 50 pints. Such urines are pale, of low specific gravity,—1.001 to 1.006,—and show a low percentage of solids, although the total amount of solids voided during the twenty-four hours may equal or exceed that of the normal. Rarely, traces of glucose are present, and inosite has been found. Albumin is usually absent.

Summary of Diagnosis.—The diagnosis is based upon the existence of polyuria without glycosuria, and the presence of intense thirst in a patient who is well nourished.

Clinical Course.—The prognosis as to life is good, diabetes insipidus seldom, if ever, causing death. The application of judicious treatment is said to give relief in selected cases.

RACHITIS (RICKETS).

Pathologic Definition.—A disease of metabolism that occurs during childhood, and that is characterized by developmental abnormalities of the bones and cartilages, with the production of physical deformities. Those portions of the bony skeleton most likely to be involved are the ends of the ribs and the long bones. An examination of the diseased long bones shows the presence of pronounced changes in the vicinity of the junction of the epiphyses with the diaphysis. A microscopic examination reveals the fact that there is an increased proliferation of the cartilage-cells, with a proportionately scanty fibroid matrix and imperfect calcification.

Exciting and Predisposing Factors.—Rickets is seen to affect the new-born, and is by no means a rare condition. **Heredity** may exert some influence, although this is doubtful when both the parent and the child have been exposed to unhygienic conditions.

Malnutrition of the mother during the period of gestation and of lactation, close confinement, and syphilis are predisposing factors.

Locality.—Rickets is found to be more common in the large cities than

in rural districts. The disease is prevalent in Russia, Italy, Great Britain, and Germany.

Race is not without influence, the African negro being especially prone to acquire this disease, and in America the half-breed is also highly susceptible.

Social Station.—Rickets is especially common among children of the poorer classes, who are surrounded by an unfavorable environment. Occasionally rickets is seen among the well-to-do classes, but in this case the disease is usually the result of improper diet, insufficient sunlight, and imperfect ventilation.

Diet.—Rickets is in a great measure dependent upon improper feeding;

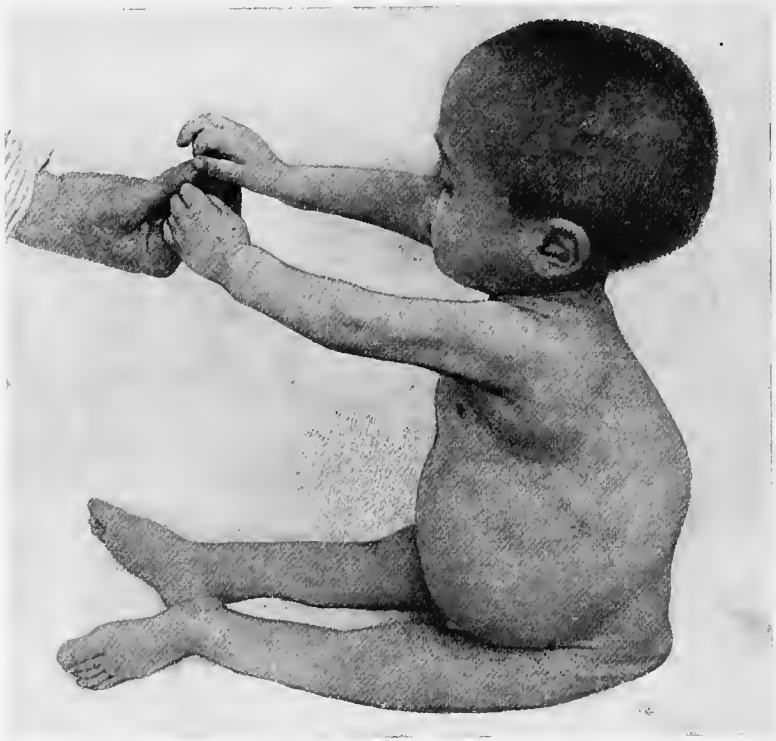


Fig. 366.—Rickets (Potter, after Dr. W. L. Stowell).
Note the size and shape of head, the rosary, Harrison's groove, kyphosis, prominent belly, bowing of legs, and the enlargement of wrists.

hence the disease is more common among artificially fed children than among breast-fed infants.

Age.—Practically 75 per cent. of all cases develop the disease before the end of the second year, although rickets may first appear as late as from the sixth to the tenth year.

Clinical Picture.—There is usually a clear history of one or more of the predisposing factors previously outlined. The mother states that the child has been less playful than usual, and has suffered from gastro-intestinal disturbances for some weeks or possibly months before true rachitic symptoms developed. The child is at first restless and irritable, sleeps poorly, and in

some cases moderate fever may be present. The mother states that the child *sweats* profusely during sleep, the pillow being wet with perspiration while the remainder of the bed-linen coming in contact with the child's body is dry. Rachitic children generally push the covers from about their head and chest during sleep, and are consequently more or less exposed to cold.

Marked **tenderness** is an early feature, and in selected instances may be localized over the bony surfaces; it is also found to affect the soft parts, and the child consequently prefers to rest rather than to be handled. The mother may very early observe that the child shows a lack of inclination to move his limbs, and whenever this condition prevails, a careful examination should be made for the possible existence of rickets or of scurvy. In chronic rickets months may elapse before definite bony deformities appear.

Owing to malnutrition the *muscles* of the extremities become soft and flabby, and there may be an apparent swelling, or at times atrophy, of such muscles, which usually goes hand in hand with impairment of function—the so-called "rachitic" paralysis.

Nervous Symptoms.—Rickets with marked deformity of the cranial bones is occasionally associated with laryngismus stridulus, and tetany of the upper extremities is also an occasional symptom. Epilepsy is quite common in rachitic children, and is probably dependent in part upon the degree of gastro-intestinal irritation present.

Enlargement of the abdomen is a conspicuous feature, and is usually due to flatulence and, in selected cases, to enlargement of the liver and spleen.

Changes in the Bony Skeleton.—Among the first bones to undergo changes are the cranial bones, the ribs, the radius, and the ulna. The cranium is large, this enlargement, however, being more apparent than real, and due to the diminished size of the facial bones. The sutures remain open, and the fontanels are large. Craniotabes is most frequently seen in infants under one year of age, and is due to pressure. It affects the surfaces on which the head of the child rests. It should be remembered that craniotabes is often a syphilitic manifestation. A rachitic head (Fig. 366) is generally of square outline, or it may present marked angularities, with an increase in the anteroposterior diameter and flattening at the top. Hyperostosis may result in prominence of the parietal and frontal eminences.

The veins of the scalp are enlarged, and the hair is scanty. On auscultating over the anterior fontanel, a systolic murmur may be audible.

The *teeth* may not be erupted until late, and may decay during childhood, although occasionally they may be misshapen and yet show no tendency to decay.

The *ribs* become beaded early, and swelling occurs at the junction of the ribs with the costal cartilages. The ribs are curved quite acutely at the lateral dorsal portion of the chest, and again curve in abruptly toward the sternum. Harrison's curve consists in a peculiar, furrow-like appearance, beginning at the anterior end of the eighth or ninth rib, and extending toward the axilla. There may be a bulging of the sternum, producing the so-called "chicken-breast."

Deformities at the junction of the diaphyses and epiphyses of the radii are among the early bone lesions of rickets, and both the radii and ulnæ are commonly found to be deformed. The clavicles may be curved and the scapulæ show marked thickening; in selected cases deformity of the vertebral column may be present. Deformity of the pelvis is also seen, and when rickets has occurred in a female child, an *x*-ray examination should be made

when the child reaches puberty, in order to ascertain whether any pelvic deformity exists.

Swelling at the lower end of the tibiæ is also an early osseous change, and curving of the femora may be present.

Laboratory Diagnosis.—Secondary anemia is commonly present, and the hemoglobin may fall to from 60 to 70 per cent. Moderate leukocytosis may be present, but is not a constant feature. A differential count of the leukocytes may show an increase in the relative number of lymphocytes, a clinical finding that may be present in children suffering from malnutrition.

X-Ray Diagnosis.—In typical cases the existence of rickets can be determined without the use of the *x*-rays, but in those cases in which malnutrition, gastro-intestinal symptoms, and profuse sweating of the head are prominent, deformity of the bones may be made out with the *x*-ray long before such osseous change can be recognized by other methods of diagnosis.

Summary of Diagnosis.—Early during the course of rickets, and before bony changes are apparent, the features of greatest diagnostic importance are: (1) The general evidence of malnutrition, together with the progressive loss of weight and the presence of secondary anemia; (2) gastro-intestinal catarrh; (3) profuse sweating of the head during sleep; (4) the application of the *x*-ray to ascertain beginning deformity of the bones of the cranium, ribs, and tibiæ. After well-marked bony deformities have developed, the characteristic alterations in the bones of the head and chest at once become apparent.

Clinical Course.—This is greatly modified by the application of judicious treatment, although in the average cases deformity of the skeleton continues to progress for a period of several months.

Complications.—Children suffering from rickets exhibit a very low grade of vitality, and consequently are especially prone to develop bronchopneumonia and the ordinary complications occurring in diseases of childhood.

MYELOMATA (BENCE-JONES ALBUMOSURIA).

Pathologic Definition.—A malady characterized by the formation of multiple new-growths (myelomata), chiefly of the flat bones, although in certain cases the long bones may also be the seat of myelomatous infiltration.

Historic Note.—Since the publication, in 1847, of Bence-Jones' case of osteomalacia fragilitas rubra associated with albumosuria, 37 similar cases have been recorded in which neoplasms of the bones, regarded as probable myelomata, were disclosed at autopsy.* In 9 of the reported cases tumor growths were observed antemortem. Von Jaksch and Fitz have each reported albumosuria occurring in connection with myxedema (?), and Askanazy calls attention to a case of lymphatic leukemia in which the urine contained Bence-Jones albumose. Albumosuria may be produced in the dog by the administration of poisonous doses of pyridin. In 1902 we reported 3 cases of Bence-Jones albumosuria, in one of which there was bone deformity, and within the course of one year from the date of our report the other two cases died. In 1902 Boston described at length a rapid reaction for the detection of this form of albumose in the urine,† and in August, 1903, he reported a case of Bence-Jones albumosuria.‡

* Anders and Boston, address before the College of Physicians, Philadelphia, June 4, 1902.

† Ibid., April, 1903.

‡ Boston, Amer. Jour. Med. Sci., October, 1902.

Predisposing and Exciting Factors.—**Age.**—From our analysis of the reported cases of questionable myelomata with albumosuria, we find that the youngest case reported was twenty-four years of age. The condition was also found in patients of thirty-two, thirty-three, thirty-six, and thirty-seven years respectively. Ten were between the age of forty and fifty, and 7 between sixty and seventy; in the remaining cases the age was not stated.

Injury.—In 15 per cent. of cases there was a definite history of severe traumatism to the bony structure.

Sex.—Of 35 reported cases, 80 per cent. of males were found to be affected.

Symptomatology.—**Pain** is an almost constant symptom, but a single exception to this being found among the reported cases. The pain is commonly described as a bone pain, or as rheumatism, lumbago, or neuralgia. The patient usually states that the pain occurs without apparent cause, is severe, and is accompanied by an apparent "giving way" of a portion of the bony skeleton. At times the pain may be dull and continuous, and again it may be cramp-like, lancinating, and pass rapidly. Pressure over the affected bones, as well as exercise, aggravates the suffering. The pain is more common over the flat bones, although the extremities may share in this agonizing symptom. *Cramps* involving the lower extremities are fairly common, and toothache, falling of the teeth, and necrosis of the maxillary bones, with well-marked salivation are occasionally encountered. Pain may be increased by the act of deglutition.

Neuralgia.—Facial neuralgia was present in 30 per cent. of the reported cases. Paralysis of the hypoglossal nerves and of the motor portion of the trifacial nerve has been recorded. In one case there were numbness and tingling of the feet and legs, and in one of our cases paralysis and twitching of the left side of the face occurred. Cephalalgia is mentioned as a prominent symptom in 70 per cent. of the cases. In 3 of our cases it was most troublesome, and in another it occurred periodically.

Painful **micturition** was present in one of our cases, and frequent urination was mentioned in 4 of the reports furnished by the literature.

Voice.—Changes in the voice were observed in 16.4 per cent. of the reported cases, whereas in 12.5 per cent. mention was made of an appreciable impairment of the auditory function.

Ocular Phenomena.—Impairment of vision occurred in 4 cases furnished by the literature, and in 2 of our cases retinal changes, consisting of hemorrhage and colloid degeneration, were detected.

Emaciation.—Progressive emaciation is an almost constant feature, and hand in hand with this go anemia, prostration, and such gastro-intestinal symptoms as nausea, vomiting, diarrhea, and constipation.

Jaundice is occasionally observed.

Glandular enlargement occurred in approximately 15 per cent. of all cases.

Laboratory Diagnosis.—In 4 cases coming under our observation the leukocytes fluctuated between 16,000 and 40,000 per c.mm.; the hemoglobin varied between 55 and 70 per cent., and the red cells numbered between 3,000,000 and 3,700,000 per c.mm. In 3 cases in which a differential blood count was made the eosinophilic cells were diminished in number or absent.

When Bence-Jones albumosuria is coexistent with myelomata, the quantity of urine voided during the twenty-four hours is likely to exceed the

normal. The specific gravity has been found to range between 1.004 and 1.040. In many of the cases recorded in the literature, and in 2 under our own observation, the urine was of the consistence of syrup. Chocolate or coffee-colored urine has been described by various writers, and in other cases the urine was said to be transparent and of low specific gravity. In applying the ordinary test for serum-albumin, a positive reaction is obtained when Bence-Jones albumose is present, and, indeed, it is customary to find both serum-albumin and albumose present in the same specimen. Casts may be present, and are ordinarily of the amyloid or hyaline varieties.

Deformities.—In an analysis of the reports of cases furnished by the literature we find that bony deformities existed in 67 per cent. Fractures were common (25 per cent. of cases), and often occurred from slight causes.

Summary of Diagnosis.—The diagnosis is based largely upon the clinical history, which usually includes pain, either neuralgic or boring in character, tenderness over the bony structures, emaciation, and prostration. A history of traumatism to the bones, together with pain at the site of injury and deformity, is of great clinical value, whereas a tendency toward fractures must also be considered. The detection of albumose in a urine of the consistence of syrup or of a coffee color makes the diagnosis fairly positive.

Complications and Duration.—In our analysis of 35 reported cases pneumonia figured as a complication in 12.5 per cent. The condition usually terminates in death within the course of two years, although one case has been recorded in which the condition existed for eight years.

GOUT.

Pathologic Definition.—A disease of metabolism, characterized by arthritic changes, including the deposit of sodium urate in the cartilages, the ligaments, and the synovial membranes. At first the deposited substance is fluid in character, but contains small masses of crystalline substance that later become hard. Secondary inflammatory changes result in fibrous overgrowth, which is accompanied by a variable degree of deformity and flexure at certain joints. Acute ulceration may follow, and gouty tophi be extruded. Gouty deposits have been found in the cartilages of the ears, nose, and larynx.

Inflammatory changes generally take place in the kidneys, and stony deposits may be found within the kidney substance, which, in turn, lead to a variable degree of destruction of the organ. The kidney of chronic interstitial nephritis (see p. 674) is present in gout, and arteriosclerosis with myocardial changes may also be present.

Varieties.—(1) Acute gout; (2) retrocedent gout; (3) chronic gout; and (4) irregular gout.

Predisposing Factors.—(1) **Age.**—Primary attacks occur most frequently during middle life. They are rare before puberty, although exceptionally they may be seen to occur even in childhood; after puberty, however, they become more frequent, this frequency decreasing rapidly after the fiftieth year is reached.

(2) **Heredity.**—The cases that develop quite early in life often show a striking hereditary taint, and it has been asserted that 50 per cent. of all cases of gout are directly traceable to a hereditary tendency.

(3) **Sex.**—The arthritic form is less frequent in women than in men, whereas in chronic gout women are more often attacked.

(4) **Alcohol** is among the chief predisposing influences.

(5) **Diet.**—Overeating and insufficient exercise doubtless constitute the most potent factor in the production of gout.

(6) **Social State.**—The majority of cases occur among members of the upper stratum of society, but there is also a well-defined form, known as "poor man's gout," that is due in part to the excessive use of alcoholic beverages.

(7) **Lead.**—We have seen a large number of cases that were clinically fairly typical of gout in which the patient had been employed in the handling of lead for from six to twenty years.

(8) **Traumatism** is a rare predisposing factor.

Symptomatology.—Clinical Picture of Acute Gout.—The onset is that of a more or less typical acute arthritis, and is at times preceded by prodromal symptoms that vary greatly with the individual cases. Slight muscular cramps and pains, dyspepsia, mental irritability, inability to sleep, and depression of spirits may antedate an attack of acute gout, following which there may be a period of relief just prior to the outbreak of an acute attack.

Attack.—This generally develops during the *morning hours*, and the patient may be awakened by severe *pain* in the region of the great toe. Pain soon becomes agonizing, and is described as though the toe were held in a vise. Within the course of a few hours the joint becomes *swollen* and *tender*, is unusually *hot* to the touch, and for some distance from the joint the skin pits upon firm pressure. Within the course of a few hours the suffering generally abates, the fever falls, and the patient becomes bathed in perspiration. After a mild attack of gout the patient may go about his usual work. One or two days after an initial attack there is a variable degree of enlargement of the joint, the edema returns, and the paroxysm recurs during the night or early morning hours. The number of paroxysms will vary greatly for a period of from six to eight days, although after the first forty-eight hours they are likely to be less severe. By the end of the first week the joint has usually returned to the normal size, and the patient suffers no inconvenience.

Acute gout returns at varying intervals, the longest of which occurs between the first and the second attack. A single joint may be affected, although after repeated attacks other joints commonly become involved.

Thermic Features.—With the onset of the paroxysm the temperature rises to from 101° to 103° F.

Retrocedent Gout.—This is a sudden transmission of the arthritic symptoms to some internal organ, and during a paroxysm the joint-inflammation may quickly disappear and intense pain develop in the region of the stomach. Faintness, nausea, vomiting, and diarrhea are now common. The pulse becomes rapid, weak, and at times compressible. Precordial pain, dyspnea, palpitation, and mental anxiety are occasionally referred to as *suppressive gout.*

Nervous Manifestations.—Severe cerebral symptoms are occasionally observed, and are most often dependent upon uremia.

Clinical Features of Chronic Gout.—This clinical type is secondary to the acute variety. The transition is gradual, the intervals between the attacks are short, whereas the attacks themselves grow milder and milder, but are of longer duration. Local inflammation subsides, and in long-standing cases disappears entirely. There is a tendency for the disease to involve other joints, and while at first corresponding joints are attacked, later there is gouty involvement of the other joints of the feet, and eventually the articulations of the hands and wrists are attacked.

Deformity ensues as the result of an accumulation of chalky deposits in and about the articular surfaces. The *skin* covering the chalky deposits may undergo ulceration, and the calcareous material be discharged.

Associated Conditions.—Among these are chronic nephritis, general arteriosclerosis, cardiac hypertrophy, myocarditis, valvular heart disease, and chronic catarrh of the gastro-intestinal tract.

Irregular Gout.—Under this head should be included those cases in which some of the symptoms of gout exist, with or without the presence of gouty deposits around the articular surfaces of certain joints. In irregular gout there is usually a history of a hereditary tendency to develop the disease, although this form of gout may be acquired.

Clinical Manifestations.—Pains are generally localized to the muscles, but may occur in any portion of the body, and are acute and lancinating in character. Certain groups of muscles are particularly prone to be affected, as, *e. g.*, the muscles of the neck, of the lumbar region, and of the thighs.

Gastro-intestinal Disturbances.—These resemble closely those seen in typical cases of lithemia. Colic, accompanied by diarrhea, is an occasional feature. During the course of irregular gout the patient may also suffer from parotitis, bronchitis, tonsillitis, and catarrh of the laryngeal mucous membrane.

Nervous Manifestations.—Cephalalgia, facial neuralgia, sciatica, a burning sensation of the skin, tingling of the extremities, pain in the hands and feet, and intense itching are among the less common nervous manifestations of this type of the disease. In selected cases eczema may occur. Gouty neuritis is occasionally seen, and in practically all cases the temperament becomes extremely irritable as the disease progresses.

Laboratory Diagnosis.—The urine is of high color, high specific gravity, often scanty, and on standing uric acid is deposited. These features are not, however, peculiar to gout alone. In many cases uric acid is in excess only at intervals, whereas at other times it may be diminished in quantity. Following the administration of atophan, Tuelzer found the uric acid in the urine increased. A heavy reddish precipitation in the urine of gouty subjects, following the use of atophan, is of diagnostic value when accompanied by amelioration of annoying symptoms. Glycosuria may develop at any time during the course of chronic gout. Albuminuria may be present, and oxaluria is also an occasional finding.

Ocular Manifestations.—Tophi in the cornea, and rarely in the eyelids, as well as conjunctivitis, keratitis, iritis, hemorrhagic retinitis, and glaucoma may accompany the other manifestations of gout.

Auditory Phenomena.—Impairment of the auditory sense may be observed within a few months after birth, and rarely occurs in long-standing cases.

Diagnosis and Differential Diagnosis.—Gout is diagnosed chiefly from the clinical history, which includes one or more of the conditions known to predispose to this disease. The onset of the initial attack, and its tendency to return at intervals and to spread to other joints, are characteristic of gout. The presence of mild fever at the onset of each attack and the development of distinct nodular masses in the vicinity of the articular surfaces go far to support the diagnosis. Disturbances of the gastro-intestinal tract are also of some value in formulating the diagnosis.

Gout is to be distinguished from **chronic rheumatism,** although the fact that the former disease displays a special predilection to attack the small joints (great toe), whereas chronic rheumatism commonly involves the larger

joints, must be borne in mind. The characteristic onset of each attack of gout is but mildly evidenced during the course of chronic rheumatism.

Acute Articular Rheumatism.—This form of rheumatism may resemble closely an attack of gout, from which it is distinguished by the general clinical picture of articular rheumatism, including not only joint involvement, but also a tendency toward the development of endocarditis.

Clinical Course.—The primary attack usually lasts for from eight to fourteen days, and the condition is likely to recur within a few weeks, months, or years. Gout is characterized by repeated exacerbations of pain in one of the small joints. The course of any clinical type of gout is decidedly modified by treatment, which varies greatly in different cases.

LITHEMIA.

Remarks.—This condition is believed to be due to disturbed cellular metabolism, and is characterized chemically by the presence of an excess of uric acid in the blood, which in turn gives rise to certain symptoms referable to the circulatory, gastro-intestinal, and nervous systems. Stockton contends that lithemia is a variety of gastro-intestinal autointoxication, and it is generally conceded that heredity and alcoholism contribute largely toward the development of lithemia. Males are more often attacked than females.

Clinical Picture.—This resembles closely that previously described under Irregular Gout, and for a description of the circulatory, respiratory, integumentary, and nervous manifestations of lithemia the reader is referred to p. 1008.

Gastro-intestinal Phenomena.—The *appetite* varies—at times it is voracious, and at others it is impaired or perverted. The *tongue* is coated, there is a metallic taste in the mouth, and the various forms of *indigestion* are common. Pyrosis, gastric oppression, and at times nausea and vomiting are conspicuous symptoms. *Flatulence* is somewhat common, and intermittent attacks of *diarrhea*, often accompanied by unusually foul-smelling discharges, are seen. Moderate *hemorrhage* from the rectum and hemorrhoids are encountered in typical cases. At times the patient complains of unusual *tenderness* along the border of the liver, and, indeed, this organ may be felt well below the costal margin.

OBESITY (POLYSARCIA ADIPOSA; LIPOMATOSIS UNIVERSALIS).

Pathologic Definition.—A disease of metabolism, characterized by the deposit of an abnormal amount of fat in the areolar tissue of the body. Not only is the adipose tissue greatly increased in localities in which it is normally found, but the various internal organs and tissues that are normally quite free from fat may show decided fatty infiltration. The condition is often accompanied by hypertrophy and dilatation of the heart. (See p. 299.) Fatty changes may also be present in the arterial system, and endarteritis with sclerosis and varicose veins are often encountered. Histologically, the fat-globules will be found to vary in different forms of obesity, the globules being larger in the plethoric variety of the disease than in the anemic or hydremic form.

Predisposing and Exciting Factors.—Among the chief predisposing factors are heredity, climate, habit, occupation, temperament, age, and sex. Among 543 cases that came under our care in which the family

history was noted, heredity was distinctly traceable in 60.7 per cent. Gout either occurred in association with the condition or was present among the antecedents in 43.2 per cent. of these cases, and the same was true of rheumatism in 35.5 per cent. In 10 the condition dated from longer or shorter periods of enforced rest, as following accidents, and infectious diseases, such as typhoid fever (in 4.7 per cent.). In 16.2 per cent. of the cases the disease dated from childbirth, and in 4.8 per cent. of 437 females it followed marriage.

Climate.—Obesity is more frequent among the inhabitants of hot, moist climates, and of low countries in the temperate and arctic regions. Hence it is commonly observed among Orientals, Dutchmen, South Pacific Islanders, southern Italians, and certain Africans.

Social Condition.—Sedentary habits and occupations constitute a common predisposing factor. A sluggish temperament also favors the accumulation of an abnormal amount of fat.

Age and Sex.—Most cases occur in persons of advanced middle life, *i. e.*, between forty and fifty years of age, but hereditary obesity often dates from infancy and early childhood; in women it may appear at puberty, and between the thirtieth and fortieth years. Women seem to be more prone to corpulence than men. Congenital monstrosities (idiots, cretins, acephali) and hemiplegics are frequently found to be excessively fat, as are also those suffering from anemia.

Exciting Factors.—An important exciting factor in the production of obesity is the ingestion of fat-building foods and the excessive use of alcoholic beverages, all of which tend to favor the accumulation of fat, irrespective of the amount of exercise taken.

Clinical Picture.—The patient may complain of inconvenience and of discomfort on walking or on working. As the viscera become involved, subjective symptoms develop. An early and annoying symptom is dyspnea upon exertion, due to a weak heart and to interference with respiration by heavy chest-walls and the upward crowded diaphragm. In plethoric subjects the face and mucous membranes are red and congested, whereas in anemic subjects the skin is pale, the muscles are flabby and weak, the pulse is small and compressible, and dyspnea, palpitation, weariness, drowsiness, and vertigo are present. In plethoric corpulent subjects the muscles are firm and strong, and the pulse and heart-beats are vigorous; late in the disease, however, the pulse becomes weak and irregular, and finally tachycardia may be seen. "Muscular power may diminish, and irregular fat masses (in the anemic variety) in the subcutaneous tissue are seen." Gastric catarrh and gastrectasia, inordinate thirst, and bulimia may be observed. Constipation may be followed by chronic diarrhea.

Sexual Peculiarities.—Sexual desire is diminished, and azoöspermia is not rare. Corpulent women often suffer from uterine displacement and prolapse, and amenorrhea, sterility, endometritis, and leukorrhea are observed.

Cutaneous Phenomena.—The skin is often irritated (intertrigo) by the excessive sweating and by the friction of cutaneous surfaces in the folds of fat, as under the breast, in the abdominal and inguinal regions, and around the scrotum and labia. This may be followed by eczema, painful excoriations, pruritus, acne rosacea, and alopecia.

Physical Signs.—The liver is commonly found to be enlarged, but owing to the presence of excessive fat and thickness of the abdominal wall this finding is best obtained by means of auscultatory percussion. The intensity of the heart-sounds is dependent directly upon the degree of cardiac hypertrophy or of degeneration of the heart muscle presented by

each individual case. The signs of fatty heart are often obtained upon physical examination.

Laboratory Diagnosis.—In the anemic variety the condition is due to chlorosis; in the plethoric form the red cells will be found to fluctuate between 6,000,000 and 9,000,000 per c.mm., and the hemoglobin will commonly exceed 110 per cent.

The urine may be normal, although at times polyuria and again oliguria may be present. As a rule, the urine is rich in urates and uric acid.

Diagnosis and Differential Diagnosis.—The existence of associated conditions, complications, and sequelæ should be carefully ascertained. In myxedema the skin is thick and inelastic, and the physiognomy is much altered, the lips, tongue, nostrils, and mouth being thickened by infiltration. Obesity is also to be distinguished from adiposis dolorosa. (See p. 1154.)

Complications.—Hernia, cardiac asthma, bronchitis, pulmonary congestion, edema, arteriosclerosis, albuminuria, glycosuria, anginal attacks, cerebral hemorrhage, and coma have all been observed.

ADIPOSIS TUBEROSA SIMPLEX.

This condition resembles adiposis dolorosa (Dercum's disease) clinically, but differs from the latter in that it is apparently dependent upon general obesity, with which it has been thus far invariably found to be associated.

"Circumscribed fat masses appear in the subcutaneous tissues; they form distinct, moderately dense, slightly movable, somewhat flattened tumors, ranging in size from a bean to that of a hen's egg. Their number varies all the way from one-half dozen to two dozen or more. These moderately firm fat-nodules are not distributed over the entire body, but in some cases are confined to the extremities, particularly the lower, and in others to the abdomen. The tumor masses show no tendency to fuse together, and are not elevated above the surrounding surface; they are sensitive to the touch, and may be the seat of pain, which varies in intensity within rather wide extremes, being moderately severe and distressing in rare cases and trivial or even absent in the majority of instances. The lymphatic glands are not involved, and the skin remains soft, flexible, and non-adherent. The mental processes are normally active, and also the muscles; asthenia is not present, and there is no more indisposition to physical exertion than is observed in cases of obesity, as a rule. The knee-jerks are present, and the cutaneous sensibility is unaltered, in some cases at least. The mammæ and abdominal panniculus adiposis may be overhanging or pendulous, but not in all cases. It is an uncommon condition, since it was noted in only 4 out of a total of 324 cases."*

From the nodular variety of *adiposis dolorosa*, the condition under discussion distinguishes itself by the absence of any psychic disturbance and asthenia out of proportion to the polysarcia, and more particularly the complete disappearance of the fat masses as the result of treatment directed to the extreme obesity. *Lipomas*, by their painlessness, soft, doughy, semi-fluctuating consistence, their more globular shape, as evidenced by the slight though distinct elevation above the surrounding surface and more or less lobulated character, may be also excluded. Moreover, these tumor-like subcutaneous growths are not dependent on associated general obesity, and are not amenable to medicinal, dietetic, and regiminal treatment.

* American Journal of Medical Sciences, March, 1908, by James M. Anders, M.D.

DISEASES OF THE NERVOUS SYSTEM.

By T. H. Weisenburg, M.D.

General Considerations.

In making a diagnosis of any organic nervous disease, each symptom should not only be appreciated but also interpreted as being associated with a definite lesion of a certain part of the nervous system. To do so it is necessary to have an accurate knowledge of its anatomy, physiology, and pathology. This is not so difficult if certain general principles are observed.

The nervous system consists of the brain, the spinal cord and the sympathetic system of plexuses, and the nerves connecting with the peripheral parts of the body and the internal organs. The brain is the principal part of this system, and in it are represented the so-called centers for every possible voluntary and involuntary movement, consciousness, and thought. The spinal cord is the pathway for fibers coming from the brain, and in it are nerve-cells which represent every part of the body, with the exception of the internal organs, which are similarly represented in the sympathetic plexuses (Fig. 367).

The brain consists of two lateral hemispheres, connected by a band of white fibers called the corpus callosum; of a system of ganglia which are in the center and between the hemispheres, this including the caudate nucleus, optic thalamus, and lenticular nucleus; and of the cerebellum, consisting of two lateral hemispheres and a central part or the vermis. Within the brain are a system of communicating cavities or ventricles which are in continuation with the central cavity of the spinal cord. These are the two lateral ventricles, occupying parts of the lateral hemispheres; the third ventricle, situated between the optic thalami; and the fourth, which is between the cerebellum and the medulla oblongata, communicating below with the central canal of the spinal cord and above with the third ventricle by means of the aqueduct of Sylvius. These cavities in life are filled with fluid. The brain is intimately surrounded by the pia, which dips between the convolutions and into the fissures, accompanying the vessels into the interior. Surrounding the pia is the dura arachnoid, which envelops all parts of the brain and subdivides the cranial cavity into two parts—that which includes the cerebrum proper, or the two lateral hemispheres, and the posterior part, which covers the cerebellum and is called the tentorium cerebelli.

Anatomically the brain consists of a system of convolutions and fissures arranged in a definite manner for a given purpose. The outer part of the convolution is called the cortex, it being from $\frac{1}{8}$ to $\frac{1}{4}$ of an inch in thickness, and consists of a system of nerve-cells arranged in layers. From these nerve-cells arise nerve-fibers. Those which go into the internal capsule are called the projection fibers and are concerned with motor, sensory,

and special functions, while the fibers which connect one part of the brain with another are called the association fibers, and are concerned with the correlation of function of the different parts of the brain.

As a result of investigation definite functions have been assigned to different parts of the cortex, these being motor, sensory, and special. The general underlying principle is that in the cortex are represented, as, for instance, in the motor, movement, and any irritation of a motor center will produce movement of the related part in the opposite side of the body, while destruction will cause loss of that movement. The same principle is true of the other portions of the cortex. Not much is known of the function of the so-called central ganglia; that is, of the caudate and lenticular nuclei and the optic thalamus. The two cerebral hemispheres are in

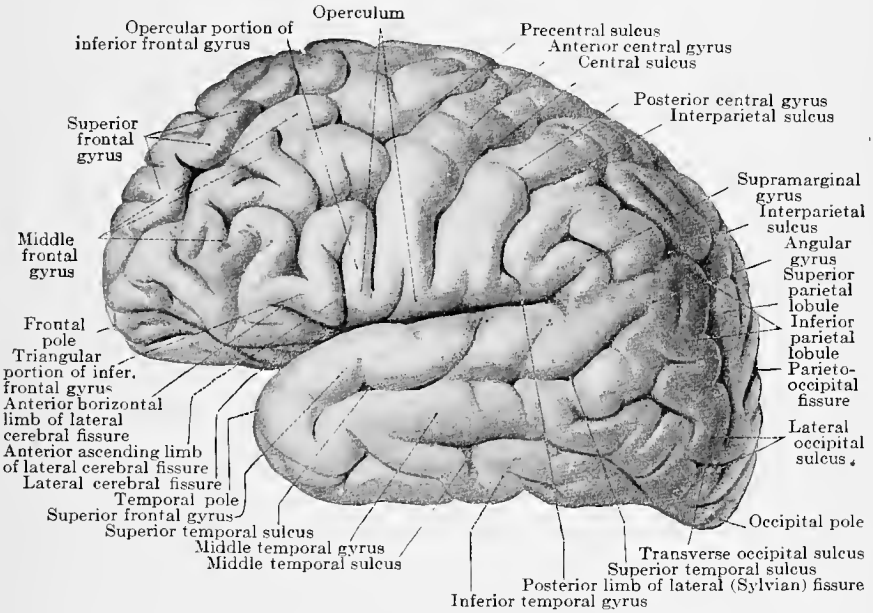


FIG. 367.—THE FISSURES AND CONVOLUTIONS OF THE CEREBRAL CORTEX AS SEEN FROM THE LEFT SIDE. THE CEREBELLUM AND BRAIN-STEM HAVE BEEN REMOVED (Sobotta and McMurrich).

intimate connection with the cerebellum, which is the great coördinating center of all voluntary and involuntary movements.

Motor Symptoms: Method of Obtaining Them and Their Interpretation.—Every part of the body has a nuclear representation in the nervous system. By that is meant that every muscle-fiber is in relation with cells which are located in the anterior horns of the spinal cord throughout its whole extent, and in the so-called nuclei of the different cranial nerves from the third to the twelfth inclusive, in the crus, pons, and medulla, these parts being really the upward continuation of the spinal cord. Thus, for instance, if there is a destruction of the nerve-cells in the facial nuclei in the pons, there will be loss of function in the muscles of the face; and if there is a disturbance of the cells in the anterior portions of the spinal cord in the lumbar region, there will be alteration of function in the corresponding muscles of the leg (Fig. 368).

In other words, there are two great forms of representation in the nervous system: That in the cortex, which is concerned purely with motion; while in the spinal cord and its prolongation, including in this the crus, pons, and medulla, are represented not motion, but the innervation of the individual muscle-fibers. There must necessarily be a connection between these two systems, and this is effected by means of the motor tracts. These have their origin in the motor centers in the cortex. From here they go through the anterior part of the posterior limb of the internal capsule, and those fibers which are concerned with the movements of the opposite side of the body below the head go through the crus, pons, cross over in the medulla, and then are transmitted by means of the crossed pyramidal tracts into the spinal cord. Some of these fibers do not decussate in the medulla, but descend on the same side of the cord in the direct pyramidal

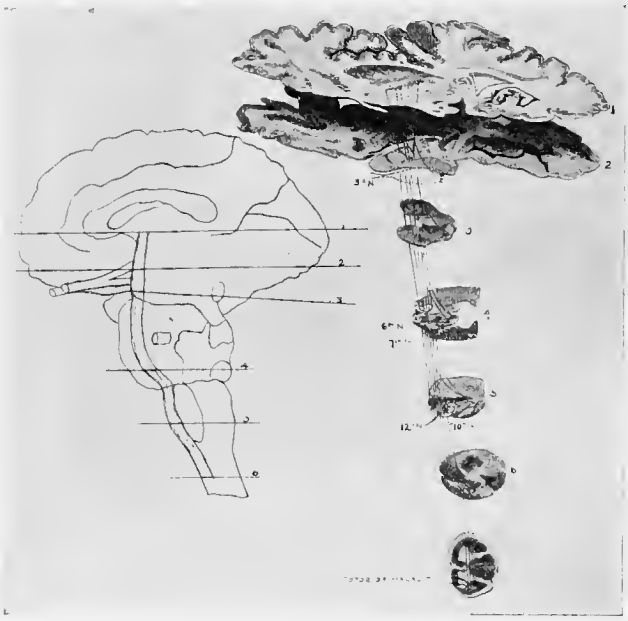


Fig. 368.—Diagram showing Course of Motor Fibers (Pickett).

tract. From here these fibers go to the cells in the anterior horn. For example, the nerve-fibers which come from the nerve-cells in the leg center first go through the internal capsule, then the crus, pons, cross over in the medulla, then go through the crossed pyramidal tract all the way down the cord to the lumbar segment, and then join the cells in the anterior horn, while the fibers in association with the arm only go as far as the cells in the anterior horn of the cervical part of the cord. Correspondingly, the motor fibers which come from the face center go through the internal capsule and leave the motor tracts in the upper part of the pons because the facial nuclei are in the lower part of the pons, while those fibers which are in relation with the muscles of the eyeball begin to leave the motor columns just below the internal capsule and above the crus because the oculomotor nuclei are in the crus and pons.

THE UPPER AND LOWER MOTOR NEURONS OR SYSTEMS.

The upper part of the motor system, that is, the cortical cells and the fibers coming from them to the nuclei of the motor cranial nerves in the crus, pons, and medulla, or to the cells in the anterior horns of the spinal cord, but not including them, is called the upper motor neuron or system, and by the lower motor neuron or system is meant the nuclei of the motor cranial nerves or of the cells of the anterior horn of the spinal cord and the fibers coming from them, this including the motor peripheral or cranial nerves as far as their ending in the periphery. We see, then, that each neuron or system, so called, has its own center or nerve-cell and a fiber continuation.

A normal relation between the upper and lower motor neurons or sys-

FIG. 369.—METHOD OF OBTAINING BICEPS REFLEX.

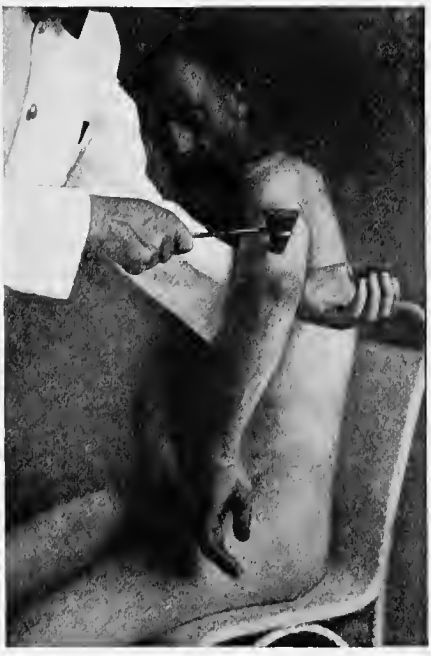


FIG. 370.—METHOD OF OBTAINING THE TRICEPS REFLEX.

tems is necessary in the performance of any movement, for while the impulse originates in the upper neuron, its performance is the result of the action of the lower, and if there is a lesion in either there necessarily results a disturbance of this relation or tone. Thus, in a lesion of the upper neuron there will be overaction of the lower, and vice versa. This overaction on the part of the lower motor neuron is manifested by an exaggeration of the tendon reflexes and spasticity or stiffness in movement, and on the part of the upper by a flaccidity or loss of tone and abolition of reflexes. In other words, in a lesion in the upper motor neuron there will be spasticity and increase in the tendon reflexes, while in a lesion of the lower, flaccidity and loss of the tendon reflexes.

REFLEXES.

It is, then, upon the normal relation or the tone between the upper and lower motor neurons or systems that the condition of the tendon reflexes will depend. Every reflex has its physiologic arc, this consisting of a sensory impulse, a center, and a motor response. The simplest example is the knee or patellar jerk, in which, after tapping the patellar tendon, the impulse is carried by the sensory nerves and posterior roots to the cells of the anterior horn in the second, third, and fourth lumbar segments, and from here the motor response is transmitted by the anterior roots and the peripheral motor nerves. If there is a lesion in any portion of this arc, there will be loss of the reflex, no matter what the condition above in the spinal cord and brain. The first principle, then, in the attainment

FIG. 371.—METHOD OF OBTAINING THE PATELLAR JERK.

FIG. 372.—METHOD OF OBTAINING THE ACHILLES JERK.

of any reflex is to have its arc intact and in normal condition. Should, however, there be a lesion in any portion of the upper motor neuron or system anywhere in its course, this disturbing the normal tone, there will result exaggeration of the reflexes because of loss of cerebral or what has often been called inhibitory influence. That every reflex has a cerebral influence is proved by the fact that if there is a complete transverse lesion, for instance, in one segment of the cervical cord, all the reflexes below are lost, even though the arcs are intact.

Reflexes are of two kinds: First, deep or tendon; and second, superficial or skin. The usual tendon reflexes employed are:

(*a*) In the Upper Limb: The biceps and triceps.

The biceps reflex is obtained by having the patient flex the arm on the forearm, at right angles, with the thumb of one hand of the examiner on the biceps tendon. Striking the thumb with the percussion hammer will produce flexion of the forearm upon the arm (Fig. 369). The center of the reflex is in the fifth cervical segment.

Triceps Reflex.—With the arm in the same position, if the triceps tendon is struck near its insertion in the elbow, extension of the forearm on the arm will result (Fig. 370). Spinal center is in the sixth and seventh cervical segments.

(*b*) In the Lower Limb:

The knee or patellar jerk is best obtained by crossing one leg over the other and striking the patellar tendon near its insertion. A forward movement of the leg will result (Fig. 371). Spinal center is in the second, third, and fourth lumbar. When the reflex is diminished, it may sometimes be brought out by reinforcement. This is done by having the patient lock his hands and then pull them apart, the examiner striking the tendon at the time of the greatest effort.

Achilles jerk is best obtained by having the patient kneel on a chair and then tapping the Achilles tendon near its insertion into the heel. A flexion of the foot on the leg will result (Fig. 372). Spinal center is in the first sacral.

Ankle and Patellar Clonus.—A clonus is obtained only when there is an exaggerated tonicity, and always indicates a lesion of the motor or pyramidal tracts. *Ankle clonus* is obtained by first flexing the leg upon the thigh. With one hand held over the calf of the leg, the other holding the foot, a sudden flexion of the foot on the leg is made, this resulting in to and fro movements which are regular in rhythm (Fig. 373).

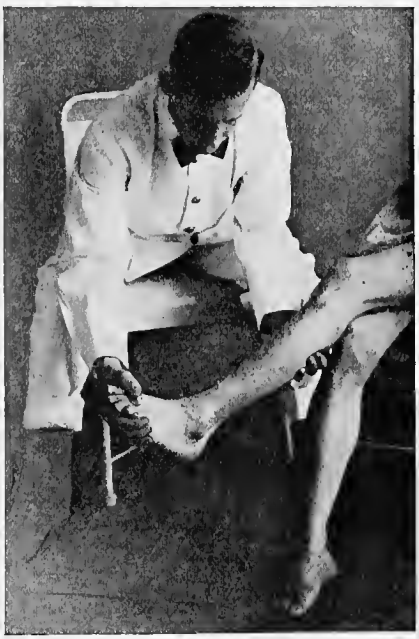


FIG. 373.—METHOD OF OBTAINING ANKLE CLONUS.

Patellar Clonus.—With the leg extended on the thigh the patella is grasped between the thumb and the forefinger and suddenly brought forward.

Biceps and triceps clonus is sometimes obtained similarly to that of the ordinary reflexes.

Rarely, ankle clonus and sometimes patellar clonus can be obtained in hysteria, but the movements are not regular and the rhythm is influenced by the will.

Skin or Superficial Reflexes.—The abdominal or umbilicus reflex is obtained by stroking on one side of the abdomen, the umbilicus moving toward the side stroked. Spinal center is in the ninth, tenth, and eleventh thoracic segments.

The cremasteric reflex is obtained by irritating the inner portion of the

upper thigh, this resulting in upward movement of the scrotum. Spinal center is in the first lumbar segment.

Plantar reflex is obtained by irritating the plantar surface of the foot, flexion of the toes resulting. Spinal center is in the second sacral.

Babinski reflex is obtained by irritating the plantar surface, extension of the toes resulting. Irritation is best produced with a match, which should first be drawn along the outer part of the foot and then across the sole. The important part of this reflex is the extension of the large toe, and the movements of the small toes may be disregarded. It is also advisable to first grasp the foot at the ankle so as to prevent any voluntary movement. This reflex is never obtained in a functional condition and is always indicative of a lesion of those motor fibers which are in relation with the leg. A lesion of the motor fibers in relation with the upper limb will not produce this reflex (Fig. 374).

FIG. 374.—METHOD OF OBTAINING THE BABINSKI REFLEX SHOWING EXTENSION OF THE LARGE TOE.

PARALYSIS.

The nature of the paralysis will also differ in lesions of the two neurons or systems. In a lesion of the upper, the paralysis will be that of movement. For example, should there be a lesion of the fibers in relation with the arm, there will be total loss of movement in the arm, while in a lesion of the lower neuron, the extent of the paralysis will depend upon the degree of involvement either of the nerve-cells or of the motor fibers innervating the arm. In the former there will be an exaggeration of tone, or spasticity, with increased reflexes; in the latter loss of tone, or flaccidity, with loss of reflexes. Besides, in the latter condition, as the cells of the anterior horn are trophic in function, there will be atrophy of the muscles and electrical reactions of degeneration. Disturbances in vasomotor function occur in both.

SENSORY SYSTEM.

The arrangement of the sensory fibers is more complex than that of the motor because of the great number and variety of sensations. The principle, however, is the same. There is still a great lack of definite knowledge regarding the course of the sensory fibers. It must be remembered that while motor impulses travel from the cortex, sensory impulses are transmitted to the cortex by means of the peripheral sensory nerves and the spinal cord. It is possible that in the cortex, sensation is represented similarly to that of motion, and that, just as in the motor area there is representation of motion, so in the sensory centers there is representation of the sensation concerned in the particular movement, and that there is a correla-

tion of function between the two. In support of that theory is the fact that the cortical motor and sensory centers concerned in a particular movement are in apposition.

Within a few years our views of the subdivisions of sensation have been considerably modified, chiefly through the work of Head, in association with Sherren and Rivers. Instead of the usually accepted subdivisions of touch, pain, and heat and cold, these authors have shown that common sensation is based upon three kinds of sensibility which are present and may be demonstrated in the peripheral system:

"(1) A system corresponding to the group of impulses which they have called deep sensibility. The end-organs of this system respond to the stimulus of pressure and to the movement of joints, tendons, and muscles. Painful impulses can also arise within this system in consequence of injury of a joint or excessive pressure. This sensory mechanism is capable of responding in such a way that the patient appreciates both the locality of the stimulus and the direction of movement in any joint which lies within an area innervated solely by this system; and yet the integrity of deep sensibility carries with it no power of appreciating a stimulus, such as that of cotton-wool, even over hairy parts. Nor does it permit of the discrimination of two compass-points applied simultaneously to the skin, even when widely separated.

"The fibers which connect these sensory impulses run mainly with the muscular nerves, and are not destroyed by division of all the sensory nerves to the skin.

"(2) The protopathic system, capable of responding to painful cutaneous stimuli and to the more extremes of heat and cold. Its end-organs are grouped in points on the surface of the body, sensitive to one only of these stimuli. Their response is diffuse, and unaccompanied by any definite appreciation of the locality of the spot stimulated.

"(3) The epicritic system. To the impulses of this system we owe the power of cutaneous localization, of discriminating two points, and of recognizing the finer grades of temperature, called cool and warm."

These three forms of sensibility are transmitted from the periphery by means of the posterior roots into the spinal cord. Anatomically, the posterior roots are supposed to contain five different sets of fibers, each having a different termination (Fig. 375).

One goes to the nerve-cells of the anterior horns of the same side and is supposed to subserve reflex functions.

The second ends in the cells of Clarke's column, this consisting of a group of cells situated at the base of the posterior horns, running along the whole extent of the spinal cord, but principally from the first thoracic to the second lumbar. From these cells new fibers arise which run to the postero-lateral portion of the same side of the cord, forming the so-called direct cerebellar tract, lateral to the motor columns and behind the tract of Gowers. This tract maintains this position in the spinal cord and enters the cerebellum by means of the inferior cerebellar peduncle and is supposed to end in the superior vermis.

The third set of fibers end in the nerve-cells of the posterior horns on the same side. From here new fibers arise, most of which cross over in the gray and white commissure, forming the so-called Gowers' tract, situated anterior to the direct cerebellar and outside of the lateral motor columns. These fibers ascend in the spinal cord and in the lateral portions of the medulla and pons, terminating in the lateral and ventral nuclei of

the optic thalamus. From here a new set of fibers arise, these ending in the sensory cortex. Some of the fibers of Gowers' tract, however, are given off along their course and end in the superior vermis of the cerebellum and in collateral nerve-cells situated in the medulla. Gowers' tract also receives a number of fibers from the cells of the posterior horn on the same side.

Not all the fibers arising from the cells in the posterior horns ascend in Gowers' column. A number cross over in the white commissure, forming the so-called anterior tract, situated in the anterior portion of the cord in front of the anterior horns. Their course is not accurately known, but some of the fibers ultimately reach the nuclei of the optic thalamus, and from here a new system is given off to the sensory cortex.

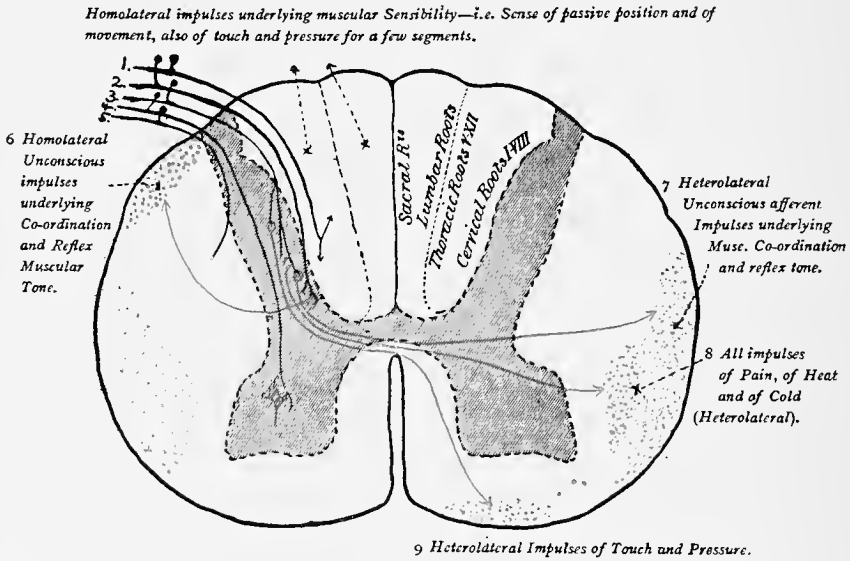


Fig. 375.—Diagram to Illustrate the Termination of Peripheral Afferent Fibers in the Spinal Cord, and the Origin of the Secondary Central Paths (partly after Edinger), with a Brief Summary of their Function.

1, Bundles of fibers passing up in the posterior column—many myelopetal (to spinal cord) and the remainder bulbopetal (to posterior column nuclei); 2, fibers terminating around the cells of Clarke's column; 3, fibers arborizing around cells in the posterior horn, and intermediate gray matter; 4, ditto around the anterior horn cells; 5, ditto swerving into the lateral column to neighboring gray matter; 6, direct, or dorsal spino-cerebellar tract; 7 and 8, Gowers' tract, *i. e.*, (7) ventral spino-cerebellar tract, (8) tract. spino-thalamic et tectalis; 9, ascending tract in the anterior column (W. Page May).

The fourth system of fibers enters the posterior columns, forming the tracts of Goll and Burdach, and ascends in the spinal cord, terminating in the nucleus cuneatus and gracilis in the medulla oblongata. Every fiber after its entry in its course upward runs mesially, the fibers from the root above forcing it inward. Thus, in the column of Goll are transmitted the fibers from the posterior roots of the sacral, lumbar, and lower thoracic from the fifth to the twelfth inclusive, while in the column of Burdach, which first becomes evident in the fifth thoracic segment, are transmitted the fibers from all the cervical and the upper five thoracic roots. The fibers of the column of Goll terminate in the nucleus gracilis, and of the column of Burdach in the nucleus cuneatus. From these nuclei new fibers arise forming the internal arcuate fibers which decussate in the medulla and run upward, forming the so-called lemniscus or median fillet. These

tracts occupy a position in the median line just back of the motor fibers, in both the medulla and pons, and terminate in the lateral and ventral nuclei of the optic thalamus. From here new fibers arise which run through the posterior part of the posterior limb of the internal capsule, terminating in the sensory cortex (Fig. 376).

The fifth arborize around the cells of the gray matter lateral to the horn. Their further course is not definitely known.

The Three Sensory Neurons or Systems.—We see, then, by tracing the course of the sensory fibers after their entry into the spinal cord, that there are three sets of neurons or systems, instead of two, as in the motor. The first or peripheral has its center in the cells of the ganglia on the posterior roots. The fibers coming from these cells divide in a "T"-shaped manner, the external divisions going to the end-organs by means

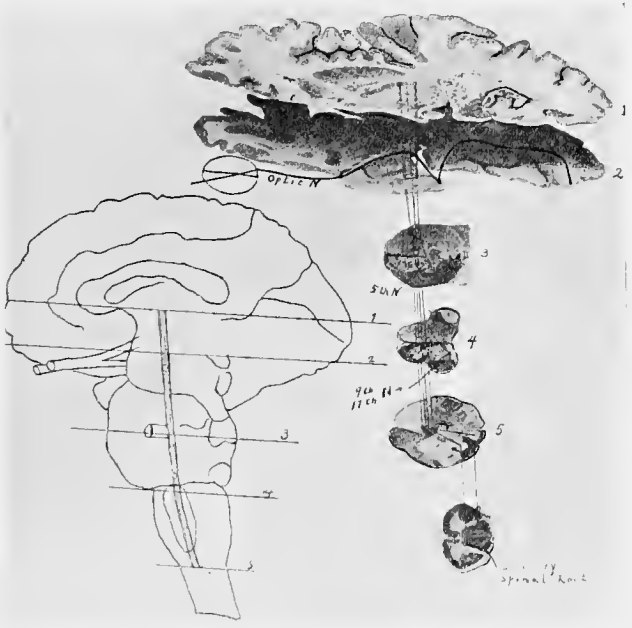


Fig. 376.—Diagram showing Course of Sensory Fibers (Pickett).

of the peripheral nerves, and the central part into the spinal cord, where they divide into descending and ascending branches. The descending branches are probably reflex in function, while the ascending transmit the different forms of sensibility, terminating in the nerve-cells in the gray matter of the same side of the spinal cord, or in the posterior column nuclei in the medulla oblongata.

The second neuron or system starts from these nerve-cells and ends in the median and lateral nuclei of the optic thalamus, it consisting of the columns of Gowers, the anterior tracts, and median fillet.

The third neuron or system starts at the optic thalamus and ends in the cerebral sensory cortex.

It is important to keep the anatomic limits of the three neurons in mind, for the sensory disturbances resulting from lesions will in each instance

differ. In the peripheral or first system the sensory symptoms will depend first of all upon the particular nerve or root diseased and its distributions, and alterations will occur in the three different forms of sensibility—the deep, protopathic, and epicritic.

Just as soon, however, as these peripheral types of sensibility arrive in the spinal cord, they are readjusted, and touch, pain, and temperature sensations arriving, no matter from what system, are transmitted in the second neuron along definite tracts, and instead of having the three peripheral types of sensibility, there will be disturbance either of touch, pain, heat, or cold in its entirety.

In the third system or neuron there is again a readjustment, and instead of separate pathways all the different forms of sensibility run together, and a lesion in any portion will cause disturbance either partially or totally of all forms.

THE TRANSMISSION OF SENSATION TO THE CEREBRAL CORTEX AFTER ITS ENTRY INTO THE SPINAL CORD.

Although we distinguish many different forms of sensibility, and while it is believed that each is transmitted along definite tracts in the spinal cord, it is probable that every type of sensation is transmitted along more than one tract.

Touch and Pressure Senses.—Sensation for touch probably ascends in the posterior columns or the columns of Goll and Burdach on the same side of the spinal cord to the respective nuclei in the medulla, and from there, after its decussation, by the median fillet to the optic thalamus, and then to the sensory cortex. It is also believed that touch sensation is transmitted by the heterolateral anterior columns. The important point is that destruction of one of these tracts will cause incomplete, while destruction of both causes complete, loss of touch sensation. The sense of pressure, which is transmitted in the peripheral nerves along different fibers from those of touch, ascends in the spinal cord with touch sensation, for nearly always loss of one will be associated with loss of the other. When considering loss of touch and pressure sensation, it must be remembered that most of the fibers concerned with these senses do not decussate in the spinal cord, and that unilateral lesions will cause a greater disturbance on the side of the lesion. This differs from the other forms of sensation because the fibers conducting them decussate in the spinal cord, and, therefore, unilateral lesions will cause the principal disturbance upon the opposite side. In testing for touch sensation it is best to employ cotton-wool or else the pressure of the examining finger. In testing for pressure, weights of various sizes should be used.

Pain and Temperature Sensations.—These are transmitted in the column of Gowers. While it is admitted that pain, heat, and cold are transmitted in these tracts, it is probable that separate fibers are concerned with each type, for it is possible to have disturbance of one without derangement of the other. In both the medulla and pons the fibers conducting pain and temperature sensations are apart from the other senses, but after their entry into the optic thalamus, they are conducted with them through the posterior limb of the internal capsule to the sensory cortex.

In testing for pain and temperature sensation test-tubes with hot and cold water should be used. It is advisable in a given case to have these always of the same temperature.

Muscle Sense.—By this term is meant every form of sensibility trans-

mitted from the muscle, tendon, capsule, and joint concerned with the movement of a part. As such it must be made up of the sense of position, of active and passive movement, of pressure, and of touch sensations. It is probable that this complex sense is transmitted on the same side of the cord in both the posterior and lateral columns. The impulses in the posterior are concerned with the conscious appreciation of this sense, while that transmitted in the direct cerebellar tract to the cerebellum is concerned with unconscious coördination.

Sense of Position.—The limbs should be absolutely relaxed and the eyes closed. When testing, for instance, the right upper limb, it should be placed in a given posture and the patient asked to put the other limb in the same position, or else be asked to describe where it is, or vice versa.

Sense of Movement.—The test is similar to that of the sense of position, the patient being asked to describe the movement or have the opposing limb moved in accordance with the movement of the examiner, or vice versa.

Ataxia.—By this is meant an incoördinate movement. In some diseases, as in locomotor ataxia, when the posterior columns are degenerated, the incoördination is increased with the eyes shut. In testing the upper limbs the patient should be asked to put his finger to his nose or to put them together. In testing the lower limbs it is best to place the patient on his back and have him move the heel to the knee, leg, or toe. In testing for the whole body, the patient should be asked to stand up and put his heels and toes together. If the patient staggers and falls, this is sometimes called *Romberg's sign.* The ataxic gait may be due to a disease of the posterior columns of the spinal cord, as in locomotor ataxia, or may be the result of cerebellar disease. These can be distinguished, however, because in the former the ataxia is increased when the eyes are shut and the incoördination is limited only to the limbs, whereas in cerebellar disease the whole body is ataxic and the patient walks like a drunken man. As a rule, closure of the eyes does not influence cerebellar incoördination.

Symptoms of Sensory Irritation.—If the irritation is in the cortical sensory area, there will be spasms of pain upon the opposite side of the body similar to the motor spasms or Jacksonian convulsions, the result of irritating motor lesions. If the irritation is succeeded by destruction, there will, of course, be disturbance of sensation or anesthesia upon the opposite side of the body, this depending upon the extent of the cortical area involved.

Sometimes, if an irritating lesion is present in the projection sensory fibers, as in the internal capsule, there may be pains upon the opposite side. These are known as *central pains.* As a matter of fact, they may result from an irritating lesion in any portion of the sensory tracts, whether in the pons or the spinal cord, as sometimes happens in syringomyelia.

If the irritation involves the posterior root or a peripheral nerve, there may be at first numbness or *paresthesia* in the related distribution. These paresthetic phenomena may be variously described, such as crawling, tingling, or pin and needle sensations. Greater irritation will cause pains which may be of a sharp, shooting character, such as are present in tabes, or a girdle sense described as a tight band around the waist or leg.

By *polyesthesia* is meant diffusion of sensation, a touch being appreciated in more than one point. By *allochiria* is meant the appreciation of sensory irritation in a corresponding part of the opposite limb.

Referred Pains.—The work of Head and Dana has demonstrated that there is a relation between superficial skin areas and diseases of the viscera and of the spinal segments. Because of this, in diseases of the

internal organs, pain is referred by means of the spinal nerves to certain skin distributions, which may be appreciated by the patient who complains

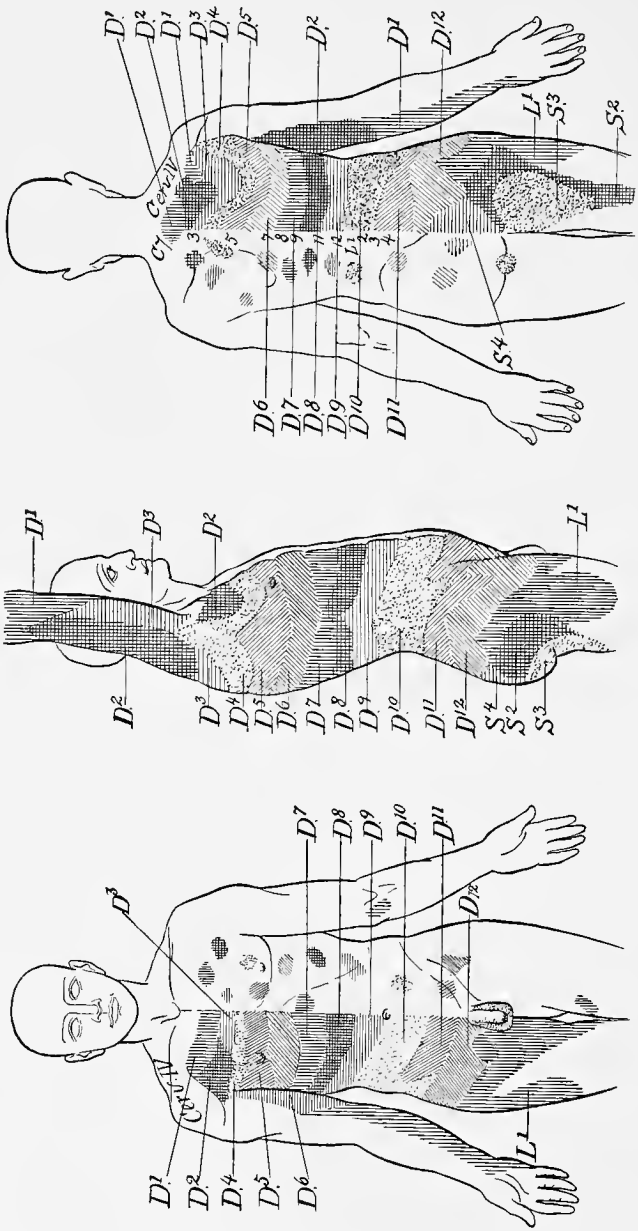


Fig. 377.—Cutaneous Areas and Maximal Points of Pain Related to the Vertebral Segments (after Head).

of a soreness or tenderness. The following table and illustrations from Head show the areas of referred pains (Fig. 377).

TABLE SHOWING AREAS OF PAIN REFERRED FROM VISCERAL DISEASE.

Heart.—First, 2d, 3d, dorsal segments.
Lungs.—First, 2d, 3d, 4th, 5th dorsal.
Stomach.—Sixth, 7th, 8th, 9th dorsal; cardiac end from 6th and 7th.
Pyloric end from 9th.
Intestines.—(A) Down to upper part of rectum.
Ninth, 10th, 11th and 12th dorsal.

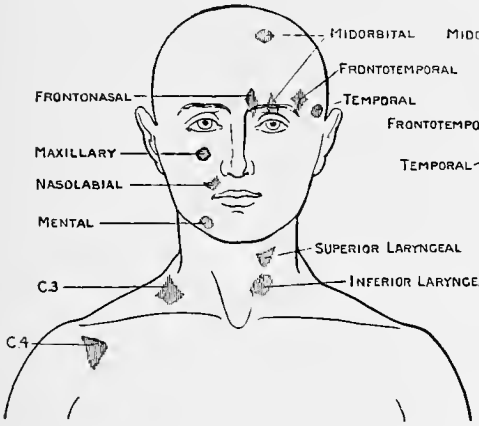


FIG. 378.

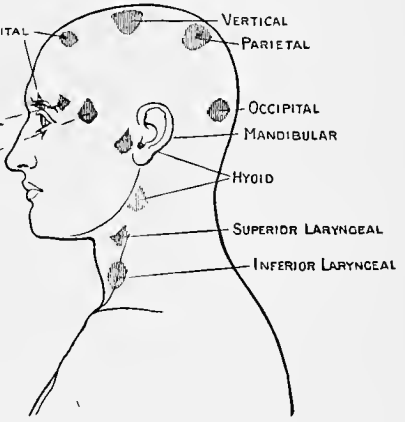


FIG. 379.

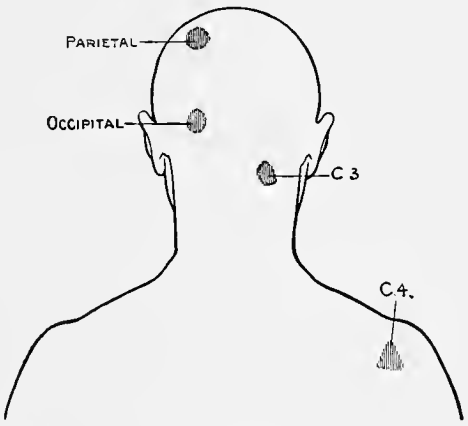


FIG. 380.

FIGS. 378, 379, AND 380.—MAXIMAL POINTS OF REFERRED AND ASSOCIATED PAIN ON THE HEAD AND FACE (after Head).

(B) Rectum.
Second, 3d, and 4th sacral.
Liver and Gall-bladder.—Seventh, 8th, 9th, 10th dorsal.
Perhaps 6th dorsal.
Kidney and Ureter.—Tenth, 11th, and 12th. The nearer the lesion lies to the kidney, the more is the pain and tenderness associated with the 10th dorsal. The lower the lesion in the ureter, the more does the 1st lumbar tend to appear.
Bladder.—(A) Mucous membrane and neck of bladder.
First, 2d, 3d, 4th sacral.

(B) Overdistention and ineffectual contraction.
Eleventh and 12th dorsal and 1st lumbar.
Prostate.—Tenth, 11th, 12th dorsal.
First, 2d, 3d sacral and 5th lumbar.
Epididymis.—Eleventh and 12th dorsal and 1st lumbar.
Testis.—Tenth dorsal.
Ovary.—Tenth dorsal.
Appendages, etc.—Eleventh and 12th dorsal and 1st lumbar.
Uterus.—(A) In contraction.
Tenth, 11th, 12th dorsal and 1st lumbar.
(B) Os uteri.
First, 2d, 3d, 4th sacral, and 5th lumbar very rarely.

In the following tables Head has also shown the relation of head pains to visceral diseases and diseases of the head and neck.

TABLE SHOWING ASSOCIATED PAINFUL AREAS ON THE HEAD RELATED TO VISCERAL DISEASE IN THE BODY.

Area on Body.	Associated Area on Scalp.	Organs in Particular Relation to these Areas.
Cervical, 3	Frontonasal(?rostral).	Apices of lungs. Stomach. Liver.
Cervical, 4	Frontonasal.	
Dorsal, 2	Midorbital.	Lung. Heart. Ascending arch of aorta.
Dorsal, 3	Midorbital.	Lung. Arch of aorta.
Dorsal, 4	Doubtful.	Lung.
Dorsal, 5	Frontotemporal	Lung. Heart.
Dorsal, 6	Frontotemporal.	Lower lobe of lungs. Heart.
Dorsal, 7	Temporal.	Bases of lungs. Heart. Stomach.
Dorsal, 8	Vertical.	Stomach. Liver. Upper part of small intestine.
Dorsal, 9	Parietal.	Stomach. Upper part of small intestine.
Dorsal, 10	Occipital.	Liver. Intestine. Ovaries. Testes.
Dorsal, 11	Occipital.	Intestine. Fallopian tubes. Uterus. Bladder.
Dorsal, 12	Occipital.	Intestine. Uterus, etc.

TABLE SHOWING ASSOCIATED PAINFUL AREAS RELATED TO DISEASE WITHIN THE HEAD AND NECK.

Organ at Fault.	Maxima of Pain and Tenderness.	Organ at Fault.	Maxima of Pain and Tenderness.
Ciliary muscle (errors of accommodation)	Midorbital.	Lower first and second molars	Hyoid and pain in the ear.
Cornea	Frontonasal.		
Iris	Frontotemporal. Temporal and maxillary.	Lower third molar	Superior laryngeal.
		Membrani tympani	Hyoid.
		Middle ear	Vertical and behind ear.
Vitreous (glaucoma)	Temporal.		
Retina	Vertical.	Tongue, tip	Mental.
Teeth (upper incisors)	Frontonasal.	Tongue, lateral part	In ear and hyoid.
Upper canine and first bicuspid	Nasolabial.	Tongue, base	Superior laryngeal. Occipital.
Upper second bicuspid	Temporal or maxillary.	Tonsil	In ear and hyoid.
		Nose, olfactory portion	Frontonasal and midorbital.
Upper first molar	Maxillary.		
Upper second and third molars	Mandibular.	Respiratory portion and posterior nares	Nasolabial.
		Larynx	Superior and inferior laryngeal.
Lower incisors, canine, and first bicuspid	Mental.		
Lower second bicuspid	Mental or hyoid.		

CEREBRAL LOCALIZATION.

Motor Centers.—The motor functions have been placed directly in front of the central or Rolandic fissure in the precentral convolution on the lateral surface of the brain, and in the anterior part of the paracentral convolution on the median surface. Every movement has its cortical representation, the head center being in the lower part of the precentral convolution, then the centers for the face, arm, trunk, abdomen, and leg, coming in order, that for the leg being on top. Thus, a man stands upside down in his motor cortex. Should there be any lesion such as would irritate any of these centers,—for instance, a tumor in the arm area on the right side of the brain,—there would result convulsive movements of the upper limb. Should this lesion destroy this center, paralysis of the limb would result. It must be remembered that while the centers concerned in the innervation of structures necessary for a movement are somewhat distinct,

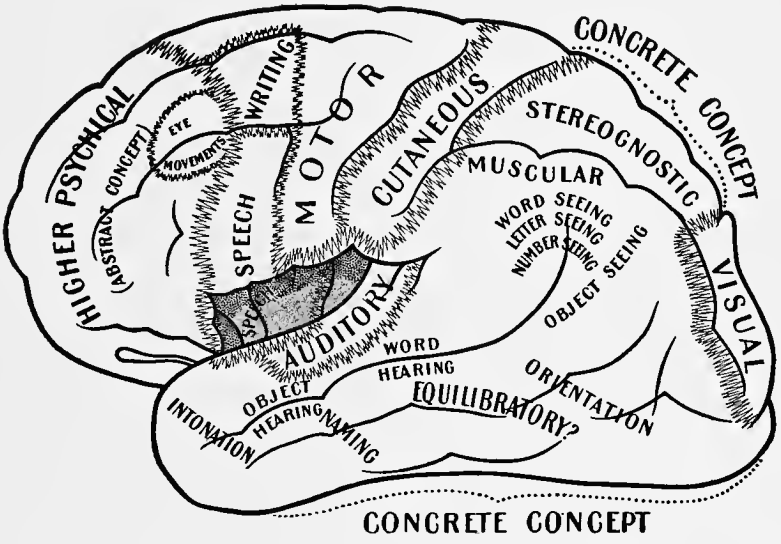


Fig. 381.—Lateral Surface of Brain, showing Localization of Function (Mills).

there cannot be and there is not a definite division, and that the nerve-cells related to different functions are in apposition and intermingle. Thus it is that irritation by an electric current or by a tumor, for instance, of the arm area, while it will cause a convulsion of an upper limb, might also cause movements of the lower limb (Fig. 381).

Sensory Centers.—Directly back of the motor centers behind the central fissure are located the sensory functions. In this area are included not only the postcentral but also the superior and inferior parietal convolutions. In the postcentral convolution itself are placed the centers for touch, pain, temperature, that is, those sensations which are primary and which develop first. Their localization is similar and in apposition to that of the motor functions, that is, those which are concerned in the innervation of the head are below and of the leg on top. In the parietal convolution have been placed the centers for the so-called acquired sensations,

that is, the senses of pressure, movement, position, localization, and stereognosis, or the ability to recognize objects placed in the hand. In the inferior parietal convolution the above sensations are localized for the upper limb, and in the superior parietal convolution for the lower limb. Should there be any irritative lesion, for instance, in the center for sensation in the upper limb on the right side of the brain, there will be numbness and pain localized in the left upper limb, these sensations being analogous to the convulsive movements which result from irritative phenomena of the motor centers. Destruction of any sensory center will also in a similar way produce loss of sensation or anesthesia.

Aphasia.—Within a few years the question of aphasia has aroused renewed interest, because of the contention of Pierre Marie that there is neither motor nor sensory aphasia, but that it is entirely due to a disturbance of general intelligence, and specifically of that concerned with language.

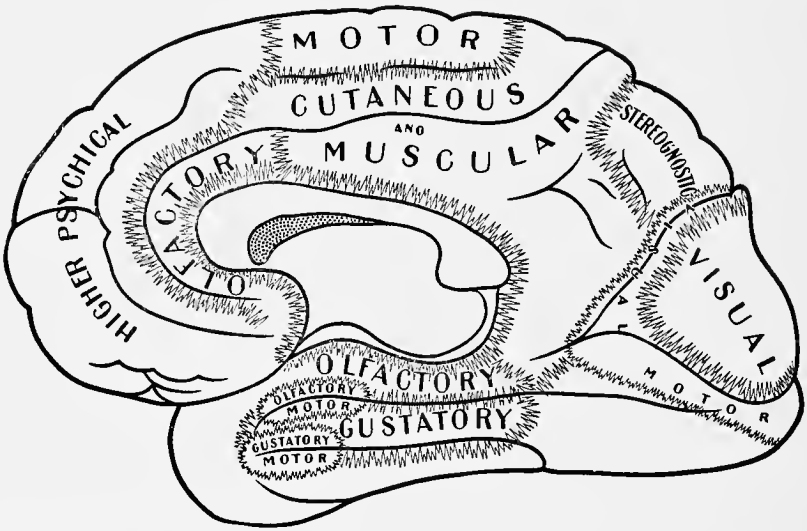


Fig. 382.—Mesial Surface of Brain, showing Localization of Function (Mills).

He rejects the old and classic view that speech has cortical localization, and contends that it is entirely an intellectual process, and that whenever there is a lesion of so-called Wernicke's zone,—in which he includes the superior and inferior parietal lobes and the posterior portion of the first and second temporal convolutions,—there will be defect of intelligence for the comprehension of spoken language, or what is ordinarily called sensory aphasia; and if, in addition, there is a lesion in the lenticular zone, in which he includes the lenticular nucleus, there will be anarthria or difficulty in articulation, or what is ordinarily called motor aphasia. The controversy is still going on, and the writer has deemed it advisable to present here the old views with some modifications.

Motor Aphasia.—In the posterior portion of the third or inferior frontal convolution is Broca's convolution, that is, the gyre which surrounds the end of the ascending limb of the fissure of Sylvius. This convolution is adjacent and in front of the head and face center, and is the part of the brain

which controls motor speech. Should there be a disturbance of this center, the patient would know what he wanted to say, would understand everything said to him, but would not be able to talk or repeat words; not because of any paralysis of the muscles which are concerned in speech, but because of destruction of the coördinating center which controls these muscles. This is motor aphasia.

As the majority of us are right-handed, the speech center is localized mostly in the left cortex. In left-handed persons, however, the speech center is on the right side of the brain. Another important point must be remembered: what act controls the right- or left-handedness of the individual? Given a person who is equally skilful with either hand, but who writes with the right, such a person will be right-handed. In other words, the function of writing, which is perhaps the highest of the developmental functions, controls the side on which the speech center is principally localized (Fig. 381).

Sensory Aphasia.—In the middle portion of the left first and second temporal convolutions in right-handed persons is the center for sensory speech. A lesion of this part will cause loss of memory for words. Such a person would be unable to understand what is said to him, but he would be able to talk because his motor apparatus is intact. His words, however, would be unintelligible and devoid of meaning.

Word and Letter Blindness.—Around the end of the first temporal convolution is what is called the angular gyrus. This convolution is directly back of the inferior parietal or sensory convolution, and between it and the visual or occipital centers. In right-handed persons this center controls the ability to recognize words, letters, and figures. In a destruction of this area the patient will be unable to write his name, or, in fact, to write anything, or to read words, letters, or figures or to write from dictation. He would, however, be able to recognize other objects, as pictures or music, or he would be able to sketch or draw or recall from memory any object in which words, letters, and figures are not concerned. This is called word, letter, or figure blindness.

Visual Centers.—The centers for vision are localized in the occipital convolutions, especially around the part surrounding the calcarine fissure or the cuneal lobe. The parts around the calcarine fissure are concerned with direct vision, while the other portions of the occipital cortex control peripheral vision. Should there be a lesion, for instance, of the right calcarine fissure, there would be loss of direct vision in the left half of each central visual field. In a lesion of the occipital lobe of the right side there will result blindness of the left half of each visual field, that is, left lateral homonymous hemianopsia, because the right occipital lobe supplies the right half of each retina, this controlling the left field of vision. In an irritating lesion of these parts, there will be flashes of light in the corresponding fields.

Psychic Centers.—The higher psychic functions have been placed in the frontal lobes, and especially in the left. In any lesion of these lobes there will result failure of memory, loss of intelligence and of reasoning, change of disposition and of character. It must be remembered, however, that there is no definite mental disturbance associated with a lesion of the frontal lobes, and failure of intelligence is not diagnostic of such lesion, for a lesion in any portion of the brain must cause some loss of intelligence, for every portion of the cortex is in constant communication with every other, and a destruction of one part must cause a disturbance of the integral whole.

Subcortical Centers.—Generally speaking the symptoms of a tumor or a lesion localized underneath the cortex will depend entirely upon what fibers are cut off. As any lesion will interfere with the fibers related to more than one function, the symptoms will never be clean-cut. For instance, a tumor localized underneath the precentral convolution will not only have motor symptoms but will also cause disturbance of sensation.

Cerebral Centers for Bilateral Acting Functions, such as Laughing, Crying, Eating, and Swallowing.—While it is acknowledged that in the cortex are localized the centers for every motion, sensation, and special act, it must be remembered that this is only in so far as simple acts, like lifting a finger or moving a limb, are concerned. Where, for instance, it is necessary to perform a complex act, such as talking, laughing, crying, eating, and swallowing, there must be some one place or center which coördinates the different functions which such an act must constitute. The speech centers, probably because of their importance, are largely localized in the cortex, but they also have representation in the subcortex. As to where the centers for laughing, crying, eating, and swallowing are, we are not certain, but we believe that they must be localized in some of the ganglia in the subcortex; among these the optic thalamus and the lenticular and caudate nucleus probably playing the most important rôle.

The Optic Thalamus.—The thalamus is anatomically divided into two halves by the lamina interna, its internal portion consisting of the anterior and median nucleus, and its external of the lateral nucleus, which is divided into anterior, middle, and posterior thirds, and dorsal and ventral halves. It also contains the center, median, and arcuate nuclei and the ventral nucleus, which are in the basal part of the thalamus. The pulvinar forms the posterior part. It is known that the continuation of the fibers of the fillet end in the anterior dorsal part of the lateral nucleus, while the continuation of the fibers of the superior cerebellar peduncle end in the posterior part of the lateral nucleus.

The functions of the thalamus are not accurately known. Lesions limited to it, however, cause the following group of symptoms: Vasomotor and trophic phenomena, principally on the side opposite the lesion, but sometimes on the same side, these consisting in burning and prickly sensations, flushes of heat, sometimes of cold, aching pains, and rarely of erythematous or acneiform eruptions. Secondly, because of the irritation of the sensory fibers, disturbance of sensation, especially of touch and muscle sense, and sometimes astereognosis and pains in the limbs of the contralateral side. Thirdly, disturbances in the emotions, the patient being able to voluntarily innervate the facial muscles, but unable to laugh or cry on the side opposite the lesion. Occasionally there is involuntary howling or crying. Fourthly, involuntary movements, consisting sometimes in deviation of the head, neck, and body to the opposite side, or of incoördinate movements, these being the result of irritation of the fibers which are the continuation of the superior cerebellar peduncle. If the internal capsule is pressed upon, there are disturbances of motion on the opposite side, but sometimes, because of pressure upon the knee of the internal capsule, disturbance in the central innervation of the face, muscles of mastication, and tongue.

Caudate and Lenticular Nucleus.—Nothing definite is known of the functions of these structures. Bilateral lesions of the lenticular nucleus sometimes cause involuntary laughing and crying, and because of the recent work of Marie the lenticula has been considered a part of the zone of speech.

Localization of Fibers in Internal Capsule.—The internal capsule is the name given to the pathway of fibers (Fig. 383) which come from the cortex. It contains an anterior limb, a knee, and a posterior limb. The anterior limb transmits the fibers coming from the frontal to the opposing cerebellar lobe, the so-called fronto-cerebellar fibers. The knee of the internal capsule transmits those fibers which come from the lowest portion of the precentral convolution, *i. e.*, the head and face centers, these being the fibers which go to the nuclei of the cranial nerves situated in the crus, pons, and medulla, *i. e.*, from the third to the twelfth nerves inclusive.

The posterior limb of the internal capsule transmits in its anterior portion the motor fibers, in its middle the sensory, and in its posterior portion the fibers which come from the occipital or visual lobes. Should there be a lesion of the posterior limb of the internal capsule, as, for instance, a hemorrhage, there would result hemiplegia, hemianesthesia, and hemianopsia on the other side. This is the only place in the brain where one lesion will always give these three symptoms.

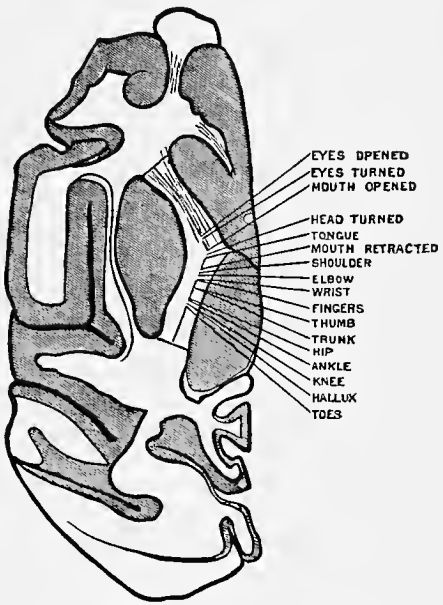


Fig. 383.—Arrangement of Motor Paths in the Internal Capsule (after Ferrier).

The Crus or Cerebral Peduncles.—The cerebral peduncles are practically the continuations of the posterior limbs of the internal capsule and transmit the fibers for motion and sensation, thus connecting the brain proper with the brain stem.

The nucleus of every cranial nerve, from the third to the twelfth inclusive, receives its innervation from the opposite cortical center. The first and second cranial nerves do not enter in this, as they are really parts of the brain. The nucleus of the third or the oculomotor nerve is situated in the posterior portion of the crus and its fibers have their exit at the foot of the cerebral peduncles.

A *unilateral lesion*, therefore, of the *cerebral peduncle* would always give oculomotor palsy on the same side, and paralysis of the arm and leg only on the other side of the body.

The Pons.—In the pons are located the nuclei of the fifth, sixth, seventh, and partially of the eighth cranial nerves, the exits of these nerves corresponding in order. In a *unilateral lesion of the upper part of the pons* there will be paralysis of the fifth nerve on the same side with hemiplegia of the opposite side. In a *lesion limited to the lower portion of the pons* there will result facial palsy on the same side and paralysis of the arm and leg only on the other side.

In discussing the symptoms of lesions in the crus and pons, it has been assumed that these are confined to the anterior portions of these structures.

Should the lesion, however, be more extensive, there would necessarily have to be involvement of the sensory fibers, which are localized directly back of the motor, and there would result, in addition, sensory symptoms on the other side.

Paralysis of Associated Ocular Movement.—Should, however, the lesions involve the median portions of the crus and pons, there would be paralysis of associated ocular movement. This is rather difficult to understand unless it is remembered that it is impossible to move one eye without the other, and therefore every movement of the eyeballs must be an associated movement. When we look to the right, we use not only the external rectus muscle on the right side, but also the left internal rectus, *i. e.*, we are receiving innervation from the nuclei of the sixth and third cranial nerves. To make this possible there must be a connection between these nuclei, and this is effected by the posterior longitudinal bundle, which is located in the posterior and median portions of the crus and pons.

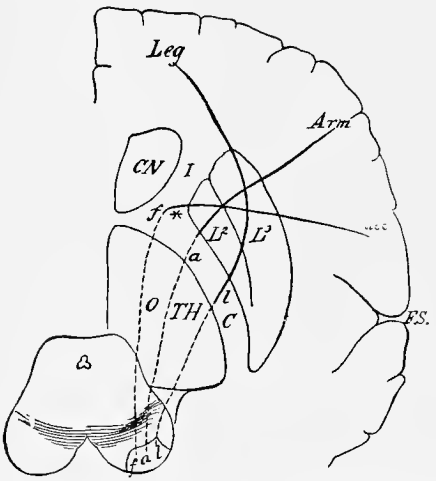


FIG. 384.—DIAGRAM TO SHOW THE RELATIVE POSITION OF THE SEVERAL MOTOR TRACTS IN THEIR COURSE FROM THE CORTEX TO THE CRUS (Gowers).

The section through the convolutions is vertical; that through the internal capsule, I, C, horizontal; that through the crus is again vertical; CN, caudate nucleus; O, TH, optic thalamus; L^2 and L^3, the middle and outer parts of the lenticular nucleus; *f*, *a*, *l*, face, arm, and leg fibers. The words in italics indicate the corresponding cortical centers.

In looking downward we use not only the muscles which are innervated by the third, but also those which are innervated by the fourth cranial nerves. In looking upward we use only the muscles which receive innervation from both oculomotor nuclei. We see then that there must be a similar connection between the oculomotor nuclei and between them and the nuclei of the fourth nerves.

Should there be a lesion, for instance, in the lower part of the right side of the pons, cutting off the posterior longitudinal bundle, there will be inability to look to the right, and a similar lesion on the left side will cause inability to look to the left. A lesion cutting off both bundles will cause inability to look to the right or left, but the ability to look upward and downward will be retained.

In a lesion of the upper portion of the pons which cuts off the connection between the third and fourth nuclei there will be paralysis of associated movement downward. A lesion still higher up will cause failure of upward movement.

Lesions of the Anterior and Posterior Corpora Quadrigemina.—The anterior corpora quadrigemina, in association with the pulvinar of the optic thalamus and the external geniculate body, is one of the so-called primary optic centers, and a lesion of it should cause disturbance of half vision upon the opposite side. The corpora quadrigemini are so close together that nearly always both are involved. As a matter of fact we know little of their functions, although it is presumed that the anterior are concerned with vision and the posterior transmit the central fibers of hearing.

Nearly always lesions of this part are extensions of tumors, either from the aqueduct of Sylvius or more commonly from the pons, peduncle, or cerebellum. Because of the proximity of the third and fourth nuclei they will be involved early and bilaterally, and as the tumor grows the sensory and then the motor fibers become affected. Nearly always the fifth nerve is either unilaterally or bilaterally diseased.

Partial or Total Lesions of the Medulla Oblongata.—Isolated lesions are unusual. As a rule, tumors occupying this part are extensions from the pons or cerebellum. Hemorrhages are rare, and nearly always cause sudden death because of involvement of the ninth and tenth nerves. Thrombosis or embolism, especially of the inferior cerebellar artery, is not at all unusual, and is especially prone to occur on the left side. The symptoms in such a case will be unilateral disease of the eighth, ninth, tenth,

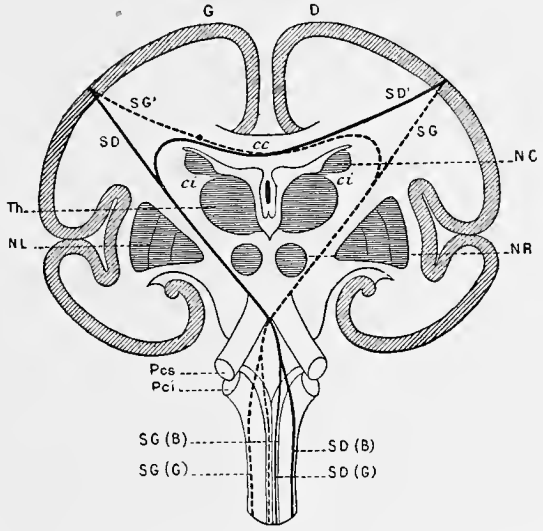


FIG. 385.—SCHEMATIC VERTICAL TRANSVERSE SECTION OF THE HEMISPHERES PASSING THROUGH THE INTERNAL CAPSULE AND REPRESENTING THE SENSORY PATHWAYS (Church and Peterson, after Brissaud).

G, Left hemisphere; D, right hemisphere; *cc*, corpus callosum; *ci*, internal capsule; SG, sensory pathway from left side of cord; SD, sensory pathway from right side of cord. Both sides are brought into intimate relation through the corpus callosum, and the sensory representation is uniformly bilateral.

eleventh, and twelfth nerves, with involvement of the motor and sensory fibers innervating the opposing arm and leg.

The Cerebellum.—Anatomy.—The cerebellum consists of a middle portion or the vermis and two lateral lobes. It is connected with the rest of the brain by three processes called the cerebellar peduncles, the superior or first connecting it with the brain proper, the middle with the pons, and the inferior with the medulla and spinal cord. In the middle are situated the dentate nuclei, the nucleus fastigii, and the nuclei emboliformis and globosus. In addition certain nuclei situated in the medulla oblongata are in direct communication with the cerebellum, and should be regarded really as part of it. These include Deiters' nucleus, the nucleus vestibularis, and the nucleus magnocellularis substantia reticularis—these being called together the paracerebellar nuclei.

The **functions** of the cerebellum are not definitely known. Experimental and clinical evidence seems to show that lesions in any portion will produce symptoms of incoördination of a definite character. It has recently been demonstrated by Sir Victor Horsley that the cortex of the cerebellum is inexcitable, but that irritation of the intrinsic cerebellar nuclei will produce

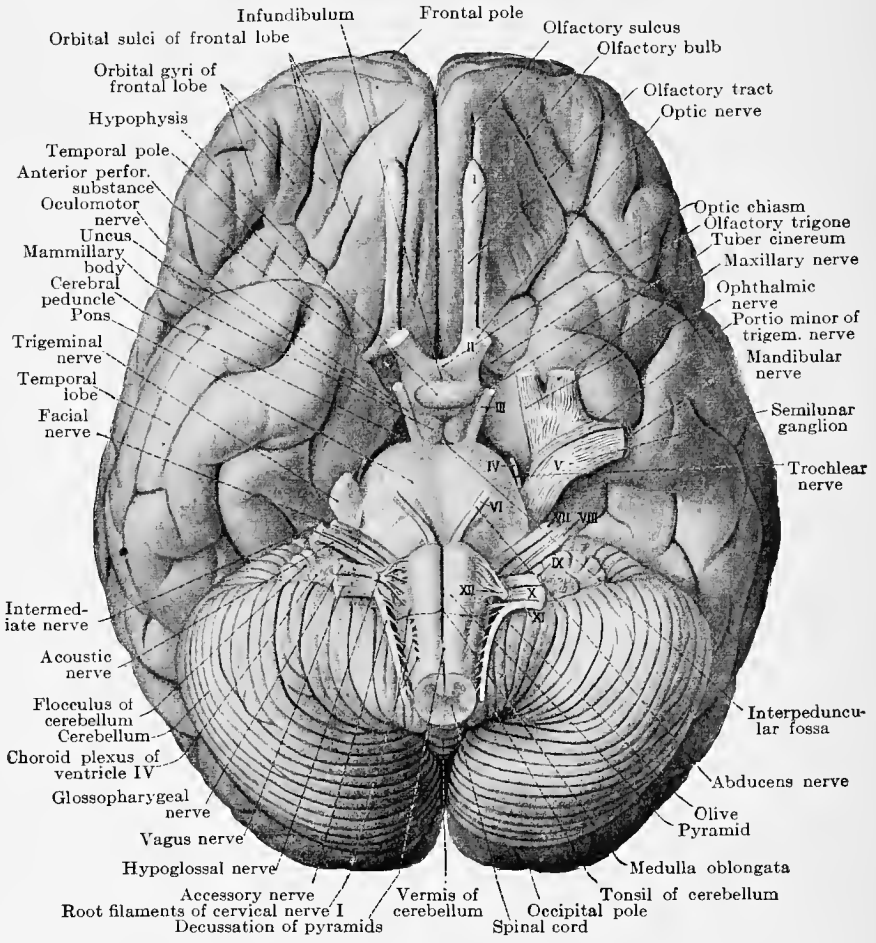


FIG. 386.—THE BASE OF THE ENTIRE BRAIN, WITH THE POINTS OF EMERGENCE OF THE TWELVE CEREBRAL NERVES UPON THE LEFT (Sobotta and McMurrich).

The entire semilunar ganglion has been retained. The hypophysis has been displaced slightly backward and compressed to expose the infundibulum. The Roman numerals indicate the number of the cerebral nerves.

conjugate deviation of the eyes and head to the same side, besides flexion of the homolateral elbow, and that deeper excitation of the paracerebellar region will produce extension of the contralateral elbow, hyperextension of the neck and trunk, with powerful extension of the lower limbs.

It is probable that the cerebellum is concerned with the coördination of every voluntary movement, whether this be of the limbs, eyes, or of those

muscles which are concerned in articulation, eating, or swallowing. It is characteristic of cerebellar incoördination that it is only apparent in voluntary movements, and that it does not increase when the object is attained or when the eyes are shut, and that it is not dependent upon any disturbance of peripheral sensation.

It has also been recently held that lesions of the cerebellum will produce weakness or paresis of the muscles of the trunk and limbs, but this is not a true weakness in the sense that it is not dependent upon the motor fibers. Besides, there may be present in the muscles a lack of tone, so that it would be possible to move the limbs like a flail. If the lesion is in the middle lobe or the vermis, the symptoms of incoördination are most marked, and will involve both parts of the body, while unilateral lesions will of course produce unilateral ataxia and atonia.

Whatever symptoms are produced by lesions of the cerebellum or by those lesions which invade the cerebellum by pressure are dependent upon this disturbance of coördination. This is apparent in every movement, whether it be in the gait, station, in the movement of a limb or limbs, or of the eyes or of those muscles which are concerned in eating, talking, and swallowing. Other localizing symptoms will be discussed under the head of cerebellar tumors.

THE CRANIAL NERVES AND THEIR DISEASES.

There are twelve pairs of cranial nerves. These are known either by special names or numerically. The first and second, or the olfactory and optic, should really be considered as parts of the brain proper and not as distinct cranial nerves (Fig. 386).

THE OLFACTORY NERVE.

The center for the olfactory nerve is not definitely known, but its function is concerned with smell, the loss of which (anosmia) is very frequent in fracture of the base of the skull. It is also lost in inflammatory conditions of the nose and turbinate bones and in any lesion which destroys the olfactory nerves or bulbs. Irritative disturbances of smell (parosmia) are present in hysteria and other functional neuroses or may sometimes form the aura of an epileptic convulsion.

THE OPTIC NERVE.

The optic or second nerve is the nerve of vision. From the orbits, in their course backward, the optic nerves enter into and form the optic chiasm, and then the optic tract, and from here the visual fibers go to the primary optic centers, this constituting the anterior corpora quadrigemina, the external geniculate body, and the pulvinar or the posterior portion of the optic thalamus. From here the fibers pass through the extreme posterior portion of the posterior limb of the internal capsule to the occipital lobe.

It must be remembered that the visual fibers coming, for instance, from the right occipital lobe, innervate the right half of each retina or the temporal part on the same side, and the nasal on the other, and thereby supply vision to the left half of each visual field. A lesion interrupting the fibers coming from the right visual centers or the occipital lobe, for instance, either in the extreme posterior portion of the posterior limb of the internal capsule or in the so-called primary optic centers or in the optic tract, must give loss

of half vision in both visual fields on the other side or left *lateral homonymous hemianopsia.*

The decussation of the optic fibers takes place in the chiasm and that of the nasal fibers in the center. A lesion, therefore, in the center of the optic chiasm will cause loss of innervation to the nasal part of each retina, or *bitemporal hemianopsia* (Fig. 387).

A lesion interrupting the fibers on either side of the optic chiasm, as, for instance, the right, will cause loss of innervation to the right temporal retina, and therefore loss of the nasal field of vision of the right eye. A bilateral lesion must give bilateral loss of vision of the nasal fields, or *binasal hemianopsia.*

A lesion destroying the whole optic chiasm, as, for instance, a tumor

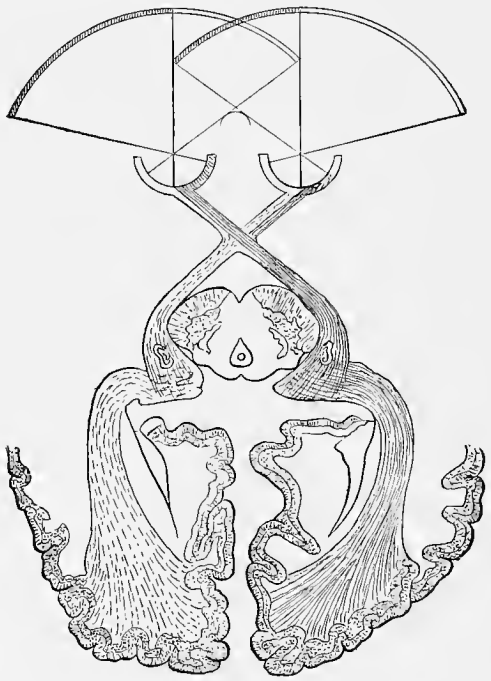


Fig. 387.—Diagram of Visual Paths (Starr).

of the hypophysis, will cause loss of vision in both eyes. Destruction of either optic nerve will, of course, give blindness in the corresponding eye.

Choked Disc or Optic Neuritis.—Whenever there occurs increase in intracranial pressure, whether because of a brain tumor, trauma, or internal hydrocephalus, pressure will be exerted upon the optic chiasm and optic nerves. This is because pressure in any portion of the brain will result in a heightened tension in the lateral and third ventricles, the latter pressing directly upon the optic chiasm and optic nerves.

In every choked disc there must be some inflammation of the optic nerve or optic neuritis, but in optic neuritis choked disc does not necessarily occur, for the latter is distinctly a pressure symptom. When the optic nerve is pressed upon, there will be first a stasis of the vessels, this resulting

in a swelling of the veins, it being so severe at times as to produce hemorrhages. There will also be retardation of the arterial flow, this causing a diminution in the size of the arteries. Because of this stasis there will result an edema, it causing a swelling of the optic nerve-fibers or of the optic nerve-head. If the pressure is continued, the nerve-fibers will become diseased, this resulting in impairment of vision. This is choked disc. If the pressure is continued for a long time there will necessarily result atrophy of the optic nerve-fibers.

Optic Atrophy.—Optic atrophy may be primary or secondary. When primary, it may be the result of an atrophic condition of the optic nerve, but it generally indicates a spinal cord disease, such as locomotor ataxia, Friedreich's ataxia, or any spinal cord disease in which the posterior columns are involved. Secondary optic atrophy is nearly always the result of an old choked disc or optic neuritis.

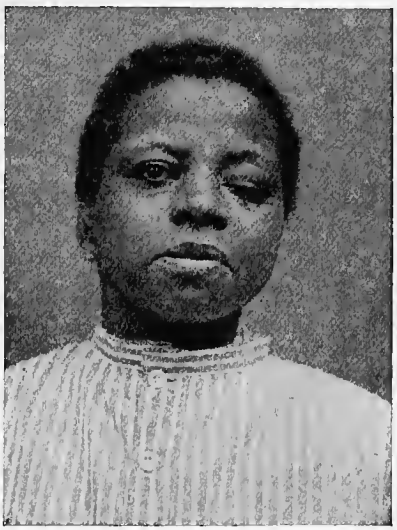


FIG. 388.—SYPHILITIC OCULOMOTOR PALSY. DROOPING OF THE LEFT UPPER LID.

Pupils and their Reactions.—The ciliary muscles react to two forms of stimulus: (1) light, (2) movement of the eyeballs. No matter what the stimulation, the contraction or dilatation of the pupil is performed by the same muscles, but the innervation differs. The ordinary light stimulation is transmitted by means of the optic nerves to the oculomotor nucleus, and from here the impulse to the ciliary muscle is carried by the oculomotor nerve. This is the light reflex arc, and if there is any disturbance anywhere in the arc, there will be impairment or loss of the reaction of the pupil to light. The fibers which are concerned with the reaction of the pupil to movement, as, for instance, in convergence and divergence, and in upward, downward, and outward movements, have probably a similar arc, with the addition that they are in connection with the nuclei of the muscles necessary to perform a certain ocular movement.

THE OCULOMOTOR NERVE.

The oculomotor or third nerve supplies all the muscles of the eyeball with the exception of the superior oblique and the external rectus. A total paralysis will cause drooping or ptosis of the upper lid, outward and downward deviation of the eye, with inability to move it in any but the outward and downward direction, and an enlarged pupil which does not react to any form of stimulation. Unilateral paralysis is nearly always due to basal syphilis (Fig. 388). It must also be remembered that just at the exit point of the third nerve at the foot of the cerebral peduncle, the different fibers which make up the nerve are still separated, and it is possible for a basal syphilitic lesion to involve only a few of the fibers. It is because of this that at times only a partial oculomotor paralysis will result from syphilis,

such, for instance, as internal rectus palsy, drooping of the upper lid, or disturbance of the iridic reflexes. Fractures of the base of the skull, basal tumors, and aneurisms may rarely cause oculomotor palsy.

The nuclei of the oculomotor nerves in the posterior portion of the crus are very close together, and a lesion or a hemorrhage in this area will nearly always cause bilateral oculomotor palsy. The disease causing such hemorrhage is known as *acute superior polioencephalitis* (of Wernicke). It comes on acutely and is usually accompanied by fever and its attending symptoms, and the pathology consists in multiple hemorrhages and areas of inflammation in the gray matter surrounding the aqueduct of Sylvius. The nuclei of the fourth and sixth cranial nerves may also be involved, and there will result what is known as complete bilateral *ophthalmoplegia*, or paralysis of all the muscles of the eyeball, causing inability to move the eyes in any direction. Areas of inflammation or hemorrhage sometimes involve the cranial nuclei in the lower portion of the pons and medulla, and we may have, in addition to the ophthalmoplegia, the symptoms of such involvement. Rarely the disease in the lower part of the pons or medulla is independent, when it is known as *acute inferior polioencephalitis* (of Wernicke). In such cases we have the symptoms of acute bulbar paralysis, with difficulty in talking, eating, and swallowing, and paralysis of the muscles innervated by the seventh, ninth, tenth, eleventh, and twelfth nerves.

Ophthalmoplegia, or paralysis of all the muscles of the eyeball, may be internal, external, or complete. By *internal ophthalmoplegia* is meant paralysis of the ciliary muscles, this resulting in rigid pupils. By *external ophthalmoplegia* is meant paralysis of the external muscles of the eye. In complete ophthalmoplegia there is inability to move the eyeballs in any direction, drooping of the upper lid, and rigid pupils. Unilateral ophthalmoplegia may result from a lesion in back of the eyeball, and commonly occurs in *cavernous sinus thrombosis*, when there will be, in addition to the ophthalmoplegia, a protrusion of the eyeball with stasis of the veins and edema of the lids. In nearly all cases of cavernous sinus thrombosis there will ultimately be bilateral involvement.

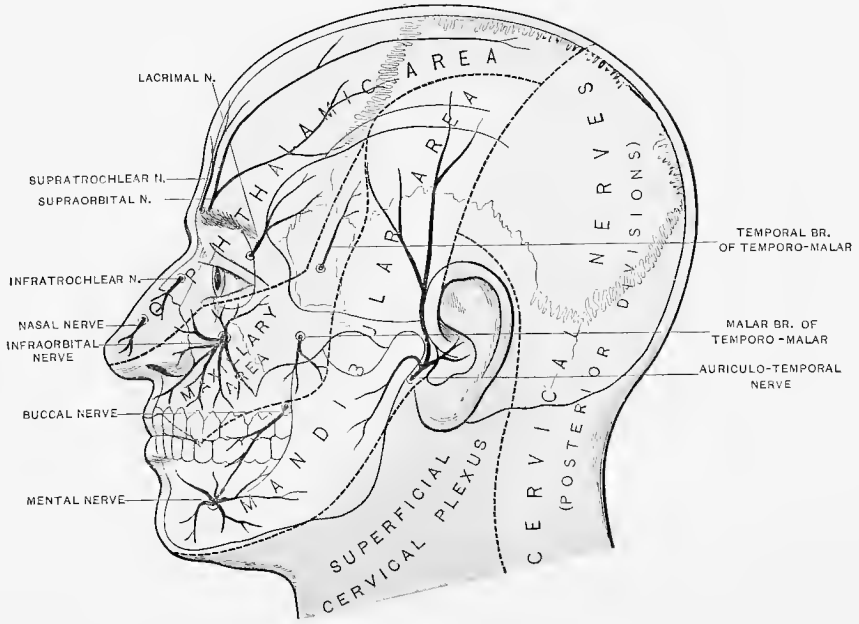
Bilateral external ophthalmoplegia may be the terminal stage of an old polioencephalitis, may be part of a chronic degeneration of the motor cranial nuclei, as in progressive bulbar palsy, or may be an independent disease coming on in childhood or in early adult life with apparently no recognizable cause, when it is called chronic ophthalmoplegia.

THE TROCHLEAR NERVE.

Isolated paralysis of the trochlear or fourth nerve is an extremely rare condition and hardly ever occurs. It is generally found in association with palsies of the other ocular muscles. This nerve supplies the superior oblique muscle, which pulls the eye downward and outward. Basal syphilis is nearly always the cause of paralysis.

THE ABDUCENS NERVE.

The abducens or sixth nerve supplies the external rectus muscle, which pulls the eye outward (Fig. 386). Temporary or permanent paralysis is a very frequent and early symptom in basal syphilis and brain tumors. This nerve is probably more frequently diseased than any of the other ocular nerves. This is partially due to the fact that it has the longest course



Sensory Areas of the Head, showing the General Distribution of the Three Divisions of the Fifth Nerve (Gerrish).

PLATE XVII

of any of the nerves in the base of the brain, and it is therefore more vulnerable to pressure, trauma, or a lesion in any portion of the skull. Its involvement with other nerves has already been discussed.

THE TRIGEMINUS NERVE.

The trigeminus, or the fifth nerve, has both a sensory and a motor function, it being mostly sensory. The motor part supplies the muscles of mastication. The sensory division supplies sensation for the face, eye, nose, palate, and pharynx, and also the anterior two-thirds of the tongue.

In **paralysis of the motor fifth** there will be inability to chew on the side of the paralysis, the contraction of the masseter and temporal muscles will be weak, and the jaw will deviate toward the affected side. Isolated paralysis of the motor fifth nerve never occurs, and when present may be one of the symptoms of chronic nuclear degeneration of the bulbar nerves. It may, of course, occur in pontile tumors, when there are, in addition, such other symptoms as paralysis of associated ocular movement and hemiplegia. When the result of a basal lesion, as syphilis, the sensory part of the nerve is involved in addition, this causing disturbance of sensation in its distribution. It is frequently temporarily paralyzed in early hemiplegia.

Fig. 389.—Arteriosclerotic Palsy of Ocular Muscles, showing Paralysis of Left External Rectus.

In an irritating lesion of the sensory part of the fifth nerve there will be pain either in its whole distribution or the subdivisions of the nerve, that is, the supraorbital, infraorbital, or mental.

Tic douloureaux, or painful spasm of the fifth nerve, is due to many causes, but in the majority of instances no ascertainable factor can be found. Repeated examinations of the Gasserian ganglia have demonstrated occasionally diseases of the nerve-cells, but this is not constant, and the real cause is not known. The disease may involve at first or be always limited to one branch of the fifth nerve, usually to the supraorbital, when it is termed *supraorbital neuralgia.* As a rule, it begins with an occasional numbness in one of the divisions, this becoming more frequent and severe, the onset of the disease lasting sometimes over a number of years, to be followed by pain which involves two and lastly all of the branches. If the disease is limited to the supraorbital nerve, pain will be marked over the forehead and brow, and there will be pain in the eye, sometimes a sensation as of a foreign body. Occasionally the pains will be so sharp as to cause closure of the eye with flow of tears. Inflammation limited to the middle or infraorbital nerve (*infraorbital neuralgia*) will cause numbness or pain in

the upper jaw and upper teeth, and sometimes in the tongue. If the disease is limited to the inferior or mental branch, the pain will be in the lower jaw, teeth, and tongue, it being aggravated by eating or talking. There will, besides, be pain on pressure over the nerves at their exits. If the disease involves all parts of the fifth nerve, the pain will come on spasmodically and cause the most excruciating pains over the whole side of the face, and associated with this contractions or spasms of the muscles. Accompanying this there will be flow of tears and pain on pressure over the exits of the nerve. At such times any irritation, no matter how slight, and talking or eating will bring on a fresh attack, and there may also be hyperesthesia in the distribution of the trigeminus.

The prognosis in a well-marked case is poor, inasmuch as operative procedure offers the only relief. In such case, whether the Gasserian ganglion

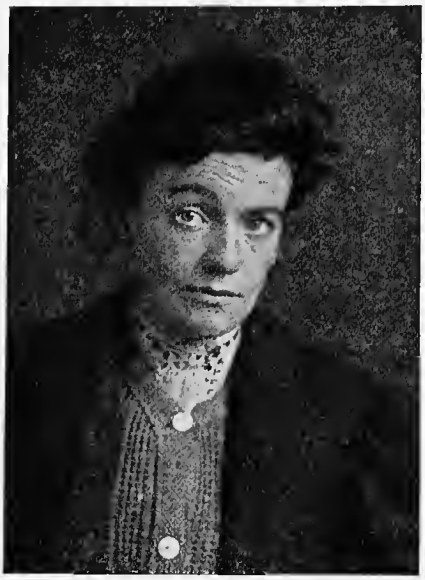


FIG. 390.—LEFT FACIAL PALSY SHOWING INABILITY TO WRINKLE THE BROW.

FIG. 391.—DROOPING OF THE LEFT ANGLE OF THE MOUTH WITH INABILITY TO SHUT THE EYE.

be excised or the sensory root cut, relief of pain will be obtained, and there will be anesthesia in the distribution of the fifth nerve. It is important to remember that sensation after a short time will return, there being a return of deep sensation, and even superficial skin sensation to some extent.

THE FACIAL NERVE.

The facial or seventh nerve supplies the muscles of the face. Its nucleus is in the lower and posterior portions of the pons, and the nerve in its course outward surrounds the nucleus of the sixth nerve. Its exit is just between the pons and medulla. Because of this anatomic relation, any gross lesion involving the seventh nucleus will nearly always involve the sixth, and vice versa. The usual form of facial palsy is that known as peripheral or Bell's palsy. Lesions causing this may be either in the pons, at the exit of the

nerve at the base of the brain, in the Fallopian canal, or in its extracranial course.

Central facial palsy is that form of facial paralysis in which the lower part of the face only is paralyzed, and is the result of a lesion in any portion of the central facial fibers between the facial centers in the cortex and its nucleus in the pons, as, for instance, in a capsular hemiplegia. The reason for the escape of the upper portion of the face in such paralysis is because wrinkling the brow, in common with other bilateral functions, such as chewing, eating, and swallowing, has bilateral cortical innervation, and to cause paralysis of such functions there must be bilateral cerebral lesions.

In a large majority of cases ordinary **peripheral facial palsy** is the result of a neuritis which may be of rheumatic origin or may follow a "cold." Rarely it is due to basal syphilis, tumors, fractures, etc. When resulting from a lesion in the pons, facial paralysis is generally accompanied by other symptoms, such as palsy of the sixth nerve, paralysis of associated ocular movement, or hemiplegia upon the other side. Abscess of the middle ear is a common cause as well as mastoid operations. When the lesion is in the Fallopian canal, we have, in addition to the usual symptoms, temporary disturbance of taste in the anterior two-thirds of the tongue because of involvement of the chorda tympani which runs along with the seventh nerve in the Fallopian canal.

The symptoms of peripheral paralysis of the facial nerve depend upon the degree of its involvement. When the paralysis is total, there is inability to wrinkle the brow, to shut the eye, to elevate the corner of the mouth, to whistle, or to pronounce labials properly (Fig. 390). Besides, there will be drooping of the lower lid and of the corner of the mouth, and the wrinkles on that side of the face will be smoothed out. Because of the drooping of the lower lid there will be widening of the palpebral fissure and excessive flow of tears because of the lack of proper conduction into the nasal cavity. Electrical reactions of degeneration will be found, their degree depending upon the extent of the neuritis. Sensory disturbances may be present at the onset, when the patient may complain of pain in the face, and there may also rarely be herpetic eruptions in the ear because of involvement of the geniculate ganglion.

Most cases of peripheral facial paralysis recover provided the cause is an ordinary neuritis such as results from "colds" or rheumatism, and that prompt treatment is instituted. In those cases in which the nerve is cut, unless an anastomosis is performed recovery cannot be expected. Sometimes, years after the occurrence of such paralysis, there may occur secondary contractures.

THE AUDITORY NERVE.

The auditory or eighth nerve has two divisions—the cochlear, which is the nerve of hearing, and the vestibular, which is concerned with equilibration. The nucleus of the eighth nerve is in the posterior and lateral portion of the inferior part of the pons and the upper part of the medulla oblongata, and may be affected by vascular lesions or tumors of this area. As a rule, deafness is due to middle-ear involvement. Rarely the eighth nerve is involved at its exit in the cerebello-pontile angle by basal syphilis or tumors. Disease of the vestibular portion of the eighth nerve is discussed under the heading of vertigo.

THE GLOSSOPHARYNGEAL, THE PNEUMOGASTRIC, AND THE SPINAL ACCESSORY NERVES.

The glossopharyngeal, the pneumogastric, and the spinal accessory, or the ninth, tenth, and eleventh nerves, have their nuclei in the posterior and lateral portions of the medulla, and their functions are intimately concerned with one another, and a lesion in the nucleus of one will nearly always involve the other. The consequences of such lesions will not be discussed, because hemorrhages in this region will nearly always cause paralysis of respiration and death. Chronic degenerative diseases, however, will often involve these nuclei, as in progressive bulbar palsy. It is only necessary to add that the *ninth nerve* supplies taste sensation for the posterior third of the tongue and the muscles of the upper part of the pharynx. The *tenth* supplies the muscles of the pharynx, larynx, heart, lungs, and other viscera, and the *eleventh nerve*, besides being an accessory nerve to the tenth, supplies the sternomastoid and the trapezius muscles, paralysis of this causing inability to turn the head to the opposite side and drooping of the shoulder.

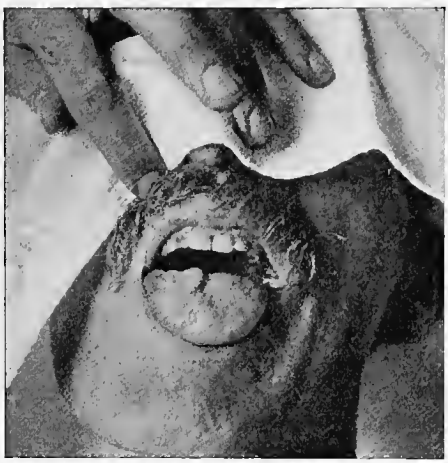


FIG. 392.—ILLUSTRATING ATROPHY OF RIGHT SIDE OF TONGUE.

THE HYPOGLOSSUS NERVE.

The hypoglossus, or twelfth nerve, supplies the tongue. Its nucleus is in the posterior median portion of the medulla. It is one of the first nuclei to be involved in such degenerative unclear diseases as bulbar palsy, when there will be fibrillary tremors, atrophy, and weakness of movement. Isolated paralysis of the twelfth nerve is very rare, but sometimes occurs as a result of basal syphilis, fractures of the base, or tumors in the posterior cranial fossa. Sometimes in hemiplegia temporary unilateral paralysis of the tongue may occur, and when the tongue is protruded it will be projected to the side of the paralysis (Fig. 392).

DISEASES OF THE BRAIN.

General Symptoms.—Certain general symptoms may be present in any disease of the brain, the degree and number depending upon the nature, extent, and location of the lesion. They are headache, nausea, vomiting, vertigo or dizziness, disturbances in motility, such as tremors, convulsions, general or focal in type, partial or total paralysis, disorders of sensation and disturbances of vision or of the other special senses, and, lastly, alterations in mentality.

Headache, as a result of any cerebral lesion, is nearly always due either to an irritation of the dura or to an increase of intracranial pressure, which causes tension of the dura. The meninges are innervated by the sensory

portion of the fifth nerve, and disease, therefore, of this part must cause pain. At times the headache is localized to the place of direct irritation, but, as a rule, it is general. The pain is usually severe and constant and is difficult to relieve by medication, and vomiting does not lessen its intensity. The location and character of the headaches as they occur in tumors and other brain diseases will be discussed under their heading.

The majority of headaches are, however, due to causes which are not a direct result of irritation of the dura, but are possibly caused by vascular changes. Under this are included the reflex headaches resulting from disturbances of the ocular, nasal, sinus, aural, buccal, pharyngeal, laryngeal, and visceral functions. In fact, there is no organ in the body disturbance of which has not been thought to be a possible cause for headache. A still greater variety of head pains are due to general disturbances, as syphilis, acute rheumatism, or diabetes, but the largest number of all occur in the so-called functional neuroses. It would be interesting to differentiate the various locations of headaches resulting from reflex and other causes, but this is impossible, for, as a matter of fact, the pains may occur in any portion of the head. The general characteristic of all neurasthenic and so-called functional headaches is that they are nearly always in the back part of the head and neck, or in the top of the head, and are described as a pressure sensation and sometimes as a band around the head. Headaches resulting from disturbance of the sensory portions of the fifth nerve or the Gasserian ganglion and from migraine will be discussed separately.

The **nausea** and **vomiting** which are present in diseases of the brain are generally indicative of intracranial pressure, for they are not present unless such be the case. They are probably due to an irritation of the ninth and tenth nerves. The nausea may appear in the morning or at any time, and may be accompanied by vomiting, but the latter, as a rule, does not relieve the nausea or the accompanying headache. The vomiting is generally projectile in character and comes on without warning. These symptoms are generally indicative of brain tumor. A greater amount of nausea and vomiting is present in cerebellar lesions because pressure is more direct upon the ninth and tenth nerves.

Vertigo or **dizziness** is also considered a pressure symptom in diseases of the brain. The dizziness may be objective or subjective, *i. e.*, the patient may either see objects move before him or may have a sensation that he moves himself. In cerebral tumors this symptom is not very common, but in cerebellar lesions vertigo appears very early and is very marked and persistent. It is probably due to pressure which is exerted on the vestibular division of the eighth nerve.

Ménière's Disease.—This is the name given to a symptom-complex the leading characteristic of which is vertigo accompanied by loud noises in the ear. The disease generally occurs in the latter end of life, and, as a rule, begins with noises in the ear accompanied by some dizziness. These first come on occasionally, and gradually the tinnitus increases, the noises sometimes resembling the shrieking of a whistle, and are accompanied by excessive vertigo, which, as a rule, terminates in nausea and vomiting. During the attacks the patient feels weak and is pale. At first the disease is unilateral, but ultimately there is bilateral involvement. Disturbance of hearing finally comes on, the deafness then becoming progressive. The tinnitus, vertigo, and deafness now become constant, sometimes preventing the patient from assuming an erect posture. Rarely when the deafness becomes complete the vertigo and tinnitus cease. It is supposed that this

symptom-complex is due to a disease of the terminal filaments of the vestibular nerve in the labyrinth, and there may also be disease of the semicircular canals.

Disturbances in motility do not occur unless there is an involvement either of the cortical motor centers or of the fibers coming from them. Because of the readiness with which motor symptoms are detected they are more quickly appreciated than any of the other symptoms. They may consist of tremors, forced movements, convulsions, either general or focal, and partial or total paralysis.

Tremors.—A tremor may be indicative of a general disease, such as *paralysis agitans*, when it is coarse, vibratory, and lessens on effort. In disseminated sclerosis the movement is made worse on effort and is called an *intention tremor*. It also differs from that present in paralysis agitans in the fact that it is not vibratory and resembles more an irregular movement.

A general tremor of the limbs may sometimes be present in old age, hysteria, and other functional neuroses.

Fig. 393.—Athetoid Movements in the Face in a Case of Old Infantile Diplegia.

Fibrillary tremors are always indicative of a chronic degeneration or atrophy of the cells in the anterior horns of the spinal cord or of the motor cranial nuclei in the medulla. The movements are fine and consist in a wave-like twitching of one or a number of muscle-fibers.

Such other movements as are present in chorea, myokymia, and its subdivisions will be discussed separately.

Forced or Associated Movements.—By this is meant the forced movement of a healthy limb when the patient attempts to move the paralyzed limb, as in hemiplegia. It is probable that this is due to the fact that one side of the cortex innervates both sides of the body.

Athetosis.—It may be present in the face or in all of the limbs, or in any one of the limbs, and is always indicative of a lesion in the motor columns or cortex, either at infancy or birth. The athetoid movement is slow, twisting, and constant (Fig. 393).

Convulsions.—These are spasmodic movements of a part of a limb, of a whole limb, of one-half of the body or of the whole body, and may be accompanied by loss or impairmemt of consciousness. If the convulsive movement is limited to a part of a limb or one-half of the body, and if it always begins in the same muscles, it is called a *focal* or *Jacksonian convulsion*, and is nearly always indicative of an irritative lesion in the motor cortex. In Jacksonian convulsions or epilepsy the spasms come on quickly and may last from a few seconds to several minutes, and are generally clonic in type, and, as a rule, are not accompanied by unconsciousness.

It is of the utmost importance to see where a Jacksonian convulsion begins, what muscles or movements it involves, and their succession. Supposing, for instance, twitchings begin in the fingers of the right hand, and from here the movements extend into the muscles of the forearm,

arm, and shoulder, and then into the muscles of the face. This would be indicative of a lesion in the left motor cortex, probably extending from the hand to the face center, from the middle to the lower portion of the precentral convolution. Should, however, the convulsion involve the leg instead of the face, it would indicate that the lesion extends from the middle of the precentral convolution upward or to the leg center. These facts are of the utmost importance when surgical procedures are considered, for upon their correct observation will rest the probable seat of operation. General convulsions are nearly always indicative of epilepsy.

EPILEPSY.

Definition.—A chronic progressive disease, characterized by periodic loss or impairment of consciousness, with or without convulsions.

There is no disease which has so many variations in its form, but in all the cardinal point of an epileptic attack is either impairment or loss of consciousness. When an attack is unusual it is either because the convulsions have been suppressed or were not present at all, that consciousness was only partially impaired or the attack consisted of so-called psychic or mental manifestations known as epileptic equivalents. These will be described separately.

It is supposedly more frequent in the male sex, although this is disputed by many. Race has no influence upon the frequency of the disease.

Predisposing Factors.—There is no other disease in which heredity plays so important a part. Epilepsy in one of the grandparents, but especially in the parents, will predispose the child toward epilepsy. Mental or brain diseases, and especially alcoholism or syphilis in the parents, are important predisposing factors.

Children in whom epilepsy subsequently develops, and who have a neuropathic history, may have certain symptoms or so-called stigmata of degeneration as an evidence of their inheritance. These stigmata may consist in microcephalic or macrocephalic heads, asymmetry in the head and face, and sometimes in deformity, irregular or poorly shaped teeth, poorly arched palates, irregular and asymmetrical ears, and at times poor sight and poor development of the limbs.

Exciting Factors.—The pathology of epilepsy is not definitely known. By many it is thought to be a functional disease, and by others the result of cortical chemical changes, but it is probably the result of a maldevelopment of the whole brain, and especially of the motor portion. In support of this, recent microscopic evidence seems to show that the motor cortical cells are undeveloped. Given a child with a bad heredity, or one who was born with a weak motor cortex, it is possible for epilepsy to develop. In a large majority of cases the disease appears in infancy, and only rarely do the convulsions appear after the twentieth year. In the early cases it is possible that reflex convulsions, such as are caused by toxemic and gastric disturbances, may be the exciting factors. The convulsions may follow regularly thereafter or may be absent for a few years, and then reappear about the age of puberty. Epilepsy, of course, may occur in any organic disease in which the motor cortex or the fibers are involved, as in hydrocephalus and tumors, and also following trauma.

Varieties and Symptoms.—There are three chief types of attacks: First, *major epilepsy*, or *grand mal;* second, *minor epilepsy*, or *petit mal;* and third, *psychic epilepsy.*

Major epilepsy, or **grand mal,** may or may not be preceded by an aura. This may consist in a disturbance of any of the special senses, such as flashes of light or temporary blurring of vision, a peculiar odor or taste, noises in the ear, or a feeling of numbness ascending one of the limbs, as from the arm to the shoulder, or the foot to the abdomen and neck, or it may be referred to one of the viscera, as a sensation of numbness rising from the stomach to the throat. Sometimes it consists in a feeling of dizziness or of a sensation or of a fear that something is going to happen. Rarely there may be a so-called dreamy stage. An aura may last from a few seconds to a minute or longer, and, as a rule, is immediately followed in a typical attack by loss of consciousness, the patient falling to the ground, sometimes hurting himself. There may be a cry—the so-called epileptic cry. The body then becomes rigid in tonic contracture, the head may bend backward, the fingers are clenched, the face is blue and livid, the eyes may roll in any direction, and the teeth are clenched. Generally the patient bites his tongue and froths at the mouth. The tonic convulsion may last from a few seconds to several minutes, and is succeeded by clonic or intermittent movements, which may also last from a second to several minutes. Relaxation then follows, and the patient may rally from the attack immediately or may not do so for several hours or longer, feeling weak and exhausted afterward. Because of the contraction of the abdominal walls on the bladder, there is usually some dribbling of urine, and there may be excretion of feces.

The above description is that of a typical attack of major or idiopathic epilepsy. There are, of course, variations, some attacks being more severe than others. An attack may consist in an aura, followed by the epileptic cry, but the tonic and clonic movements may be very slight, although there may follow just as severe a period of exhaustion as that which succeeds the more severe attack. Sometimes the spasms may be very limited, such as movements of the jaw, smacking of the lips, or twitching of a limb, but the important point is that in all of these incomplete attacks there is impairment or loss of consciousness, and nearly always there is a preceding aura and the succeeding period of exhaustion which characterize the more severe attacks.

By **minor epilepsy,** or **petit mal,** is meant a condition in which there is either a partial or an incomplete loss of consciousness and no appreciable or a mild convulsive movement. The milder forms consist only in an aura, followed by a slightly dazed feeling which may last a second or so, but in which there is no actual loss of consciousness. In the more severe attacks, besides the aura the patient will fall to the ground and the loss of consciousness is more complete, the patient feeling dazed for a moment or so. It can be easily recognized from this that there may be many varieties of petit mal, depending upon the presence of the different forms of aura and the extent of the impairment of consciousness.

By **psychic epilepsy** is meant a condition in which certain mental symptoms take the place of the convulsion or spasm with loss of consciousness. By many these attacks are likened to the incomplete form of the minor epileptic attacks, as, for instance, when there is only an aura accompanied by loss or impairment of consciousness. It is probable that these so-called psychic attacks take the place of the spasm, and as such are known as *epileptic equivalents.* They resemble the mental conditions which sometimes occur before and after a spasm. By describing, then, the different forms of psychic epilepsy or epileptic equivalents we will be describing the mental conditions which occur before and after an epileptic attack.

The commonest form is that in which the patient loses consciousness a

moment or so and performs mild, automatic movements, such as unbuttoning his clothes, or making use of some exclamation, and then resuming his previous occupation or what he had been previously engaged in. The patient has no recollection of the occurrence.

When the automatic movement is prolonged for some time, it is called *epileptic ambulatory automatism.* The patient may be in this condition for an hour or longer or for several days, and rarely weeks, and he may wander or travel over considerable distances and behave himself in an apparently orderly manner and after it is over have no recollection of what he has done. He may in the interim have had typical convulsive attacks. Ambulatory automatism sometimes occurs in hysteria, and the differential diagnosis will depend upon the history and the occurrence of other forms of epileptic attacks.

Acute maniacal conditions sometimes take the place of epileptic attacks. They usually come on suddenly, with a feeling of irritability and exhibition of temper, and suddenly the patient becomes maniacal. These attacks sometimes take the form of homicide, suicide, or pyromania or the desire to burn objects. The attacks may last for an hour or longer or for some days, and may finally terminate in stupor, sometimes the patient dying as a result.

Instead of acute mania there may be *temporary delusional* conditions which may last only a short time, or the patient may become stuporous or in a *catatonic* condition, or he may get into the so-called *dreamy state,* during which he has a feeling of unreality, especially regarding himself and objects surrounding him. Again, the attacks may take the form of *paroxysmal laughing,* and *crying* or of *narcolepsy* or periodic sleeping.

Frequency of Attacks.—It is impossible in the beginning of this disease to foretell the frequency of the attacks. As a rule, epilepsy occurs periodically, and in ancient times it was thought that it occurred nearly always once a month, at the time of the full moon. There is no question that in the majority of cases proper treatment will lessen the number of the spasms. The frequency of the attacks will to some extent depend upon the time of the onset and the type of the spasm. In those cases in which they come on in early life, it is probable that they will occur with more frequency and that they will be more severe, while in those in which the attacks begin late in life they will probably not occur so often. Heredity also plays an important part, for in a child in whom there is a neuropathic disposition, and especially one in whom the stigmata of degeneration occur, it is probable that the attacks will become frequent. In those cases in which there are, first of all, minor attacks or attacks of so-called psychic epilepsy, there is also no rule regarding the frequency of the attacks, for most of these cases will finally terminate in typical major epileptic attacks.

It is not at all unusual for epilepsy to occur at night, when it is called *nocturnal epilepsy,* and it may be a number of years before the patient himself becomes aware of his disease, and it may not be until some one sees him in the fit or he has an attack in daytime that the disease is really suspected. Sometimes after the attacks have been manifested in daytime they may for a time come on at night.

Sometimes one attack follows another without the patient regaining consciousness in the interim. Such a condition is known as *status epilepticus.* The patient may be in this state for hours, sometimes for two or three days, and may have as many as seventy attacks a day. They sometimes terminate in death.

Summary of Diagnosis.—History of epilepsy, insanity, or nervous

disease in parents; convulsions which began in childhood and which have progressively become more frequent, and which may be ushered in by an aura and may be followed by an epileptic cry; and then convulsions which are first tonic and then clonic. These are characterized by rigidity of the head, back, and limbs, blueness of the face, frothing at the mouth, biting the tongue, and dribbling of urine, the whole lasting about two or three minutes, and which is followed by a period of exhaustion. Consciousness is recovered in from a few minutes to an hour or a number of hours, with a feeling of weakness.

Differential Diagnosis.—The cardinal point of epilepsy is loss of consciousness and inability to recall anything which has occurred during the attack. The convulsions are characteristic. Sometimes true idiopathic epilepsy may simulate Jacksonian convulsions, but in these cases there is an entire absence of the usual accompanying symptoms, such as headache, nausea, vomiting, vertigo, and choked disc. It is sometimes difficult to differentiate from the convulsions occurring in hysteria, but in the latter the movements do not have the tonic and clonic succession, the tongue is never bitten, and there is never dribbling of urine, and, most important, there is no loss of consciousness.

Clinical Course and Complications.—From the very nature of the disease the prognosis is poor, for the convulsions nearly always become more frequent, more severe, and longer in duration. It has been estimated that in about 10 per cent. of the total number of epileptics cure can be expected, but these cases are nearly all mild and treatment has been early and vigorously instituted. Psychic epilepsy is nearly always followed by petit mal, or later by grand mal, or, what often happens, there may be the three varieties of attacks in one person. The earlier the onset and the frequency of the attacks, the worse the prognosis. When the attacks come on after the twentieth year, the convulsions will not be so frequent and the disease will not make such rapid progress. In nearly all cases the mental functions become ultimately impaired, this resulting partially from the lack of development of the other portions of the brain, from the result of attacks and the constant medication which nearly all patients undergo.

Besides the usual mental enfeeblement which accompanies the disease, the degree of which depends upon the early onset, severity, and frequency of the attacks, there may be total loss of intelligence, this resulting in what is known as chronic epileptic insanity or dementia. The patient usually dies as the result of some intercurrent disease, and only in rare instances does death occur in a fit. It is probable that epilepsy in itself has some influence in shortening the tenure of life, for most epileptics do not reach an old age.

PARTIAL OR TOTAL PARALYSIS.

It must be borne in mind that in the motor cortex are represented the centers for movement, and if these are destroyed paralysis of movement will occur, the extent and completeness depending upon the centers destroyed. Paralysis of one limb, the result of a cortical lesion, is very unusual, and if present is always due to a small tumor, or more probably to an injury. If the whole motor cortex is destroyed, hemiplegia will result.

HEMIPLEGIA.

Definition.—A paralysis of one-half of the body, this including the leg, arm, and lower part of the face, with only a temporary involvement of

such bilaterally acting muscles as are concerned with looking upward, eating, talking, swallowing, and respiration.

Most hemiplegias result from the bursting of a cerebral vessel or apoplexy. Unusual paralyses are those in which the regular order of symptoms is not present or some unusual symptom occurs. These will be explained later. It is more often present in the male, probably because of the greater frequency of early arteriosclerosis and syphilis.

Predisposing and Exciting Factors.—Hemiplegia sometimes runs in families, but in most instances heredity plays no important part, with the exception that if a patient inherits a disease such as syphilis, which gives an early arteriosclerosis, hemiplegia is more liable to occur. Any lesion which interrupts the motor fibers between the motor cortex and the decussation in the medulla will give a hemiplegia on the other side, the form of the specific paralysis and the accessory symptoms depending upon the seat and the extent of the lesion. In most instances this is a hemorrhage; but the other frequent causes are thrombosis, embolism, tumors, injuries of the motor cortex, uremia, and other toxic causes.

Varieties.—Hemiplegia may come on as the result of a lesion at birth, at the infantile period, or that period at which the child cannot walk, in the time of mature development or between the time when the child is fully able to walk and early adult life, about the twenty-first year, and from this time on. These subdivisions have been made because the clinical type of the paralysis will differ according to the time of life it comes on.

Hemiplegia Resulting from Injuries at Birth.—This occurs only when, as a result of difficult or instrumental labor, there is an injury to the motor cortex either of one side or of both sides. Pathologically, meningeal hemorrhages are most frequently found. If the injury is one-sided, a hemiplegia will result, and the child from its earliest life will be unable to use the limbs of one side. The characteristic of this palsy is that the paralyzed limbs will never fully develop and will always be smaller than those on the healthy side, and there will be present athetoid movements. If the meningeal hemorrhage is removed early, it is possible to obtain considerable return of power.

Should there be *bilateral meningeal hemorrhage*, there will be paralysis on both sides of the body, or a so-called *infantile diplegia*. In such case there will be bilateral spasticity, increased reflexes, and the Babinski reflex. Besides, the limbs will never become fully developed, and there will be present athetoid movements of the upper and lower limbs and in the muscles of the face, head, and neck. In most instances, also, there will be inability to talk.

Paralysis Coming on During the First Two Years of Life or in the Infantile Period.—To understand this it is necessary to have a knowledge of the development of the motor system. The child when it is born cannot walk because of lack of development of the motor fibers. This can be readily seen when a cross-section is made of the spinal cord of a newly born child, for the myelin sheaths of the motor fibers will not stain. On the contrary, if the spinal cord of a chicken, which walks immediately after birth, be stained it will be found that the myelin sheaths are fully developed. That is why the chicken can walk and the child cannot. Ordinarily it takes from one to two years for the myelin sheaths to obtain full development, and when this is reached the child will be able to walk. It can be readily seen from this why it is an error to force or urge children to walk before they are able to do so themselves, and also is an evidence of the cause of deformities of such children. Should, therefore, there occur a lesion of the motor centers or fibers in this

period, there will result a hemiplegia, and, similar to the paralysis which occurs as a result of meningeal lesions, there will be spasticity, increased reflexes, Babinski reflex, and lack of development of the limbs, but this will not be as great as in lesions at birth. It can also readily be seen why a lesion occurring near infancy will cause a greater lack of development. Athetoid movements, as a rule, do not occur, and if the lesion should be bilateral, it is probable that there will not be much impairment of speech (Fig. 394).

Paralyses Which Occur from the Second Year or the Infantile Period to Full Maturity.—A child grows up and does not reach full development until about the twenty-first year, sometimes later. The hemiplegia which occurs in this period will differ from the paralysis occurring later only in the fact that there will be lack of development of the limb, this being greater the earlier the lesion. The causes are generally injury to the head, early syphilis of the nervous system, and embolism.

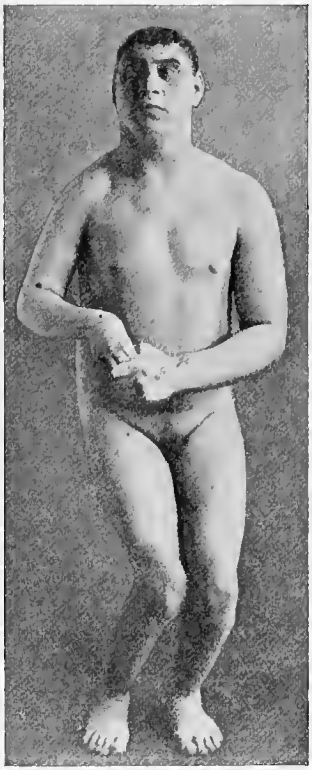


Fig. 394.—Right Infantile Hemiplegia with Contractures, Lack of Development of Limbs, and Idiocy.

Hemiplegia Occurring after the Twentieth Year.—The hemiplegias which occur after the twentieth year do not differ as to type, but they do as regards their etiology. It is a safe rule to assume that when hemiplegia occurs in early adult life, before the fortieth year, the cause is syphilis. The other causes may be embolism, uremic or toxic conditions, brain tumors, or injury to the head. If the result of syphilis, there may or may not be present the indications or early history of such disease.

Hemiplegia Coming on after the Fortieth Year.—Hemiplegias coming on after the fortieth year are usually the result of apoplexy. The other causes are also operable.

Apoplexy.

By apoplexy is meant the bursting or occlusion of a blood-vessel, the usual seat of hemorrhage being in the lenticulo-striate artery. Such a lesion will usually injure the posterior limb of the internal capsule, thereby causing hemiplegia on the opposite side; and if the sensory and visual fibers are also involved, hemianesthesia and hemianopsia. Hemorrhages in the other portions of the brain and brain stem will give various symptoms according to their localization. (See section on cerebral localization.)

When apoplexy occurs, there is usually an accompanying shock, the patient being rendered unconscious. It is somewhat difficult to tell which side is paralyzed, because in the period directly following the attack there is so much shock that there is complete loss of tone in all of the limbs, and it will be impossible to recognize by the resistance which limb is paralyzed. Later on, of course, tonicity will become apparent in the sound side. It will be found, however, that on the side of the paralysis there will be drooping

of the lower part of the mouth, dribbling of saliva, and stertorous respiration. The paralytic will bring to aid all of the accessory muscles of respiration, and in expiration the cheek on the paralyzed side will be puffed out, and because of this there will be dribbling of saliva from the paralyzed to the healthy side. This is an important sign. Again, if the patient is stuck with a pin, there will be reflex movement on the sound side and not on the paralyzed side.

As a rule, the patient regains consciousness within a few hours, and from then on there will be a progressive return of power. If, however, the patient does not regain consciousness in twelve hours or more, the prognosis is invariably fatal.

When hemiplegia is due to *embolism,* there is always an accompanying heart disease and the signs of a valvular lesion. The onset is generally abrupt and unconsciousness is nearly always present. It may come on at any age, but generally in the young.

Hemiplegia due to *thrombosis* nearly always has a slow onset and occurs after the fortieth year. In such cases there is nearly always a cerebral arteriosclerosis, and there is usually a history of some disease, such as syphilis or rheumatism, these being prone to produce arteriosclerosis. There is hardly ever unconsciousness.

When hemiplegia is the result of *syphilis,* the onset is usually abrupt, and, as a rule, there is no unconsciousnsss. It generally occurs in persons before the fortieth year.

Hemiplegia sometimes occurs in the course of or following an *infectious disease,* such as typhoid fever, scarlet fever, measles, diphtheria, whooping-cough, etc. In most of these cases the paralysis is the result of multiple areas of cortical inflammation, and there is produced what is known as *encephalitis.* Again, the disease instead of producing multiple areas of inflammation in the brain may cause thrombosis, this resulting in hemiplegia. The extent and character of the paralysis will depend upon the cortical areas involved.

In the course of a *uremic condition* it is possible to have paralysis of one side of the body. It is characteristic of this disease, however, that the paralysis is never complete, that it does not last long, and that it is always accompanied by convulsions, which may be either Jacksonian or general in type. There are also present the accompanying symptoms of uremia, such as unconsciousness, urinous odor, dropsical condition of the limbs, and albumin and casts in the urine.

The paralyses which come on in the course of *an injury to the head* are usually accompanied by the surgical symptoms of the injury. This subject will be discussed separately, as will also paralyses resulting from *brain tumor.* In the latter condition the hemiplegia comes on very gradually, and there are always the accompanying symptoms of an irritating lesion of the brain.

Symptoms.—Premonitory symptoms are sometimes present in those cases in which hemiplegia follows apoplexy, thrombosis, or cerebral arteriosclerosis. The reason for this is that in these instances there is either a weakening of the cerebral vessel walls or there may be, as in apoplexy, a congestion of the vessels or heightened arterial tension, which results finally in the bursting of its walls. In thrombosis the premonitory symptoms may last for a long time, because in this condition we have a gradual closing up of the lumen of the vessel, and in arteriosclerosis there is usually the same condition. These premonitory symptoms generally consist in headache of an indefinite character, sense of dizziness or fullness of the head, sometimes

noises in the ears, and in arteriosclerotic conditions a more or less complete failure of memory, and occasionally tingling or numb sensations in the limbs which subsequently become paralyzed. Sometimes there may even be a temporary weakness in one or both limbs. As a rule, however, these preliminary symptoms are not present. Most hemiplegias occur at night, the patient going to bed in good condition, to find himself paralyzed in the morning.

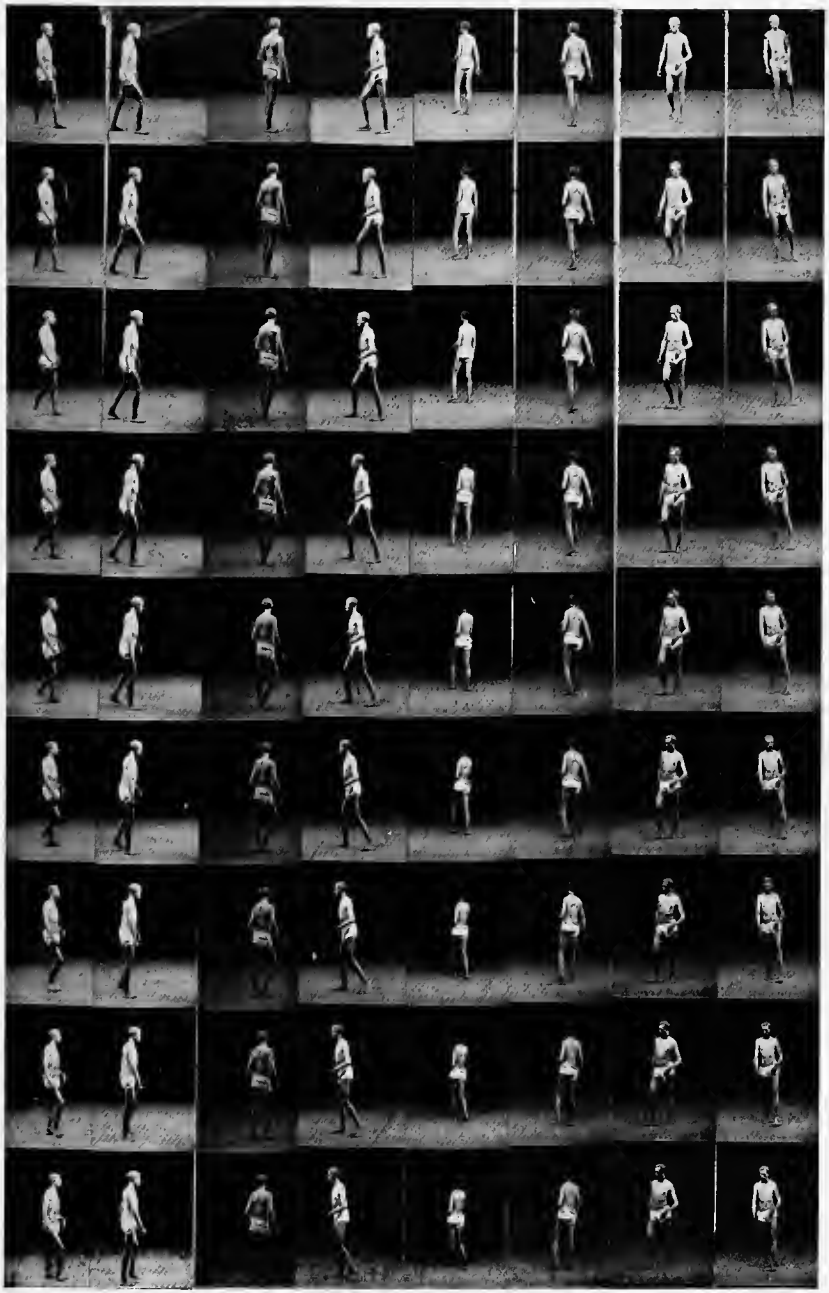
If there is an accompanying loss of consciousness and stupor, which is especially true in hemiplegia resulting from apoplexy, the patient may not rally at all and die within a few to twenty-four hours. In most cases, however, consciousness is not lost very long, the patient feeling fairly well within a half hour or an hour. At first the paralysis will be complete and flaccid, there being inability to move the arm, leg, and lower part of the face on one side. Within a few hours, or a day or two at the most, recovery of movement begins. The earlier this recovery, the better the prognosis; and it is a safe presumption that if there is no recovery of function within two or three days after the attack, the paralysis will remain as it is. The return of power is always greater in the lower than in the upper limb, and it is for this reason that most hemiplegics are able to walk. Because of the fact that, naturally, the flexor muscles are stronger in the upper limb and the extensors in the lower, there will be greater return of function in these groups, and it is because of this greater recovery that there develops what are termed *contractures*, and in nearly all hemiplegics this contractured condition of the limbs will be of the flexor type in the upper and extensor in the lower, there being inability to move the shoulder, the arm is held closely adjusted to the side, the forearm flexed on the arm and slightly turned inward, and the fingers clenched. In the lower limb the foot will be turned in and extended and the leg adducted, the knee being held near the median line. When such a patient walks, he has what is called unilateral spastic or hemiplegic gait, dragging the toes along the ground, the leg being only partially bent at the ankle and knee and swung around the body like a pendulum. The arm is usually held close to the side of the body (Plate **XVIII**).

Of course, the degree of recovery will depend upon the extent of the destruction of the motor fibers. If that is complete, there will be no recovery at all, and the paralyzed limbs will become stiff or spastic and contractured. Whenever there is a lesion of the motor columns, as has been explained in a previous section, the involved limbs will become stiff or spastic, contractures will result, all of the tendon reflexes will be increased, and there will be present the Babinski reflex, which always indicates a lesion in those motor fibers in relation with the leg, and possibly ankle and patellar clonus, depending upon the degree of the spasticity and involvement of the motor tracts.

Atrophy of the muscles of the paralyzed limb always occurs following hemiplegia, and this can be noticed first especially in the muscles of the shoulder, and later in the muscles of the hand and leg. This is due partially to disuse, and principally to a disturbance of trophic function of the cells in the anterior horns of the spinal cord whose innervation is disturbed. This atrophy is general and not very marked, and must be distinguished from the lack of development of the limbs occurring as a result of a lesion at birth or soon after. The limbs, also, because of the disturbance of trophic function, have poor circulation and are generally cold, and there may also be disturbance in the growth of the nails and hair, and the skin, as a rule, does not react as well as that of the healthy side.

Motor aphasia, or difficulty or inability to talk, although the patient

PLATE XVIII



Moving Picture of Left Hemiplegia. (Courtesy of Mr. Sigmund Lubin of Philadelphia, Pa.)

understands what is being said to him, occurs when the lesion in right-handed persons is on the left side of the brain. The completeness of the aphasia will depend upon the extent of the destruction of the motor cortex concerned with motor speech. A similar condition occurs if the lesion is on the right side in left-handed individuals. In most cases, except when the lesion is very large and cortical, there is some recovery in the function of motor speech, but occasionally the aphasia may be absolute, so that the patient can utter only one or two words, as "yes" or "no." This subject has been more fully discussed under the head of aphasia. Sometimes in lesions of the right side of the cortex in right-handed individuals there may be a temporary aphasia, this only lasting for a day or two. This is partially due to shock, and also to the fact that in most individuals the motor functions have some bilateral representation. Of course, sensory and other forms of aphasia occur if the relative centers are destroyed.

As has already been mentioned, in an ordinary hemiplegia there is paralysis only of the lower and upper limb and lower part of the face, and there is no disturbance of such bilaterally acting muscles as those concerned with looking upward, mastication, eating, swallowing, and respiration. That is the rule; but if the patient is carefully examined immediately after the attack, there may be a little weakness, which lasts only for a short time, as, for instance, of the masseters, in looking upward, the tongue may be protruded to one side or the respiratory muscles may not act as well on the paralyzed side as on the other. To have a permanent disturbance of function there must be bilateral cerebral lesions. Such a condition is known as *pseudo-bulbar palsy*, and will be discussed under a separate heading.

The above description of the symptoms and course is that of the usual hemiplegia occurring in the middle or latter part of life as a result of an interruption only of the motor fibers. If there should be, in addition, an involvement of the sensory fibers, especially in hemorrhages destroying the posterior limb of the internal capsule, there will be, in addition to the motor symptoms, alteration of sensation on the other side of the body, and also disturbance of vision because of the interruption of the visual fibers, or hemiplegia, hemianesthesia, and hemianopsia.

The symptoms of *hemorrhages occurring in the cerebral peduncle, pons, and medulla* have been discussed in the section on cerebral localization when discussing the symptomatology of lesions in these areas. In addition to the specific symptoms of such lesions, there will be the usual symptoms of shock, such as described under apoplexy.

Summary of Diagnosis.—A man past middle life, with or without headache, dizziness, or fullness in the head, goes to bed at night and is found unconscious with a paralysis of one side of the body. When examined, he does not respond to questions, the limbs are flaccid, and there is no resistance or tonicity of movement. There is stertorous respiration, puffing out of the cheek on one side, and drooping of the mouth on the same side, dribbling of saliva from this side, and if he is stuck with a pin he will move the limbs of one side only, the paralysis being on the side of the drooping of the face. The patient usually recovers consciousness within an hour or longer, and if right-handed and the hemiplegia is on the right side, will have an accompanying difficulty in talking, or motor aphasia, although he will understand everything that is said to him. He will be able to begin to use the limbs within a few hours, and in a week or so there will be considerable recovery of movement, especially in the flexor groups in the upper and extensor in the lower, and the limb will become stiff, spastic, and contractured

and there will be increase of reflexes with the Babinski phenomena, with or without ankle clonus, besides drooping of the corner of the mouth and inability to show the teeth. He will be able to wrinkle the brow and shut his eyes without any difficulty and he will have a hemiplegic or unilateral spastic gait.

Differential Diagnosis.—There is no difficulty in diagnosticating a hemiplegia the result of an organic lesion, because of the usual symptoms of weakness, spasticity, increased reflexes, and the Babinski reflex. It is sometimes, however, difficult to recognize what the exact cause of the hemiplegia may be, and this is important, especially as regards treatment. These points, however, have been considered when discussing the causes of hemiplegia. Sometimes hemiplegia occurs in hysteria, but in the latter condition there will always be the accompanying symptoms, the paralysis is never complete, the contractures are not typically of the flexor and extensor group in the upper and lower limbs, and the Babinski reflex is never present.

Clinical Course and Complications.—The prognosis in any given case of hemiplegia will depend upon the extent of the destruction of the motor fibers. If that is complete, there can be no return of function. In most cases, however, there is always enough recovery to enable the patient to walk and to use his upper limbs somewhat. The prognosis in cases of apoplexy has already been discussed under that head. In hemiplegias resulting from such toxic causes as uremia and diabetes, the prognosis depends upon the ability to overcome such toxemia, and if that is accomplished, the paralysis will disappear.

The complications occurring in hemiplegia depend upon the extent of the involvement other than that of the motor fibers, and the specific symptoms will depend upon the location of the lesions. These symptoms have been discussed under cerebral localization.

In nearly every hemiplegic there will be some impairment of mental functions. This is due to the interruption or destruction of some of the association brain fibers. The ordinary course of life of a hemiplegic need not necessarily be shortened, except that it must be remembered that whenever there is a destruction of the motor fibers there is lessened general resistance, especially on the hemiplegic side.

Diplegia.

Definition.—A paralysis or paresis of both upper and lower limbs and of the lower portions of the face resulting from bilateral lesions of the motor fibers either in the cortex or anywhere in their course between the cortical motor centers and the decussation in the medulla.

Varieties.—Diplegia may result: (1) from lesions which are either congenital or occur before birth; (2) from lesions or injuries at birth; (3) from lesions occurring in early infancy or in the first few years of life; and (4) as a result of lesions occurring in the latter end of life. The clinical picture in each instance differs, and therefore will be discussed under separate headings.

Diplegia Resulting from Lesions which Are Either Congenital or Occur Before Birth. Congenital Spastic Rigidity of Limbs (Congenital Hypertonia). Little's Disease.—Under this classification will be included only those cases in which, as a result of premature birth, there is a weakness of all four limbs, although many writers include under the term Little's disease spastic paresis of the limbs resulting from lesions at birth.

To better understand the symptomatology of so-called congenital spasticity of the limbs, it is necessary to remember that the motor tracts are not fully developed at birth and do not become so until after the first year. Therefore, the earlier the interruption of the motor fibers, the greater will be their lack of development. A child born prematurely will not only have undeveloped motor fibers, but will also have lack of development of other fibers concerned with sensation and association of ideas and of thought. It is because of this that there is, in addition to the spastic symptoms in the limbs, lack of mental development, which sometimes is very great.

In such a child the limbs are undeveloped and poorly shaped and walking will either be impossible or delayed many years. The upper limbs are never as much involved as the lower, and in most instances it will be possible to use them. There is hardly ever any weakness of the lower part of the face. As in all cases where there is a lesion of the motor columns, there will be stiffness or rigidity of the limbs with increased reflexes and the Babinski phenomenon. Besides, the contracture in this disease is typical. The thighs are flexed on the abdomen, the knees adducted, almost touching each other, the legs extended from the knees at an acute angle, and the feet are held in a position of equinovarus. There is hardly ever any contracture in the upper limbs. The rigidity in these cases is excessive, and is more so than in lesions of the motor fibers occurring later in life. Convulsions with spasms in the limbs are very frequent, as are also athetoid movements both in the limbs and face. The mentality in most cases is poor, although rarely the child is capable of considerable development. The disease is progressive and no cure can be expected.

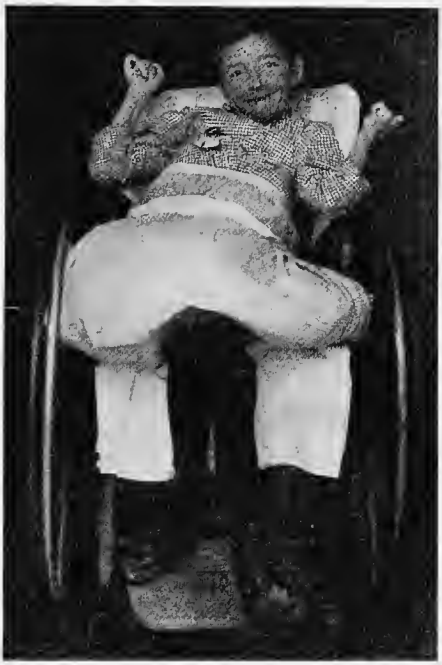


Fig. 395.—Infantile Diplegia showing Contractures and Athetoid Movements in the Limbs and Face.

Diplegia Resulting from Lesions or Injuries at Birth.—Most diplegias appearing at birth or soon after are the result of cortical injuries sustained in difficult labor, as bilateral meningeal hemorrhages from the use of forceps. In these cases there may or may not be present the surgical evidences of injury. The subsequent history consists in the development of convulsions, which may be either Jacksonian or general in type, with sudden or gradual development of paresis or paralysis of the limbs of one or both sides, accompanied by a gradually increasing rigidity, exaggeration of reflexes, and the Babinski phenomenon. Such is the course if there is no operative interference with removal of the hemorrhages. Besides, there will be considerable mental impairment, and as the case progresses there may develop athetoid movements, and deformed and contractured limbs with considerable lack of

development. The speech functions in most of these cases never become developed (Fig. 395).

Internal Hydrocephalus.—Another frequent cause of infantile diplegia is internal hydrocephalus, by which is meant an increase of cerebrospinal fluid in the ventricles of the brain. The cerebrospinal fluid is probably secreted by the choroid plexus, and if there is any interference with the normal outflow of the fluid, or if there is an overproduction of fluid, there will necessarily result a dilatation of the ventricular walls, or internal hydrocephalus. These causes may be congenital, such as closure of the foramen of Magendie or of the aqueduct of Sylvius, or an aberrant secretion of the choroid plexus. When this disease appears later in life, the closure of one of these foramen may result from a basilar meningitis, which is usually of a tuberculous nature. Whatever the cause, the gradual increase of fluid in the ventricles will increase the size of the cranial cavity and cause pressure of the brain substance, with consequent atrophy and loss of function (Fig. 396).

Fig. 396.—Moderate Hydrocephalus, showing Enlargement of Head.

If the causes are congenital, the child may be born with a very large head, but in most instances it does not become apparent until after birth, when it will be noticed that the development of the child both physically and mentally is delayed. The head gradually becomes large, especially in the frontal and middle portions, the fontanels do not close and bulge, and the head sometimes assumes an enormous circumference. The face does not show any deformity, with the exception that the eyes may bulge. Coincident with this it will be noticed that the limbs do not become developed and soon show an increasing weakness, with rigidity and exaggerated reflexes and the Babinski phenomenon with contractures. The mentality is poor, although sometimes there may be considerable development. Associated with internal hydrocephalus there may be a rachitic condition of the chest.

There is a form of hydrocephalus known as *external hydrocephalus*, by which is meant an accumulation of fluid in the cortical meninges. This occurs nearly always in association with chronic meningitis, and will be discussed under that head.

Diplegia Resulting from Lesions in Early Infancy or in the First Few Years of Life. Acute Encephalitis.—These result mostly from inflammation of the brain, such as occurs in the course of the various infectious diseases and is known as encephalitis. Pathologically there is usually found congestion of the vessels with round-cell infiltration with destruction of the nerve-cells and fibers. Occasionally the meninges are also diseased. The foci of inflammation are nearly always multiple and usually involve the motor fibers. If the lesions are limited only to one side of the brain, unilateral

paralysis will occur. Sometimes the inflammation is purulent in nature and may be in association with a purulent meningitis.

Paralysis resulting from such encephalitis does not differ from the usual form of hemiplegia, with the exception that the onset is acute and there is present a history of an infectious disease with fever and its accompanying symptoms. The paresis or paralysis comes on immediately after this, and the degree of the diplegia will depend upon the extent of the motor involvement. The limbs become increasingly spastic and rigid, the reflexes are exaggerated, and the Babinski phenomenon is present. The lower limbs are always more involved than the upper. The undevelopment of the limbs will depend upon the time of the occurrence of the lesion. The nearer birth, the greater will be the lack of development. Besides, there will be contractures, and possibly athetoid movements.

Acute inflammations of the brain occur elsewhere than in the cerebrum. They are frequently present in the gray and white matter surrounding the aqueduct of Sylvius, when it is called *superior polioencephalitis*, or it may be present in the medulla, where it is known as *inferior polioencephalitis*. This has been more fully discussed on page 1038. In association with the latter diseases and with cerebral encephalitis there may be similar inflammations in the spinal cord, such as *acute poliomyelitis* (page 1088), or there may be at the same time inflammations in many or all of these areas.

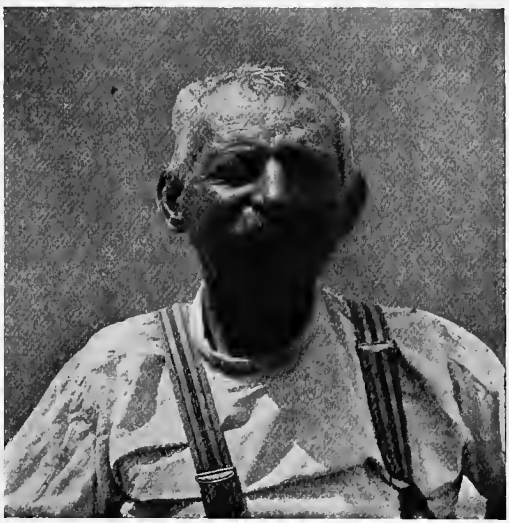


FIG. 397.—INVOLUNTARY CRYING IN PSEUDOBULBAR PALSY.

Diplegias Resulting from Lesions Occurring in the Latter End of Life. Pseudo-bulbar Palsy.—This usually comes on after the fortieth year and results from multiple areas of softening on both sides of the brain. Rarely, however, because of syphilitic inflammation the disease may be present in young adults. There is usually an attack of hemiplegia on one side, with its attending symptoms, and in the course of a few years—possibly one or two or longer—there is another attack, which causes a hemiplegia on the other side of the body. Immediately after the second attack the symptoms of so-called pseudo-bulbar palsy appear. Because of the bilateral motor involvement there is a varying weakness or paralysis in the lower and upper limbs. In most cases there is a preponderant paralysis on one side, or the involvement of the limbs may be very small, and there is usually present spasticity with increased tendon reflexes and the Babinski phenomenon.

This symptom-complex is called pseudo-bulbar palsy because the symptoms, other than those in the limbs, resemble those present in true bulbar palsy, but the lesions differ in each. As has been previously explained, whenever there occurs a lesion on one side of the brain, as in apoplexy, there is

usually paralysis only of the limbs and lower part of the face, with an escape of the bilaterally acting muscles concerned in eating, talking, swallowing, and respiration, because these functions have bilateral cerebral centers. If, however, there are bilateral cerebral lesions interrupting the motor fibers anywhere in their course between the cortex and the medulla, there will necessarily be involvement of these bilateral functions, and there will be difficulty in talking, eating, and swallowing. The speech will be thick, indistinct, and "bulbar" in character; eating, chewing, and swallowing will be difficult, the patient choking very frequently, it being a common cause of death; besides, there will be disturbance in the emotional qualities, as in laughing and crying, and there is present what is called involuntary or forced laughing and crying (Fig. 397).

There should be no difficulty in distinguishing this disease from true bulbar palsy, for in the latter, besides the so-called bulbar symptoms, there will be, in addition, fibrillary tremors and atrophy in the muscles of the face, tongue, palate, pharynx, and larynx. The involvement will be gradual and there will be no history of bilateral hemiplegia.

BRAIN TUMORS.

Under this head will be considered tumors, abscesses, areas of softening, or whatever else may give the symptoms of a neoplasm in the brain. The most common form of tumor is the glioma, next in order being sarcoma, endothelioma, fibroma, fibrosarcoma, carcinoma, tuberculoma, adenoma, etc.

Glioma.—This form of tumor is almost always primary and single, although metastasis may rarely occur. The tumor may be as small as a cherry or as large as a hen's egg. It always grows from the brain substance itself and is of slow growth. It is not sharply defined, but infiltrates into the brain substance, and it is difficult to tell it from normal brain tissue, although sometimes there is an increased consistency to pressure and there may be a slight swelling. The border zone of the tumor may present an increased number of blood-vessels and there may be islets of new tissue.

Sarcoma.—The growth may be small, flat, or nodular or may be of large size. It is primary and usually solitary. Sarcomata always grow from the meninges, periosteum, or cranial bones, or from the pial covering of the blood-vessels. It never grows from the brain substance, and therefore, unlike the glioma, it always compresses the brain tissue and may be distinct from it, although not infrequently it infiltrates the latter. Even when growing within the brain a distinct margin may sometimes be found, due to the softened area surrounding it. It is usually harder in consistency than the glioma, slower in growth, and very vascular.

The tumor may soften or caseate, and myxomatous, hemorrhagic, and cystic changes are not uncommon. Cystic changes are especially common in the cerebellum, not only in sarcomata, but also in gliomata. If the fibrous tissue is very marked, we have a fibrosarcoma.

Sarcoma may manifest itself as a diffuse multiple sarcomatosis. This may involve, first, the nervous substance and the meninges; and, second, the membranes only, when they may appear in the form of small tumors or as a diffuse infiltration. When the brain or its meninges are implicated in sarcomatosis in about two-thirds of the cases, a tumor of the cerebellum is found. They may also be found in the fourth and lateral ventricles, Gasserian ganglia, and pituitary body; in fact, almost anywhere.

It is important to remember that when sarcomatosis is present the soft tumor masses grow in the pia about the cranial nerves and spinal roots and may produce little or no compression or destruction of the nervous tissue. It is because of this that few clinical symptoms may appear, although there may be extensive alterations in the nervous tissue. A correct diagnosis of sarcomatosis of the brain and the pial covering is often impossible.

Endothelioma.—This is a form of sarcoma which grows either from the endothelial lining of the dura or from the perivascular spaces. It differs from sarcoma only in that the cells are arranged in clumps or columns and that it is more vascular. It never infiltrates the brain tissue, but compresses it, and is a very favorable growth for removal. When it is present, there may be an accompanying overgrowth of the cranial bones covering it.

Osteosarcoma.—Occasionally a sarcoma will grow from the cranial bones, or it may involve the cranial bones secondarily. In such cases the tumor is called an osteosarcoma.

Glioma, sarcoma, and cysts of various kinds are more frequent in the adult, and tuberculous growths are more common in persons below the age of twenty years.

Tuberculoma.—Tuberculous growths occurring in childhood are more frequently located in the cerebellum than in any other portion of the brain. In the adult they are found with equal frequency in this region and in the pons and cerebral cortex. They are nearly always multiple and secondary to a tuberculous process elsewhere in the body. A tendency to symmetrical arrangement is also sometimes observed. Their size varies from a small nodule to a large fist. Macroscopically it is hard to distinguish a tuberculoma from a syphiloma. Both have poor blood-supply and a tendency to caseate; the tuberculous growth, to pus formation. Again, both have a tendency to grow from the meninges, although the tuberculous growths are found in the substance of the brain and may have granulation areas and miliary tubercles about their border.

The growth of a tubercle may be either rapid or slow. Tuberculous tumors may give no clinical symptoms. This has been explained by the slowness of the growth, the brain tissue gradually accommodating itself to increased pressure. It is possible, however, to demonstrate by certain silver stains the persistence of the axis-cylinders in these growths, this explaining the persistence of function. Surgically it is not advisable to operate upon tuberculous tumors, as they are multiple and cannot be removed. A tuberculous growth may be part of a general tuberculous meningitis or there may exist tuberculous meningitis alone. If the symptoms of meningitis arise, it is always a wise procedure to look for a tuberculous process in the lungs as an aid to diagnosis.

Syphilitic Growths.—Gummata are rarely found postmortem, although they are usually thought to be the most common form of brain tumor. The usual results of syphilis in the nervous system are an endarteritis of the blood-vessels, round-cell infiltration, and meningitis. The endarteritis is usually general, and because of the weakening of the blood-vessel walls, early hemorrhages may result.

Syphilitic meningitis usually involves the basal membranes, but may also involve those of the cortex. In the latter instance the meninges may be half an inch in thickness and thereby compress the brain and give focal symptoms of tumor. Occasionally the syphilitic process involves the brain substance itself, causing a diffuse cellular infiltration, or the bones may be involved, causing a carious condition of a part or many of the cranial bones.

When basal meningitis occurs, it may involve the whole extent, or, what is more often the case, only the meninges near the chiasm, thus involving the second, third, fourth, and sixth cranial nerves.

At times, instead of meningitis there may be diffuse areas of softening throughout the brain, these areas being yellowish-red in color, soft in consistency, and well defined from the surrounding brain tissue. Syphilitic growths are usually rapid in development, but it must be remembered that the various pathologic conditions which lead on to the growths are long present.

Fibromata.—These tumors are rare, but they are relatively more frequent in the cerebellum than in the cerebrum, and especially in the cerebello-pontile angle.

A fibroma invading the cerebello-pontile angle or the angle between the pons and the cerebellum may be only part of a general neurofibromatosis. This, however, is rare, and usually a tumor in this area is the only expression of this process or a central neurofibromatosis. The growth is slow and generally unilateral, although in rare instances it may be present on both sides. Experience has shown that they are more common on the left side in the ratio of three to two.

The fibroma may be as small as a cherry or the size of a large egg. The growth is firm, hard, nodular, and has a distinct capsule surrounding it. When located in the cerebello-pontile angle, they are generally loosely attached to the brain by an atrophic nerve-trunk and a few blood-vessels or a meningeal process. These attachments may be easily ruptured. These tumors are in organic relations, especially with the acoustic nerve, and more rarely with the trigeminus and facial nerves. They nearly always grow from the endoneurium and rarely from the perineurium or epineurium. Consequently it is possible to find medullated nerve-fibers either in the periphery of a tumor or in its center. As a rule, if many cranial nerves are involved, there is a general neurofibromatosis.

These tumors compress greatly the lateral lobes of the cerebellum, the pons, and the medulla oblongata. At times even the temporal lobes may be compressed. Because of the slow growth and nature of the tumor clinical symptoms may not appear at all or only late in the disease. Tumors of the cerebello-pontile angle are among the most favorable for surgical removal.

Carcinoma.—Carcinoma of the brain is always secondary to growths elsewhere in the body, generally from the stomach, lungs, or breast. The tumor may grow in the substance of the brain, but mostly grows from the dura or the cranial bones. Carcinomata may be as small as millet-seeds or of large size, and may occur anywhere in the brain substance. At times there may be an infiltration of cancer cells in the pia covering the whole brain substance. This, however, is a rare occurrence. The possibility of toxic changes must be considered, as it is not improbable that through intoxication caused by carcinoma elsewhere in the body symptoms of tumor may be present.

Such other tumors as *adenoma*, *cholesteatoma*, *lipoma*, and *psammoma* very rarely occur in the brain, and as they do not differ from similar growths elsewhere, will not be considered.

Cysts.—Cystic degeneration of gliomata and sarcomata is very common. Other tumors, as fibroma and carcinoma, are prone to undergo cystic change, but more rarely. It is possible for the whole tumor to disappear and only the cyst remain, so that microscopic examination will be necessary to detect the small tumor mass in the walls of the cyst.

Congenital cysts may occur in the fourth and lateral ventricles or in the substance of the brain. This, however, is a rare occurrence.

The most common cystic changes found in the brain are due to parasitic growth, the *cysticercus cellulosæ* and the *ecchinococcus*. These, however, are so rare in this country that they will not be considered.

Cysts due to traumatism may occur, but their genesis is by no means clear. It is probable, however, that they are the result of a hemorrhage which has occurred at birth or soon after. As the brain tissue at this time of life is not fully developed, cystic changes or porencephalus may result.

Cystic tumors occasionally grow from the choroid plexus in any of the ventricles. These may not give any symptoms, but if sufficiently large will compress the ventricular walls and the surrounding brain tissue.

The Influence of Brain Tumors upon the Surrounding Structures.—At operation when the dura is removed there is nearly always increased tension and the parts may bulge. The surface of the brain is flat and the fissures may be abolished, and the pia covering the neoplasm is generally poor in blood-supply. The tissues near the growth may be softened. Pressure symptoms nearly always result, this depending upon the nature of the tumor, the extent of its growth, and its location. The greatest pressure is nearly always exerted in the nearby structures, but often a tumor of the cortex may exert pressure upon the cranial nerves at the base of the brain.

The cerebrospinal fluid may be increased in brain tumor, but this is especially so when the growth is in the posterior cranial fossa, because pressure here is exerted directly upon the communication between the lateral and fourth ventricles, or upon the veins of Galen, which convey the blood from the choroid plexus to the sinus rectus. Because of this, increased tension results in the lateral and third ventricles, the latter causing direct pressure upon the optic nerves.

The Diagnosis of Tumors of the Cerebrum.—In the preceding pages the individual symptoms and pathology of tumors have been considered and analyzed, and continuous reference will be made to their contents. The collective symptoms as they occur in brain tumors will now be discussed.

The general symptoms of brain tumor are headache, nausea, vomiting, vertigo or dizziness, and choked disc. As a rule, all these symptoms are present in a tumor of fairly large dimensions, but a growth may exist without the presence of any of these. Such a growth, however, must be small and of such character as not to cause pressure. The symptoms which are present in the great majority of cases are headache and choked disc.

The *headache* may be localized to the site of the lesion, but, as a rule, it is general.

Choked disc occurs in about 90 per cent. of cases, and may be greater on the side of the tumor. The swelling of the optic nerves is always greater in cerebellar lesions and comes on earlier than in cerebral lesions.

Very recently Cushing described inversion and interlacing of the color fields, which has been usually thought to be characteristic of hysteria, as an early pressure phenomenon.

Nausea, vomiting, and *vertigo* are especially prone to be present when the growth is large and great intracranial pressure exists. These symptoms are also more liable to be present in tumors which press upon the medulla, as is the case in occipital lesions and in tumors of the cerebellum.

When considering the symptoms of brain tumors, the side on which such

a growth may occur must be taken into consideration. Tumors which are located on the left side of the brain can be more readily detected because our knowledge of localization is better on this side. This, of course, is in a right-handed individual. The contrary would be true of a left-handed person.

It is, of course, impossible to tell exactly, when the symptoms of a brain tumor are present, what the nature of the growth may be. If there are present elsewhere in the body certain conditions, such as carcinoma, tuberculosis, and abscess of the lung, or if there is a history of syphilis, the presumption is justifiable that similar conditions exist in the brain, provided the symptoms come on in regular order. Growths in the cerebral cortex are more likely to be sarcoma or glioma, while in the brain stem glioma and tuberculoma are more common.

Fig. 398.—Facies of a Brain Tumor, showing some Exophthalmos with Weakness of Right Internal Rectus.

It is also important, from a diagnostic standpoint, to differentiate between a *cortical* and a *subcortical* growth. A *tumor which is cortical* may in time involve the subcortex, and a subcortical tumor may in time involve the cortex. Generally speaking, a cortical lesion will always give irritative phenomena. If in the motor cortex, there will result Jacksonian convulsions on the opposite side, to be followed later by paralysis. If in the sensory cortex, there will first be numbness and pains of the Jacksonian type, this to be followed later by anesthesia. If the location is in the occipital lobes, there will first be irritative visual phenomena, such as scintillating scotoma in the visual fields related to these areas, and later loss of vision. Generally speaking, then, in a cortical growth there will be irritative phenomena, to be followed by paralytic symptoms (Fig. 398).

In a *subcortical growth* the symptoms will depend entirely upon what fibers are cut off, as in the subcortex a tumor, no matter how small, will always involve fibers concerned with more than one function. The symptoms of a lesion in this area will always be greater than in a cortical lesion, for in the latter instance a tumor may involve only a very limited portion of the cortex, this giving only a few symptoms. Again, in a subcortical lesion irritative symptoms are not likely to occur, and the earliest symptoms are those of loss of function.

It is well known that tumors have a predilection for certain areas. These are the frontal, central or motor, parietal, occipital, and temporal. The symptoms which occur in growths of these parts will be taken up in order.

Tumors of the Frontal Lobe.—Tumors in this area are not very com-

mon. The growths more often are of the sarcomatous variety, and in most instances grow from the frontal bones or from the orbital plate. When the neoplasm is limited to the frontal lobe itself, there are, as a rule, very few localizing symptoms. In the frontal lobes have been placed the centers for higher psychic functions, this being especially so in the left; but it cannot be said that any special mental symptoms occur from destruction of these areas. The usual mental symptoms are gradual change in disposition, gradual loss of memory, of intellect, and of the power of reasoning. As can be readily seen, these symptoms may occur from tumors in any portion of the brain. Headache may and may not be present, and is prone to be localized to the orbit and frontal bones. Nausea, vomiting, and dizziness are not very common, and choked disc is a rather rare occurrence except in those instances in which the tumor is mostly localized to the orbital part of the frontal lobes and direct pressure is exerted upon the optic nerve, in which case the choked disc is unilateral. In the latter instance the olfactory nerve is also pressed upon, causing loss of the sense of smell on that side. If the tumor is of large size and involves the precentral convolution, motor symptoms will be present.

In the middle portion of the third convolution have been placed the centers for the movement of the head and eyes, and if a lesion irritates these centers conjugate deviation of the head and eyes, or of both, will result to the opposite side. If the lesion, however, destroys these parts, the head and eye will be directed to the side of the lesion. This is because of the unrestrained action of the muscles which are innervated by the opposite cortical centers. If the lesion involves the inferior posterior portion of the third frontal or Broca's convolution, motor aphasia will be present.

The frontal lobes are in direct connection with the opposite cerebellar lobe by the so-called frontocerebellar fibers. In a growth of the frontal lobe in which the tumor is largely subcortical, cerebellar symptoms may result, and it is difficult to differentiate the symptoms from those of a cerebellar lesion. This, however, is a very rare occurrence.

Another symptom which is sometimes supposed to be present is an abnormal tendency for poor jokes, or the "Witzelsucht" of the Germans. This, however, is of questionable value.

The symptoms, then, of a tumor in the frontal lobe are headache, localized mostly to the frontal bones, occasional nausea, vomiting or vertigo, and occasionally optic neuritis, which is mostly unilateral and confined to the side of the lesion. The special symptoms are loss of memory and change in disposition.

If the tumor is of large size and presses upon the adjoining motor areas, the above symptoms are accentuated, and there may be, in addition, motor symptoms which may be either of an irritative or a paralytic character, and which are confined to the limbs of the opposite side, conjugate deviation of the head or eyes or of both, and motor aphasia.

Tumors of the Motor Area.—Growths in this location are more common than in any other portion of the cerebrum, and are mostly sarcomata or gliomata. The symptoms will depend upon the location and extent of the lesion. If the growth is limited, for instance, to the center for movement of the upper limb, the symptoms will be referred to this part. If the lesion is of large extent, the symptoms, of course, will be referred to the related parts. Jacksonian or focal convulsions nearly always result from a cortical lesion. A tumor, for instance, involving the center for the upper limb will give Jacksonian convulsions beginning in this limb. If the growth extends downward, the movement will extend from the upper limb to the muscles of the head and

face. If the growth extends from the middle to the upper portion of the precentral lobe, the convulsions will extend from the arm to the lower limb. It must be remembered that a convulsion which is first Jacksonian may become general in character, and that in an epileptic convulsion there may sometimes be Jacksonian manifestations. If the tumor destroys the motor areas, paralysis in the related parts will result.

Lesions in the motor area are rarely only confined to the precentral convolution, and mostly also involve the postcentral convolutions or the sensory centers, in which case sensory symptoms will be present in addition to the motor. If the lesion is irritative, there will be pains of a Jacksonian type in the limbs of the opposite side; or if the lesion is destructive, disturbance of sensation or anesthesia in the related limbs on the opposite side will result.

If the tumor involves the frontal lobes, and especially the head and eye centers, conjugate deviation will result to the opposite side if the lesion is irritative, and to the same side if the lesion is destructive. If the tumor is on the left side of the brain in a right-handed individual, motor aphasia will also be present.

As a rule, tumors in the motor area give symptoms of great pressure, and headache, nausea, vomiting, and choked disc are present in most instances. Some of these symptoms may, of course, be absent, but, as a rule, headache and choked disc are present.

The symptoms, then, of a tumor confined to the motor area are headache, nausea, vomiting, choked disc, and Jacksonian convulsions on the opposite side, to be followed later by paralysis depending upon the extent of the lesion.

If the tumor invades the postcentral convolution, there are, in addition, sensory symptoms, such as pains and disturbances in sensation for touch and pain. If the growth invades the frontal lobes, there may be at first conjugate deviation of the head and eyes to the opposite side, and later to the same side. A lesion in the left side of the brain in a right-handed individual will also always give motor aphasia. The contrary is true in left-handed persons.

Tumors of the Sensory Area.—This includes the postcentral and superior and inferior parietal convolutions. Growths involving only this part are very rare, for in most instances the adjoining motor centers are also diseased. As has already been stated, most tumors of the motor area involve the postcentral convolution.

Isolated tumors involving either the superior or inferior parietal convolutions may rarely occur. In such case a lesion of the superior parietal convolution will give disturbance in the sense of localization, of position, of movement, of pressure, and ataxia in the lower limb, with inability to recognize objects placed against the sole of the foot.

A lesion involving the inferior parietal convolution will give the above symptoms in the upper limb instead of the lower. In addition, in both there may be headache, nausea, vomiting, and choked disc.

In most parietal lesions the adjoining postcentral convolution is involved, and there are, in addition to the symptoms already enumerated, disturbances in touch and pain. Very often in irritative lesions of the sensory areas there may be numbness and spasms of pain in the related limbs similar in character to the Jacksonian spasms, the result of motor irritation.

If the growth involves the adjoining occipital convolution, disturbance in vision will result. If the lesion is left-sided in a right-handed person and the angular gyrus is involved, there is, in addition, word and letter blindness, this causing inability to read or write voluntarily, or from dictation.

Tumors of the Occipital or Visual Area.—Growths in this area are not very common. They cause early pressure symptoms, as a rule, and disturbances of vision are among the first manifestations. There may be flashes of light or scintillating scotoma in the related visual fields, to be followed later by disturbance of vision, either for light or for colors, and lastly loss of half vision, or hemianopsia. Visual hallucinations are common, and usually occur on the side opposite the lesion, but may be on the same side. They may occur in the blind fields. Besides there may be a dissociation of the color sense; that is, while the patient may be able to recognize and match all colors, and the form of objects, he is unable to recognize the particular color of an object. If the lesion is right-sided, there will be left lateral homonymous hemianopsia, and vice versa. Occipital headache is always marked, as is also nausea, vomiting, and vertigo, and choked disc will come on early. This is because direct pressure is exerted upon the cerebellum. There may be, in addition, the symptoms of cerebellar incoördination.

If the growth involves the adjoining parietal or angular gyres, their related symptoms will occur.

Tumors of the Temporal Lobes.—Growths in this area are of rare occurrence, in most instances the adjoining parietal lobes being also involved. If the lesion is on the left side of the brain in right-handed individuals and the growth is confined to the temporal lobes, the symptoms will be those of pure sensory aphasia, *i. e.*, the patient will be able to talk, but he will have loss of memory for words as to their meaning and his speech will be unintelligible.

If the lesion is on the right side of the brain in right-handed individuals, no localizing symptoms will be present. This is the so-called "silent area" of the brain. There may, of course, be headache, nausea, vomiting, and choked disc. If the growth involves the adjoining parietal lobes, their related symptoms will occur.

Tumors of the Subcortex.—In the process of growth nearly every cortical tumor will become subcortical, so that practically every growth of the cerebral hemispheres will have symptoms the result of interruption of cortical fibers. The specific symptoms will depend upon what fibers are interrupted. The differential diagnosis between growths in the cortex and subcortex has already been referred to on page 1062. It is only necessary to add that subcortical growths nearly always are gliomata or sarcomata, that they are slow in growth, and that, as a rule, the first symptoms are those of intracranial pressure, with the symptoms depending upon whether the motor, sensory, or special fibers are involved.

Tumors of the Lateral, Third, and Fourth Ventricles.—Growths in these cavities are rare, and are relatively more frequent in the fourth than in the third or lateral ventricles. They may grow either from the ependymal walls of the ventricles—so-called ependymal glioma—or from the choroid plexus, and may be sarcomatous or glandular in nature. Cysticerci are common abroad, but not in this country. Tumors in these cavities may be secondary to growths of surrounding structures, or the growths in the ventricles may involve the surrounding tissue, but, as a rule, a ventricular tumor does not grow into the brain substance.

Tumors of the Fourth Ventricle.—The symptoms will depend largely upon the size of the growth and upon the pressure symptoms exerted on either the cerebellum, medulla oblongata, or pons. If the tumor is small, it may give no symptoms; but as it grows it may block up the normal flow of fluid, and thereby cause internal hydrocephalus, and give the usual pressure symptoms, as headache, nausea, vomiting, vertigo, and marked choked disc.

These general symptoms vary because the closure of the fourth ventricle may at times be incomplete. Generally speaking, the specific symptoms of tumors in the fourth ventricle are those of a lesion in the posterior cranial fossa; that is, there will be, besides the general symptoms, cerebellar incoördination, and if pressure is exerted upon the floor of the fourth ventricle, there may be involvement of the twelfth, and especially of the ninth and tenth nerves; and as a result of this, sudden death, because of respiratory or cardiac failure, is very frequent. Because of pressure upon the pons there may be paralysis of the seventh, and especially of the sixth nerves.

Tumors of the Third Ventricle.—It is extremely difficult to diagnose tumors in the third ventricle, and it is doubtful whether a small tumor in this area can ever be diagnosticated in life. If, however, the growth is large, besides the general symptoms of headache, nausea, vomiting, vertigo, and choked disc, which may or may not be excessive, depending upon the closure of the foramen of Monro and the general intracranial pressure, the specific symptoms will depend largely upon the direction of the growth. In most instances tumors of the third ventricle grow backward and extend into the structures surrounding the aqueduct of Sylvius involving the region of the oculomotor nuclei. If such be the case, besides oculomotor palsy, because of involvement of the posterior longitudinal bundle, there will be paresis or paralysis of associated ocular movement upward. For the same reason involvement of the superior cerebellar peduncle or the red nucleus will cause cerebellar incoördination. If the growth is very large, it may press upon the optic chiasm, and, besides causing excessive choked disc, it may produce paralysis of the sixth and third nerves. Lateral pressure upon the internal capsule may cause paretic symptoms either of one or both sides, and pressure upon the thalamus, disturbances in vasomotor and trophic functions on the side opposite the lesion, of mimetic expression, and sometimes involuntary howling, circulatory movements, or deviation of the body to the side opposite the lesion, disturbances of sensation, and pains upon the other side of the body.

Tumors of the Lateral Ventricles.—Growths in these areas are difficult to diagnosticate, because in most instances the symptoms will be those of pressure upon the internal capsule, which will cause hemiplegia on the opposite side. It has been thought that lesions of the lateral ventricles produce convulsions, but this is questionable. The general symptoms of brain tumor are here also very marked.

Tumors of the Crus or Cerebral Peduncles.—Growths limited to the cerebral peduncles are very uncommon, and in most cases are either extensions of tumors of the pons or of the third ventricle. The specific symptoms, if the lesion is unilateral, will consist of oculomotor palsy on the same side, with hemiplegic involvement on the other.

Tumors of the Pons.—Growths in this area are usually tubercular or gliomatous in nature, of slow growth, and usually occur in young adults. The symptoms will depend upon what fibers are involved, but as the pons is small, and as in most cases there is involvement of all of the structures, the specific symptoms will be those of involvement of the different cranial nerves in the pons, of the fifth, sixth, and seventh, and, because of disease of the posterior longitudinal bundle, there will result paralysis of associated ocular movement, and as the motor and sensory fibers are involved, their associated symptoms. There will, of course, be the usual general symptoms of headache, nausea, vomiting, vertigo, and choked disc. Cerebellar symptoms will be present if the growth involves either the superior, middle,

or inferior cerebellar peduncles. The symptomatology of lesions in the pons has been discussed on page 1031.

Tumors of the Medulla Oblongata.—These are uncommon and are usually gliomatous in nature. The specific symptoms will be those of involvement of the ninth, tenth, and eleventh cranial nerves. In such cases, however, there will be interference with cardiac and respiratory functions and death.

Tumors of the Cerebellum.—When considering the relative size of the cerebrum and cerebellum, it is probable that tumors are more frequent in the latter. Growths in the posterior cranial fossa may involve either the substance of the cerebellum or the surrounding structures, the latter giving the symptoms of cerebellar disease because of pressure or involvement of this organ. It is also necessary to consider growths which occur in the cerebrum, but which, because of pressure, give symptoms of cerebellar disease.

The general symptoms of tumors of the cerebellum are headache, nausea, vomiting, vertigo, choked disc, and incoördination.

Headache, as a rule, is present, and is more severe in lesions of the cerebellum itself than in extracerebellar lesions, and is generally localized to the back part of the head and neck. Sometimes the pain is so severe as to cause retraction of the former. Occasionally no headache is present.

Nausea and *vomiting* are, as a rule, present early, and are more intense in intracerebellar lesions.

Vertigo is present nearly always, and is one of the prominent symptoms. It may consist in a feeling of dizziness in which objects may swim before the eyes and the patient feels as if he were losing consciousness, or in a feeling of rotation of objects before the eyes or of rotation of self. Vertigo, as a rule, is more marked in extracerebellar lesions, and is probably dependent upon involvement of the vestibular branch of the eighth nerve. It is the opinion of some that when there is a sensation of rotation of objects before the eyes, whether the lesion be intracerebellar or extracerebellar, it is always from the diseased to the healthy side. When there is a sensation of rotation of self, the direction is the same in intracerebellar lesions, but opposite in extracerebellar lesions. This symptom, however, is by no means certain.

Occasionally a sense of dizziness is obtained when the eyes are deviated to one side, generally to the side of the lesion, but there is no dizziness when the head is deviated. In such case the vertigo may be due to a weakness of one of the ocular muscles, and is not a true cerebellar vertigo.

Choked disc is one of the early and most constant symptoms of lesions in the posterior cranial fossa. As a rule, it comes on earlier and is more marked than in lesions of the cerebrum. It may be greater on the side of the lesion. Sometimes choked disc comes on after the appearance of other cerebellar symptoms, and when it does so, its development is usually very rapid. Tumors of the substance of the cerebellum itself usually give a greater choked disc because of the direct pressure exerted upon the fourth ventricle.

Incoördination results from a lesion in any portion of the cerebellum or its connections. As has already been stated, it is probable that the cerebellum is concerned with the coördination of every voluntary movement, and therefore whatever symptoms are produced are dependent upon this.

A lesion in the middle portion or the vermis will produce the greatest amount of incoördination, this being apparent on either side of the body, whereas lesions involving only a lateral lobe will produce a preponderance

of symptoms on the side of the lesion. Tumors outside of the cerebellum will produce mostly unilateral symptoms unless the middle lobe or the vermis is involved, in which case bilateral ataxic symptoms will be present.

The incoördination of cerebellar disease is manifested only when an effort is made, and is not dependent upon peripheral symptoms, *i. e.*, there is never disturbance of sensation and no involvement of muscular sense. This incoördination becomes apparent in the gait, station, position of the head and limbs, movements of the eyeballs, head and limbs, and in talking, eating, and swallowing. These will be taken up in order.

When considering the ataxia present in cerebellar diseases, it is necessary to consider also the possible influence of the *weakness and the atonia* which sometimes result from lesions of the cerebellum. This question is by no means settled, but there is no doubt that in lesions of the vermis itself there may be paresis or weakness in the muscles of the limbs, and especially those of the trunk, and in lateral lobe lesions weakness has been found in the limbs and trunk muscles of the same side. This can be readily seen after operations upon the cerebellum in which this organ has been injured. The weakness is not prominent and is not always present. It is also characteristic of cerebellar disease that the limbs, especially on the side of the lesion, lose their accustomed tone and are rather flaccid. This symptom is also by no means constant, and is present especially in lesions of the vermis.

The *gait* in cerebellar diseases resembles that observed in a drunken person. The patient will make a few steps and then will totter or lurch to one side or the other, or backward or forward, and, recovering, will repeat this. In lesions of the vermis this is most marked, but in lateral lobe, and in extracerebellar lesions in which the former is pressed upon, it will not be so prominent. Generally the patient will have a tendency to walk to one side, usually to the side of the tumor, and will occasionally have a tendency to fall to this side. If such a patient's gait were not corrected, he would tend to walk in a circle, the center of the circle being the side of the tumor. The patient is generally aware of this tendency to walk to one side, and in his effort to correct this will sometimes walk to the opposite side.

As a rule, the closure of the eyes will not tend to increase the incoördination if the lesion is in the vermis, but sometimes in lateral lobe and extracerebellar lesions the gait is distinctly made worse when the eyes are closed. If the motor columns are pressed upon, as is not infrequent in extracerebellar lesions, there is added a spastic condition on the side opposite the tumor. A bilateral spastic condition is also often present when there is a complicating internal hydrocephalus. This spasticity to a certain extent will modify the incoördinate gait.

The *station* and *attitude* of a patient with cerebellar disease depend largely upon the position of the growth. In lesions of the vermis itself there may be retraction of the head and extension of the lower limbs with flexion of the upper. There may also be lordosis in the lower portion of the spinal column. It has been supposed that the attitude and position of the trunk and head are considerably modified by the weakness which is supposed to be present in the erector spinæ and other trunk muscles. This is questionable, for the alternate contraction of these muscles is probably only an effort to keep the parts above in their proper position and is only a part of the general incoördination. Sometimes in tumors of the cerebellum the head is held in certain positions in such a way so that the growth would avoid pressing directly upon the vermis. In tumors, for instance, of the left lateral lobe, the patient will be inclined to lie on his left side, for when

he lies on the right pressure may be exerted upon the vermis. This symptom, however, is not by any means constant. Very often also patients with cerebellar tumors will hold their heads in abnormal positions, not because of the possible influence the change of position would have upon the vertigo and dizziness, but because they see double, and by holding their heads in certain positions they are able to avoid this.

If the patient is placed with his feet together, he will have a tendency to fall, generally to the side of the lesion. As a rule, if the eyes are closed the ataxia will be increased, and this is especially so in extracerebellar lesions.

The incoördination or ataxia which is present in the limbs is of two types, *i. e.*, it may be made worse with the eyes shut, or this may have no influence upon it. This ataxia is dependent upon the lack of coördination in the muscular contractions, and is not dependent upon any sensory disturbances. As a rule, it is greatest on the side of the lesion, but it may also be observed on the opposite side. If the upper limb on the side of the tumor is moved in any direction, for instance, as in supination and pronation, it will be found that the movement will not be as well or as rapidly performed as upon the other side. The same thing is true if the lower limb is moved. These symptoms are dependent upon the lack of coördinate contraction of the muscles concerned in the movements.

Incoördination in the movement of the eyeballs, or nystagmus, is present nearly always in lesions of the cerebellum. This incoördination of the eyeballs is similar to that observed in any other movement, and is present only when the eyeballs are moved, and is greater when they are directed to the side of the lesion. This nystagmus may consist in to and fro jerkings, and is greater in lateral deviation.

Incoördination of the muscles which are concerned in talking, eating, and swallowing sometimes occurs in lesions of the cerebellum and its connections. This, however, is not a very common occurrence. Disturbance in these functions dependent upon the incoördination of the muscles concerned must be differentiated from the difficulty observed in these functions when an extracerebellar tumor presses upon the cranial nerves innervating the muscles necessary to perform these acts.

Cranial Nerve Symptoms.—The cranial nerves, as a rule, are not involved in lesions of the middle lobe of the cerebellum. In tumors of the lateral lobe it is possible to have involvement of the fifth, sixth, seventh, and eighth cranial nerves on the same side, but, as a rule, such cranial nerve involvement indicates a tumor in the angle between the pons and cerebellum or the cerebello-pontile angle.

The *first* or *olfactory nerve* is hardly ever diseased. The same is true so far as the *third* and *fourth cranial nerves* are concerned.

The *fifth cranial nerve* may sometimes be involved, especially in extracerebellar lesions. Very rarely a tumor may grow from this nerve.

Unilateral involvement of the *sixth nerve* is a very common symptom in extracerebellar lesions. Bilateral sixth nerve paralysis may sometimes be present in unilateral lesions, but, as a rule, this indicates a tumor in the middle lobe.

The *seventh nerve* is nearly always involved in tumors of the cerebello-pontile angle, and a fibroma may grow from this nerve. A lateral cerebellar tumor may sometimes cause involvement of this nerve by pressure.

Tumors of the cerebello-pontile angle, as a rule, grow from the *eighth nerve*, and are generally fibromata. At first there may be such subjective symptoms as roaring, hissing, or buzzing noises in the ear, and later com-

plete nerve deafness. This nerve may also sometimes be involved by pressure from a growth in the lateral lobe of the cerebellum.

The *ninth, tenth, eleventh,* and *twelfth nerves* may be involved in extracerebellar lesions, this resulting from pressure, thus causing difficulty in talking, eating, and swallowing. Bilateral involvement is uncommon, and, as a rule, indicates lesions in the medulla itself.

Pupillary Symptoms.—Tumors of the cerebellum probably have no direct effect upon the condition of the pupils, alterations in them probably depending upon the presence of optic neuritis or choked disc.

Motor Symptoms.—The weakness or paresis which is sometimes present in cerebellar lesions has already been discussed, and is not dependent upon pressure on the motor columns. An extracerebellar tumor, as a rule, compresses the motor fibers of the pons, and this causes the spastic condition on the side opposite, with the consequent weakness, increased reflexes, and the presence of the Babinski phenomenon. In complicating internal hydrocephalus this condition may be bilateral.

As a rule, lesions of the cerebellum have no influence upon the state of the reflexes, for they may be increased, diminished, lost, or in normal condition.

Convulsions.—Convulsions sometimes occur in the course of cerebellar disease. These may be general or limited to certain parts. If general, as sometimes occurs in lesions limited to the vermis, there is retraction of the head, extension of the lower limbs, and flexion of the upper, and the whole body is held in tonic contracture.

Tumors which involve the seventh nerve may cause tremors in its distribution, and sometimes convulsions which are limited to this nerve and are focal or Jacksonian in character.

Instead of this there may occur irregular fainting spells, during which time the patient feels giddy and has a tendency to fall. These are not really convulsions, and are dependent upon the vertigo common in this disease.

In diagnosing, then, tumors of the posterior cranial fossa it is necessary to consider whether the growth is limited to the cerebellum or whether it is extracerebellar. Not only that, but it is necessary, when limited to the cerebellum, to recognize, if possible, whether the tumor is localized to the center or to the lateral lobe.

Summarizing the symptoms of a tumor in the vermis, we have as follows: headache in the back of the neck, excessive nausea and vomiting, intense vertigo, bilateral early and marked choked disc; marked incoördination in every movement of the body, whether in the limbs, trunk, movements of the eyeballs, and sometimes in articulation, in eating, and in swallowing; sometimes weakness in the limbs and the muscles of the trunk, with atonia, an ataxic gait, poor station, and rarely so-called cerebellar fits, during which time the head is retracted, the legs extended, and the arms flexed—all in tonic contracture.

Tumors of the lateral lobe of the cerebellum give headache, nausea, vomiting, intense vertigo, bilateral early and marked choked disc, which may be greater on one side, incoördination in all movements of the limbs, but which is greater on the side of the lesion, a staggering gait with a tendency to lurch to the side of the lesion, nystagmus, more marked in looking to the side of the lesion, sometimes paresis and atonia in the limbs of the same side, and, if the tumor is large, it may press upon the cranial nerves on the same side.

Extracerebellar lesions may be either in the angle between the pons

and the medulla, *i. e.*, the so-called cerebello-pontile angle, or may grow from the occipital or temporal bone primarily, and secondarily involve the structures in the cerebello-pontile angle and the cerebellum itself.

Tumors of the cerebello-pontile angle are usually fibromata, and grow from the eighth, seventh, fifth, and sixth nerves in order of frequency, and the first symptom will depend upon what nerve is involved. If the growth is on the eighth nerve, there is first a roaring, buzzing, or hissing noise on the side of the tumor, to be followed by deafness, and then the symptoms of paralysis of the seventh and sixth nerves as these nerves are pressed upon, and more rarely of the fifth nerve. There are, besides the general symptoms, headache, nausea, vomiting, vertigo, which may be excessive if the eighth nerve is diseased, and choked disc, which, as a rule, is greater on the side of the tumor. When the cerebellum is pressed upon, there are, in addition, incoördinate symptoms in the limbs, greater on the side of the lesion, paresis and atonia, only rarely on the side of the tumor, a staggering and incoördinate gait to the side of the tumor, and less frequently nystagmus, which is greater when the eyes are deviated to the affected side. If the tumor grows from the seventh nerve, spasms in its distribution may be observed. As a rule, the growth will press upon the motor fibers of the pons, giving, in addition, weakness and spasticity, with increase of reflexes in the limbs of the opposite side.

If the tumor grows from the dura covering the occipital or temporal bones, the symptoms may be a little more diffuse, and may give not only the symptoms above enumerated in tumors of the cerebello-pontile angle, but, in addition, there may be involvement of some of the cranial nerves on the same and opposite side.

Sometimes *diffuse syphilitic lesions* in various portions of the brain or a pial infiltration at the base of the brain may give the symptoms of a cerebellar tumor to such an extent that it is almost impossible to make a differential diagnosis. There may be present all of the general symptoms of a cerebellar lesion, but there will be, in addition, almost always a greater involvement of the cranial nerves, such as that of the third—a very unusual condition in pure cerebellar or extracerebellar lesions. Multiple sarcomatous tumors may also give the symptoms of a tumor in the cerebellum or of the angle, and in such instances it is almost impossible to differentiate the symptoms from those resulting from basal syphilis.

ABSCESS IN THE BRAIN.

Chronic otitis media is the most frequent cause of abscess in the brain. It may be due to such other causes as traumatism, or may be a part of a general process, or it may follow an abscess in the lung. When it is the result of middle-ear disease, the abscess is generally localized to the temporal lobe or to the cerebellum on the same side, but it may cause an abscess in the parietal or other lobes. This, however, is uncommon. Pus may be transmitted along the facial and acoustic nerves from the middle ear and cause an extradural abscess. Occasionally an abscess in the pia will result.

In most instances a localized abscess is only a part of a general purulent cerebrospinal meningitis. Sometimes a localized pus cavity may rupture and cause a general purulent meningitis. The abscess is usually surrounded by a thick wall and considerable inflammation surrounds it. The pus itself is very thick and contains the usual microörganisms.

Symptoms.—The localizing symptoms of an abscess in the brain are

similar to those of any other lesion or growth. Because of the fact that most abscesses occur as a complication of middle-ear disease or extension of such inflammation, most pus cavities or abscesses are to be found either in the temporal area, in the cerebello-pontile angle, or in the cerebellum, or, what often happens, besides a lesion either in the temporal or cerebellar areas there may also be a meningitis, with its accompanying symptoms. Specifically it cannot be said that there are any general symptoms which indicate an abscess in the brain. The inference is that if there is a history of, or if there is an abscess in the middle ear, and if following it there are symptoms which are referred to either the temporal or cerebellar areas or the meninges, the lesion is purulent and secondary to middle-ear disease. There may be, as is usually the case in any growth, headache, nausea, vomiting, vertigo, and sometimes choked disc, these depending upon the extent of the lesion and the pressure exerted in the cranial cavity. There may or may not be changes in the temperature, such as result from pus elsewhere. The other symptoms will depend upon the location of the lesion, whether temporal or cerebellar.

INJURIES TO THE BRAIN.

The general symptoms of injuries to the brain will depend largely upon the character and extent of the injury. It is possible to have a fracture of the skull without any injury to the brain tissues, or a severe injury to the brain without involvement of the enveloping bones. It is impossible in any given case to foretell what the results of an injury may be, but an effort is made here to classify the symptoms which may occur. It should be understood, however, that this classification cannot be, and is not intended to be, a definite one, for any and all symptoms may occur in any given case.

Classification of Injuries.—There may be (1) fracture of the vault of the skull, with or without injury of the brain; (2) fracture of the base of the skull with or without injury of the brain; (3) hemorrhages from the vessels of the meninges, either extradural or intradural, either with or without involvement of the brain itself; (4) injuries to the brain, which may consist, first, of large hemorrhages which are either single or multiple; second, of multiple small hemorrhages which cannot be seen except under the microscope; and, lastly, so-called contusion of the brain; (5) injuries of some of the cranial nerves without any other involvement; and (6) the functional neuroses.

General Symptoms.—Inasmuch as certain general symptoms occur no matter what the injury, these will be first discussed. As a rule, if the injury is severe enough there will be impairment of consciousness. If this is complete, so that the patient cannot be aroused, it is called a *coma*. If the patient can be aroused so that questions can be answered, it is called a *stupor*, whereas an expression of wandering ideas accompanied by stupor is called a *delirium*.

In most instances the period of unconsciousness will not last long and the patient will rally within a few hours, but sometimes the stupor may persist for a number of days and even longer. It is possible for the patient to regain consciousness and then to again lapse into a period of stupor. As a rule, if the patient rallies within a few or less than twenty-four hours, the prognosis is good, whereas stupor lasting for more than a day will make the prognosis very grave. It is necessary in a great many instances to diagnose

such a comatose condition from those arising in alcoholism, uremia, diabetes, and hysteria. There should, however, not be much difficulty in making a differential diagnosis if the underlying causes are considered.

Certain general symptoms may always be present whenever considerable *compression* of the brain, no matter from what cause, exists. This, of course, can only be apparent after the patient has rallied from whatever mental condition the injury has placed him in. These are headache, which may be localized to the point of injury or may be diffuse, nausea, vomiting, sometimes vertigo, choked disc, stertorous respiration, and slow pulse. These symptoms occur only when there is great compression, such as occurs from hemorrhages or depressed fractures.

The *physical evidences of injury*, such as contusion and laceration of the scalp, swelling of the injured tissues, a subconjunctival and subcutaneous ecchymosis, and the escape of cerebrospinal fluid from the ear or nose, may be present. It is necessary, however, to remember that severe hemorrhages or destruction may occur within the cranial cavity without the slightest external evidence of injury.

Fracture of the Vault of the Skull.—Cause and Symptoms.—This, as a rule, results from direct injury, such as are caused by stab, sword, and bullet wounds or blows upon the head. The fracture may be at the point of injury, or the effects of this may be so diffuse that the fracture is on the other side of the skull, or at times at the base. There may be in all cases a visual point of injury, such as contusion of the scalp, and if there is a fracture, the accompanying depression, which can be felt unless the swelling of the tissues is too great. In most instances the fracture will be depressed and will injure the meninges and the brain tissue underneath. In all instances an incision should be made for the purpose of diagnosis. The general symptoms will be impairment or loss of consciousness, depending upon the force and extent of the injury, and the focal symptoms will depend upon what part of the brain is injured. If the frontal lobe is injured, there will be no focalizing symptoms; if the motor part, Jacksonian convulsions or paralysis on the other side of the body; if Broca's convolution, motor aphasia if the patient is right-handed and the injury is on the left side of the brain; if the temporal convolutions, sensory aphasia under the same conditions; if the parietal areas, sensory symptoms on the other side of the body; and if the occipital lobes are involved, hemianopsia on the other side.

Very often a direct injury to the brain will cause no apparent contusion at the point of insult, and even though a fracture is present it may be of such character as not to cause depression. It is hardly possible, however, for a fracture to be present without some symptoms, for if the injury is severe enough to cause a solution of continuity in the bone, it is severe enough to cause an injury of the meninges, this resulting in a laceration of some of its vessels. It is, then, from the focal symptoms caused by the resulting hemorrhage that we are able to make a diagnosis, the symptoms depending upon the part of the brain which is compressed.

Sometimes if the blow is severe enough, or even if the injury is very slight, there may result an accompanying fracture of the base, the symptoms of which will be discussed later. Again, it must be remembered that if an injury is severe enough to cause a fracture, it will also cause a severe contusion of the brain itself, this resulting in multiple areas of small hemorrhage, which later on may become absorbed and no symptoms remain, or this may be replaced by connective tissue. The occurrence of these multiple

areas of small hemorrhage will be largely influenced by the state of the blood-vessels in the given individual, for if there is present an arteriosclerosis, such weakening of the blood-vessels may result as to cause a secondary hemorrhage into the brain substance.

Fracture of the Base of the Skull.—Cause.—It is impossible to tell just when and what kind of injury will produce a fracture of the base of the skull. It may result from a fall on the back or buttock or from a blow upon the head. Whenever there results such a fracture, there will be, as a rule, severe injury to the brain, or there may be an accompanying fracture of the vault. In about two-thirds of the cases there will be loss of consciousness, from which the patient may rally in a few or less than twenty-four hours, although it is possible to have a stupor lasting a week or longer with full recovery of the patient, and in those cases in which no unconsciousness results there may be a momentary stupor.

Symptoms.—Accompanying the stupor there may be the physical evidences of injury, such as bleeding or the escape of cerebrospinal fluid from the nose, throat, or ears, rupture of the membranes of the ear, and subconjunctival or subcutaneous ecchymosis back of the ear. There may also be stertorous respiration, a slow, weak pulse, and either a dilatation or contraction of the pupils. Of these, the most important symptom is the condition of respiration, and especially of the pulse. The pupillary symptoms may be absolutely disregarded, for while it is held by a great many that a dilatation of the pupils will always result on the side of the injury, this is probably fallacious.

It is upon the focal symptoms that the diagnosis of fracture of the base must be made. This will depend upon the line of fracture and upon the possible existence of a hemorrhage. In nearly all cases some of the cranial nerves will be involved, and of these, the optic and the sixth, seventh, and eighth cranial nerves are most commonly the seat of injury.

The *first or olfactory nerve* is frequently involved from a fracture in any portion of the skull, probably because of injury to the ethmoid, unilateral or bilateral loss of smell and impairment of taste resulting.

The *second or optic nerve* is very frequently diseased, either on one or both sides. This may be either because of a hemorrhage in or about the optic chiasm, or, what is more frequently the case, because of fracture through the optic foramen. The impairment of sight will depend upon whether one or both optic nerves are diseased and upon the part of the nerve which is injured. Very frequently there will be neither fracture through the optic foramen nor hemorrhage involving the optic nerve, but a momentary pinching of the nerves. Whether this causes a hemorrhage into the sheath or into the nerve itself, or whether it causes a destruction of fibers with a consequent atrophy, is not known; but the fact remains that such pinching will in many cases result in a diminution and sometimes total loss of vision. In rare instances it is possible to have such an injury of the optic nerve with consequent optic atrophy, without the accompaniment of any other symptom, and more rarely still this impairment of vision may be in the form of irregular hemianopsia.

The *third or oculomotor nerve* is rarely involved, and occurs especially when there is a fracture through the orbit and the middle cranial fossa. It may be unilateral or bilateral. In most instances only part of the distribution of the oculomotor nerve is paralyzed, this resulting in drooping of the upper lid, or possibly a weakness of some of the ocular muscles.

The *fourth nerve* is only rarely diseased in injuries of the brain. The

fifth nerve is sometimes involved in fracture of the middle cranial fossa, but its occurrence is also rare.

The *sixth, seventh,* and *eighth nerves* are probably more frequently involved in fractures of the base than the other cranial nerves, and in most instances together. This is because the exits of these nerves at the base are so close together.

Very rarely the *ninth, tenth, eleventh,* and *twelfth cranial nerves* are diseased, this causing difficulty in eating, talking, and swallowing, and irregularity of the pulse and respiration. These are only present in severe cases, which nearly always result fatally.

It is characteristic of these cranial nerve palsies that they are not of permanent duration, for in most instances, if the patient lives, a partial and sometimes total recovery may be expected.

Sometimes there results in fracture of the base of the skull hemorrhage from one of the basal arteries. The symptoms of this will depend entirely upon the place of hemorrhage and upon the structures compressed. In most instances the hemorrhage is in or about the optic chiasm, this causing paralysis of the ocular muscles and impairment of vision, and if the hemorrhage is large enough, the general symptoms of compression, such as headache, nausea, vomiting, and choked disc.

Summarizing, then, the symptoms of fracture of the base of the skull, there may be either coma, stupor, or delirium, which may last from a few to twenty-four hours or a number of days, and from which the patient may or may not rally, stertorous respiration, slow, irregular pulse, bleeding from the nose, throat, or ear, sometimes the escape of cerebrospinal fluid, subconjunctival or subcutaneous ecchymosis, and paralysis of some of the cranial nerves, with irregular pupils. If there is an accompanying hemorrhage into the substance of the brain, the symptoms will depend upon whether the motor, sensory, or special fibers are involved; if there is a fracture of the vault, the additional symptoms of this.

The **prognosis** will depend upon the extent of the cranial nerve involvement, whether or not there are hemorrhages in the brain substance, and upon the stupor and the state of the respiration and pulse. The prognosis is always best where the patient rallies within a few or twenty-four hours, and the state of the pulse is the best indication of the results to be expected.

Injuries of the Meninges.—Causes and Symptoms.—Under this head will be considered traumatic diseases of the dura and the pia-arachnoid and rupture of its vessels. In most cases of fracture of the skull there will result some injury of the underlying meninges. This may be a contusion or a laceration of the dura, which may be followed in time by adhesions, the whole giving the picture of an external pachymeningitis. If the internal surface of the dura is involved, there will be adhesions to the pia-arachnoid and the brain itself. The symptoms of external pachymeningitis will depend upon the extent and location of the lesion, the focal symptoms depending upon the part of the brain involved. In all cases there should be some headache localized to the diseased part.

More commonly, however, as a result of injuries to the brain there may be an inflammation of the pia-arachnoid which may involve not only the injured parts, but the meninges of the whole brain and cord, giving the symptoms of a cerebrospinal meningitis. One of the most frequent causes of this is infection through the wound. The symptoms will depend upon the severity of the disease. If the meningitis is of septic character, there will be fever, sweats, chills, coma, stupor or delirium, retraction and rigidity of

the head, stiff neck and back, rigidity of the extremities, various cranial nerve palsies, sometimes choked disc, occasional convulsions, either focal or general in character, and paralysis of the limbs which may be hemiplegic in type. Lumbar puncture will always demonstrate the presence of pus and various pyogenic bacteria.

Sometimes an abscess of the brain will follow a septic injury, or it may occur in the course of a purulent meningitis. The symptoms of the former have been sufficiently dealt with under the head of tumors of the brain, while in the latter instance the additional symptoms will depend upon the focal lesions resulting from the location of the abscess.

Meningeal Hemorrhages.—Rupture of the blood-vessels of the meninges is one of the commonest results of injuries of the head, and of these the middle meningeal artery is usually involved. It contains three branches, and, as a rule, an injury will produce laceration only of the central or the principal branch, the focalizing symptoms of which will be convulsions, Jacksonian in character, of the other side of the body, accompanied by paralysis of the hemiplegic type, and if the lesion is on the left side in a right-handed person motor aphasia.

The anterior portion of the middle meningeal artery supplies the frontal convolutions, and in a rupture of this vessel there will be motor aphasia if the lesion is on the left side of the brain in right-handed persons, with stupor, and no focalizing symptoms unless the motor cortex is also involved. The posterior branch of the middle meningeal artery supplies the occipital and parietal convolutions, and hemorrhage of this part will produce hemianopsia plus sensory symptoms on the opposite side of the body. In all these instances there will be the symptoms of the accompanying shock of the hemorrhage, as stupor or coma, irregular pupils, stertorous respiration, and slow pulse. There may or may not be an accompanying fracture of the skull.

Injuries to the Brain Substance.—The brain may be severely injured without any external evidence of fracture of the skull. The occurrence of hemorrhage in the brain tissue in conjunction with the latter condition has already been discussed. There may result in any injury to the skull either one hemorrhage or multiple hemorrhages of large size, or, what very frequently occurs, multiple small hemorrhages which can only be detected under the microscope. As a rule, whenever an injury is severe enough to cause a hemorrhage into the brain substance there will be multiple hemorrhages throughout the brain, and the symptoms will depend largely upon the greatest point of hemorrhage, this in most cases involving the motor fibers. There will be either total or partial unconsciousness or stupor, convulsions, and hemiplegia of one side, with conjugate deviation of the head and eyes and sometimes paralysis on both sides of the body. In such cases the prognosis is almost always hopeless.

Whenever there results multiple microscopic areas of hemorrhage, the symptoms present will be those of cerebral contusion, the patient being in a mentally irritable condition, complaining of diffuse headache, dizziness, inability to concentrate, lack of energy, and a general nervousness. In such cases the prognosis in the young is excellent, for these multiple small areas of hemorrhage will practically almost always disappear leaving no symptoms; but if they occur in elderly persons, they may be the starting cause of a slow hemorrhage into the brain tissue.

By *contusion* of the brain is meant that condition which results from a shaking up of the cranial contents. There is usually a dazing or a confusion

of the intellect which may be momentary or last from a few minutes to an hour, and from which the patient recovers, the symptoms being entirely of a mental character. As a matter of fact, it is really the condition described in the previous paragraph as resulting from multiple microscopic areas of hemorrhage or softening.

Terminal Effects of Injuries to the Brain.—These will depend largely upon the character of the injury and its effects and the benefit of whatever therapeutic measures have been employed. Injuries to the skull such as those which involve the meninges and brain are among the most frequent causes of traumatic epilepsy. If the injury is over the motor area, Jacksonian convulsions may result, but very often injury anywhere in the brain, especially if this occurs in the young, may be followed by general or idiopathic epilepsy. Such other effects as hemiplegia or diplegia and impairment of vision and sensation need no further discussion.

The mental symptoms are by far the most important. Very often a trivial injury will cause a change in the disposition of the individual and produce more or less irregular headache, dizziness, lack of attention to business details, with the addition of many functional symptoms which will be discussed later.

It is also a mooted question whether injuries to the brain can produce insanity. It is probable that in very rare instances injury may cause the earlier appearance of insanity where there has been a predisposition for it, but it is hardly possible that direct injury to the brain may cause insanity. There is no denying, however, that mental impairment is not an infrequent occurrence.

CHRONIC BULBAR PALSY (Glosso-Labio-Laryngeal Paralysis).

Definition.—A disease of the motor cranial nerve nuclei of the medulla and pons, usually involving the fifth to the twelfth inclusive, and characterized by progressive weakness, atrophy, and fibrillary tremors in their distribution, with progressive difficulty in talking, eating, and swallowing.

The pathology is similar to that of chronic poliomyelitis, and consists in a progressive degeneration of the motor cranial nuclei. The disease is slow in its onset, and usually begins in the nuclei of the twelfth nerve, gradually involving the other motor nuclei, and only rarely those concerned with the movements of the eyes. Sometimes this degeneration occurs at the end of an amyotrophic lateral sclerosis, or it may be the starting-point of such disease.

Predisposing and Exciting Factors.—The disease occurs in the latter end of life, and is probably the result of an early death of the parts concerned. It may be a manifestation of a lack of resistance or of maldevelopment. Usually the disease starts without an exciting cause.

Symptoms.—Because of the fact that the nuclei of the twelfth, eleventh, and the motor parts of the tenth and ninth cranial nerves are first diseased, the early manifestations nearly always consist in a slowly increasing difficulty in pronunciation of certain words, especially those in which action of the tongue and lips are prominent, as R, L, G, B, P. At the same time or soon after there will be some difficulty in swallowing, and there may be very early regurgitation of food, and eating becomes slow. Speech becomes more and more difficult, typical bulbar speech being slow, nasal in type, monotonous, indistinct, and hard to understand. Soon after there will be difficulty in chewing, and eating of meat will become almost im-

possible, the patient living nearly always on soft or milk diet. Choking spells are very common, and may come on with the slightest form of irritation of the pharyngeal muscles or independently of the swallowing of food. Coincident with the above symptoms weakness and atrophy will develop first in the tongue, its surface becoming furrowed and irregular, and fibrillary tremors will be prominent. The weakness of the tongue gradually increases until it will be impossible to move it even from side to side. The lips become thin and droop, and with the atrophy of the cheeks produce an expressionless countenance, the so-called bulbar face. Dribbling of saliva is a common symptom, and is probably caused by the inability of the facial and orbicular muscles to retain the secretion. The palatal, pharyngeal, and laryngeal muscles are next involved, and their reflexes are lost early. If the disease progresses, there may be at the very last involvement of the ocular nuclei, causing inability to move the eyes in any direction; but, as a rule, the disease terminates before this, the patient usually choking to death. The mentality is hardly ever involved, although the patient becomes somewhat weak-minded. Sensory symptoms are never present (Fig. 399).

FIG. 399.—CHRONIC BULBAR PALSY IN A CHINAMAN, SHOWING TYPIC FACIES, LACK OF EXPRESSION, AND DROOPING LIPS.

Summary of Diagnosis.—A person past thirty with gradually increasing difficulty in articulation, this terminating in a slow, thick, monotonous, indistinct speech; increasing difficulty in swallowing and chewing, with choking spells, dribbling of saliva, weakness, atrophy, and fibrillary tremors in the facial muscles, lips, and tongue, and absence of the palatal, pharyngeal, and laryngeal reflexes.

Differential Diagnosis.—There should be no difficulty in diagnosing this disease. Occasionally, however, besides the symptoms above enumerated there may be weakness, spasticity, increased reflexes, with atrophy and fibrillary tremors in the muscles of the upper and lower limbs, such as occur in amyotrophic lateral sclerosis. Again, bulbar palsy may occur at the end of an amyotrophic lateral sclerosis. This subject has been more fully discussed on page 1090.

In pseudobulbar palsy besides the difficulty in eating, talking, and swallowing, there is always a history of preceding attacks of hemiplegia occurring on one and then the other side, with the accompanying symptoms, and, most important of all, there are no fibrillary tremors or atrophy in the face, tongue, and lips.

Acute Bulbar Palsy.—Sometimes as a result either of a thrombosis of one of the bulbar vessels or a hemorrhage there may be an acute involvement of the nuclei of the medulla. Thrombosis of this area nearly always involves the *inferior cerebellar artery* of one side. The symptoms may come on acutely, with or without unconsciousness, and there will be difficulty in eating, talking, and swallowing, which subsides somewhat in a few days, and there are usually, in addition, motor and sensory symptoms, gener-

ally referred to the side opposite the lesion, and unilateral cranial nerve paralysis.

Sometimes as the result of or during the course of infectious diseases, alcoholism, or ptomain poisoning, there may occur areas of hemorrhage or inflammation in the medulla or pons. These have already been discussed under the head of superior and inferior polioencephalitis on page 1038.

Myasthenia Gravis (Asthenic Bulbar Palsy).—By this is understood a disease which is characterized by rapid fatigue and exhaustion in certain muscles. It may be limited to movement of the limbs or may be referred to the distribution of the motor cranial nuclei, especially those concerned with the movement of the eyeballs. It usually occurs in young adults without any apparent cause. When the patient rises in the morning or on first effort there may be no apparent weakness, but gradually, if the symptoms are limited to the cranial nerves, there will be drooping of the upper lids, with

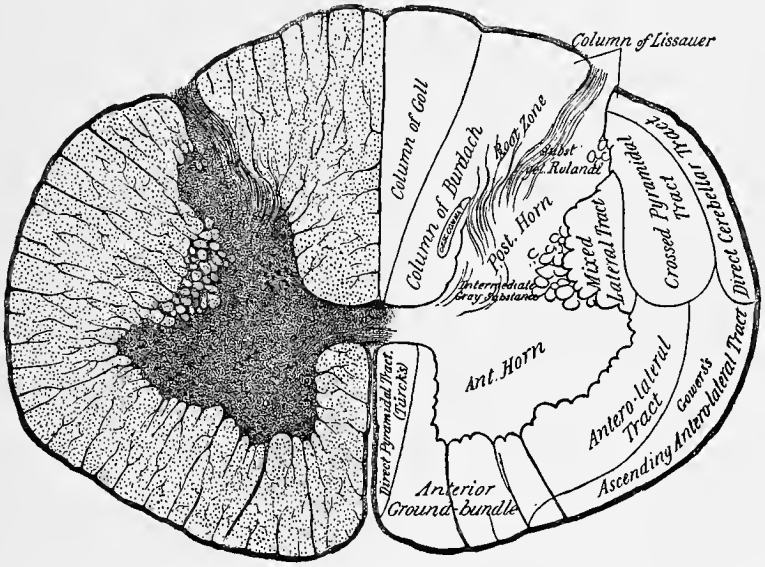


Fig. 400.—Cross-section of Cervical Spinal Cord, showing its Anatomic Subdivisions (Schaefer).

closure of the eyes, or weakness in the muscles of the face. Sometimes there may be difficulty in talking and in eating and swallowing. If the patient lies quietly, there may be a temporary recovery of function with weakness again as effort is made. When these symptoms are referred to the limbs, the movement may at first be normal, the patient gradually tiring. It has also been found that the electric reactions to the faradic current, which are normal at first, soon become diminished—the so-called myasthenic reaction. The prognosis in most cases is not very good. Pathologically a disease of the thymus gland has been found in some cases. The motor cranial nuclei are not diseased.

Clinical Course and Complications.—The disease hardly ever lasts more than two or three years, the symptoms gradually increasing, the patient usually choking to death.

Diseases of the Spinal Cord.

The spinal cord is situated in the spinal canal, and extends from the lower portion of the medulla oblongata to a point opposite the upper border of the second lumbar vertebra. It consists of eight cervical, twelve thoracic, five lumbar, and five sacral segments. The cord is composed of gray and white matter, the former being in the center and surrounded by the white matter. The gray matter is divided equally on both sides of the spinal cord and is connected by a commissure and consists of an anterior and a posterior horn. It is composed of nerve-cells and their dendritic processes, axis-cylinders, nerve-fibers, and neurogliar tissue which holds these structures in place. The white matter consists of nerve-fibers and neurogliar and connective tissue, besides arteries, veins, and lymphatic vessels throughout the whole spinal cord. The nerve-fibers which are situated in the white matter are bound together in bundles or tracts, each of which has a definite function. Normally these cannot be differentiated, and it is necessary to have pathologic processes, or what is called secondary degeneration, to bring out the different tracts (Fig. 400).

Fig. 401.—The Figures Indicate the Relations of the Vertebral Bodies and Spines to the Corresponding Spinal Segments of the Cord (Church and Peterson).

From the nerve-cells situated in the gray matter of the anterior horn come the so-called anterior roots, which are motor in function. The posterior roots enter into the spinal cord in an area called the entrance root zone, median to the inner surface of the posterior horn of the gray matter. The fibers transmitted by the posterior roots come from the periphery and ascend into the spinal cord, and are sensory in function. On each posterior root is situated a collection of nerve-cells called the posterior root ganglia. The anterior and posterior roots join together to form one nerve which goes through the dura. Each spinal segment has a pair of anterior and posterior roots which form two nerves, one coming off from the right and one from the left side of the cord.

The spinal cord is surrounded by the pial sheath, and is held in place by the anterior and posterior roots and connective-tissue septa (ligamenti denticuli) and by the cerebrospinal fluid, these structures being attached to and surrounded by the dura, which in turn is held in place in the spinal canal by the attached peripheral nerves and bands of connective tissue from the anterior surface of the dura to the vertebra.

Spinal Roots.—The anterior and posterior roots travel within the dura for various lengths before they join to form a peripheral nerve. It is necessary to know the place of exit of each nerve-root, and an easy way to remember it is that every nerve-root leaves the spinal canal at the bottom of the corresponding vertebra; thus, the second lumbar root leaves at the bottom of the second lumbar vertebra, etc. There is an exception, however, so far as the cervical roots are concerned. There are eight cervical segments and only seven cervical vertebra, so that

the eighth cervical root leaves at the bottom of the seventh cervical vertebra. As the end of the cord is opposite the upper border of the second lumbar vertebra, the course of the cervical roots in the spinal canal before their exit is very short. It is longer for the thoracic roots and still greater for the roots from the lowest portion of the spinal cord; thus the second lumbar root has a course of three or four inches within the spinal canal.

Spinal Segments.—It is also necessary from a diagnostic standpoint to know the relations of the different spinal segments to the vertebra. This, however, is not definite and cannot be fixed by any rule, and reference therefore must always be made to charts. It should be remembered, however, that the spinal cord ends opposite the upper border of the second lumbar vertebra, and that sometimes in children it is a little lower. The end of the spinal cord is called the conus medullaris, and its fibrous prolongation the filum terminale (Fig. 402).

Fig. 402.—Showing the Relation of the Spinal Cord to the Body Surface (Church and Peterson).

Functions.—The spinal cord has two functions: one, to conduct impulses to and from the brain; and, second, to supervise and control the motor and trophic functions of the limbs, chest, and abdomen. A better understanding of the cerebrospinal system will be had if it is remembered that there are two sets of centers in the nervous system, and that in the higher or in the cerebrum is represented the center for every motion, sensation, and special act, in this being included also the cerebellum; and that in the lower centers, in which are included the crus, pons, medulla, and spinal cord, are represented the whole surface of the body. For instance, in the crus, pons, and medulla there are collections of nerve-cells or nuclei which are concerned with the innervation of the movements of the face, eyes, nose, throat, and eating, talking, and swallowing, whereas in the spinal cord the collections of nerve-cells in the anterior horns are concerned with the movements of the limbs, trunk, and abdomen, and that the peripheral nerves which connect the peripheral musculature with the spinal cord have exactly the same function that the cranial nerves have which connect their musculature with the nuclei in the crus, pons, and medulla.

Localization.—There are two enlargements in the spinal cord—the so-called cervical and lumbar. This is necessary because the enormous musculature of the limbs requires a large number of nerve-cells. The cervical enlargement begins in the fourth and includes the fifth, sixth, seventh, and eighth cervical and first thoracic segments, whereas the lumbar enlargement begins in the first lumbar segment and includes the second, third, fourth, and fifth lumbar. From here on, the spinal cord gradually tapers off. That part of the cord which includes the second, third, fourth,

and fifth sacral is called the *conus medullaris*, and just above this, and including the fifth lumbar and first and second sacral segments, is the so-called *epiconus*. The nerve-roots coming from the lumbar and sacral cords, when taken together, have been called the *cauda equina*, from their resemblance to a horse's tail.

MOTOR AND REFLEX FUNCTIONS OF THE SPINAL-CORD SEGMENTS.—(*After Starr and Edinger.*)

Segment.	Muscles.	Reflexes.
Cervical	Sternomastoid.	
	Trapezius.	
2–3	Scaleni.	
	Small rotators of head.	
	Diaphragm.	
	Lev. ang. scap.	Dilatation of pupil by irritating side of neck, 4–7 cervical.
	Rhomboids.	
4	Spinati.	
	Deltoid.	
	Supinat. long.	Scapular reflexes, 5 C.–1 D.
5	Biceps.	Supinat. long., 5 C.
	Supinat. brev.	
	Serrat. mag.	
	Pectoralis (clav.).	Biceps, 5–6 C.
	Teres minor.	
6	Pronators.	Posterior wrist, 6–8 C.
	Brachialis ant.	
	Triceps.	
7	Long extensors of wrist and fingers.	Anterior wrist, 7–8 C.
	Pectoralis (costal).	
	Latiss. dorsi.	Palmar, 7 C–1 D.
	Teres maj.	
8	Long flexors, wrist and fingers.	Epigastric, 4–7 D.
	Extensors of thumb.	
Dorsal 1	Intrinsic hand-muscles.	Abdominal, 7–11 D.
2–12	Dorsal and abdominal muscles.	
Lumbar	Abdominal muscles.	Cremaster, 1–3 L.
1	Iliacus.	
	Psoas.	Patellar, 2–4 L.
2	Sartorius.	Bladder, 2–4 L.
	Flexors of knee.	
	Quad. femoris.	
3	Int. rotators of thigh.	
	Adductors of thigh.	Rectal, 4 L.–2 S.
4	Abductors of thigh.	
	Tibialis ant.	Gluteal, 4–5 L.
	Calf-muscles.	
	Ex. rotators of thigh.	
5	Extensors of toes.	Achilles, 5 L.
	Peronei.	
Sacral	Long flex. of toes.	Plantar, 1–2 S.
1–2	Intrinsic foot-muscles.	Anal, } 3–5 S.
3–5	Perineal muscles.	Virile, }

Motor Functions.—The nerve-cells situated in the gray matter of the anterior horns innervate directly the peripheral musculature, and it is probable that a number of nerve-cells are concerned with each nerve-fiber. It is necessary to know what cells are concerned with the innervation of every muscle (see table above). It will be seen from this that we do not know

exactly this location, and that approximately every muscle has a representation in the nerve-cells of one or two segments. Should there be a lesion destroying the cells supplying any muscle or group of muscles, there will necessarily be loss of power, and as these nerve-cells are also trophic in function, there will be, in addition, atrophy and loss of tone or flaccidity in the related parts. Besides, in the performance of every movement we have a sensory irritation or impulse, a center which is in the nerve-cells and a peripheral or motor response; this is the so-called physiologic reflex arc, and an interference with any part of it will cause a loss of any form of reflex.

Summarizing, then, the symptoms of a lesion destroying the cells of the anterior horn, there will be loss of power or paralysis in the related muscles, atrophy, loss of tone or flaccidity, loss of reflexes, and electric reactions of degeneration. Such is the case in acute anterior poliomyelitis or acute infantile spinal palsy. Should there be a slow or chronic degeneration of the cells in the anterior horn, such as occurs in chronic poliomyelitis, there will result fibrillary tremors in the related muscle-fibers, gradual atrophy and loss of power, loss of reflexes, and gradual reactions of degeneration.

The second function of the spinal cord is that of conduction of impulses, either from or to the brain. These are transmitted by means of the different tracts situated in the white matter of the spinal cord. The motor functions are transmitted from the motor cortex by means of the crossed and direct pyramidal tracts. For instance, the right crossed pyramidal tract comes from the left motor cortex, the decussation having taken place in the medulla. From the pyramidal tracts these fibers probably go to the cells of the anterior horn of the spinal cord of the same side, and from these cells come the anterior roots, and from the anterior roots the motor part of the peripheral nerves. A lesion of the motor columns causes weakness, spasticity, increased reflexes, and the Babinski phenomenon. If the lesion involves the pyramidal tracts above the cervical cord, these symptoms are present in both the upper and lower limbs, but if the lesion is below the cervical enlargement, it is only possible to have these symptoms in the lower limb on the same side.

Reflexes.—The reflexes to be considered are the biceps and triceps in the upper, and the patellar or knee jerk and the Achilles jerk in the lower limbs. Whenever there is an exaggerated spasticity, there may be ankle and patellar clonus. In every lesion of the motor columns involving the big fibers there will be obtained the so-called Babinski reflex.

Electric Reactions of Degeneration.—A normal nerve or muscle will respond to any form of electric stimulation. If it is diseased, it will not respond to a faradic current, but will give an increased response to a galvanic current, but the reaction obtained will be slow and sinuous, in opposition to the quick and prompt response obtained when a nerve is normal. The usual method of testing is to first apply a slowly interrupted current to the corresponding normal nerve, and then try the same current on the diseased nerve. If the nerve is completely diseased or sclerosed, no reaction will be obtained to either current. The galvanic current is then tried and a minimum current applied to the diseased nerve first, and the response will be slow and sinuous. The same current applied to the healthy nerve will not cause any reaction, and to obtain a response it will be necessary to increase the current to such an extent that it will be painful. Reactions of degeneration are not obtained until about one or two weeks after the severance of the nerve, and should never be sought for as long as a nerve is inflamed or there is pain on pressure. Its presence makes the prognosis doubtful; its absence, good (Figs. 403–407).

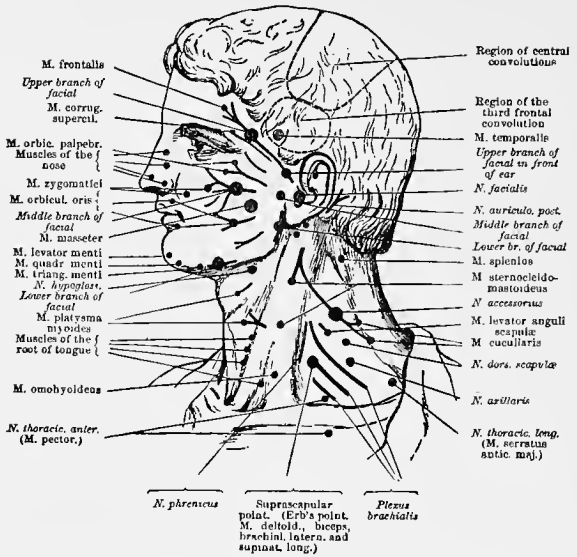


FIG. 403.—NERVES AND MOTOR POINTS IN FACE AND NECK.

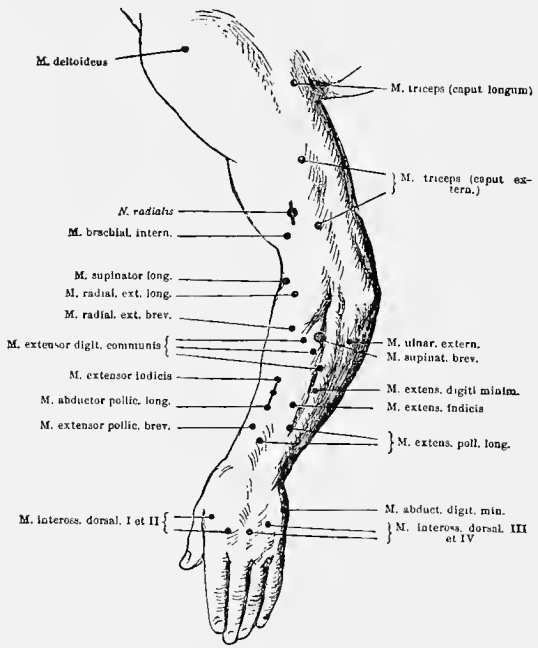


FIG. 404.—NERVES AND MOTOR POINTS IN UPPER EXTREMITY.

Sensory Functions.—The sensory fibers which enter the spinal cord by means of the posterior roots take various courses after their entrance. This has already been discussed on page 1080.

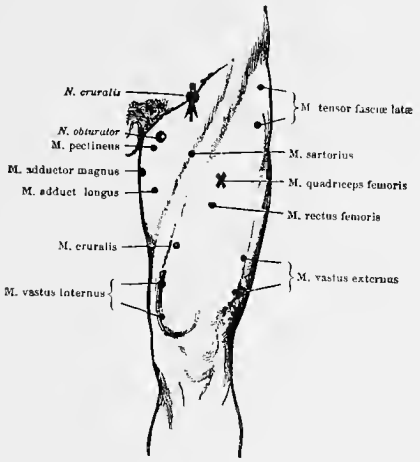


Fig. 405.—Nerves and Motor Points in Lower Extremity.

If a disease involves a posterior root and destroys its fibers, there will be loss of all forms of sensation in the parts from which these fibers come. The

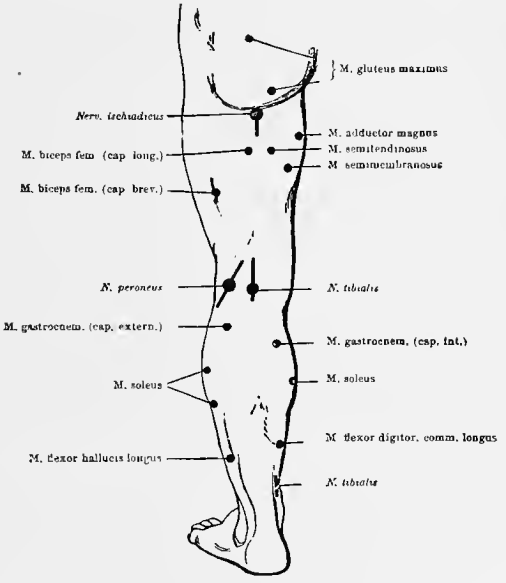


Fig. 406.—Nerves and Motor Points in Lower Extremity.

skin areas of sensation which are in relation to a posterior root are fairly well known, and run in bands lengthwise in the limbs and horizontally in the chest and abdomen. It is necessary to distinguish the area of sensation

in relation with a certain root from that of the segment which this root supplies. In the former the disturbance of sensation will always be unilateral, while in a lesion involving any segment of the spinal cord the disturbance of sensation must be unilateral (Plate XIX). It is probable that sensation in any part of the limbs or of the chest and abdomen is in relation with more than one root or segment, and in a lesion which destroys one root or segment the disturbance of sensation will be very limited.

Bladder, Rectal, and Sexual Centers.—In the second, third, and fourth sacral segments are situated the centers for bladder, rectal, and sexual functions, and a destruction of this part of the cord will cause a loss of these functions. It seems also that the fibers concerned with the bladder and rectal functions descend in the lateral columns of the spinal cord, and that lesions in these tracts may cause an impairment in these functions.

Influence of Secondary Degenerations.—Whenever there is a

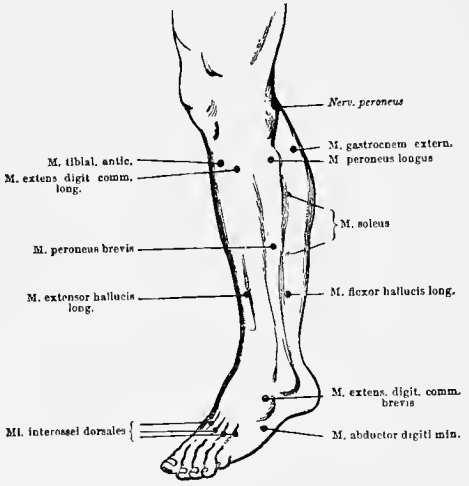


Fig. 407.—Nerves and Motor Points in Lower Extremity.

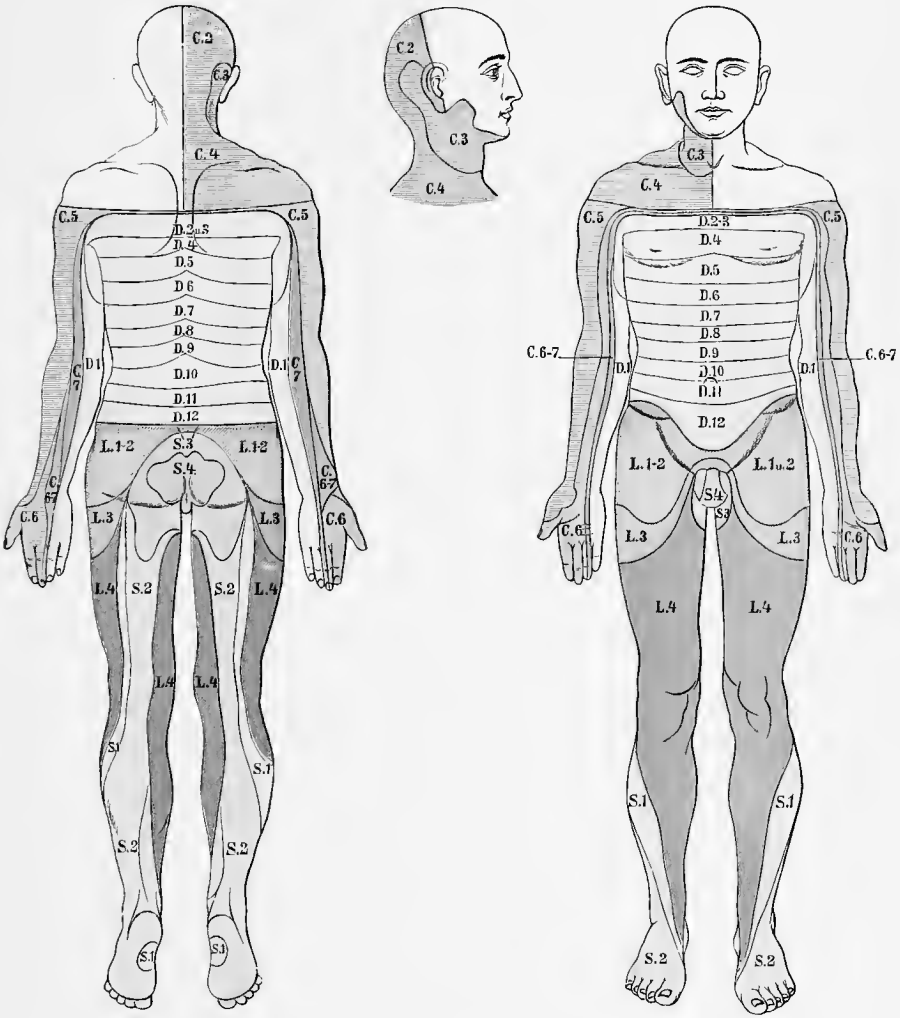
lesion in any portion of the spinal cord, there will necessarily be secondary degeneration. If the motor columns are involved, the degeneration will be downward; if the sensory, upward. Secondary degenerations do not cause active symptoms, for whatever produced the original lesion has also caused the secondary degeneration, and this is no more than a mechanical death of the part.

ACUTE ASCENDING PARALYSIS (Landry's Paralysis).

Definition.—An acute disease, characterized by a rapidly ascending flaccid paralysis, with loss of reflexes, beginning in the muscles of the foot and involving successively the muscles of the leg, thigh, buttocks, abdomen, thorax, and upper limbs, with no sensory symptoms, and terminating in most cases in death.

In the original description of Landry no alterations in the nervous tissue were discovered. Since then, while we still accept the view that there is a type of acute ascending paralysis as originally described, it is a fact that

PLATE XIX



The Sensory Innervation of the Body by the Spinal Segments, according to Kocher.

Red: Cervical segments.
Brown: Dorsal "
Violet: Lumbar "
Blue: Sacral "

} The intensity of the color depends upon the level of the segment.

C_2, D_2, L_2, S_2, etc. = Second cervical, dorsal, lumbar, sacral segment, etc.

most of these cases are either types of acute anterior poliomyelitis or multiple neuritis. There are, however, true cases of Landry's paralysis, and in these there has been found pathologically alterations in the nerve-cells of the anterior horns, an engorgement of the vessels of the cord, and a slight inflammation of the meninges. Diffuse inflammation of the spinal cord is rarely observed.

Contributing and Exciting Factors.—The etiology of the affection is still obscure. It has been known to follow a cold, influenza, various infectious diseases, and other similar causes. It is probable that it is due to an acute intoxication, and that this may be caused by various agents, and that the toxin acts upon the entire central nervous system. The toxic theory is supported by the uniform finding of an enlarged spleen and the febrile symptoms which usually usher in and accompany the paralysis. Bacteriologically a number of bacilli have been found, but none are characteristic.

Symptoms.—The onset is acute and there is usually a rise of temperature with its accompanying symptoms. There is first a diminution of power in the muscles of the lower limbs, nearly always of the foot and leg. This ascends rapidly, and in the course of one, two, or a number of days there is successive involvement in the muscles of the buttocks, loins, abdomen, chest, arms, and neck. The limbs are flaccid, the reflexes are lost, and there may be some paresthetic phenomena, such as numbness, but no other sensory disturbances. The bladder and rectum are never involved and the mind remains clear to the end. The course is rapid and may be fatal in a few days, through either respiratory or cardiac paralysis.

This symptom-complex has been modified to a considerable degree, and while we still recognize a rapidly ascending form of paralysis, other symptoms may be present. It can be assumed that the symptoms just described are to be found in the early stages of the disease, and if it be not fatal we may have added anesthesia of the limbs, abdomen, and chest, pain on pressure over the nerve-trunks, atrophy of the muscles, reactions of degeneration, and sometimes paresis of the functions of the bladder and rectum. If the medulla oblongata is involved, there are added the symptoms of bulbar palsy.

Summary of Diagnosis.—Acute onset with rapidly ascending flaccid paralysis of the muscles of the leg, thigh, abdomen, chest, arms, and neck, with loss of reflexes; no disturbance of sensation, with the exception of occasional numbness in the limbs; and no involvement of the bladder and rectum, the disease terminating in death in a few days. The mind remains clear to the end. If death does not ensue, the subsequent symptoms are those of an acute ascending myelitis.

Differential Diagnosis.—Most cases of so-called Landry's paralysis are either types of acute anterior poliomyelitis or of multiple neuritis. From the former the disease can be distinguished by the rapidity of its course, and by the fact that the paralysis is ascending and involves both sides equally, whereas in acute anterior poliomyelitis the paralysis is partial and involves only part of a limb. From multiple neuritis Landry's disease can be distinguished by the fact that there is absence of pain on pressure over the nerve-trunks and of anesthesia, while in multiple neuritis the paralysis is limited only to the muscles supplied by the different peripheral nerves.

Clinical Course and Complications.—The disease is usually of rapid progress, terminating in death in a few days. If, however, the patient lives, there will be present, as described, the symptoms of acute ascending myelitis, with remaining disturbance of sensation and motion and bladder and rectal symptoms.

CHRONIC POLIOMYELITIS (SUBACUTE ANTERIOR POLIOMYELITIS); PROGRESSIVE MUSCULAR ATROPHY (PROGRESSIVE SPINAL MUSCULAR ATROPHY).

Definition.—Under the above headings are discussed those diseases in which there is progressive or chronic degeneration of the cells in the anterior horns of the spinal cord. Different symptoms are described in each, but in all the pathology is the same, the difference in the clinical symptoms being due to the extent and rapidity of the chronic degenerative process. All are characterized by weakness, atrophy, fibrillary tremors, and gradual loss of reflexes and of the normal electric reactions. In all the disease comes on in adults, more especially after the fortieth year, and nearly always the pathologic process involves predominantly the cervical cord, causing the symptoms to be referred largely to the upper limbs.

Contributing and Exciting Factors.—The causes that produce a chronic degenerative disease of the cells in the anterior horns are not definitely known, but inasmuch as the process begins in the latter end of life it may result, first, because of a primary defect in the development of these structures, or what has been termed an abiotrophy; and, secondly, in the spinal cord as well as in the other parts of the cerebrospinal system there is constantly going on a process of wear and repair. If the repair is not equal to the wear, there may result a premature degeneration or destruction of certain elements, and if, added to this, there is some defect in development, there may result chronic degenerations in the cells of the anterior horn, as in the subject under discussion, or in other portions of the cord, as in lateral and amyotrophic lateral sclerosis.

The exciting causes of the degenerative process are not known, but it is possible that severe injuries of the cord, an old inflammation, or possibly syphilis, may be the starting-point of the process.

Varieties and Symptoms.—Inasmuch as the terminal picture in all the varieties is the same, the symptoms known under the different headings enumerated will appear in the beginning of the disease. These depend upon the extent and rapidity of the involvement of the cells in the anterior horns, and are discussed separately.

Chronic Poliomyelitis (Subacute Poliomyelitis).—Under these headings are included those diseases in which the degeneration of the cells in the anterior horns comes on subacutely or more rapidly than in progressive muscular atrophy, and in which the first symptom is that of weakness, to be followed by atrophy and fibrillary tremors. In the latter disease the first symptom is that of atrophy, followed by weakness or paralysis.

The disease always comes on in adult life, and the patient first notices a weakness in one upper limb, nearly always the right, which is soon followed by a similar weakness in the left. The diminution in power is not limited to a few muscles, but nearly always involves the whole hand, arm, or forearm. Very soon the muscles in the right limb gradually atrophy and fibrillary tremors make their appearance. The tendon reflexes gradually diminish and electric excitability, both by faradic and galvanic currents, becomes diminished. This atrophic weakness continues, and may finally involve the lower limbs, and also the muscles of the head and neck and the chest and abdomen. In the terminal stage the reflexes are lost and there is no response to electric currents because of the absence of muscle.

Progressive Muscular Atrophy (Progressive Spinal Muscular Atrophy).—This comes on nearly always after the thirtieth year, and the patient first notices a wasting in the small muscles of the palm of the hand,

soon followed by fibrillary twitchings, or the tremors may appear first. The muscles of the thenar and hypothenar eminences and of the interossei are nearly always the first to be affected, and as the atrophy, tremors, and weakness increase, the patient will experience increasing difficulty in adducting and abducting the fingers and the thumb, or flexing or extending the phalanges. This may be first noticed by a lessened ability to write or sew or to approximate the thumb with the tips of the other fingers. As the disease progresses, because of the involvement of the interossei and lumbricales, there appears what is known as the *claw hand*, because of the hyperextension of the metacarpal joints and flexion of the phalanges. Because of the wasting of the dorsal thumb muscles there may be what is known as the *monkey hand.*

In most cases the right hand is involved or there may be a coincident involvement of both hands. The progress of the disease is slow and it may take a year or more for the atrophy to extend above the wrist. Gradually the muscles of the forearm, and then the arm, become atrophic and weak, and fibrillary tremors are seen everywhere. As the atrophy involves more and more of the hand, the so-called claw hand disappears and there is extension of all the fingers, or what is called the *skeleton hand.*

As a rule, the disease next involves the muscles of the shoulder and neck, that is, the trapezoid, rhomboid, and sternomastoid muscles, causing the head to fall forward, or it involves, instead, the muscles of the shoulder. The ganglion cells in the lumbar region next become involved, causing progressive atrophy, tremors, and weakness in the lower limbs. As this progresses there may be involvement of all the anterior horn cells in the spinal cord, causing wasting, tremors, and weakness in every part of the body. If the patient does not die because of involvement of the respiratory muscles, the disease may progress upward and involve the motor nuclei in the medulla and pons, causing symptoms of bulbar palsy, with difficulty in eating, talking, swallowing, and chewing, with atrophy and tremors in the related muscles. As a rule, however, the patient dies before bulbar paralysis comes on.

The tendon reflexes become gradually diminished as the disease progresses, and finally will be lost, either because of destruction of the reflex arc or absence of muscle and tendon to produce the reflex. The excitability to both faradic and galvanic currents is diminished from the start and finally is lost. Sensory symptoms are never present and the bladder and rectum are never involved until the last, when the sphincters become weak. This is probably because their innervation is in the spinal cord. The different bones and joints in the limb take part in the general atrophy. Mentality does not become seriously involved until the last.

Summary of Diagnosis.—Chronic Poliomyelitis (Subacute Poliomyelitis).—A previously healthy adult develops increasing weakness in one or both upper limbs, or more rarely in one or both lower limbs. This is followed by gradually increasing atrophy, with fibrillary tremors, gradual loss of tendon reflexes and of excitability to both faradic and galvanic currents. Sensation is not impaired and bladder and rectal involvement does not occur.

Progressive Muscular Atrophy (Progressive Spinal Muscular Atrophy).—A previously healthy adult develops wasting, with fibrillary tremors in the muscles of the thenar and hypothenar eminences, and in the interossei and lumbricales muscles of one hand, usually the right. Atrophy and tremor precede the weakness. These progress and involve finally all the muscles of the hand, forearm, and arm, usually of both sides, causing in their progress

the so-called claw and monkey's hand. The muscles of the back, shoulder, and neck next become diseased, and finally the lower limbs and the muscles of the abdomen and trunk. If the patient lives long enough, bulbar symptoms supervene. The tendon reflexes and the excitability to the electric currents are gradually diminished and finally lost. Sensation and mentality are never impaired. Bladder and rectal impairment does not occur until the last.

Differential Diagnosis.—In their onset these diseases must be differentiated from amyotrophic lateral sclerosis and syringomyelia. In amyotrophic lateral sclerosis there will be, in addition, spastic symptoms in the lower limbs, with increase of tendon reflexes and the Babinski reflex, while in syringomyelia there is the typical dissociation of sensation, that is, the ability to recognize touch and not pain and temperature sensations.

Clinical Course and Complications.—The progress of the disease is slow and bulbar involvement is not very common. Usually the patient dies from some intercurrent cause.

AMYOTROPHIC LATERAL SCLEROSIS.

Definition.—A progressive disease, characterized by gradual atrophy, fibrillary tremors, and weakness, usually beginning in the small muscles of one hand, and finally involving all the muscles of both upper and sometimes of the lower limbs and of the chest and abdomen, with spasticity, and increased reflexes of the lower and later of the upper limbs, terminating in bulbar palsy.

The pathology of the disease consists in a gradual degeneration of the cells of the anterior horns of the spinal cord, with a primary degeneration of the motor or pyramidal tracts throughout their whole extent. The disease is allied to progressive muscular atrophy, and probably has the same etiology, that is, an abiotrophy or lack of vital endurance of both the motor columns and the cells of the anterior horns. The cells of the anterior horns in the cervical cord are nearly always predominantly affected, and their symptoms develop first. The degeneration of the motor columns usually begins at the same time or soon after, and first involves the spinal portions only; but as the disease progresses the whole cortico-spinal motor tracts are involved, and in not a few instances the degeneration can be traced into the motor cortical centers. The degeneration of the cells of the anterior horns extends finally into the thoracic and lumbar cords and the motor nuclei of the medulla and pons. Degenerations of the anterior motor roots coming from the cells of the anterior horns have also been found. The peripheral nerves show typical atrophy.

Contributing and Exciting Factors.—These are similar to those discussed under progressive degenerative diseases of the cells of the anterior horn.

Symptoms.—The onset of the disease is similar to that of progressive muscular atrophy. There is usually first wasting, with tremors in the muscles of the thenar and hypothenar eminences, and then in the dorsal interossei and lumbricales, followed by weakness. These symptoms progress slowly and involve the muscles of the forearm and arm or one or both upper limbs. Coincident with this progressive wasting, tremor, and weakness, the patient experiences difficulty or stiffness in walking, stumbling over slight objects. The weakness and stiffness of the lower limbs increase, and if the patient is examined at this time there will be found spasticity with increased tendon reflexes, with ankle and patellar clonus and the Babinski reflex. As the disease progresses the tendon reflexes in the upper limbs be-

come increased and contractures may develop in both the upper and lower limbs. Besides the spastic paralysis of the lower limbs the muscles gradually atrophy and fibrillary tremors are prominent.

As the disease progresses not only may there develop atrophy, weakness, and tremors in the muscles of the abdomen and chest, but because of the involvement of the motor nuclei in the medulla and pons there supervene the symptoms of bulbar palsy, with difficulty in eating, talking, swallowing, and chewing, the patient finally choking to death. If the patient lives long enough, nearly every case of amyotrophic lateral sclerosis terminates in bulbar palsy. Sometimes the bulbar symptoms may be the very beginning of the amyotrophic lateral sclerosis, but this is rare.

Sensory symptoms are never present. Bladder and rectal impairment is not an uncommon symptom. This is because the cortico-spinal sphincter fibers are probably transmitted in the lateral columns. The mental functions are not impaired until the last.

The tendon reflexes in this disease are nearly always increased, but may be lost in the latter end because of the complete atrophy of the muscle and tendon. Electric reactions are first normal, but later may become diminished.

As in all cases where there is a degeneration of the lateral columns, there may occur spasmodic contractures of one or of both limbs which are more or less painful.

Summary of Diagnosis.—A previously healthy adult develops wasting, fibrillary tremors, and weakness in the small muscles of the hand, usually the right, which gradually increases, and involves the hand, forearm, and arm of both sides. Coincident with this, or soon after, the lower limbs become weak, stiff, the gait is slow and spastic, the reflexes are very much exaggerated, and the Babinski phenomenon is present. The upper limbs soon become spastic and stiff and their tendon reflexes are exaggerated. The atrophy and tremors involve the lower limbs and finally the muscles of the abdomen and chest, terminating in bulbar palsy, with difficulty in eating talking, swallowing, and chewing, the patient finally choking to death.

Differential Diagnosis.—In the onset of this disease the symptoms resemble those of progressive muscular atrophy and syringomyelia. From the first it can be distinguished by the presence of spastic symptoms, with increased reflexes in the lower and upper limbs, and from syringomyelia by the fact that there are no sensory symptoms, such as inability to recognize pain and temperature with preservation of touch sensation.

Clinical Course and Complications.—The progress of the disease is usually slow, the length of life depending upon the bulbar involvement. Usually after this appears the patient dies in from one to two years, either because of disturbance of the vagi, or, what is commonly the case, he chokes to death.

LATERAL SCLEROSIS (PRIMARY).

Definition.—A progressive disease, characterized by weakness, spasticity, and increased tendon reflexes of the lower and upper limbs.

Contributing and Exciting Factors.—It is probable that the degeneration or sclerosis of the motor columns is primary, and not dependent upon a previous disease, and is the result of a lack of vital endurance. The degeneration involves equally the direct and crossed pyramidal tracts of both sides, and affects first the thoracic and lumbar regions. The sclerosis

is progressive, and in the course of many years it may involve the whole cortico-spinal motor tracts. Its isolated occurrence is rare.

Symptoms.—The disease nearly always begins in early adult life or about the twentieth year. The patient first notices that he does not walk as quickly as formerly and that there is a tendency to stumble over slight objects. This may be apparent in one or both lower limbs. Gradually the gait becomes stiff and slow and the limbs weak, until finally the patient can only walk with difficulty, dragging both toes, feet scraping the ground. The limbs become rigid and there is considerable resistance if an attempt is made to move them, it being likened to the resistance offered in bending a lead pipe. Spasms or cramps of the muscles are common, and often when walking is attempted spasm of the adductors of the thigh will cause the so-called scissors gait, in which one leg is placed directly in front of the other (Plate XX).

The patellar and Achilles jerks become gradually increased and ankle and patellar clonus is common. Plantar irritation will always produce the Babinski reflex. There is never disturbance of sensation, but the patient may complain of numbness. The muscles are firm and rigid and their nutrition good. Atrophy never occurs.

For many years the rigidity and weakness may be confined to the lower limbs, but gradually the upper become similarly involved, and there will develop an increasing rigidity with exaggeration of the triceps and biceps reflexes, and sometimes clonus of these reflexes. If the sclerosis involves the motor columns above the pyramidal decussation, there will be increase of the jaw jerk, and, what is not at all infrequent, spasms of the muscles concerned with the emotions or involuntary laughing and crying.

Hereditary or Family Spastic Paralysis.—There is a form of lateral sclerosis which is hereditary, several or all the members of the same family being affected. The symptoms may appear soon after birth or in early childhood, or may not appear until early adult life, and in no other way differ from the usual form.

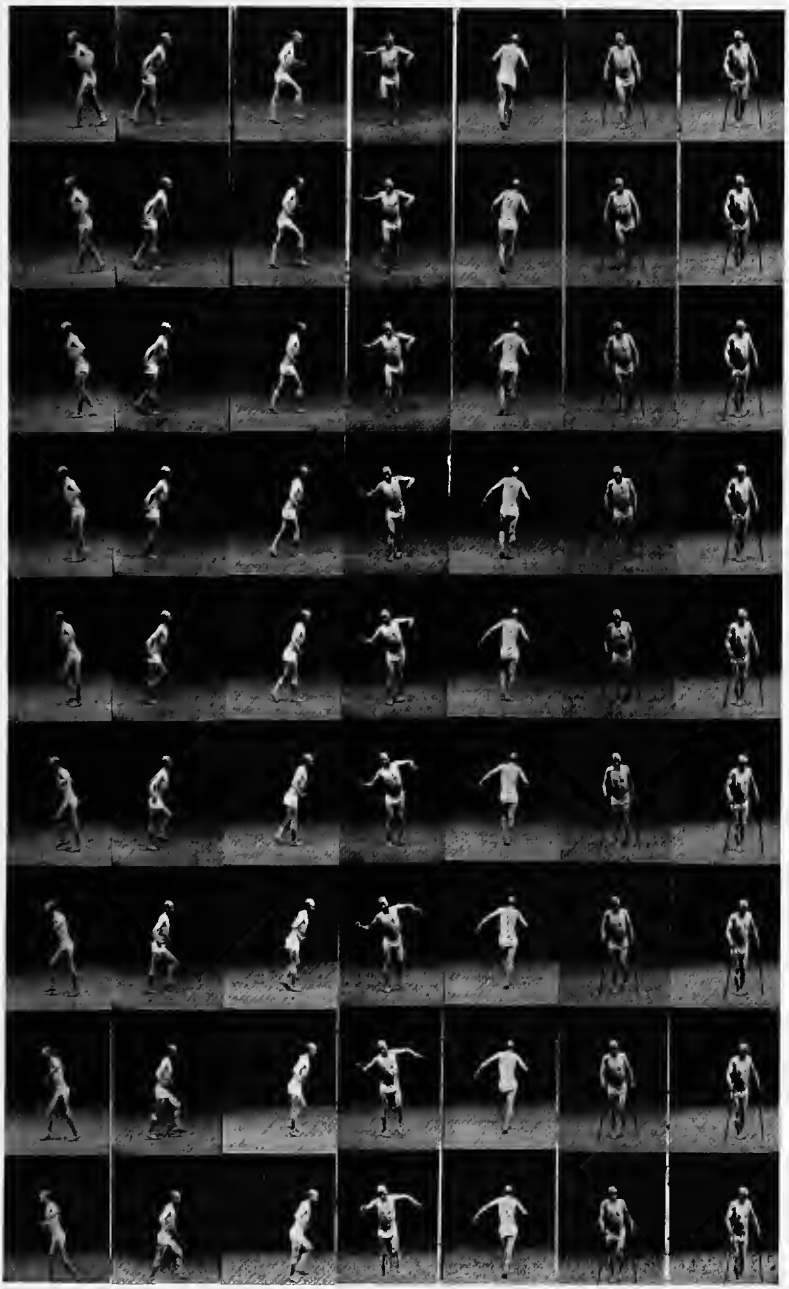
Unilateral Ascending or Descending Sclerosis of the Motor Columns.—Only recently Mills described a form of lateral ascending or descending degeneration of the motor columns limited to one side. The disease may first affect one lower limb and in the course of time involve the upper, or vice versa. It may be the beginning of a bilateral sclerosis or the starting-point of a multiple sclerosis.

Summary of Diagnosis.—Weakness or stiffness of one or both lower limbs, progressive in character, with a stiff spastic gait in which one foot is dragged after the other, sometimes causing the so-called scissors gait, in which one foot is placed in front of the other; exaggerated patellar and Achilles jerks, with patellar and ankle clonus and the Babinski phenomenon. Pain and disturbance of sensation never occur. The muscles are tense and rigid, cramp-like contractures are common, and atrophy is never present.

Differential Diagnosis.—Primary lateral sclerosis is rare. Inasmuch as sclerosis of the lateral columns may occur in amyotrophic lateral sclerosis, and in any disease in which the motor columns are interrupted, as, for instance, in a myelitis, such a diagnosis should not be made until after a number of years have elapsed and there is either no beginning atrophy and fibrillary tremors common in amyotrophic lateral sclerosis, or there is no history of previous paralysis with disturbance of sensation such as occurs in myelitis.

Clinical Course and Complications.—The disease lasts for a

PLATE XX



Moving Picture of Spastic Gait in a Case of Lateral Sclerosis. (Courtesy of Mr. Sigmund Lubin of Philadelphia, Pa.)

long time, and it is possible for the sclerotic process to proceed for thirty or forty years. As the spastic symptoms increase the rigidity may become so extreme that the patient will be unable to walk and becomes bedridden, and there may develop contractures in which the thighs are drawn up on the abdomen and legs on the thighs. Contractures in the upper limbs are not so common, but may occur. Sometimes there may be a slight disturbance of bladder functions, because of the fact that the cortical sphincter fibers are probably transmitted in the motor columns.

SYRINGOMYELIA.

Definition.—A chronic disease, characterized principally by typical dissociation of sensation, that is, ability to recognize touch, with loss or disturbance of pain and temperature sensations, combined with atrophy, fibrillary tremors, weakness in the upper and sometimes in the lower limbs, with spasticity and exaggerated reflexes, especially in the lower limbs.

Pathologically there is usually found a cavity in the central portion of the spinal cord. It is usually largest in the cervical region and diminishes gradually as the thoracic and lumbar segments are approached, and may extend upward into the medulla and pons. The cavity usually involves the gray matter and may extend into the posterior and lateral columns, and may rarely affect only one side of the cord. In life it is filled with fluid.

Contributing and Exciting Factors.—The syringomyelic cavity in most instances results from lack of normal development of the spinal cord. Sometimes there is first an overgrowth of neuroglia tissue, a central gliosis, or a tumor which breaks down, forming a cavity. More rarely traumatism may sometimes produce hemorrhages into the cord, these breaking down and producing cavities. Sometimes the normal central canal is widened, producing what is called a hydromyelia; but unless it is very large, there may be no symptoms.

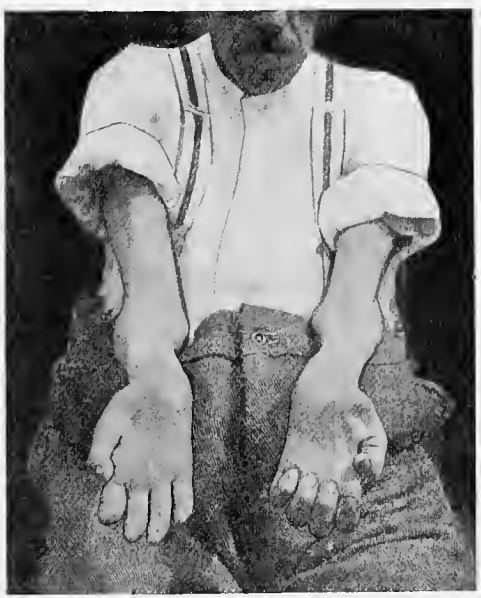


Fig. 408.—Atrophy and Contractures in Syringomyelia.

Symptoms.—The whole symptom-complex of this disease depends upon the interruption of the fibers concerned with pain and temperature sensations, with preservation of touch sensation and the involvement of the anterior cornu and lateral columns. This is because the pain and temperature fibers cross over in the central gray matter, and as the cavity is nearly always in this area, these functions are interrupted. If the cavity is limited only to the central gray matter, there may be present only the dissociation of sensation which is referred to the related peripheral part, usually in the upper limb, but in most cases the cavity

also involves the adjacent cells of the anterior horns plus the lateral columns, their related symptoms developing, such as fibrillary tremors, atrophy, weakness with spasticity, and increased reflexes of the lower limbs. It can be readily seen, then, that the symptoms in different cases may vary.

The disease usually begins in a young adult, the patient sometimes becoming aware of it by the fact that he burns himself without pain. If examined, touch sensation will be found to be normal, but heat or cold or both will not be recognized as such. Sometimes one or the other temperature sensations are alone disturbed, or heat may be as cold and cold referred to as hot. The disturbed areas are usually in the upper limbs, chest, and back, depending upon what spinal segments are destroyed. Coincident with this dissociation, or soon after, atrophy, tremors, and weakness in the small muscles of the hand develop, and there may be present a typical claw hand, its progress being very much like that of either progressive muscular atrophy or amyotrophic lateral sclerosis. Soon after there may develop weakness and spasticity of the lower limbs, with exaggeration of the tendon reflexes and the Babinski phenomenon (Fig. 408).

The progress of the disease is usually slow, and may last for twenty or thirty years, with gradual increase of the wasting, tremors, and loss of power, finally involving all of the upper limbs, shoulders, and chest, and sometimes the lower limbs. The areas of sensory dissociation also gradually increase. If the cavity involves the gray matter of the lumbar and sacral cords, besides the sensory dissociation in the lower limbs and buttocks there will be impairment or loss of bladder, rectal, and sexual functions, and sometimes there may be loss of the knee or Achilles jerks because of interference with the central portions of the reflex arcs.

Fig. 409.—Trophic Enlargement of Thumb in Syringomyelia.

If the cavity extends into the medulla and pons, the symptoms depend upon the extent of the involvement. Usually in the medulla the cavity is unilateral, and there may be partial difficulty in eating, talking, and swallowing; or if bilateral, typical bulbar symptoms develop with tremors, atrophy and weakness in the tongue, facial, masseter, and pterygoid muscles. If the cavity involves the sensory fibers, there may be dissociation of sensation in the face. Rarely primary optic atrophy occurs, and more rarely still pupillary symptoms because of involvement of the cervical sympathetic.

Trophic symptoms are very common in syringomyelia. These may consist in different forms of skin eruption or a destruction of the joints either of the fingers or of the wrist and elbow or shoulder, resembling very much

the so-called Charcot joint of tabes dorsalis. Occasionally there may be sharp shooting pains in the limbs and girdle sense (Fig. 409).

Summary of Diagnosis.—A young adult suddenly burns himself without being aware of it, or there may develop tremors, wasting, and weakness in the small muscles of the hand, with claw-like contractures. Examination demonstrates preservation of touch, with loss or disturbance of pain or temperature sensations, or of both, in the upper limbs and chest. Atrophy, tremors, and weakness may develop in the lower limbs, and there may be, in addition, spasticity with exaggerated reflexes and the Babinski phenomenon. The dissociation of sensation increases and may involve considerable areas of the back, front of the chest, and upper limbs. If the disease progresses into the thoracic and lumbar cords, similar dissociation will be present in the chest, abdomen, and lower limbs. Rarely dissociation of sensation in the face and bulbar symptoms may supervene.

Differential Diagnosis.—There should be no difficulty in recognizing this disease because of the typical dissociation of sensation. In its early progress, however, it may be necessary to differentiate it from progressive muscular atrophy and amyotrophic lateral sclerosis, but this can be usually done by the sensory symptoms.

Clinical Course and Complications.—The disease is of long duration and the patient may live for many years. The pathologic process is progressive, and if the cavity extends into the medulla and pons there may be death from bulbar involvement, but, as a rule, in this disease the patient dies of some intercurrent cause.

POSTEROLATERAL SCLEROSIS.

Under this heading will be discussed all of the different spinal cord diseases in which the posterior and lateral columns are affected. In this are included *ataxic paraplegia, subacute combined sclerosis, diffuse sclerosis,* and the *degenerations which occur in wasting diseases* and *pernicious anemia.*

In all the pathology is the same. There is involvement always of the posterior columns, especially of the columns of Goll, of the lateral or motor columns, and to a less extent of the direct cerebellar tracts. The degeneration seems to be greater in the thoracic than in the cervical or lumbar segments. The difference in the clinical symptoms depends upon the rapidity of the onset, the preponderant involvement of either the lateral or the posterior columns, and the extension of the disease into the direct cerebellar or the anterior tracts.

In *ataxic paraplegia* the degeneration seems to involve principally the columns of Goll and Burdach and the motor columns, with very little involvement of the direct cerebellar tracts.

In *subacute combined sclerosis* and *diffuse sclerosis* there is, besides the involvement of the posterior and motor columns, a degeneration of the direct cerebellar and the tracts anterior to the pyramidal. Besides there may be isolated areas of degeneration in the gray matter.

In the spinal cord changes occurring in *pernicious anemia* the degeneration may involve the posterior and lateral columns equally or may be greater in either. It may also involve the surrounding white matter.

Symptoms.—Ataxic Paraplegia.—Ths disease nearly always begins in the latter end of life, about the fortieth year, without any apparent cause. The patient experiences a gradual weakness in the lower limbs, with increasing spasticity and exaggeration of the patellar and Achilles jerks. The Babinski phenomenon is present. Besides there is ataxia of both lower

limbs. Only rarely are sensory symptoms present, but when this is the case there is only slight disturbance of touch and pain sensation, especially in the soles of the feet and anterior part of the leg. Girdle sense is uncommon and there is hardly ever any disturbance of bladder and rectal functions, although these may appear late. Because of the ataxic weakness, the gait becomes slow, stiff, and slightly incoördinate, this increasing when the eyes are closed. The disease is of long duration and of gradual progress, and may after many years involve the upper limbs and produce increased reflexes and ataxia.

The etiology of the disease is not known, but it is probable that it is the result of a lack of vital endurance or early death of the fibers. In some cases there is an antecedent history of syphilis.

Subacute Combined Sclerosis and Diffuse Sclerosis.—The symptoms in this disease are similar to those of ataxic paraplegia, with the exception that they are much more rapid and there is greater involvement. The specific symptoms will depend upon the preponderant involvement of the posterior or lateral columns. If the posterior columns are preponderantly diseased, there is considerable ataxia, with numbness in the lower limbs, occasionally pains and girdle sense and disturbance of the bladder and rectum, loss of reflexes, and the only expression of degeneration of the lateral columns may be the Babinski reflex. If the lateral columns are preponderantly involved, there is considerable weakness with spasticity, increased reflexes with the Babinski phenomenon and ataxia of the limbs, rarely some numbness, pain and girdle sense, and occasionally involvement of the bladder and rectum. In the course of a few months there rapidly develops complete paralysis of both lower limbs, flaccid in type, with loss of reflexes but retained Babinski phenomenon, and considerable disturbance of sensation with involvement of the bladder and rectum, giving the appearance of a diffuse myelitis, which the disease really is. The progress of the affection is rapid and rarely lasts more than one year.

Sclerosis Occurring in Pernicious Anemia.—The symptoms of the spinal degeneration may occur coincident with the anemic changes, but, as a rule, alterations in the blood are demonstrated first. Occasionally it is possible to diagnose anemia from the spinal cord symptoms. These consist, besides headache, weakness of the limbs, paleness of the skin and mucous membranes, and changes in the blood, of extreme numbness and a tingling feeling in the lower and upper limbs. Sometimes these are very great and may be the first indication of the disease. There may be also pains of an indefinite character, or there may be sharp shooting pains in the lower or upper limbs and occasionally girdle sense. If the posterior columns are preponderantly diseased, there is considerable ataxia, with loss of the tendon reflexes in the lower and upper limbs, and there may or may not be some disturbance of sensation. As a rule, besides the ataxia and pains the tendon reflexes are increased and the Babinski phenomenon may be demonstrated. The weakness may become so marked that the patient is bedridden. The spinal cord degenerations progress according to the rapidity of the blood changes, and it is possible for the symptoms to ameliorate provided the blood condition improves.

Sometimes in simple anemia, such as results from acute gastric or other hemorrhages or in wasting diseases, as carcinoma and phthisis, there may develop a diffuse sclerosis of the posterolateral columns. The symptoms will consist in weakness, increased reflexes, the Babinski phenomenon, ataxia, and occasionally numbness with disturbance of sensation.

Summary of Diagnosis.—In all the different diseases above de-

scribed under posterolateral sclerosis the preponderant symptoms are present in the lower limbs, and consist in weakness, increased reflexes, spasticity, the Babinski phenomenon, ataxia, occasional numbness and pain in the lower limbs, and more rarely disturbance of sensation and of bladder and rectal functions.

Differential Diagnosis.—With the symptoms above enumerated there should be no difficulty in diagnosing the type of posterolateral sclerosis. Occasionally, however, there may be such symptoms in myelitis or in multiple sclerosis. In the former there is nearly always a history of an acute onset, with paralysis of the lower limbs, which gradually lessens, leaving the symptoms of a posterolateral sclerosis, while in disseminated sclerosis there is, besides, intention tremor, nystagmus, and scanning speech.

FRIEDREICH'S ATAXIA.

Definition.—A hereditary or family disease characterized by progressive ataxia of the limbs and body, diminution of power, especially of the lower limbs, and loss of reflexes, but no disturbance of sensation or of bladder and rectal functions.

Pathologically there is degeneration or sclerosis of the posterior columns,

FIG. 410.—FRIEDREICH'S ATAXIA—BROTHER AND SISTER.

especially of the columns of Goll and of the lateral or motor and direct cerebellar tracts of both sides. Occasionally there is, in addition, degeneration in the cells of the columns of Clarke and some atrophy of the cerebellum.

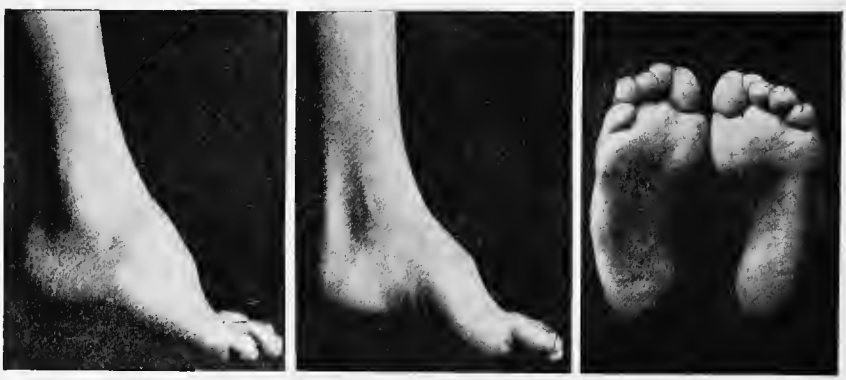
Contributing and Exciting Factors.—The disease is hereditary, and occurs, as a rule, in several members of the same family. Occasionally sporadic cases are observed. It is probable that the disease is congenital and is due to a maldevelopment of certain tracts of the spinal cord.

Symptoms.—As in every hereditary and congenital disease, the symptoms begin early in life, about the age of puberty. The early development of

the child is usually slow, and in most cases it has taken more than the usual time for the child to learn to walk. As a rule, it is noticed very early that the lower limbs are not normally developed and that there is present a peculiar deformity of the foot and toes of both sides which is characteristic of Friedreich's ataxia. It consists in a diminution in the length of the foot, the dorsum is prominent, the arch of the sole is deeper than it should be, the large toe is hyperextended at the metatarso-phalangeal and flexed at the phalangeal joint. The position of the foot is that of a talipes equinovarus. There is usually also a deformity of the spine—either a scoliosis or kyphoscoliosis (Fig. 410).

The above developmental symptoms may or may not be prominent. It is then noticed that about the age of puberty the child begins to stagger in walking, the incoördination involving especially both lower limbs and the trunk, resembling that seen in cerebellar disease, and soon walking becomes impossible. Ataxia of the upper limbs does not, as a rule, become prominent until late in the disease.

Coincident with the ataxia there is an increasing weakness of the lower limbs, and the patellar and Achilles jerks become lost, although the Babinski phenomenon may be present on both sides. The reflexes later on also become lost in the upper limbs. There is never any spasticity. Sensation is only rarely disturbed and there is hardly ever numbness or pain. Bladder and rectal disturbances are never present.



Figs. 411, 412, and 413.—Typical Deformity of Foot in Friedreich's Ataxia.

There is often present a peculiar nodding or to-and-fro movement of the head, and sometimes of the whole body, which resembles to some degree the movements of multiple sclerosis, but these differ in the fact that they do not become very much worse on excitement. There is nearly always also some nystagmus, this consisting in to-and-fro or oscillatory movements of the eyeballs, or there may be only few jerkings on lateral deviation.

Speech is nearly always somewhat disturbed and becomes slow, the voice becoming dry, thin, and high-pitched and each word is syllabized. There is never, however, the muffling of the words or difficulty in enunciation that is so common in multiple sclerosis (Figs. 411–413).

Summary of Diagnosis.—Several members of the same family may have lateral curvature and a peculiar deformity of the foot and toe, consisting in a prominence of the dorsum and in an extension of the large toe on

the metatarso-phalangeal and flexion of the phalangeal joint. The disease begins early in life, about the age of puberty, with ataxia of both lower limbs and the body, resembling the incoördination of cerebellar disease, weakness of both lower limbs, loss of the tendon reflexes with occasional presence of the Babinski phenomenon, no involvement of the bladder and rectum or of sensation, a peculiar hesitating speech, nystagmus, and tremor of the head.

Differential Diagnosis.—The disease may be confounded with juvenile tabes dorsalis, but can be differentiated from it by the absence of numbness, the characteristic lightning pains or girdle sense, and no involvement of the bladder and rectum. Besides, pupillary symptoms and the Argyll-Robertson pupil are uncommon.

Hereditary Cerebellar Ataxia.—In this disease there is a congenital atrophy of the cerebellum, and the symptoms consist in a gradual ataxia of all the limbs, disturbances of speech, nystagmus, and some tremor of the head, all these resembling the symptoms of Friedreich's ataxia, but the disease differs in the fact that there is hardly ever any deformity of the foot or of the spine, and that there are, in addition, increased reflexes, optic atrophy, and occasionally the Argyll-Robertson pupil.

Clinical Course and Complications.—The disease is progressive and the ataxia and weakness increase, the patient becoming chair- or bed-ridden, but may live for a long time. There may be in the latter end of the disease bladder, rectal, and sensory disturbances, but this is uncommon. The mind, as a rule, is not affected, but there may be some diminution of intelligence.

TABES DORSALIS (LOCOMOTOR ATAXIA).

Definition.—A chronic progressive disease, characterized first by numbness in the lower limbs, then by pains of a sharp, shooting character, girdle sensation, difficulty in walking and in execution of any movement, this being especially made worse with eyes shut, absence of reflexes, disturbance in the functions of the bladder and rectum, irregular pupils, with failure of the reaction to light, and later optic atrophy and disturbance of sensation in various portions of the body.

Tabes is more frequent in males, and it does not occur in negroes except where there has been an intermingling of white blood. Its distribution is also interesting when one considers that in certain nations, as among Asiatics, and especially the Chinese, although syphilis is very common, tabes is frequent, while paresis is rare.

Contributing and Exciting Factors.—It is the belief of many neurologists that every case of tabes dorsalis is due to syphilis. The statistics of Erb show that in men in about 97 per cent. of cases there is either a specific history or that there have been present symptoms indicating such disease. In only from 1 to 5 per cent. of the large number of syphilitics does tabes dorsalis occur. There must be, therefore, other contributory causes or exciting factors. These may be either a special syphilitic infection or a predisposition to this disease by the infected person, the latter probably being the more potent cause. What this may be has not as yet been determined, but it is probable that exhaustion contributes to it largely. It has been shown in animals that fatigue will produce the characteristic pathologic changes of early tabes dorsalis. While it is probable that the exhaustion itself will not produce this disease in man, occasional cases are seen in which the symptoms follow injuries to the back or are consequent to severe falls. This, however, is of rare occurrence.

Given a person who has been specifically infected, and even one who has been treated by the proper anti-specific remedies, the symptoms of the disease may appear anywhere from five to twenty years after the infection, as a rule, before the tenth year. It is a curious fact that so far as the patient is concerned no symptoms are apparent for some years. It is probable, however, that were the patient carefully examined, some symptoms would be found, for it is difficult to believe that a toxin will be dormant for a number of years and that its effects will not manifest themselves upon any portion of the economy. However that may be, it has been recently proved that examination of the cerebrospinal fluid will also show an increased number of lymphocytes in all cases in which there has been a syphilitic history. Besides, a positive Wasserman reaction is obtained in nearly all tabetic cases. It can be assumed, therefore, that the toxin of syphilis is present in the blood and cerebrospinal fluid.

Pathology.—Method of Infection.—It has only recently been shown that in the peripheral nerves, spinal roots, and cranial nerves there is a constant stream of lymph ascending toward the central nervous system whose main current lies in the inner meshes or lymph-spaces of the fibrous perineural sheath. Any toxins such as would follow a specific infection would reach the spinal cord and brain by this channel; and although they spread to some extent in the lymph-spaces of the pia-arachnoid, and so affect structures at a distance from their point of entry, for the most part they pass in the main current of the lymph along the nerve-roots into the substance of the central nervous system. Here they apparently follow the nerve-paths of the affected roots, and show little tendency to diffuse among the neighboring fibers. It has been shown that just as long as these nerves are protected from the influence of the toxins by the vital action of their neurilemma sheath, the nerve itself will not degenerate, but will on losing this. This is a very important fact. Just before the posterior roots enter the spinal cord they lose their neurilemma sheaths. Should, therefore, there be any toxins circulating in the cerebrospinal fluid or in the nerve, here would be a point of least resistance, provided, of course, there is a contributing cause to weaken these roots which may be fatigue or a lessened amount of resistance of this particular part. Should this be the case, there would begin a degeneration of these posterior roots, and here would start the pathologic process. Just why the posterior roots are taken for this selected action is difficult to explain, but not more so than that other portions of the nervous system are selected by the same poison and that the posterior roots escape.

Microscopically in the early stages of tabes there is found a mild meningitis, especially in the posterior part of the cord, and a beginning degeneration of the posterior roots. As the disease progresses there will be a consequent ascending degeneration in the posterior columns or the columns of Goll and Burdach, the cells of the columns of Clark and of the fibers coming from them, or the direct cerebellar tracts. The degeneration finally involves all the posterior roots, although it is probable that those of the lumbar and sacral are first involved. The degeneration of the cells in the posterior ganglia is probably secondary to the posterior root degeneration. Late in the disease it is common to find a slight degeneration of the peripheral nerves.

Early Symptoms.—Numbness and Pain.—As the lumbar roots are the first to be diseased, the primary symptoms of which the patient complains will be referred to their distribution, that is, the lower limbs. As a rule, the patient will complain of a feeling of numbness in his feet, sometimes of a sensation as of walking on leather or of a dead feeling. This at

first will only appear for a short time and then will become chronic. Very soon these paresthetic phenomena are succeeded or are accompanied by pains of a sharp, shooting, jagging character. At first they will come on only at intervals, and will appear in various portions of the lower limbs, lasting from a few minutes to at least a half hour and leaving the muscles very tender. Gradually, however, they will become more frequent, more lasting, and of much sharper intensity, acquiring the lancinating character typical of this disease. The pains are not limited to any one nerve distribution, but appear at irregular places, and as the disease progresses, they are to be found in the upper limbs and other portions of the body. Very rarely the patient will complain of pain in the face, in the distribution of the fifth nerve, resembling very much the pains of tic douloureux, and more rarely still pains will appear in the cervical and occipital distribution. The pains seem to be influenced by the weather, the patient first likening them to rheumatism.

Girdle Sense.—One of the earliest symptoms the patient complains of is girdle sense, or a feeling of constriction around the waist. The patient very often likens this feeling to that of a band tied around the waist or of a drawing sensation. This symptom after it appears is very liable to become permanent, although its intensity varies. Often these feelings of constriction appear in other places, as around the thighs, knees, ankle, and sometimes around the chest or parts of the upper limbs.

Disturbance of Reflexes.—Necessarily, as the degeneration of the posterior roots progresses there must be some interference with the reflexes, as these roots are integral parts of the reflex arcs. There is, therefore, very early in the disease a diminution of the patellar or knee jerks and the Achilles jerks. As the degeneration progresses in the posterior roots and in the corresponding portions of the posterior columns, these reflexes later become totally lost, and cannot be obtained even under reinforcement. The reflexes in the upper limbs—that is, the biceps and triceps—will in the same way be first diminished and then lost.

Bladder, Rectal, and Sexual Symptoms.—As these roots also transmit the fibers which are in relation with bladder, rectal, and sexual functions, these are necessarily first diminished and then lost. Constipation is a very early symptom, while difficulty in the starting of the urine may not make its appearance until later. Sexual functions may be retained until late in the disease, but, as a rule, are lost early.

Alterations of Sensation.—It is to be expected that from the very beginning there should be alterations in sensation, and that the very earliest symptom of tabes would be a disturbance in touch, pain, temperature, and the sensations in the muscles, ligaments, tendons, joints, and bones. As a matter of fact, this is the case, but these symptoms are not demonstrated, principally because they are not looked for. The patient himself will not call attention to his disease or will not be aware that anything is the matter with him until he finds either that he has pains or that he has some difficulty in coördination.

Diminution in the sensation for touch is first to be found in the soles of the feet and over the anterior portion of the legs just in front of the tibia. Another very common location is in the front part of the chest and along its side. Here careful testing will denote a diminution of the sensation for touch and pain sense. Heat and cold, as a rule, are properly interpreted at first, but later in the disease one may be taken for the other, or there may be either diminution or inability to recognize these sensations. As the disease progresses the areas of hypesthesia for touch and pain become a little

more general and there appears what seems to be a very characteristic symptom of tabes, that is, that sensations are not as quickly appreciated as they should be, and sometimes it will take a number of seconds for a pin-prick to be recognized. Again, sensations are misinterpreted, and a pin-prick will feel as a touch or it will be described as being in the other limb. In the very last stages of the disease touch, pain, and temperature sensations may be lost over most of the body.

Very early in the disease and among the first symptoms it will be found that the sense of pressure is diminished over the calves and feet. This symptom is of value only in the early stages, for it will only then be possible to compare with the pressure sense of the upper limbs. The sense of position and movement will also be diminished in the early stages. If, for instance, with the eyes closed and the patient's body totally relaxed, a toe is moved the patient will be unaware of the position in which it is. As the disease progresses it will be possible to demonstrate these symptoms in the joints and limbs. It is to be remembered that total relaxation must be obtained, or otherwise these tests will be unsatisfactory.

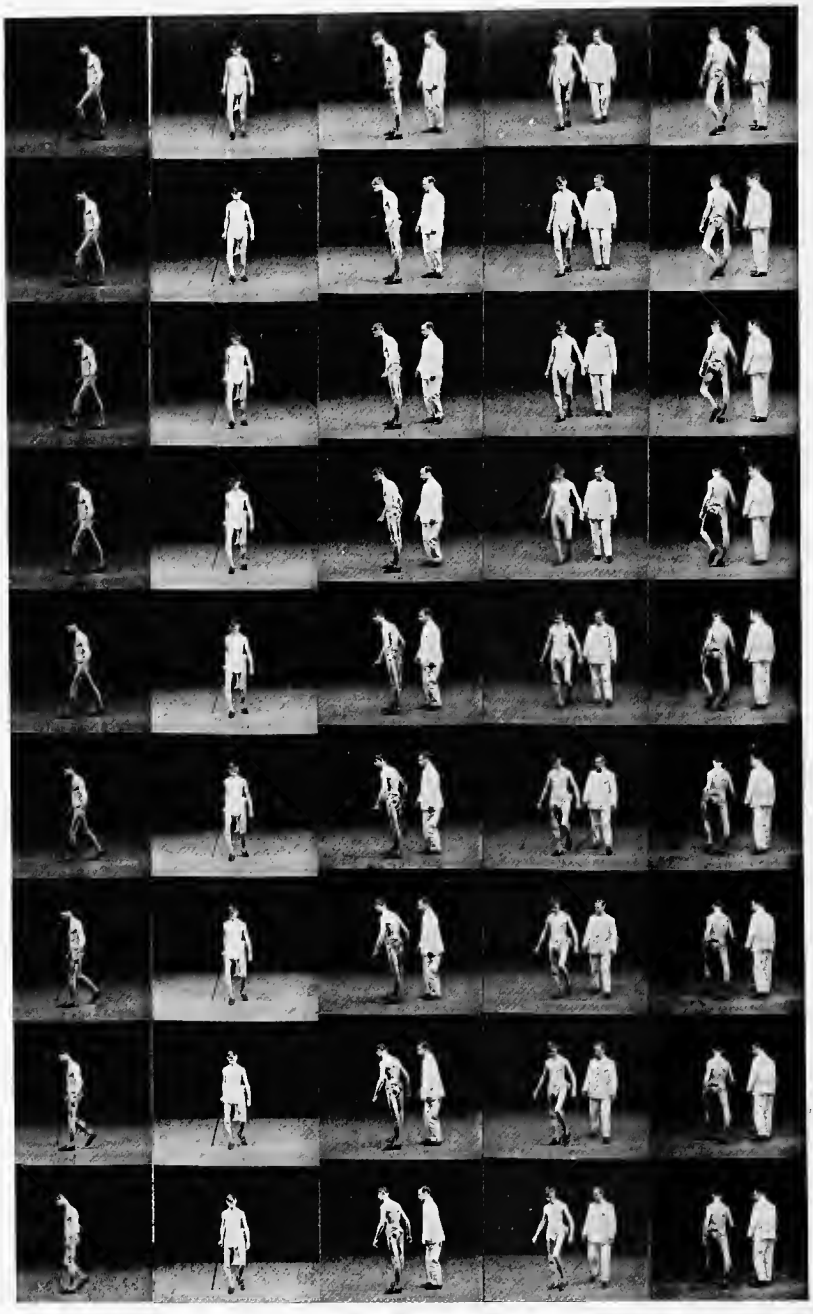
We see, then, that because of the pathologic process present in this disease there is a gradual diminution and finally a destruction of all forms of sensation in the muscles, ligaments, tendons, and joints, and apparently less so of the skin. Because of this, the normal relation that these structures bear to each other or their tone is disturbed, and in the performance of any movement the peripheral sensory impressions cannot be normally transmitted, interpreted, or performed, and because of this we have the symptoms of ataxia or incoördination of motion.

Physiology of Locomotion.—To understand why disturbances of locomotion appear it is important to understand the normal mechanism concerned in walking, standing, and, in fact, in all our movements. The child when it is first born is very ataxic, and has to be taught, for instance, to eat, and then later on to walk. This process of education, while it appears perfectly simple because it goes on in a rather slow, indifferent manner, is really a very complex process, as can be readily understood when one considers the utter helplessness present either in advanced tabes or cerebellar disease.

Take, for instance, the process concerned in educating one to play the piano. The movements at first are coarse, irregular, and in a high sense incoördinate. To be able to play this instrument, it will not only be necessary to develop the muscles concerned in this act, that is, the motor part of the arc, but it will be necessary to so control them that the proper amount of pressure, accuracy, and coördination can be acquired. Not only that, but these movements will have to be so promptly performed as to enable the player to coördinate with the peripheral ocular impression obtained by reading notes, or a central impression of playing from memory. We see, then, that to perform any movement it is necessary to have a motor and a sensory arc, and a coördinating center which controls and properly maintains the relation between these two, and also with such higher functions as will and intelligence.

In a healthy individual this normal relation between the motor, sensory, and coördinating arcs is constantly maintained, and the individual is always aware of the position of any portion of the body and its relation to other parts. Physically this is manifested by a normal relation or tone between the muscles, ligaments, joints, and bones. Should there be any disturbance of the sensory portion of the arc, this normal relation or tone will be disturbed, and instead of these parts acting in relation to each other, there will be an incoördination.

PLATE XXI



Moving Picture of Gait in Locomotor Ataxia. (Courtesy of Mr. Sigmund Lubin of Philadelphia, Pa.)

Hypotonia.—This disturbance of normal relations between the structures that make up the joint produces the symptom called hypotonia, and is one of the earliest found in the disease. Because of the fact that the muscles do not check one another, and that there is not the normal play between the antagonists, and that normal resistance is not offered in the joints, there is a tendency for any movement to continue beyond the previously fixed maximum. The knee- and ankle-joints are among the first to become involved, and because of this, these joints can be moved in abnormal directions. Clinically this is demonstrated by the backward giving of the knees, and in the ankles by the abnormal tendency of this joint to give way.

As hypotonia is present in the muscles surrounding the spinal column, and also later on in the other joints of the body, it can be readily understood what an influence this will have upon the attitude and gait of the patient. This symptom in itself will not cause ataxia, but it undoubtedly contributes to it. Space does not allow us to take up separately the methods for detecting hypotonia of the different joints. It can be easily demonstrated, for it will be found that it is possible to bend more than usual the leg on the thigh and the thigh on the abdomen. In fact, sometimes it is possible with the whole leg extended to flex it and place it in apposition with the trunk. Again, in testing for abduction and adduction of the thighs, it is sometimes possible to completely stretch the limbs in a straight line, while the flabbiness of the muscles of the back will enable the patient to bend in any direction.

Ataxia.—Principally, then, because of the slow alteration of sensation in the peripheral parts, the patient will begin to have difficulty in performing fine movements, and these will become more apparent either when the eyes are closed or when the patient is in the dark. This is because ocular impressions have a great influence upon whatever movement is performed. It may become apparent when the patient attempts to walk in the dark, or when he tries to balance himself with eyes covered, as in washing the face; or very frequently the patients are not aware that anything ails them until, when attempting to dance, their lower limbs suddenly give way. Unconsciously the patient will begin to walk with his feet a little wider apart, and he will walk more slowly or carefully, and in the performance of any movement it will be found that either the muscles concerned in the movement are contracted abnormally, or the contraction is too long or the movement too rapid. This difficulty in walking will gradually become more manifest, and the patient will not only walk with his feet wide apart, so as to give himself more base, but he will bend his knees a little higher than he should, and in replacing them, because of the ataxia and hypotonia, the leg will be thrown in what seems to be an aimless way. With this the patient will gradually acquire a tendency to stoop over, the head bent down and the eyes to the ground, because he is uncertain of the position of his feet, and of his desire to bring to his aid the sense of sight (Plate XXI).

Methods of Testing for Ataxia.—While no difficulty will be experienced in demonstrating ataxia in the later stages, considerable skill is required in developing the symptom very early in the disease. Each limb should be tested separately, for it will be found that the degree of ataxia will vary in the different extremities. The patient should be made to stand on one leg with the eyes opened and closed, and then with the knee bent. Romberg's sign, which is obtained by placing the heels and toes in apposition with closed eyes, the patient swaying, can usually be demonstrated. In testing for ataxia of either lower limb, the patient should be placed on his back and asked

to place his heel on the opposing knee with eyes open or shut, and it will be found that a greater degree of ataxia will be developed as the patient places his heel away from the knee to the distal parts. In testing for ataxia of the knee-joint, the patient should be placed upon his abdomen and the leg bent on the thigh. The usual finger to nose or the finger to finger tests are employed in testing for incoördination of the upper limbs. Ataxia is often present in the muscles concerned in respiration and in the abdominal muscles, but this is not easily demonstrated. The facts to be remembered are that when a movement becomes incoördinate it will be either executed too rapidly, or there will be exaggerated muscular exertion, or an unduly prolonged state of muscular contraction, which continues long after the maximum of excursion has been reached.

Pupillary Phenomena.—Pupillary signs are among the earliest and the most constant phenomena of tabes dorsalis. Very early in the disease the pupils will have a tendency to become smaller than normal, the so-called miotic pupils, the margins to be irregular, and at the same time the reaction to light will be unequal and diminished. As the disease progresses the pupils will become still smaller, the margins more irregular, and the light reaction will be lost, although reactions to movement will be retained. This is the Argyll-Robertson pupil, and is one of the most valuable symptoms present in this disease.

Paralysis of Cranial Nerves.—One of the commonest and sometimes one of the earliest symptoms of tabes is double vision due to paralysis of one external rectus. This diplopia is not continuous and will last only for a short time, that is, from a few hours to several days, and will reappear at intervals. Just why this involvement of the motor nerve should occur is difficult to explain. Another occasional early symptom is paralysis of a vocal cord, causing the hoarse, stridulent tones sometimes found in tabetics. The involvement of these two nerves in a sensory disease may be explained by the theory that weakness may occur in those parts which are constantly used, or by the less satisfactory one of *locus minoris resistentiæ*.

Crises.—So far we have attempted to trace the symptoms and signs step by step as they occur in analogy with the pathologic findings. As this progresses all the symptoms above enumerated will grow in intensity and new ones appear. The numbness and pain will become more persistent and of a sharper intensity, and may appear in almost any portion of the body. Pains may be localized in various viscera of the body, and are then called crises; and of these, the gastric crises are the most common. They may appear at any time, and the pains in the stomach may become very severe, and, as a rule, are associated with nausea and vomiting. They may, however, appear without these symptoms, or with one of them, and may last from a few minutes to several hours or longer. Vomiting, as a rule, does not relieve the pains. Crises next most frequently occur in the larynx, where they are associated with difficulty in breathing and stridulous respiration. Intestinal crises are next most frequent, and are accompanied by violent pains and evacuations of the bowel. Almost any organ may be the seat of these phenomena, and there may be renal, bladder, rectal, genital, ocular, and oral crises. No satisfactory explanation has ever been given for their occurrence, but it is presumed that there is a disturbance of function of the organ brought about by derangement of the sympathetic plexus. These crises are not as frequent as is usually inferred.

Involvement of the Trigeminus Nerve.—A not unusual symptom is paresthesia of the face, the patient complaining of objects crawling over his

eyebrows, or that there is a mask drawn over his face. These phenomena may be succeeded by pain in the same distribution, and are probably the result of degeneration of the sensory or descending root of the fifth nerve, which is found as low down as the third cervical segment, its involvement being an excellent indication of the upward progress of the disease. Associated with this, there necessarily will be a diminution of sensation in the distribution of one or both fifth nerves, one of the early manifestations of this being a tendency to looseness of the teeth, it being possible to pull out a tooth without the slightest pain. Later there may be complete loss of sensation in the distribution of the fifth nerves.

Optic Nerve Atrophy.—Atrophy of the optic nerve is one of the most serious complications to be found in this disease, and is present, according to Gowers, in about one-tenth of the cases. It is usually a late manifestation, but if it appears early it seems to have a beneficial tendency upon the other symptoms, for these, as a rule, will diminish. Among the other ocular complications is paralysis of part or the whole of the oculomotor nerve and of the sixth nerve.

Deafness.—Deafness is a very frequent symptom in late tabes, and some diminution of hearing would be found in every case of early tabes, were it carefully searched for. Very rarely the vestibular branch is involved, causing vertigo, tinnitus, and cerebellar gait. Among the other cranial nerves which may be involved are to be mentioned the facial nerve, and those which supply the organs of the voice.

Vasomotor and Trophic Phenomena.—Vasomotor and trophic disturbances are not unusual, and when it is considered that the sympathetic plexus takes part in the degeneration found in tabes, and that some of these fibers enter the spinal cord by means of the posterior roots, it is not difficult to explain their occurrence. Local sweating in the palms or soles or in the hands, and alterations in the pigment of the skin, are not uncommon. Occasionally there may be herpetic eruptions, accompanied by pain, or there may be alteration in growth of hair following pains. Because of the hypotonic condition of the joints and of the altered amount of sensibility, there occurs what is called the tabetic joint and the tabetic foot. This is nothing more than a giving way of the parts concerned.

As has been previously shown, alteration in the sensation of the structures beneath the skin is one of the earliest symptoms of the disease. Necessarily, then, the bones enter in this, and microscopic examination has shown alteration in their structures. Because of this fractures are very common, and must be guarded against. Should there be any laceration of the tissues, as by stepping on a nail, because of the lessened resistance of the tissues infection will be easy and extensive wounds may result, the healing of which is very difficult, an example of this being the perforating ulcer which is found on the soles of the feet of tabetics.

Charcot Joints.—Considering, then, the lessened amount of resistance in the tissues and the alterations in the bones, should there be any injury of a joint, there may result what was first described by Charcot and named after him. These Charcot joints are, as a rule, found in the knee, but may be located in the ankle, elbow, shoulder, or, in fact, even in the joints of the vertebra, and are characterized by looseness of the parts and ability to move them in any direction, this not being painful. Pathologically, an erosion of the parts of the joints is found (Fig. 414).

Unusual Varieties.—Juvenile or Hereditary Tabes; Optic or Cervical Tabes; Sacral Tabes.—In the juvenile or hereditary form the

disease begins very early in life, and there are always present other signs of congenital syphilis. This type is very rare. In the usual form of tabes the symptoms, as a rule, come on anywhere from five to ten years after infection with syphilis, but may not appear until twenty years after. In the optic form the symptoms come on in the usual chronologic order, but optic atrophy is early manifested and the principal symptoms are confined to the upper limbs, the disease being mostly localized to the cervical cord. After the optic atrophy has become complete the ataxia nearly always diminishes, leaving only the general symptoms of the disease with pains. In the sacral form the pathologic process is first limited to the lowest portion of the spinal cord, giving as early symptoms disturbances of bladder and rectal functions, and also of sensation and locomotion for a long time only in the lower limbs.

Summary of Diagnosis.—Early Tabes.—Numbness in the lower limbs, followed by sharp, shooting pains; beginning girdle sense; diminution and later loss of patellar and Achilles jerks; beginning hypotonia and looseness of the knee-, ankle-, and hip-joints, with some diminution in the sense of movement, position, and pressure in the lower limbs; some difficulty in

Fig. 414.—Charcot Joint in Tabes.

walking, which is increased with the eyes shut; a tendency to constipation and possible loss of sexual functions; irregularity of the pupils with miosis and a slow reaction of the pupil to light, but prompt contraction to movement of the eyeballs.

Late Tabes.—Constant numbness in the feet; sharp, shooting, lancinating pains all over the body; girdle sense, crises; an ataxic gait which is increased with the eyes shut; considerable hypotonia, loss of sense of position, of pressure, and of movement; diminution or loss of touch or pain sense and bone sensation; absence of all reflexes; failure of the reaction of the pupil to light, with optic atrophy and possibly oculomotor and abducens nerve palsy; perforating ulcer; Charcot joints.

Differential Diagnosis.—The only disease with which tabes is liable to be confounded is the so-called ataxic form of multiple neuritis or pseudo-tabes. The following symptoms are common in both: Numbness in the limbs; sharp, shooting, lancinating pains; absence of reflexes and ataxia. In multiple neuritis, however, we have a history of either alcoholism or arsenical poisoning, plumbism, etc., and their symptoms, the rapid onset

following the intoxication, wrist drop and toe drop, pain on pressure over the nerve-trunks, reactions of degeneration, and marked disturbances of sensation over the limbs different from the type found in tabes. The following symptoms of tabes are also never present: irregularity of the pupil or the Argyll-Robertson pupil and disturbances of the bladder and rectum. Again, in multiple neuritis the patient will in most instances fully recover.

Occasionally it will be difficult to diagnose this disease from general paresis. In both there is a history of syphilis, and there may be the same pupillary, sensory, and reflex phenomena; but there are, in addition, in general paresis mental symptoms, such as change in disposition and ideas of grandeur, tremor of the facial muscles and sometimes of the limbs, and difficulty or tremulousness in speech. As a rule, there is not in paresis the regularity of symptoms which is so characteristic of tabes, and not much difficulty should be experienced in differential diagnosis.

Clinical Course and Complications.—The prognosis in any given case must depend upon the character of the onset, whether the patient has been given anti-specific treatment, and the severity with which the symptoms appear. As a rule, if the patient has been thoroughly treated for syphilis the disease will be longer in its appearance. There are, however, exceptions to this. The earlier the symptoms appear, the more severe is the disease likely to be. While, as a rule, the symptoms of tabes appear in regular order, sometimes there may develop an undue amount or an early hypotonia and ataxia. If this be the case, the probabilities are that the patient will be so incoördinate that it will be difficult to educate him to walk. Again, in the so-called form of optic tabes blindness will appear early, but the ataxic symptoms will almost subside.

It must be remembered that the disease is chronic and progressive in most instances, but occasionally cases are met with in which there has been an undoubted arrest of symptoms. It is not unusual for the symptoms to subside in from five to twenty years. The prognosis in large part is influenced by the treatment, for should this be carefully instituted, a subsidence of symptoms is more likely to occur.

The complications which may arise in the course of this disease are those which are liable to occur as a result of any syphilitic infection. There may be added to the tabetic process, a wide degeneration of the whole cerebro-spinal axis, thus producing the disease, general paresis. Again, there may be a diffuse syphilis or a spinal meningitis. In rare instances a hemorrhage may occur, causing hemiplegia, and still more rarely there may be a cavity formation in the central portion of the spinal cord, adding the symptoms of syringomyelia. There may also rarely be an inflammation of the peripheral nerves.

MYELITIS.

Definition.—By this is meant inflammation of the substance of the spinal cord. It may be produced by a great variety of causes, such as direct injury the result of bullet or stab wounds, pressure from a dislocated or diseased vertebra, and tumors of the meninges whether intradural or extradural. Inasmuch as in these instances the myelitis is secondary, its symptoms will be discussed under separate headings, and under myelitis will be discussed only those forms which are the result of causes not already mentioned.

Pathologically there are many different forms of myelitis. The inflam-

mation may involve the whole transverse section of the cord (transverse myelitis), it may affect only the central gray matter (central myelitis), or irregular areas in the white or gray matter (disseminated myelitis). Histologically the inflammation may be confined principally to the parenchymatous structures or blood-vessels, or it may assume the characteristics of both. Myelitis also occurs as the result of a thrombosis or embolism of the vessels, and will produce a necrosis or death of the parts from which the blood-supply has been cut off. Such a condition is known as myelomalacia.

If the meninges of the spinal cord are involved, the disease is known as meningomyelitis. As a rule, such a condition is the result of a syphilitic inflammation or an extension of the pathologic process from the vertebra, as in vertebral carcinomatosis. It is possible for the pia alone to be diseased or there may be inflammation also of the dura.

Myelitis may also be divided, according to the onset of the symptoms, into acute, subacute, and chronic.

Contributing and Exciting Factors.—There are many causes for acute myelitis. It may result from the transmission of the purulent process from small abscesses in the periphery, from gonorrheal inflammations, general septic processes, exposure to cold and wet, or may occur in the course of or follow many infectious diseases, such as typhoid fever, scarlet fever, influenza, grippe, etc. It sometimes occurs without any apparent cause.

Symptoms.—Acute Transverse Myelitis; Thoracic Cord.—The onset is usually rapid, and there may be a rise in temperature with headache and a general feeling of malaise. There is usually first a feeling of numbness or a tingling in the lower limbs, which is followed by weakness, which may proceed in a few days to total paralysis. As most cases of acute myelitis occur in the dorsal area and are completely transverse, there will be, besides paralysis of both lower limbs and of the lower abdominal and gluteal muscles, at first retention, followed by dribbling and later complete loss of urinary function, with incontinence of feces and total anesthesia for all forms of sensation in both lower limbs and lower part of the abdomen, the skin area corresponding to the acute transverse lesion. There may also be a band-like area of hyperesthesia corresponding to the segment of the spinal cord which is diseased. In most cases the acute loss of motion and sensation is followed by improvement, and very soon there will be return of sensation over the lower limbs and abdomen, and later return of power, and instead of complete flaccidity with loss of reflexes, which first occurs, there develops spasticity with a gradual increase of tendon reflexes, patellar and ankle clonus, and the Babinski phenomenon. The bladder and rectal functions also improve. The further course of the disease is chronic, but the patient may recover sufficient power to be able to walk; as a rule, atrophy and contractures develop, and there may sometimes be electric reactions of degeneration. If the patient is bedridden, bedsores may develop and skin eruptions may occur because of the general impairment of nutrition and of trophic functions.

If the acute transverse myelitis occurs in the *cervical cord*, there will be, besides the symptoms above enumerated, paralysis of motion and loss of sensation in the upper limbs; and if the lesion is high enough, paralysis of some of the muscles of the neck and diaphragm.

If the lesion occurs in the *lumbar cord*, the symptoms will be similar to those in the dorsal region, with the exception that there will not be any weakness or anesthesia in the abdominal muscles. The subsequent course is the same.

If the meninges are involved, as in meningomyelitis, there will be, in addition to the symptoms above enumerated, pains of a sharp, shooting character, and especially girdle sense. As a rule, however, a meningomyelitis is nearly always of syphilitic origin, and will be discussed under that head (Fig. 415).

Disseminated Myelitis.—Because of the fact that in this form the areas of inflammation are diffuse, and may occur in any portion of the spinal cord, there can be no regularity of symptoms, and these necessarily depend upon the parts of the cord diseased. As a rule, disseminated myelitis follows some infectious disease, pyemic process, or abscess of the periphery. The onset is nearly always gradual, and there may or may not be premonitory or febrile symptoms. Because of the fact that nearly always there is a predominant involvement of the motor columns, and that motor symptoms are promptly appreciated, the first symptoms may be those of weakness of motion of one or both lower limbs. The weakness increases, and is nearly always accompanied by exaggeration of the tendon reflexes, sometimes with ankle and patellar clonus and the Babinski phenomenon. Rarely the reflexes may be absent, provided the areas of inflammation are located so as to interfere with their arcs. As a rule, there will be disturbance of sensation, irregularly distributed over the lower limbs, abdomen, and chest, and sometimes the upper limbs, and there may be numbness and pain, sometimes shooting in character, and girdle sense. Vesical and rectal disturbances are nearly always early in their onset. Gradually the weakness, spasticity, and increased reflexes are followed by atrophy and contractures. The upper limbs may be similarly involved in time.

Fig. 415.—Contracture with Inability to Move the Limbs in Myelitis.

As in disseminated sclerosis, there is nearly always involvement of different portions of the brain, the relative symptoms depending upon the extent and location of the pathologic process. The duration of the disease varies, but, as a rule, it is fairly rapid; that is, it may extend over a number of months, but hardly ever longer than a year. As a rule, disseminated myelitis terminates in death, but sometimes the pathologic process may subside and leave multiple areas of sclerosis.

Subacute and Chronic Myelitis.—Under the former is understood a form of myelitis which develops in a number of months, while under chronic

myelitis is understood inflammation of the spinal cord which comes on over a longer period—a year or more. As a matter of fact, these classifications are arbitrary, and it is better to class myelitis, not according to the length of the onset, but according to the pathology or the extent of the involvement of the spinal cord. There is, however, a form of myelitis in the aged known as senile myelitis which comes on slowly and will be discussed under that head.

Senile Myelitis.—Sometimes in the latter end of life, especially in persons who are arteriosclerotic, there occurs a gradual diminution of power in the lower limbs, with numbness, pin or needle-like sensations, or a dead feeling, and some disturbance of bladder function. These may be accompanied by increase of reflexes, and only rarely by the Babinski phenomenon. It is characteristic of this form of myelitis that the symptoms grow better and worse, and extend over a long period of years, terminating in more or less complete weakness of both lower limbs, with increase of reflexes, some disturbance of sensation, and pains of varying character.

The symptoms here described are similar to those which occur under the symptom-complex known as *intermittent claudication*, and are dependent upon a gradual lessening of the blood-supply of the spinal cord because of the closing up of the lumen of the vessels due to a gradual developing arteriosclerosis. The intermittent symptoms depend upon the occasional shutting off of the blood-supply. When the arteries are finally closed up, there develops myelomalacia or death or softening of the part, because of abolition of the blood-supply.

Caisson Disease or Diver's Palsy.—By this is understood a form of paralysis affecting those persons who work under a heightened atmospheric pressure, as divers, and who suddenly return to the normal pressure. It usually affects those persons who are old or alcoholic or who return too suddenly to normal conditions. There may be headache, dizziness, flashes of light with pains in the limbs or abdomen, or difficulty in breathing, or there may be complete coma and unconsciousness. In the course of an hour or more there may develop weakness of one or both lower limbs, which may at first be flaccid in character with loss of reflexes, and later become spastic with increase of reflexes. There may also be involvement of the bladder and rectal functions and disturbance of sensation. Sometimes the paralysis only lasts for a short time, and there may only result little disturbance of motion, but rarely the paralysis is permanent. Pathologically are found diffuse areas of inflammation and softening in various parts of the spinal cord and sometimes in the brain and air-bubbles in the myelin.

Serous Myelitis.—Sometimes all of the above symptoms of myelitis may be caused by pressure due to an increase in the cerebrospinal fluid, the result of a serous meningitis. It is, however, impossible to recognize this, for the symptoms are identical. Lumbar puncture, however, will demonstrate a great increase in the quantity of fluid and a heightened tension in the spinal canal.

Summary of Diagnosis.—The symptoms will depend upon the form of the myelitis, whether transverse or disseminated, and upon the location of the lesion, whether in the cervical, thoracic, or lumbar cords. If acute, transverse, and in the cervical region, there will be complete paralysis of movement of all four limbs, of the thoracic and abdominal muscles, and of the diaphragm, and loss of sensation in an area corresponding to the cervical segment involved, with incontinence of urine and feces.

If in the thoracic region, the paralysis will involve only the lower limbs,

abdominal and part of the thoracic muscles, and the sensory disturbance will only extend up to the thorax.

If the lesion is in the lumbar region, the paralysis of motion and sensation will only involve the lower limbs.

If the myelitis is disseminated, the symptoms will be gradual in onset, disturbance of motion and sensation may involve first one limb and then the other, or both at the same time, this gradually increasing and lasting over a number of months, either terminating in death or persisting with the symptoms of spastic ataxia of the lower limbs and sometimes of the upper.

The diagnosis of senile myelitis can be made upon the gradual onset of weakness and disturbance of sensation with numbness in the lower limbs coming on in an old man.

Differential Diagnosis.—There should be no difficulty in diagnosing acute transverse myelitis. It is sometimes necessary, however, to diagnose this from a similar involvement resulting from pressure backward of the vertebra, or a myelitis resulting from tumor. In disease of the vertebra, however, there will always be a history of an injury or previous disease, whereas in tumor there will be the gradual onset of the symptoms with pains suddenly terminating in acute myelitis.

Disseminated myelitis is sometimes difficult to diagnose from a similar pathologic process occurring in syphilis. In the latter, however, there may be a history of the disease, and there are nearly always irregularities of the pupil with disturbance in their reactions, and sometimes early and transient ocular palsies.

Clinical Course and Complications.—These have been indicated in the description of the various diseases, and need no further discussion.

TUMORS OF THE SPINAL CORD.

In comparison with tumors of the brain they are rare. They may be extradural, intradural, or involve the spinal cord, but are mostly intradural, tumors of the cord being most rare. Pathologically they may be sarcoma, fibroma, glioma, carcinoma, or cystic. Tubercular and such other tumors as psammoma, myxoma, endothelioma, and lipoma rarely occur.

Sarcoma.—Sarcoma of the cord itself is rare, and is secondary to that of the vertebra when it involves the anterior and especially the posterior roots or may infiltrate in the pia. Generally the lower portion of the spinal cord, and especially the cauda equina, is the seat of multiple sarcomata. Their characteristics have already been discussed, and it must only be remembered that sarcomata may be soft and infiltrating, and because of this may give only few symptoms.

Fibroma.—These tumors are mostly intradural and grow in the pia or about the roots. As a rule, they are not multiple, and are favorable for operative removal.

Cysts.—These may be limited to the meninges and be simple, or may be multiple, as occurs in cysticercus cellulosa and in echinococcus cysts.

Circumscribed Serous Meningitis.—Recently there has been observed a circumscribed serous collection of fluid within the pia-arachnoid which may at times be of large size. The differential diagnosis between such cases and tumor is very difficult, the only difference, perhaps, being in the variability of the symptoms, inasmuch as the pressure of the fluid upon the different roots may alter from day to day, this giving especially a vari-

ability of sensation and reflexes, whereas in tumor these changes are permanent.

Symptoms.—These will depend upon the location of the tumor and extent of the involvement, either of the meninges, root, or cord. As a rule, most tumors are located in the thoracic cord, and are generally situated about the lateral and posterior surfaces. It is impossible to state definitely what symptoms may occur in tumors, but they are due to involvement either of the roots or of the spinal cord itself.

Root Symptoms.—Numbness, pain, or girdle sensation, to be followed by pain, are usually among the first manifestations, these being referred to the parts in relation with the posterior root diseased. As a rule, the pains are sharp, shooting, and agonizing in character, and may be of such intensity as to prevent the patient from moving. If the growth involves several posterior roots, and is large, there may be tenderness and pain on pressure over the involved part, and jarring may sometimes cause excruciating pains. Later there may be an accompanying disturbance of sensation.

Cord Symptoms.—As the spinal cord itself becomes involved, its accompanying symptoms will develop, such as disturbance of sensation if the posterior part of the cord is affected, and if the lateral columns are compressed there will be weakness, spasticity with increased reflexes, and the Babinski phenomenon in the parts below. Of course, if the tumor is around the anterior part of the cord the symptoms will be purely motor. This, however, is uncommon.

Tumors of the Cauda Equina.—These are generally sarcomatous and multiple. The symptoms will depend upon what roots are involved. There will usually be pain, referred to the sciatic distribution of one or both sides, or possibly a localized pain in the lower part of the back; but the most distinguishing feature is the segmental disturbance of sensation around the buttock, perineum, and anus, and the anesthesia in the genital organs. There may also be involvement of bladder, rectal, and sexual functions. Paralysis is not very common, but if it occurs will usually be in the distal portions of the limbs.

DISEASES OF THE VERTEBRA.

These may be divided into diseases of the vertebra itself and those the result of growths. Diseases of the vertebra are generally tubercular, starting either in the spongiosa, periosteum, lamina, spinal processes, or ligaments. The process may grow from one or several locations at the same time, and may involve the spinal cord secondarily in three ways: first, by direct extension of the tuberculous process from the vertebra, the tuberculous masses involving the dura and then the spinal cord; second, by direct pressure the result of a backward displacement; and, lastly, by so-called toxic action. It is probable that in the last classification the spinal cord itself becomes diseased through the disturbance of the lymphatic and arterial circulation. The symptoms caused by tubercular disease of the vertebra or Pott's disease are identical with those caused by growths, and will be discussed under that head. Syphilitic caries rarely occurs.

Tumors of the Vertebra.—These are generally sarcoma or carcinoma, or may be the result of a growth of the bone-marrow, when they are called myeloma. The earlier mentioned tumors are nearly always secondary to growths elsewhere, generally from the lungs, stomach, breast,

or uterus. Such benign growths as osteoma or enchondroma may sometimes occur.

Symptoms.—These will be divided into, first, those which are the result of diseases of the vertebra itself; second, those resulting from involvement of the spinal roots and cord.

Symptoms Due to Affection of the Vertebra.—In nearly all cases, whether the tumor affects the vertebra, lamina, or processes, there should be some displacement and deformity of the vertebra, especially in caries. It must be remembered, however, that both in this condition and in tumors there may not be the slightest evidence of deformity, and the first symptoms will be those the result of pressure on the roots. In most cases besides this evidence of deformity there will be pain over the affected parts, this causing a stiffness and a rigidity of the back and neck with accompanying awkwardness in movement.

Root and Spinal Cord Symptoms.—As the disease progresses, pressure will be first exerted on the posterior roots, and the first symptoms will be those of numbness, to be followed by sharp, lancinating pains with girdle sense, referred to the distribution of the diseased roots. Any jarring of the back or pressure will bring on a fresh attack or exaggeration of pain. As the disease involves the dura and the cord there will be added disturbance of sensation and of bladder and rectal functions, weakness in the limbs with increased reflexes, spastic condition, and Babinski phenomenon. The extent of the paralysis will, of course, depend upon the amount of involvement, sometimes there resulting complete myelitis and total paralysis.

INJURIES OF THE SPINAL CORD.

The result of any injury, no matter how trivial, cannot be foretold. There may be, first, a sprain or injury to the ligaments of the vertebral column, either with or without injury of the cord; second, fracture or dislocation of the vertebra or both, either with or without involvement of the spinal cord; third, injuries to the cord itself; and, lastly, the so-called traumatic neuroses, which may enter into all the above classifications, and also are independent of these. The symptoms will be discussed in order.

Sprain or Injury to the Ligaments of the Vertebral Column with or without Involvement of the Cord.—This generally results from overstretching of the vertebral column or from some severe muscular effort. A direct injury to the back may cause a contusion of the ligaments. As a rule, the cord itself will not be involved, and the symptoms will be those of pain localized to the affected parts with accompanying rigidity of the back and pain on movement. There should be no difficulty in making this diagnosis, were it not for the fact that in medico-legal cases there may be present the symptoms of a traumatic hysteria which may resemble injury of the cord, or there may be, what not infrequently occurs, multiple small hemorrhages or areas of softening in various portions of the cord, and sometimes hemorrhages into the substance.

Fracture or Dislocation of the Vertebra or Both, Either with or without Involvement of the Spinal Cord.—Fractures of the vertebral column are usually associated with dislocation and are the result of severe injuries. They generally occur either between the atlas and axis, the fifth and seventh cervical, or the first and second lumbar vertebræ. In nearly all cases the spinal cord is involved, the only exception being when the frac-

ture is of mild nature and causes no deformity, or if there has been a fracture of limited degree involving the lamina or processes.

Symptoms.—There are usually present the surgical evidences of injury, for here, as elsewhere, the fracture may be simple or compound. Besides there will be evidences of deformity of the vertebral column with rigidity and pain in the back. The symptoms will depend largely upon the place of injury. If in the cervical cord, the principal symptoms will be in the upper limbs; if further down, as in the lumbar region, they will be limited only to the lower limbs.

Injuries to the Cord.—In nearly all cases where the injury has been severe enough to cause a fracture or dislocation of the vertebra, the cord itself will be severely damaged. This may be either because of a direct pressure exerted upon the cord, the result of backward displacement of the vertebra, or, as sometimes happens, there will be at the time of injury a sudden torsion or twisting of the spine, this causing momentary pressure upon the cord with destruction of its elements. Again, there may be severe injury to the cord, but no evidence of fracture or dislocation, or even sprain or contusion of the ligaments, with either multiple small hemorrhages or softening, or one large hemorrhage.

The symptoms of compression of the cord will not differ from those described under the head of myelitis. As a rule, the destruction will be intense, and transverse myelitis and sometimes complete severance of the cord may result, this causing complete loss of power and of sensation in the parts below, with the bladder, rectal, and trophic symptoms. It must also be remembered that, besides the direct destruction of the cord, the result of the injury, there will also be multiple small areas of hemorrhage above and below the point of injury. If there is only a partial destruction there will be, after the initial complete paralysis of motion and sensation, return of sensation, and then of motion, with increased reflexes, spasticity, Babinski phenomenon, and disturbance of bladder and rectal functions.

Hemorrhages into the cord, or **hematomyelia,** may occur with contusion of the substance of the cord, or independently of this as a result of injuries, without an accompanying fracture or dislocation of the vertebra. As a rule, hemorrhages occur into the substance of the cord, mostly in the central gray matter, and only very rarely in the outer or inner surface of the dura. . The gray matter of the cord seems to be easier to infiltrate than the white matter, and as a consequence any hemorrhage may involve considerable length of the cord. The symptoms will, of course, depend largely upon the location of the lesion, whether within the cervical, thoracic, or lumbar parts, and upon its extent. As the hemorrhage involves principally the middle portion of the cord, it will interrupt the fibers concerned with transmission of pain and temperature sensations, and there will result the so-called syringomyelic disturbance of sensation in the lower limbs; *i. e.*, loss of pain and temperature sensations with preservation of touch. Besides, there will be weakness with spasticity, increased reflexes, and the Babinski phenomenon, and if the hemorrhage involves the cells of the anterior horn, loss of power with atrophy and reactions of degeneration in the related parts.

If, however, there should be multiple microscopic areas of hemorrhage or softening, no definite symptoms will result, because there has not been sufficient injury to cause disturbance in function unless the injury occurs where marked arteriosclerosis is present, when severe hemorrhages or

softening may be brought on any time through the weakening of the vessel walls.

The Prognosis of Injuries of the Spinal Cord.—This will, of course, depend upon the nature and extent of the injury. If the cord has been severely crushed for several segments, there can be no hope for return of function. If the injury has been partial, some return of power will always result. If a hemorrhage has occurred in the central gray matter, there should be some return of power; and if there are multiple microscopic areas of hemorrhage or softening, complete recovery may ensue. In all of these instances the prognosis depends entirely upon the possible regeneration of fibers in the spinal cord, and this has been the subject of controversy for a long time. It is probable that this cannot occur, and whatever improvement results is because the fibers which have been injured have recovered from whatever traumatism they have undergone. In every injury there is a certain amount of shock which will temporarily injure the cord, but unless a complete severance or myelitis ensues there should nearly always be some return of function.

UNILATERAL SPINAL CORD LESIONS, OR BROWN-SÉQUARD PARALYSIS.

Sometimes tumors or injuries resulting from bullet or stab wounds will cause a unilateral lesion of one or two segments of the spinal cord. The symptoms will depend upon the part of the cord involved and the extent of the lesion. Should, for instance, there be a unilateral lesion in the eighth cervical and first thoracic segments of the right side of the cord, there will be the following symptoms: Because of the destruction of the nerve-cells in the anterior horns, inability to flex or extend the right wrist or move the fingers, besides atrophy and electric reactions of degeneration in these parts. Because of the involvement of the right motor or pyramidal column, weakness, spasticity, increased reflexes, and the Babinski phenomenon in the right lower limb. Because of the destruction of the sensory roots, loss of all forms of sensation along the under surface of the whole right arm. As the posterior columns transmit the fibers for touch sensation and muscle sense, there will be disturbance of touch in the right lower limb and right abdomen and chest with impairment of muscle sense and ataxia in the right leg. In the left lower limb there will be disturbance of pain and temperature sensations only, because of the destruction of the right column of Gowers (Fig. 416).

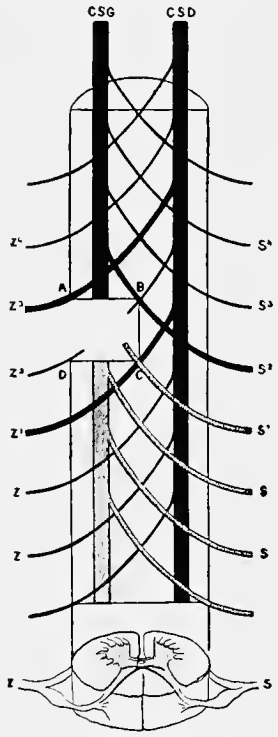


FIG. 416.—SCHEME REPRESENTING CORD-LESION AND EFFECTS IN BROWN-SÉQUARD PARALYSIS (after Brissaud).

CSG, Left sensitive tract; CSD, right sensitive tract; A, B, C, D, lesion involving the left half of the cord; S, S, S, sensory roots from right side of body; Z, Z, Z, sensory roots from left side of body; Z^1, Z^3, and S^2 are irritated only at the points A, B, C, and their peripheral area is hyperesthetic; Z^2 is divided and its skin area is anesthetic on the same side as the lesion. Corresponding to S^1 and all the roots below arising from the right side of the body, there is anesthesia.

SPINA BIFIDA.

A defect in the closure of the posterior vertebral arches, especially in the lumbar and sacral region. It is of embryonal origin, and is usually detected at birth or very soon after, and rarely may interfere with it. The defect may consist only in a lack of union of the posterior vertebral arches, but, as a rule, there is a tumor-like projection in the lower spine, which may consist only in a protrusion of the dura, which may be from the size of a nut to that of an orange or larger and be filled with cerebrospinal fluid, or there may be, in connection with the dural protrusion, an involvement of the spinal cord itself, consisting either in an enlargement of the central canal, a hydromyelia or attachment of the lumbosacral cord or its roots to the walls of the sac.

Symptoms.—Most cases of spina bifida die either at birth or soon after. When there is only a dural involvement, there may be no symptoms besides the physical evidences of protrusion. Pressure, however, upon the sac will cause bulging of the fontanels with the symptoms of cerebral compression. If, however, the cord itself be involved, there will be paralysis of both lower limbs and disturbance of bladder and rectal functions and of sensation. The disease is of long duration, the symptoms having a tendency to increase, and the prognosis is not very good. There are usually in association embryonal defects elsewhere, such as cleft palate or harelip.

DISSEMINATED OR MULTIPLE SCLEROSIS.

Definition.—A disease of the brain and spinal cord characterized by progressive weakness, spasticity, increased reflexes and ataxia of the limbs and trunk, with tremors which become worse on effort, scanning speech, and nystagmus.

Pathologically there are multiple areas of sclerosis throughout the whole brain and spinal cord. The sclerotic patches involve equally the gray and white matter and the cranial nerves. The sclerosis in this disease differs from that of any other in the fact that there is no resulting secondary degeneration. The myelin sheaths only are involved and the axis-cylinders escape. It is because of this that there may be little alteration of function although the sclerosis is extensive.

Predisposing and Exciting Factors.—There are many theories as to the origin of the multiple areas of sclerosis, but it is probable that they are due to faulty development of the nervous system. Sometimes the disease appears in several members of a family, and in rare instances in father and son. The exciting causes are not known, but in not a few cases the symptoms seemed to have developed after exposure to cold and wet. Rarely the symptoms are due to multiple syphilitic lesions. The disease is rather rare in this country, but on the Continent it is as common as tabes.

Symptoms.—Inasmuch as the multiple areas of sclerosis may occur in any portion of the nervous system, and that in no two cases is the pathologic process alike, the symptoms must necessarily vary in each case. There are, however, certain general symptoms which are present in nearly all, and these are a tremor which is intention in type, scanning speech, nystagmus, spastic ataxia of the lower and upper limbs, and less commonly optic atrophy.

The disease nearly always begins in early adult life or around the twentieth year, with weakness or spasticity of one or both lower limbs, which gradually increases. Coincident with this or soon after there may develop

PLATE XXII

Moving Picture Illustrating Tremor of Head, Body and Limbs in Multiple Sclerosis.
(Courtesy of Mr. Sigmund Lubin of Philadelphia, Pa.)

ataxia of one or both lower limbs, and the gait, which at first may have been spastic, will become somewhat ataxic, the patient staggering from one side to the other much like a drunken man. The tendon reflexes of the lower limbs become progressively exaggerated, and the Babinski reflex is common. Often patellar and ankle clonus is present. It is not at all unusual for the spastic symptoms to be greater in one lower limb, and these symptoms may also be found in the upper limbs.

Sometimes numbness and pains are present in one or both lower limbs, and rarely girdle sense and disturbance of sensation.

Coincident with the spastic ataxia of the limbs there develops a tremor which is intention in type, that is, it becomes worse on effort. This tremor may, in mild cases or at first, involve only the upper limbs, but later all the limbs may be affected. In fact, in severe cases the tremor involves the head, neck, and the whole body, interfering with locomotion, eating, and talking. When the patient is quiet, there may be no tremor present, but the slightest excitement or movement brings it out (Plate XXII).

Disturbance of speech is an early symptom, and is usually described as scanning. The words are uttered slowly, sometimes explosively, the patient having a manifest difficulty in getting a start, although after beginning there is not so much hesitancy. The speech becomes slower and more difficult as the tremor progresses. Often it resembles the speech of a person shivering with cold.

Nystagmus is common and may consist of only a few to-and-fro movements, and sometimes may not be apparent except on deviation. It is less constant than the scanning speech or the intention tremor.

Disturbances of vision are very common. These may consist in a central scotoma for colors or a contraction of the visual fields, and optic atrophy frequently results. Pallor of the optic discs, especially on the temporal side, is characteristic of this disease, and is found in probably one-half the cases.

If the areas of sclerosis are limited to the spinal cord, there may not be present the intention tremor of the head, nystagmus, ocular symptoms, or the scanning speech. If the sclerosis involves the gray matter of the lumbar and sacral cord, there may be loss of the knee and Achilles jerks and interference with bladder and rectal functions, but this is unusual.

In not a few instances the first symptoms may be those of hysteria, and it is difficult to establish a diagnosis. Gradually with the hysterical symptoms there develops spasticity of the lower limbs with increased reflexes and the Babinski phenomenon, and later the intention tremor, scanning speech, and nystagmus.

Summary of Diagnosis.—Weakness, spasticity, increased reflexes, and the Babinski phenomenon in both lower limbs, spastic ataxic gait, intention tremor of one or all the limbs and of the head and neck or of the whole body, scanning speech, nystagmus, optic atrophy, and pallor of the temporal side of the discs. The symptoms differ in each individual case, but the cardinal symptoms are spastic ataxia of the limbs, intention tremor, scanning speech, nystagmus, and optic atrophy.

Differential Diagnosis.—In the onset of the disease it is difficult to differentiate the spastic symptoms from those occurring in lateral or amyotrophic lateral sclerosis or from myelitis, but as the disease develops the intention tremor and scanning speech establish the differential diagnosis. The hysterical symptoms may for a long time mask the disease, but later the cardinal symptoms develop.

Clinical Course and Complications.—The course of the disease is usually slow. Usually the spastic symptoms increase slowly, but the intention tremor and the scanning speech grow progressively more and more marked, and the tremor in some cases may become extreme. Remissions in the disease are very common, and it is because of this that it is sometimes difficult to establish a diagnosis. Later in the disease the patient becomes bedridden, and the paralysis of the limbs will become extreme. Death usually occurs from some intercurrent cause.

SYPHILIS OF THE NERVOUS SYSTEM.

It is of the utmost importance to make the diagnosis of syphilitic lesions of the nervous system as early as possible, for it is in the primary stages that therapeutic measures are of most benefit. That the diagnosis of such lesions presents many difficulties is evident, since there is no part of the nervous system that may not be attacked. Certain symptoms are present, however, in nearly all cases, and it is principally by the recognition of these that we are able to make a proper diagnosis. To better understand what symptoms are possible in syphilis, an effort will be made to trace the course and nature of the disease and the method of involvement of the nervous system.

Method of Infection of Nervous System.—In only a few of the large number of persons affected does the nervous system become secondarily diseased. The important question, then, is, Why should one person have his nervous system affected and not the other, and what influence, if any, does treatment have upon the prevention of such involvement?

Repeated instances have been recorded in which a number of persons, not at all related, have become infected from the same source, and subsequently developed general syphilis, and, later, specific disease of the nervous system. Here there are two possibilities: one, the most probable, that the specific infection was of such character that it had a special affinity for these structures; and, second, that there was lessened resistance of the nervous system to the infection. It has always been one of the old theories that only certain forms of specific intoxication will cause disease of the nervous system; the best proofs of this have been such instances as quoted above. That such is the case there can be no doubt, but such a theory will hardly answer for all cases. There is no question that specific manifestations at the present time are not as severe as they have been. This is due partially to the better recognition and earlier and better treatment, and also to the fact that all diseases have a tendency to lessen in intensity in the course of time.

It is probable that the most important factor which determines whether the infected person will have future involvement of the nervous system is the lessened resistance of these structures, or possibly a special predisposition. Just what determines this is, of course, difficult to tell, but it must be that certain parts of the anatomy are less resistant than others. Hereditary and family influences contribute to this to a large degree. Erb records instances of specific infection in families in which all had tabes dorsalis following infection from the same source.

Frequency of Disease.—It is important to know in what percentage of cases of syphilis the nervous system becomes diseased. This can never be determined with accuracy, and we must rely principally upon the statistics to be obtained from institutions. Erb, for instance, collected some years ago a history of many cases of specific infection, and of these,

in about 1 to 5 per cent. tabes followed. In how many, however, there were other involvements of the nervous system is not recorded, but judging from the fact that tabes is no more frequent than other cerebrospinal diseases, it is probable that a fair percentage would be about from 5 to 10 cases in 100. This, of course, is only conjecture.

It is also a question whether or not early treatment of the disease will prevent such future involvement. That it does so in the majority of instances there is no doubt, but in many it is of no avail. It is, however, in the more chronic cases, or in those in which the symptoms of cerebrospinal involvement appear many years after the infection, that this question arises. In many, anti-specific treatment had been instituted and carried out vigorously, and the patient pronounced well, only to have some part of the nervous system become diseased years afterward. The conclusion is forced upon us that in some forms of syphilis the cerebrospinal system will become diseased whether or not treatment is instituted, and no matter how vigorously.

After an infection, the nervous system may be involved almost immediately, in the midst of treatment, or may not for some years, sometimes as many as twenty. It is generally supposed that the earlier the cerebrospinal symptoms appear, the more severe the infection. Considering, however, that the symptoms produced years after the infection are about of the same severity and character as they are in the early periods, there is probably little difference.

Pathology.—Pathologically, in the early forms, no matter where the nervous system is involved, there is always a round-cell infiltration about the blood-vessels and in the pia, this being especially true of the base of the brain and the posterior part of the spinal cord; and there is also a secondary endarteritis and permanent thickening of the meninges. Rarely, small gummas will be found, but these are not as frequent in the nervous system as is commonly supposed. More rarely, there is also an involvement of the substance of the cord or brain. When such is the case, in the former there is very often an extensive inflammation, this resulting in a myelitis; if in the brain, the cortex and subcortex are diseased, this causing the symptoms of diffuse encephalitis. It is characteristic of these acute inflammations, such as myelitis and encephalitis, that the symptoms appear suddenly.

When the disease comes on many years after the infection, the usual pathologic findings consist in a sclerosis of the vessels and thickening of the meninges, and sometimes in gummas, but it is principally to the results of the endarteritis that the symptoms are due. There are, however, other forms of disease resulting from specific infection which are caused, not by inflammation, but by gradual changes in the nervous structures, thought to be the result of toxins. These changes mostly occur in structures which have more or less functional relation, and are called systemic, the best example of this being locomotor ataxia or tabes dorsalis.

Symptoms.—It can be readily understood, then, that, so far as the symptoms of specific disease of the nervous system are concerned, the majority of cases will show an involvement of both the brain and spinal cord, and that the focal symptoms will depend upon what particular portions of these structures are most diseased. No matter what the extent of the affection, there is in every case an infiltration of the meninges, and this in itself will nearly always give certain symptoms. There is a tendency, however, for certain forms of involvement in those cases in which the symptoms manifest

themselves within a few years after the infection, and these will be discussed first.

Early Symptoms.—*Myelitis.*—Among the earliest is a myelitis, this usually coming on suddenly, and in most instances affecting the lower thoracic or thoracic-lumbar region, and often giving the symptoms of a complete transverse myelitis, there being loss of power in both lower limbs, of the bladder and rectal functions, and of sensation. In the majority of cases some return of power can be hoped for, especially if vigorous treatment is promptly instituted. Added to the symptoms of the myelitis there will be those of meningeal involvement, such as girdle sense and pain.

Cerebrospinal Involvement.—Other common early manifestations, sometimes appearing in the midst of treatment, are those the result of multiple lesions of the brain and spinal cord, giving diffuse symptoms depending upon the location of the lesions. There will nearly always be motor and sensory involvement of irregular character, with the addition of meningeal symptoms of the base of the brain and of the cord.

Hemiplegia.—Hemiplegia is also common in early syphilis, this resulting from early endarteritis. In fact, hemiplegia occurring in young adults below forty years of age is nearly always syphilitic in origin; but the most common manifestations of syphilis are those the result of meningeal involvement of the base of the brain or of the spinal cord.

Meningitis.—Meningitis of the base of the brain is nearly always greatest around the chiasm, and it is because of this that the optic, oculomotor, and sixth nerves are commonly diseased in syphilis. Whether it is because of this involvement of the optic nerves, or because the specific toxin has a special predilection for the iridic muscles, disturbances in the reactions of the pupils with irregularities in their margins are the most common and constant symptoms of syphilis. Such general diseases as syphilis nearly always affect those structures which are most commonly used, and it is, perhaps, because of this that the iridic muscles are so early diseased. In most instances there will be found irregularities in the margins, with a loss of the reaction of the pupils both to light and to movement of the eyeballs, and sometimes, in the later stages, a failure of response of the pupils to light, with preservation to movement of the eyeballs, or the Argyll-Robertson pupil. There is no disease which will so constantly give these symptoms, and there are no symptoms which are so constant or which can be so much depended upon.

Oculomotor palsy, unilateral in type, occurs most frequently in syphilis. In fact, it has been termed its *sign manual.* Of the cranial nerves, however, the sixth nerve is probably the most frequently diseased, it causing double vision. This, as a rule, lasts only a very short time, appearing and reappearing, until finally it will be permanent. Paralysis of any of the other cranial nerves is a rare manifestation. Meningeal involvement in the spinal cord is nearly always greatest in its posterior portion. Because of this, girdle sense and pains referred to various parts of the limbs are common.

Late Symptoms.—Of the diseases which have a tendency to become manifest many years after the infection, that is, after the fifth year, while there is in every case an affection of both the brain and spinal cord, there is nearly always a preponderance of the disease in one of these structures. When the brain is mostly involved, there will be, besides the irregularity of the pupils and the slowness of reactions to light and movement, other symptoms depending upon the location of the lesion. These may consist in a gummatous deposit in most any portion of the brain, or of a basal infiltration, besides those diseases resulting from the early endarteritis.

It is characteristic of brain symptoms, just as it is of those the result of basal lesions, that they may appear for a time and then disappear, only to reappear again and remain more or less permanently. In those instances in which the spinal cord is mostly involved there may be diffuse symptoms, but in nearly all there is some meningitis, this causing girdle sense and pains.

Disturbances in bladder and rectal functions, especially the former, are very common in spinal infections, and sometimes may be the only symptoms indicating such disease.

Cases are observed in which there seems to be an equal involvement of the brain and the spinal cord, but even here one of these structures may be more diseased. The symptoms will, as a rule, be multiple, and there will be, besides the specific pupillary phenomena, disturbance of mentality, irregular motor and sensory symptoms, and involvement of the bladder and rectum.

So far, only those cerebrospinal syphilitic diseases have been discussed which are the result of the direct pathologic processes produced by such infection, while other diseases, as general paresis, tabes dorsalis, and posterolateral sclerosis, are supposedly produced by its toxins. Their symptoms, as a rule, come on many years after the infection, at least so far as their appreciation is concerned. It is curious that a person may be infected with syphilis and have no symptoms for a number of years, and then may develop posterior sclerosis. This does not seem logical, and it is probable that if our methods of observation were better, we should be able to demonstrate some symptoms, for the syphilitic toxin does cause early and constant changes.

Serum Tests for Syphilis.—Wassermann Reaction.*—Wassermann in 1905 described a reaction, known by his name, which is of the greatest help in the establishment of diseases of syphilitic taint. Since the original description many modifications have been made, the most important being by Noguchi. At the present time these tests are cumbersome and can only be made by the experienced laboratory worker and cannot be applied by the general practitioner. In view of this fact, and also because the technic is constantly changing, it has been deemed by the writer inadvisable to give a description of such tests, but only to discuss its significance and applicability to therapeutic measures.

These tests can be applied both to the blood and to the contents of the cerebrospinal fluid, and while it is possible to give an opinion in reactions applied to either, it is important, if possible, to give consideration to the cellular elements in the cerebrospinal fluid, the globulin content, and the specific reaction of blood and serum found in the spinal fluid.

It must be understood that there is not always parallelism between the conditions found in the blood and spinal fluid or between the Wassermann reaction and the lymphocytosis of the cerebrospinal fluid. At times the reactions are alike, but not always.

In brief, it is the present opinion that a positive Wassermann reaction means not only tabes, general paresis, or any of the so-called metasyphilitic diseases, but it also indicates active syphilis. This reaction has been obtained in a number of instances in leprosy, in sleeping sickness, and in other diseases, but this by no means lessens its value.

It must be remembered that the application of mercury negatives the reaction, whereas potassium iodid has no such influence, this being an important indication of the value of mercurial treatment of syphilitic diseases.

* Details of the technic of the Wassermann Reaction are given on p. 345.

DISEASES OF THE MENINGES.

The meninges, which envelope both the brain and spinal cord, are divided into the outer coat, or the dura, and the inner, or the pia and the arachnoid. Inflammation of the dura is called pachymeningitis, and of the inner coats leptomeningitis. With the exception of localized inflammations and those following injury, inflammation of the meninges nearly always involves the coverings of both the brain and spinal cord.

CEREBRAL PACHYMENINGITIS.

This may affect either the outer or the inner coat, when it is called external or internal pachymeningitis. *External pachymeningitis* nearly always results from injury to the skull, and is not as common as is usually thought. It may be secondary to a growth of the overlying bone, especially in syphilitic, tubercular, and carcinomatous conditions.

Internal pachymeningitis is rare. It sometimes is hemorrhagic in nature, there being accumulations of blood between the dura and pia, and usually occurs in old persons, especially in those who are either arteriosclerotic or alcoholic. It is rarely found in some forms of insanity. It may be present in conjunction with external pachymeningitis, especially in purulent, syphilitic, and tubercular inflammations.

Symptoms.—The symptoms of pachymeningitis, whether external or internal, are definite, and depend on the pressure exerted on the brain. Generally the patient complains of headache, and rarely of tenderness localized to the inflammatory area, the specific symptoms depending upon the part of the brain involved. If in the motor area, there will be irritative symptoms such as Jacksonian convulsions, which may be followed by more or less paralysis; if over Broca's convolution, motor aphasia; if over the parietal areas, where the pachymeningitis is most common, there may be irritative pains or paresthesia on the other side of the body, accompanied sometimes by disturbance of sensation; if over the temporal lobes, aphasia; if over the occipital convolutions, disturbance of vision on the other side. There may sometimes be loss of consciousness, delirium, or stupor, or there may be no symptoms at all.

Summary of Diagnosis.—History of a preceding injury to the head or of alcoholism, or syphilis, or the presence of symptoms indicative of tumor of the bones of the skull. The symptoms may come on rapidly or slowly, and consist of diffuse headache, possibly unconsciousness, coma or delirium, with irritative phenomena, such as convulsions on the other side, paralysis of various kinds, either partial or complete motor or sensory aphasia; or, what is important, there may be no symptoms at all. It is evident that the diagnosis of a pachymeningitis is very difficult and depends upon the location and character of the inflammation.

SPINAL PACHYMENINGITIS.

Isolated inflammation of the spinal dura without involvement of the membranes underneath is very unusual and its occurrence is doubtful. In some instances, however, the dura is preponderantly involved. As a rule, inflammations of the dura are secondary to disease of the vertebra, as in tubercular, syphilitic, carcinomatous, or sarcomatous inflammations. It is possible to have a syphilitic pachymeningitis without involvement of the

vertebra, but in most of these cases the pia and spinal cord are also diseased. The symptoms of a spinal pachymeningitis secondary to vertebral inflammations have already been discussed under the latter heading.

HYPERTROPHIC CERVICAL PACHYMENINGITIS.

Definition.—An inflammation of the dura of the upper portion of the spinal cord, localized principally to the cervical region, and characterized by thickening of the membranes with pressure upon the inclosed roots and spinal cord.

Pathologically there is a thickening of the dura, which comes on without any apparent cause, and is not secondary to vertebral disease. The dura is thickened throughout its whole circumference and gradually causes a pressure myelitis. An antecedent history of syphilis is sometimes present.

Symptoms.—The disease is progressive, and the symptoms appear gradually. Because of the fact that both the posterior and the anterior roots are involved, and that the spinal cord is ultimately pressed upon, the symptoms will be both sensory and motor. There will be at first pain and paresthetic phenomena referred to the back of the neck, shoulder, and upper limbs, the pains sometimes becoming sharp and shooting. Any sudden jarring in the back of the neck or vertebra will increase the pain, and there will also be tenderness on pressure over the cervical area. The pains and paresthesia increase, and examination may demonstrate segmental disturbance of sensation in the neck, shoulder, and upper limbs. In conjunction with these sensory symptoms, or soon after, there will be fibrillary tremors in various portions of the upper limbs, to be followed by wasting and loss of power. Because of the fact that the ulnar and median portions are principally involved, there will develop a contracture of one or both upper limbs, which is characteristic of this disease, consisting of an acute extension of the wrist upon the hand, flexion of the metacarpal, and extension of the terminal phalanges. Gradually as the spinal cord is pressed upon, there will develop weakness, spasticity, increased tendon reflexes in one or both lower limbs, and ultimately the Babinski phenomenon will be demonstrated. Because of pressure on the posterior and lateral columns there may develop sensory symptoms in both lower limbs, trunk, and abdomen, and ultimately disturbance of bladder and rectal functions. As the pressure upon the spinal cord increases, the tremors, atrophy, and weakness involve all of the upper limbs and the patient becomes helpless.

Summary of Diagnosis.—A gradually progressive disease characterized by numbness or pain in the neck, shoulder, and upper limbs, with segmental disturbance of sensation, to be followed by fibrillary tremors, atrophy, and weakness in the upper limbs, with typical contracture consisting in an acute extension of the hand upon the wrist and flexion of the fingers. This is followed by spastic paresis of the lower limbs, with increased reflexes and the Babinski phenomenon. Later, disturbance of sensation and of bladder and rectal functions.

Differential Diagnosis.—It is somewhat difficult to diagnose this disease, because any inflammation of the dura which ultimately involves the spinal cord will cause the same symptoms. Its principal characteristic is the typical contracture in the hands mentioned. In those cases in which the pachymeningitis follows disease of the vertebra, as in tubercular, syphilitic, and malignant growths, there may be a spinal deformity, and in tuberculous and carcinomatous conditions there may also be a history of similar

growths elsewhere; and in syphilis, involvement of the brain and some pupillary and ocular phenomena.

INFLAMMATION OF THE PIA-ARACHNOID.

Cerebrospinal Meningitis.—In most cases the pia of the brain and spinal cord are involved at the same time, and it is only rarely that either is involved alone. Inflammations may be of various kinds. The epidemic form has already been discussed on page 836. The other varieties are purulent, tuberculous, and serous. Syphilitic meningitis has been discussed under the head of syphilis, on page 826.

PURULENT MENINGITIS.

In most instances purulent inflammation of the meninges is secondary to septic processes elsewhere, such as infected wounds of the scalp or cranium, middle-ear disease, localized abscess of the brain or pia, and general pyemic processes or abscesses in the various parts of the periphery, or secondary to a septic endocarditis or one of the infectious diseases, as pneumonia or typhoid. As a rule, the process involves equally the membranes of the convexity, base of the brain, and spinal cord.

Symptoms.—If the meningitis occurs in the course of an infectious disease, as typhoid, pneumonia, septic endocarditis, or is secondary to pyemic processes, injuries to the head, or middle-ear disease, their accompanying symptoms will be present, and very often the early symptoms of meningitis are masked. As a rule, they come on rapidly, with headache which at times is excessive, and a rise of temperature, the patient becoming delirious, stuporous, and then unconscious. The pulse generally at first is rapid, and then slow and somewhat irregular, and respiration becomes more or less embarrassed. The head is retracted, the back held rigidly, and often the patient assumes a position of opisthotonos. The arms are retracted, the legs are flexed on the abdomen, and any attempt to extend the limbs is met with resistance (Kernig's sign). About this time the irritative phenomena become prominent, and there may be general convulsions, or the spasms may be limited to one or more limbs, which may be followed by partial paralysis or hemiplegia. The reflexes may be exaggerated, diminished, or lost. Because of basilar involvement the pupils become irregular, their reactions impaired, and there may often be swelling of the optic nerve-heads or choked disc. Cranial nerve palsies are common, especially of the sixth, causing diplopia; the third, resulting in ptosis of the upper lid and inability to move the eyeballs; the seventh, paralysis of the face; and of the vagus, interference with the action of the cardiac and respiratory functions and ultimately death. Vasomotor phenomena may be present, consisting in a flushing up of the skin after stroking, described as tache cerebrale.

Summary of Diagnosis.—Headache with gradually developing stupor and unconsciousness, rise of temperature, retraction and rigidity of the head and back with opisthotonos, rigidity, and retraction of the lower and upper limbs, pains and tenderness in various portions of the body, with convulsions which may be general or local, followed by paralysis of various kinds or increased or lost reflexes, pupillary irregularities, choked disc, drooping of one or both upper lids, ocular and facial paralysis, and disturbance of cardiac and respiratory functions. Lumbar puncture will demonstrate pus-cells and increase of lymphocytes and sometimes the specific bacillus.

Differential Diagnosis.—There should be no difficulty in diagnosing cerebrospinal meningitis. It is sometimes impossible, however,

to demonstrate the type of inflammation. This, however, can be readily demonstrated by lumbar puncture. In the tubercular form of meningitis the inflammation is nearly always limited to the base of the brain. In serous meningitis there is not, as a rule, unconsciousness, and the irritative and paralytic phenomena are mild. In the epidemic form the symptoms are like those of the purulent type, but, in addition, there are skin eruptions and the history of an epidemic, although occasionally sporadic cases are found, and the specific bacillus may be isolated by lumbar puncture.

TUBERCULOUS MENINGITIS.

In this type the inflammation is nearly always confined to the membranes of the base of the brain, although there is some involvement of the convexity and of the spinal cord. In nearly all cases the tubercular meningitis is secondary to similar processes elsewhere, especially of the lung, pleura, intestines, or glands. It may occur in adults, but in most instances it affects children below the fifth year.

Pathologically there is found tuberculous inflammation with small miliary nodules. Besides, there is nearly always some serous effusion.

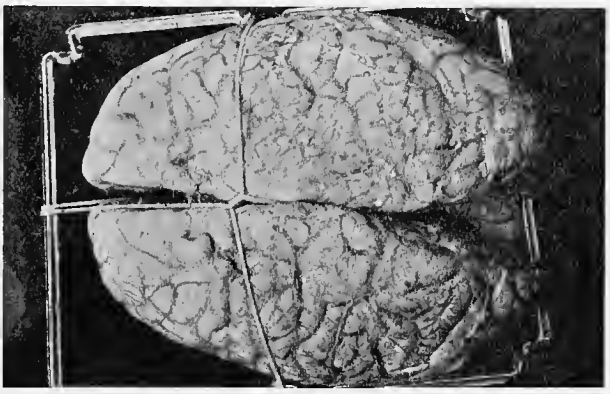


FIG. 417.—BRAIN OF A PATIENT WITH TUBERCULOUS MENINGITIS SHOWING NODULES WITHIN THE PIA.

Symptoms.—When occurring in an adult, there are always the accompanying symptoms of a tubercular inflammation elsewhere, either in the lung, pleura, or glands. There gradually develop headache, irritability, vomiting and nausea, rigidity of the head and neck, some disturbance of consciousness, and then the symptoms of involvement of the cranial nerves at the base of the brain. These are choked disc or optic neuritis, irregular pupils with disturbance of their reactions, ocular palsies, drooping of the upper lid, facial paralysis, disturbance of hearing and of cardiac and respiratory functions. Sometimes there may be convulsions or paralysis of the limbs of one or both sides. In most instances the disease is fatal.

Tuberculous Meningitis in Infants.—When it occurs in infants, there is usually a slow onset with general restlessness, loss of weight, rise of temperature, and gastro-intestinal disturbances with delirium, unconsciousness, and retraction and rigidity of the head, neck, and back, retraction of the upper and lower limbs, and the symptoms of basal involvement which have been described above. Usually the disease terminates in death, but if the patient lives there will be closure of some of the ventricular connections with a consequent internal hydrocephalus. Because of this there will be an in-

crease in the size of the head, bulging of the fontanels, paralysis of one or both sides of the body, diminution of intellect, and a general rachitic condition of the body, with its accompanying symptoms of maldevelopment.

Summary of Diagnosis.—The symptoms of meningitis occurring in the course of a tuberculous condition in an adult, such as retraction of the

Fig. 418.—Case of Tuberculous Meningitis, Showing Marked Emaciation.

head with cranial nerve palsies, with increase in temperature, cardiac and respiratory abnormalities. In the infant, generally before the fifth year, with the typical symptoms of meningitis with special involvement of the cranial nerves, and, if the patient lives, the symptoms of internal hydrocephalus. Lumbar puncture will nearly always demonstrate the tubercle bacillus, although sometimes cultures may be sterile. The fluid will always

Fig. 419.—Different View of Case of Tuberculous Meningitis in a Child Ten Years of Age.

be increased and turbid and the lymphocytes, especially of the mononuclear variety, are increased in number.

The **atropin test,** which is applicable to both meningeal and certain cerebral conditions, may be found of value: Inject subcutaneously 2 mgms. of atropin, noting first the frequency of the radial pulse or of the heart-beats. In event of meningitis being present, the number of heart-beats per minute is appreciably and often decidedly increased. The acceleration of the pulse is observed within twenty minutes after administration of the atropin, and continues becoming most marked at the expiration of one hour.

SEROUS MENINGITIS—MENINGISM.

This is a form of meningitis only recently described in which there is an effusion into the meninges, but in which there is no exudation such as occurs in the purulent variety.

Under the term *meningism, meningismus,* or *pseudomeningitis* has been described that clinical variety in which the symptoms of meningitis are present, but in which pathologically and by lumbar puncture nothing is found perhaps beyond a congestion and edema of the vessels. It is probable that it is nothing more than the primary stage of a serous meningitis. If the disease goes further, into the second stage or stage of effusion, there will be what is commonly termed serous meningitis.

Meningeal processes, whether of an irritative or of an effusive nature, can be likened to similar pathologic conditions occurring in the internal organs, as in the various stages of a pleurisy or pericarditis.

Pathologically, in meningism there will be found a congestion of the blood-vessels with either little or no edema, and rarely the bacillus of the disease may be found in the meninges. In serous meningitis a similar condition is present, with the addition that there will be a serous effusion with an increase of the lymphocytic elements, and only rarely will a specific organism be found.

Symptoms.—Meningism.—This may occur in the course of or follow any infectious disease, such as pneumonia, typhoid, rheumatism, scarlet fever, measles, or grippe. It is not difficult to recognize, for there will be present those symptoms which are commonly termed meningeal, such as pain along the back or limbs, which may be of a numb character, or may be described as sharp and shooting, but the principal complaint is headache, especially in the back of the head. Besides there will be rigidity of the head and back, and unwillingness to move the limbs because of fear of increasing the pain. Sometimes there will also be hyperesthetic areas in different parts of the body. There may rarely be muscular twitchings in the limbs and a general increase of the reflexes. Lumbar puncture is negative. The onset is generally acute. The temperature may or may not be increased, and the pulse and respiration are not much altered. The duration of the disease is usually short, and the prognosis always favorable. Sometimes, however, there may be a complicating serous effusion.

Serous Meningitis.—This may involve either the brain or spinal cord alone, or both. There will be, in addition to the symptoms enumerated above, which may occur first, pressure symptoms resulting from the presence of fluid, their intensity depending upon the degree of the pressure. When the spinal cord is principally involved, there will be, in addition to the meningeal symptoms, pains in the limbs, girdle sense around the waist, and, because of pressure upon the anterior and posterior roots, and later on the spinal cord, disturbance of sensation, increased reflexes which are later lost, and bladder and rectal phenomena. Lumbar puncture will always demonstrate an increase in the intraspinal pressure and there will be considerable exudation of fluid. In most cases the disease only lasts a few weeks, the patient getting well.

In the cerebrospinal forms, besides the symptoms enumerated, there will be, in addition, some disturbance of consciousness and greater rigidity of the head and neck and of the limbs, and sometimes a swelling of the optic nerve-heads. More rarely there may be temporary diplopia and disturbances in the temperature, pulse, and respiration. Lumbar puncture will, of course, demonstrate increased intraspinal pressure with increase of fluid. In most instances the symptoms will subside in a few weeks, the patient getting well.

If, however, they persist, there will develop a serous effusion in the cerebral ventricles, with its accompanying symptoms of intracranial pressure.

Circumscribed serous meningitis may occur in the spinal cord, and generally involves the lower portions, although it may be found in almost any part. It has already been discussed.

Cerebral Serous Meningitis.—A serous effusion into the ventricles may be the beginning of a general serous meningitis, or may be confined only to them. The same causes which are active in the production of a serous meningitis may produce an internal hydrocephalus.

Pathologically there will always be found an internal and sometimes also an external hydrocephalus, or an increase of fluid in the cortical meninges. Histologically there may be cloudy swelling and proliferation of the ependyma, accumulation of cells under the ependyma, and cellular infiltration in the brain and spinal cord substance, and in its meninges, especially along the blood-vessels. The choroid plexus is nearly always diseased, as its over-action is supposed to be the cause of increase in fluid.

Circumscribed serous meningitis may occur in the pia-arachnoid of the cortex. Its symptoms will in no way differ from those of tumor of that part.

Internal hydrocephalus resulting from serous effusion, as a rule, comes on in early childhood, and is not difficult to recognize if the process is active. Very often, however, there may be only mild symptoms such as have been described under meningism, only to have later in life either an acute or a chronic serous meningitis or internal hydrocephalus. In fact, many writers consider that serous meningitis or serous effusion in the ventricles in the adult is only an acute exacerbation of an old process which had its origin in childhood. However that may be, there is no question that in the adult a serous effusion may develop either acutely or gradually in the ventricles and cause symptoms which are usually recognized as occurring in brain tumor, and from which it is sometimes almost impossible to make a differential diagnosis.

If internal hydrocephalus develops acutely, there will be, as a rule, an accompanying high fever, and the course of the disease will be rapid, it resulting either in cure or death. Headache, nausea, vomiting, vertigo, and disturbance in vision and choked disc, sometimes of high caliber, are prominent symptoms. Besides there may be paralysis of some of the cranial nerves, especially of the sixth, either on one or both sides, and there may also develop cerebellar ataxia. Consciousness is nearly always clouded. The disease may last a week or two, terminating in quick recovery, leaving behind slight atrophy of the optic nerves, but no other symptoms. Sometimes there may be a recurrence of the disease, this terminating also either in recovery or death. The diagnosis from a brain tumor can usually be made by the rapid onset, the high fever, and the quick recovery or termination in death.

If, however, the symptoms of internal hydrocephalus come on gradually, the differential diagnosis from brain tumor will be very difficult. There will be present all the pressure symptoms, such as headache, nausea, vomiting, vertigo, and choked disc, and because of the pressure exerted upon the motor fibers in the internal capsule, there will result weakness and spasticity of the limbs, with increased reflexes and sometimes the Babinski reflex. There may also be paralysis of the external rectus, either on one or both sides. Because of pressure on the cerebellum there will result incoördination in walking and sometimes incoördination of the eyeballs or nystagmus. The differential diagnosis from cerebellar lesions is sometimes very difficult, but can be made principally upon the fact that in cerebellar tumors there is

hardly ever involvement of the limbs on both sides and the ataxia is more acute and much more marked.

It must also be remembered that internal hydrocephalus may also accompany tumors of either the cerebrum or the cerebellum, and in such case there will be, in addition to the symptoms resulting from the tumor, spastic paresis of the limbs with increased reflexes and the Babinski phenomenon. The prognosis in most cases of uncomplicated internal hydrocephalus is not very good, but sometimes complete recovery ensues, either as a result of operative interference, anti-specific treatment, or sometimes spontaneously, leaving behind nothing but a slight atrophy of the optic nerves.

MUSCULAR DYSTROPHY.

Definition.—A progressive hereditary and family disease, usually beginning in childhood, characterized by gradual weakness and atrophy of the muscles. Under the general term of muscular dystrophies have been described many clinical types, but while in the beginning there is a difference in the method and the seat of the involvement, the terminal stages are alike in all.

Until very recently it has been thought that in the muscular dystrophies

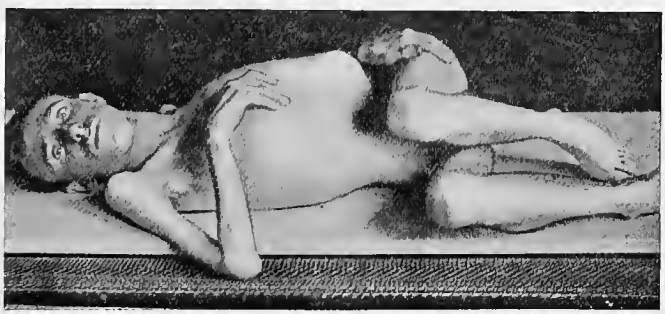


Fig. 420.—Muscular Dystrophy, Last Stage, showing Contractures and Atrophy.

the nervous system itself is never diseased, and that the cause is entirely in the muscles. Recently, however, there has been found in a number of cases a chronic degeneration of some of the peripheral nerves and atrophy of some of the motor nerve-cells in the spinal cord. The usual findings in the muscles consist in a gradual atrophy of the fibers with an increase of their nuclei and of interstitial connective tissue. In the pseudo-hypertrophic type there is swelling of the muscle-fibers with a large accumulation of fat-cells. The hypertrophy and fatty infiltration is succeeded by gradual atrophy, and there may be found both an increase of fatty tissue and atrophy of the muscle-fibers.

Symptoms Common to all Dystrophies.—It is probable that the disease is due to a maldevelopment of the muscular structures. Sometimes a number of members of the same family may be affected, and it is usually hereditary. In all types the symptoms begin before the twentieth year, and mostly between the ages of five and puberty. There is usually a history of slow muscular development, the child taking a longer period than normal to learn to walk. The weakness and atrophy progress at the same time. There are never fibrillary tremors, the reflexes become gradually

diminished, and in the last stages are absent, and there are no electric reactions of degeneration. Sensory and bladder and rectal symptoms are never present. In the terminal stages there can be no differentiation made between the various clinical types.

Pseudo-hypertrophic Form.—In this type males are more frequently

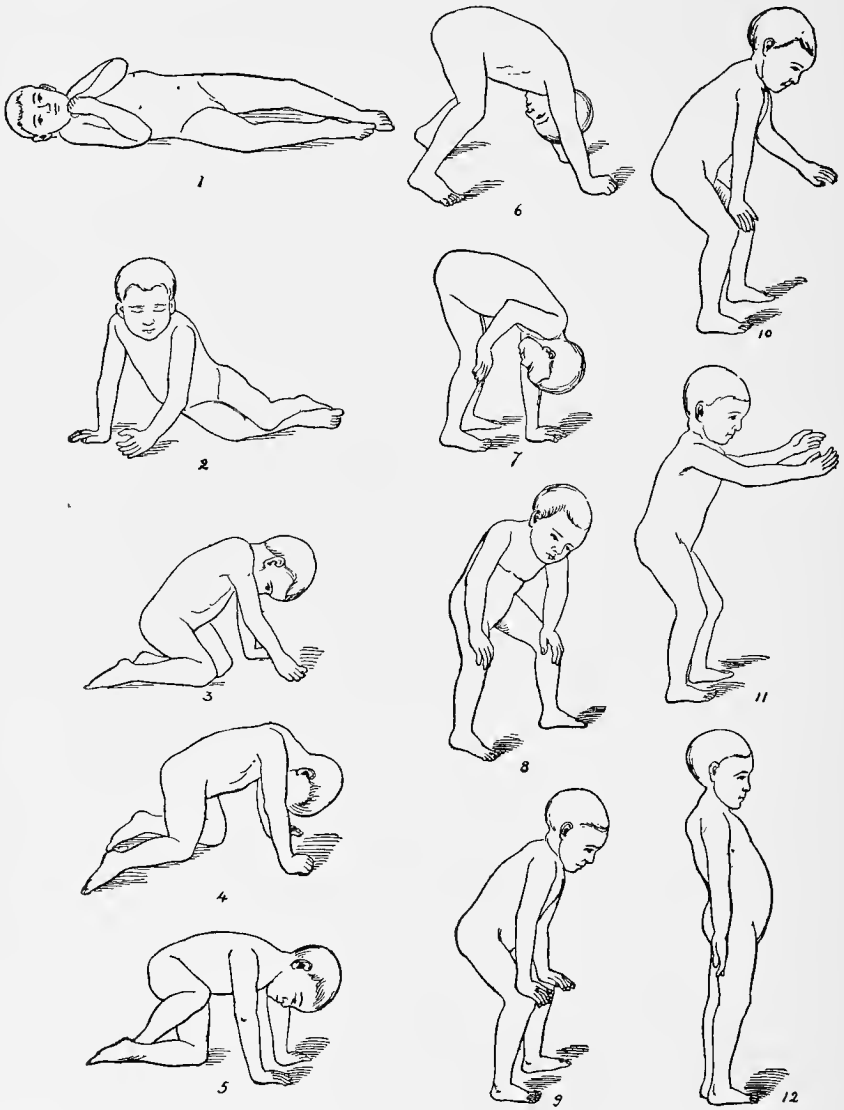


FIG. 421.—MANNER OF ARISING IN PSEUDO-HYPERTROPHIC MUSCULAR DYSTROPHY.

diseased and the symptoms usually begin between the fifth and tenth year. It is first noticed that the muscles, especially of the calves, thighs, buttocks, and shoulders, are disproportionately large, but gradually there develops difficulty in running, the child tiring easily, especially in going up- and down-

stairs; the gait becomes slow, with a tendency to lift the hips from side to side, and later characteristically waddling. At the same time the shoulders are retracted, the abdomen protruded because of the weakness of the gluteal, lumbar, and abdominal muscles, and there is present a deep spinal frontal curve. Gradually the weakness increases and the gait becomes progressively more difficult, and when the child is placed upon the ground there is usually a characteristic method of rising. The child first turns on his face, extending one and then the other leg, bracing his toe against a stationary object, supporting the weight of his body and legs by both hands. When the legs are firmly extended and braced, the hands are brought nearer and nearer to the legs until finally the hands are braced against the ankle, then the leg, knee, thigh, and then with a supreme effort the shoulders and body are finally elevated and held erect. The diagnosis can be usually made upon the characteristic waddling gait and climbing-up method of rising. The muscles are soft to the touch, and gradually this pseudo-hypertrophy is succeeded by atrophy, until finally walking is impossible. In the terminal stages the patient becomes chair- or bedridden, the atrophy becomes general and involves all parts of the limbs, trunk, and abdomen, and in the last stages the muscles of the face. The bones also take part in the general atrophy (Fig. 421).

Infantile Form.—In this type the disease nearly always begins before the fifth year. The muscles of the face, scapula, and humerus are preponderantly involved, and this form is often known as the facio-scapulo-humeral. It may sometimes be in association with the pseudo-hypertrophic type, but, as a rule, the atrophy begins in the muscles of the face and shoulder girdle, and there is no preliminary hypertrophy. The atrophy of the face usually involves the oral and palpebral orbicularis muscles, and it becomes impossible to shut the mouth or close the eyes, and there is a peculiar drawn expression of the face, which is known as the myopathic facies. Articulation, whistling, and laughing are sometimes interfered with. The atrophy gradually extends and involves the muscles of the neck, shoulders, and upper arm, and will finally involve the muscles of the shoulder, chest, abdomen, and then of the limbs.

Juvenile Form.—In this type the disease appears nearly always after puberty, and begins with an atrophy and weakness of the muscles of the shoulder and upper arm. In the final stages the muscles of the chest, abdomen, limbs, and face become similarly involved.

Summary of Diagnosis.—A family or hereditary disease, beginning nearly always before the twentieth year, and especially before puberty. In the pseudo-hypertrophic form, a hypertrophy and weakness of the muscles of the thigh, shoulder, and buttock, with gradually increasing difficulty in walking, the gait becoming waddling, with protrusion of the abdomen, retraction of the shoulder, and a peculiar climbing method of rising from the ground. In the infantile form the muscles of the face and shoulder girdle are first involved, with inability to close the eyes and shut the mouth or move the shoulder and upper arm. In the juvenile form the disease begins after puberty, and first involves the muscles of the shoulder and upper arm. In all the types the atrophy gradually involves all the muscles, the patient becoming helpless. There are never fibrillary tremors, the reflexes become gradually diminished, electric reactions of degeneration are not obtained. There are no sensory or bladder and rectal symptoms.

Differential Diagnosis.—There should be no difficulty in diagnosticating the muscular dystrophies in their terminal stages. It is of no prac-

tical importance to make a diagnosis of the different types, inasmuch as they all terminate alike.

Clinical Course and Complications.—The course of the disease is progressive, and the patient may live for many years. Usually death results from intercurrent disease. Sometimes muscular dystrophy may complicate other spinal cord diseases, such as chronic degeneration of the cells of the anterior horn.

PERONEAL OR DISTAL MUSCULAR ATROPHY.

(Charcot-Marie-Tooth-Hoffman-Sachs Type.)

Definition.—A progressive disease characterized by gradual atrophy and weakness beginning in the distal portions of the lower and upper limbs, with tremors, loss of reflexes, and some sensory disturbances.

Pathologically, besides the degeneration of the muscle-fibers, which consists in a gradual atrophy, increase of muscle nuclei and of interstitial tissue, there is some degeneration of the nerve-cells of the anterior horns and of the column of Goll, and more rarely a diffuse degeneration of the lateral columns and peripheral nerves.

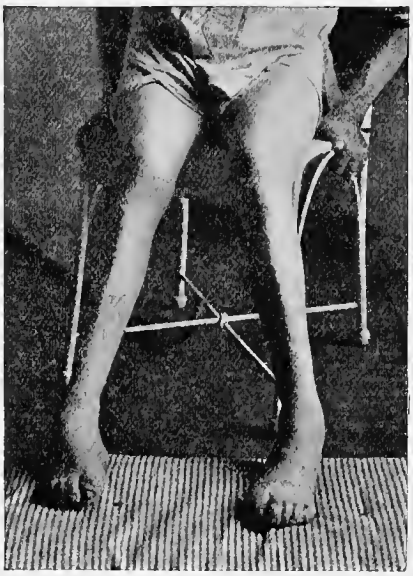


Fig. 422.—Primary Neurotic Atrophy, showing Wasting and Contracture of the Lower Limbs.

Contributing and Exciting Factors.—The disease is hereditary, and occasionally occurs in families. It usually appears in young adults without any exciting cause. It is probably a manifestation of maldevelopment of the parts involved.

Symptoms.—The onset is gradual, and begins with atrophy and weakness of the small muscles of the foot and toes, it involving especially the distribution of the peroneal nerves, the tendons of the small toes becoming prominent. As the disease progresses there may develop deformity of both feet, such as equinovarus, or the patient may become flat-footed. Gradually the muscles of the peroneal and anterior and posterior tibial groups atrophy, and then the muscles of the thigh, especially the vastus internus. Locomotion becomes difficult, and usually the patient in walking spreads his feet wide apart, and because of the foot-drop the toes are dragged on the ground, and the knees elevated more than they should be.

Coincident with the distal atrophy of the lower limbs there may develop a similar atrophy in the small muscles of the hand, especially in the thenar and hypothenar eminences and the interossei, this gradually progressing and involving the muscles of the forearm, especially the extensors, and then the muscles of the arm and shoulder. As a rule, the involvement of the upper limb follows the lower, but sometimes the atrophy in the upper limb appears first (Fig. 422).

Fibrillary tremors are common in the involved limbs, the reflexes be-

come gradually diminished and finally lost, and there may rarely be pain on pressure over the nerve-trunks. Disturbances of touch, pain, and temperature are sometimes found in the limbs. The disease is slowly progressive, and may ultimately involve the muscles of the trunk, buttocks, and face, but usually the patient dies before this occurs.

Summary of Diagnosis.—A family or hereditary disease occurring in young adults, atrophy and weakness beginning in the distal portions of the leg or arm, gradually increasing and extending upward, with fibrillary tremors, gradual loss of reflexes, and some disturbance of sensation.

Differential Diagnosis.—It should not be difficult to diagnose this disease if the nature and progress of the atrophy and weakness are clear. Sometimes, however, it is necessary to diagnosticate from multiple neuritis and progressive spinal muscular atrophy. In the former there is always pain on pressure over the nerve-trunks, with considerable disturbance of sensation and a history either of alcoholism, lead, or some similar cause, and the prognosis is good, the patient usually getting well. From progressive spinal muscular atrophy the disease is sometimes difficult to differentiate, especially when the distal form of atrophy begins in the upper limb, for there is present in both tremors, atrophy, weakness, and gradual loss of reflexes, but in the distal type there will be found occasionally pain on pressure over the nerve-trunks with some sensory disturbance, and the progress of the disease is different, inasmuch as the lower limbs become diseased early, this first involving the distal portions.

DISEASES OF THE PERIPHERAL NERVES.

Every peripheral nerve-fiber consists of an axis-cylinder, of an enveloping substance called the myelin, and of a surrounding membrane—the neurolemma sheath. The nerve-fibers in the brain, spinal cord, and sympathetic system do not have this sheath, and some of those of the sympathetic system have no myelin. The individual fibers are bound together by interstitial tissue called the endoneurium. Every nerve-trunk consists of a number of these bundles held together by interstitial tissue, the perineurium, the whole being surrounded by the epineurium.

Pathology.—Inflammation of a peripheral nerve may be limited to a part or the whole extent of a nerve, when it is called a simple neuritis, or may involve several or most of the peripheral nerve-trunks in the body—a multiple neuritis. Inflammations may be limited to the interstitial tissue—an interstitial neuritis, or to the myelin substance and the axis-cylinders, when it is called a parenchymatous neuritis. The disease, of course, may affect both the interstitial tissue and the parenchymatous substance. Inflammations are further divided into acute and chronic, depending upon the onset. Most acute inflammations involve both the parenchymatous and interstitial substance, while chronic inflammations will have a tendency to be interstitial in character, with a secondary degeneration of the parenchymatous tissue, and are commonly called degenerative.

Microscopically, acute interstitial neuritis is characterized by swelling of the connective-tissue fibers and congestion of its vessels, with edema, round-cell infiltration, and swelling and breaking up of the myelin substance and of the axis-cylinders. Most cases of acute interstitial neuritis subside. If the disease becomes chronic, the acute inflammatory symptoms disappear, the interstitial substance will increase, and the myelin substance and axis-cylinders atrophy slowly. In the parenchymatous form both the myelin

substance and axis-cylinders swell and break up into nodules, and there is a congestion of the vessels, with some round-cell infiltration, and, unless regeneration occurs, the fibers will be replaced by scar tissue. Degeneration of the peripheral muscles in which the nerves end results. Besides, there is often found, especially in multiple neuritis, a degeneration of the nerve-cells in the anterior horns of the spinal cord, and sometimes even in the cranial nuclei.

Functions.—The peripheral nerves have a threefold function: (1) Motor, transmitting impulses from the anterior roots of the spinal cord to certain muscles; so if a nerve is cut, there will be loss of motion in its distribution; (2) they transmit sensation from the periphery as touch, pain, heat and cold, vibratory sensations, and what is known as muscle sense, this including the sense of pressure, localization, position, and movement; and, lastly (3), they conduct vasomotor and trophic fibers which concern nutrition of the hair, nails, skin, deeper structures, and joints, and control sweat secretions.

A nerve may be either purely motor or sensory, but in most cases combines both functions. In a mixed nerve it is impossible to tell what fibers transmit motion, sensation, or vasomotor functions; but the recent work of Head, which has revolutionized our ideas of peripheral sensation, has demonstrated the important point that deep sensibility, such as is capable of answering to pressure, and even producing pain when this is excessive, is transmitted mostly in the deep muscular nerves, and is not destroyed by division of the sensory nerves to the skin. This deep sensibility is also concerned with movement of the muscles and the extent and direction of passive movements of the joints. It is important to remember this, for these sensations are always preserved when only the peripheral nerves are cut.

In Head's second division of sensation, or what he calls protopathic sensibility, there is capability of responding to painful cutaneous stimuli and to extremes of heat.

His third classification, or epicritic sensibility, is concerned with the power of cutaneous localization, of the appreciation of light touch, and the discrimination of two points (compass test), and of the finer grades of temperature (particularly from 25° to 40° C., that is, those called cold or warm).

"The above-mentioned sensibilities are often dissociated in an area affected by the disease or operative procedure. According to the completeness of the lesion, the kind of nerves affected, and the state of repair, all forms of sensibility (superficial and deep) may be absent or more or less partially present. Thus an area may present protopathic sensibility and not epicritic sensibility, or even the epicritic and not the protopathic form. Moreover, the nerves subserving these two forms of sensibility do not coincide in their areas of distribution. For, provided the peripheral nerves are divided into certain groups, it may be said that, as regards light touch, and other forms of epicritic sensation, very little overlapping occurs, whereas in the case of protopathic sensibility enormous overlapping is found, and it becomes evident that while the unit of supply for epicritic sensibility, looked at broadly, lies in the peripheral nerves, the unit of protopathic supply lies in the posterior roots.

"Further, the two systems regenerate with unequal facility; for, during the process of regeneration in a divided peripheral nerve in man, protopathic sensibility may begin to return in the subserved area in about seven weeks (average eighty-nine days), and be complete in twenty-nine weeks (average one hundred and seventy-eight days). At this stage of the more primitive

form (protopathic sensibility) there is the power to appreciate in the affected parts pin-pricks, extremes of heat and cold (above 45° C. and below 20° C.), but no power to appreciate or respond to light touches, small differences of temperature, and no accurate localization is possible; in fact, fine discriminating power is absent. The return of protopathic sensibility brings a cessation of all those destructive nutritive changes that occur in parts where the skin is insensitive, such as ulcers, etc., which form as the consequence of burns or cuts, and do not heal so readily as on normal skin. Such trophic changes are confined to parts deprived of protopathic sensibility. With the return of the latter, ulcers and sores heal as readily as on the normal skin. Moreover, when a peripheral nerve to the hand is divided, it is noticeable that the palm begins to sweat at a time after union which coincides approximately with that of the return of protopathic sensibility. This sweating is innervated and controlled by the motor fibers of the sympathetic (the autonomic fibers of Langley and Anderson) that supply the skin.

"As the regeneration proceeds the higher and more discriminating form, viz., epicritic sensibility, begins to return in about three hundred and twenty-one days after the lesion, and, though it varies somewhat, may be complete in about three hundred and sixty-four days. Its return brings a power to respond to light touches, to localize accurately the sites of application of stimuli, and to appreciate correctly small grades and differences of temperature."

It is only necessary further to add that after a peripheral nerve is cut or diseased, regeneration is possible provided the conditions are favorable, and there is no continuation of the pathologic process. After a nerve is cut there is degeneration or physiologic death of the parts peripheral to the cut, while only a small portion of the central stump degenerates. This is because the central part still has its trophic supply from the nerve-cell from which it originates, while in the peripheral part this is absent.

DISEASES OR INJURIES OF THE SPINAL NERVES.

There are thirty-one pairs of spinal nerves, corresponding to their respective spinal segments—eight cervical, twelve thoracic, five lumbar, five sacral, and one coccygeal. Because some of these nerves innervate the upper and lower limbs, two principal plexuses have been formed, the brachial and lumbo-sacral. The individual distribution of the peripheral nerves, in so far as the skin areas are concerned, is shown in Figs. 423 and 424. Only the common inflammations or injuries of the peripheral nerves will be discussed. The general pathology, regeneration of function, and peculiar sensory disturbance resulting from neuritis or injury have already been discussed.

Symptoms.—These will depend upon the extent and degree of the neuritis, and whether the nerve is motor, sensory, or mixed.

Motor Symptoms.—If a nerve is totally diseased or severed, there will be paralysis in its distribution, and within a week or two electric reactions of degeneration, which become complete in three weeks. As regeneration appears the reactions gradually improve, until finally normal responses are obtained. Atrophy appears about the same time as the reactions of degeneration, and has about the same course. The reflexes are, of course, lost in the distribution of the nerve.

Sensory Symptoms.—The disturbances of sensation will, of course, depend upon the particular distribution of the nerve. Its extent and character

have already been discussed on page 1134. It is important to remember that sensation returns before motion.

Vasomotor Symptoms.—These include disturbances in nutrition of the skin, hair, nails, and joints, and deeper structures, their degree depending upon the nerve involved and the extent of the paralysis.

Duration.—An ordinary neuritis may last from four to six weeks, and then will gradually subside. If it involves a number of nerves, as a plexus,

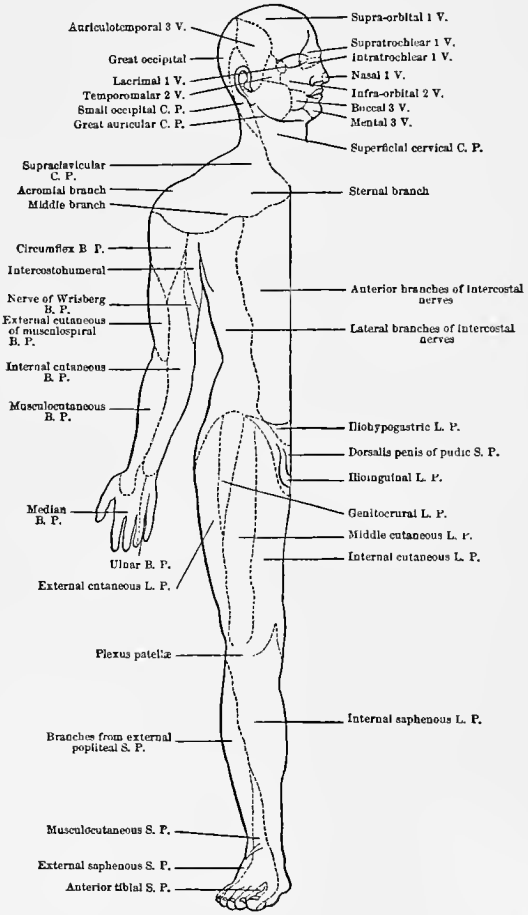


FIG. 423.—CUTANEOUS DISTRIBUTION OF NERVES (after Flower).

the duration is longer. In most cases the prognosis is good, this depending largely upon the extent of the paralysis, the removal of the cause, and the effects of treatment.

BRACHIAL NEURITIS.

The brachial plexus is composed of the fifth, sixth, seventh, and eighth cervical and first thoracic roots, and supplies motion and sensation to the

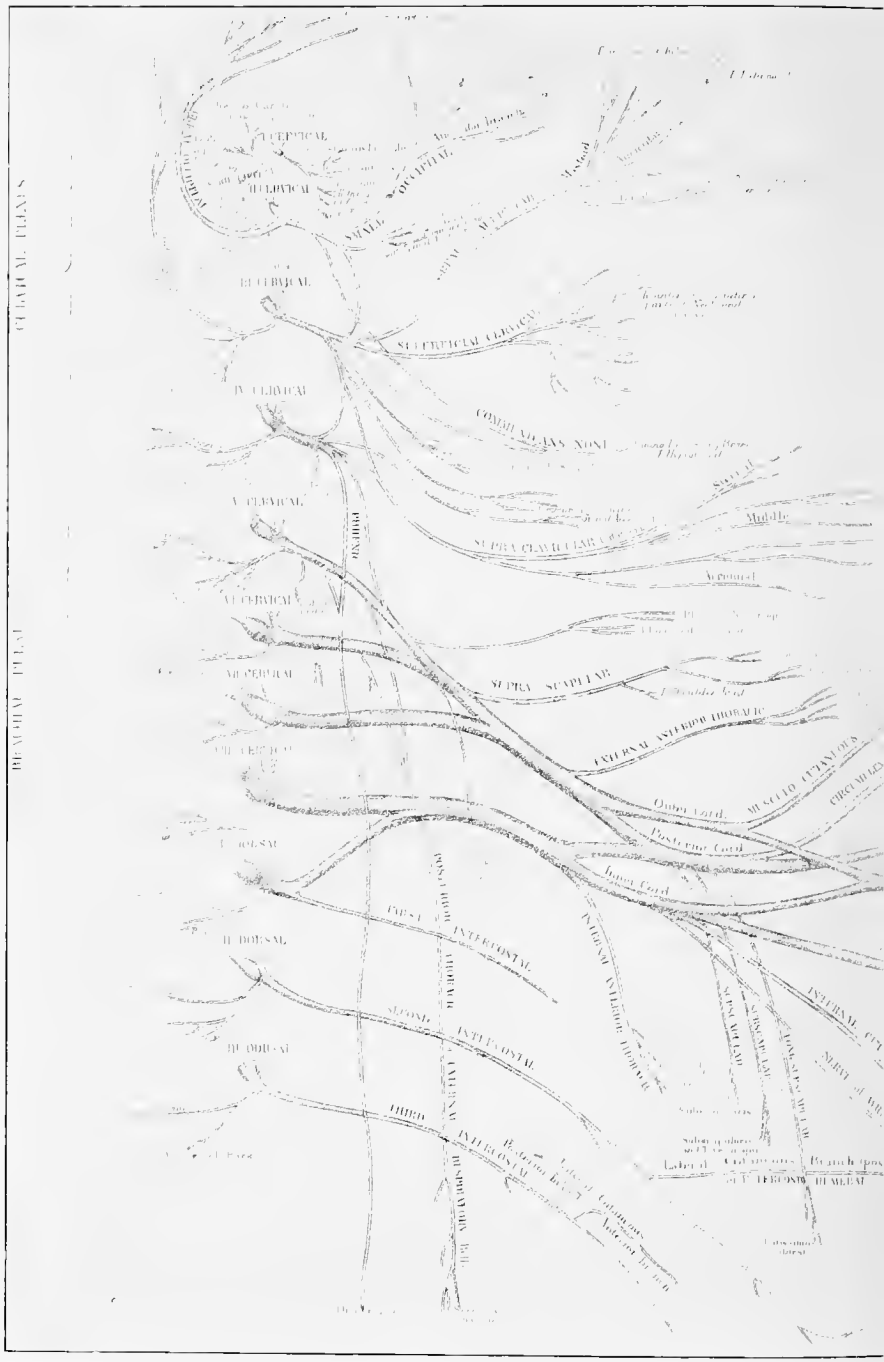
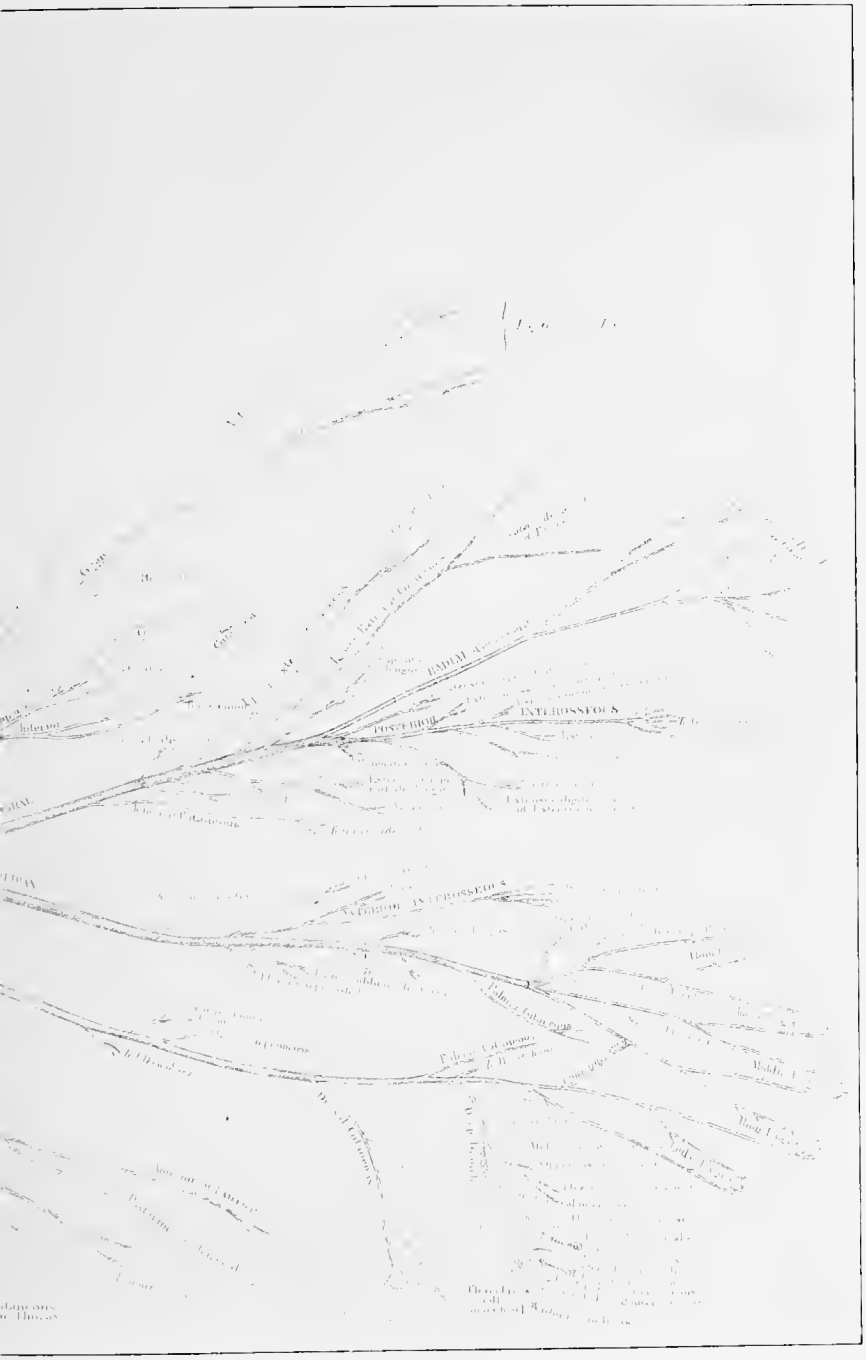


Diagram Showing Relations and Distrib

III



Cervical and Brachial Nerves (Flower).

upper limbs. A neuritis may involve all the branches of the plexus, or be limited to its upper part, this including the fifth and sixth cervical, or the lower, involving the seventh and eighth cervical and first thoracic roots, or their continuations. If in association with the brachial neuritis there is disease of the first four cervical nerves, it is called a cervico-brachial neuritis.

Most cases of brachial neuritis appear in adults without any apparent cause, although it is probable that such general diseases as gout, rheumatism,

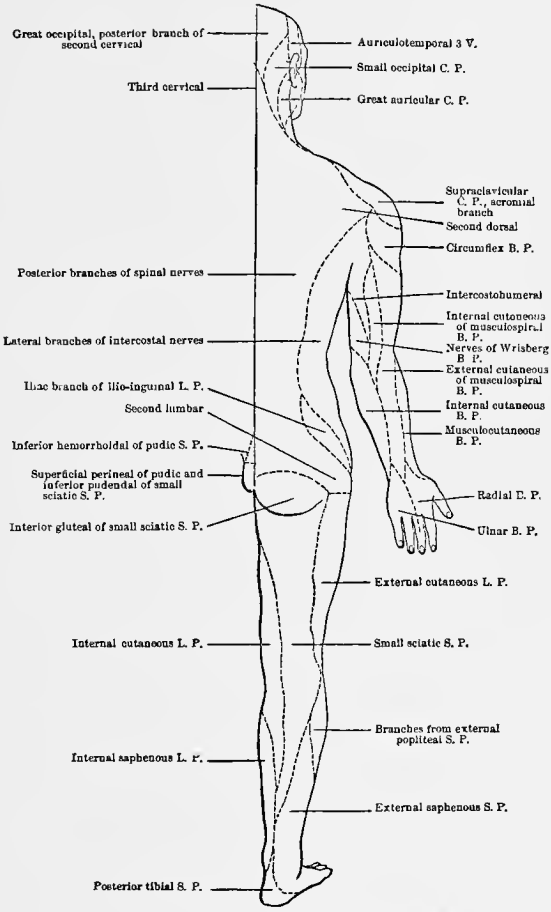


Fig. 424.—Cutaneous Distribution of Nerves (after Flower).

and anemia are often the causal factors. Direct injuries to the shoulder or plexus, dislocations, caries of the vertebra, birth palsies, cervical rib, and aneurisms are frequent causes.

Symptoms.—Whether the neuritis involves all or only part of the brachial plexus, the most important symptom is pain. This may appear gradually or acutely, and is usually sharp and shooting in character, it being aggravated by movement of the arm. If the whole plexus is diseased, the

pain involves all parts equally; if the upper cords, it will be limited to the neck, shoulder, and arm as far as the elbow; if the lower, to the arm, forearm, and hand. The nerve-trunks and muscles are tender and painful to pressure, and vasomotor and trophic disturbances are common. Associated with the neuritis there will always be some paralysis, with consequent atrophy of the muscles and loss of reflexes. The specific paralytic symptoms depend upon whether all or part of the plexus is diseased. In the upper form the paralysis will be limited to the deltoid, biceps, coracobrachialis, and supinator muscles; if the lower, to the muscles of the forearm, and especially of the hand. In association with the lower type of neuritis there may be certain pupillary phenomena, which will be discussed under the lower arm type of paralysis.

The duration of the neuritis will depend largely upon its etiology. If due to causes which can be removed, the prognosis is excellent. If idiopathic or due to constitutional diseases, the neuritis is of long duration and difficult of cure.

The diagnosis of brachial neuritis should not offer much difficulty. It is important to determine the etiology and remember that in the beginning of a rheumatoid arthritis the pains may be referred to the shoulder and upper arm.

BRACHIAL PALSY.

Paralysis of the brachial plexus may be total or partial, unilateral or bilateral. If *total*, the arms hang limply by the side, no movements being possible, the muscles soon atrophy, the reflexes become lost, and electric reactions of degeneration are early obtained. Partial brachial paralysis may be either of the upper plexus type, the so-called Duchenne-Erb form, in which the fifth or sixth cervical roots or their continuations in the plexus are involved; or the lower plexus or Klumpke's type, in which the eighth cervical and first thoracic roots or their continuations are diseased.

Upper Arm Type of Brachial Palsy; Birth or Obstetrical Palsy.—This form of paralysis is mostly traumatic in origin, and occurs most frequently at birth when abnormal traction is made upon the head or arm or pressure exerted upon the brachial plexus either by forceps or in breach presentation. Sometimes it may result from abnormal stretching of the arm in the course of etherization. The paralysis may be noticed immediately after birth, and involves the deltoid, triceps, brachialis anticus, supinator longus and brevis, and infraspinatus muscles. It will be impossible to adduct the arm, and the forearm is extended and pronated. The muscles soon become atrophic, the reflexes are lost, and electric reactions of degeneration are obtained. Sensation, as a rule, is not destroyed.

Lower Arm Type of Brachial Palsy.—This is sometimes called Klumpke paralysis, and involves the eighth cervical and first thoracic roots or their continuations. It is a very rare form, and usually results from injury. There will be paralysis of the small muscles of the hand and forearm, especially of the flexors, resulting in inability to move the fingers or hand. Atrophy and reactions of degeneration follow, as well as sensory disturbances, especially in the ulnar distribution.

Involvement of the Sympathetic System.—Our knowledge of the sympathetic system is by no means exact. We know, however, that in the lower part of the cervical and upper part of the dorsal cord (in the eighth cervical and first thoracic segments) there is a so-called ciliospinal center, and that the rami communicantes of the anterior roots of the

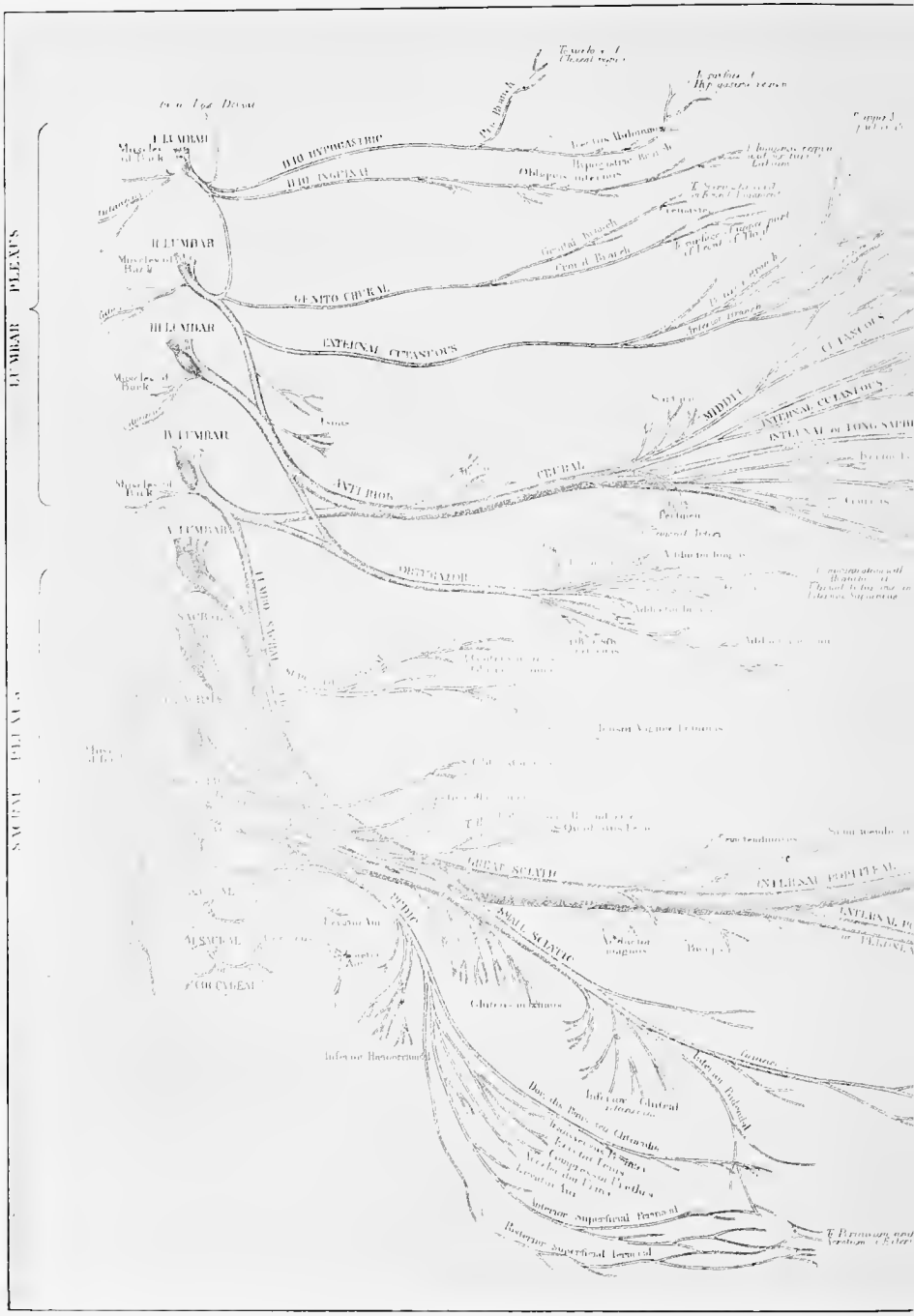
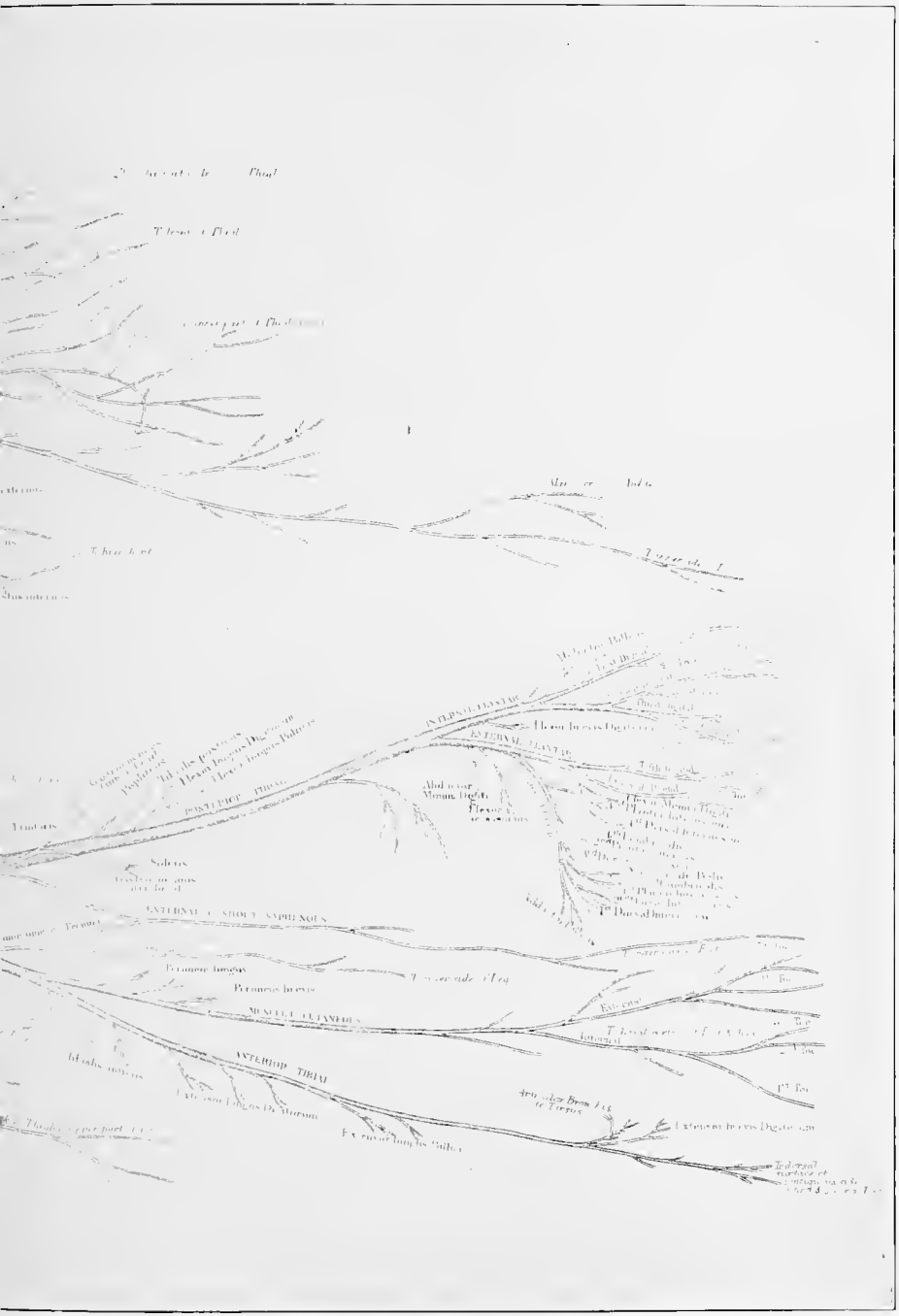


Diagram Showing Relations and Distribu



mbar and Sacral Nerves (Flower).

first dorsal segment contain the so-called oculopupillary or sympathetic fibers.

The classic symptoms of irritation of the cervical sympathetic—by this being meant either the ciliospinal center in the spinal cord, the anterior root-fibers of the first dorsal segment, or the cervical sympathetic plexus—are enlargement of the pupil, widening of the palpebral fissure, a slight exophthalmos, delayed descent of the lid in looking downward, paleness of the face, and increase in the sweat secretion. Paralysis of the cervical sympathetic produces a small pupil, narrowing of the palpebral angle, a slight enophthalmos, warmness or coldness of the face, and disturbance of sweat secretion. It is only rarely, however, that all of these symptoms are present, the most consistent being disturbance in the size of the pupil, and either protrusion or retraction of the eyeball with alteration in the width of the palpebral fissure.

It can be readily understood, then, that sympathetic symptoms occur from injuries either of the cervical portion of the spinal cord, the cervical sympathetic plexuses, or in brachial neuritis or palsy in which the rami communicantes of the first dorsal root are involved. Therefore, in the lower arm type of paralysis, in which the eighth cervical and the first dorsal roots are diseased, there are always oculopupillary symptoms. It is possible, however, to have this type of paralysis without sympathetic involvement, if the fibers coming from these roots in the brachial plexus, and not the roots themselves, are diseased. It is difficult, however, to make such a clinical differential diagnosis, because the symptoms are identical, but it can always be assumed that, if the oculopupillary symptoms are present, the first dorsal root is diseased.

In the Duchenne-Erb or upper type of paralysis, due to a birth palsy, or the paralysis occurring in the course of etherization, the traction upon the arms may cause an abnormal stretching and tearing of the rami communicantes of the first dorsal root, this causing sympathetic paralysis without the first root itself being diseased.

If all the roots of the brachial plexus are diseased, there may be oculopupillary symptoms. As a result of gunshot or stab wounds there may be forms of paralysis which do not conform to any of the regular types with sympathetic symptoms. In these cases either the first dorsal roots are involved, or the oculopupillary fibers in the cervical sympathetic are injured.

Paralysis of the Circumflex Nerve; Deltoid Paralysis.—This usually results from dislocation or direct injury to the shoulder, and produces paralysis of the deltoid muscle, there being inability to raise the upper arm from a hanging position. When the anterior part of the muscle only is affected, it will be impossible to adduct the upper arm or place the hand to the opposing shoulder. When the posterior part is involved, the patient will be unable to put his hands in his pockets. Following the weakness there will be atrophy, reactions of degeneration, and triangular disturbances of sensation.

Paralysis of the Long or Posterior Thoracic; Serratus Magnus Paralysis.—This results sometimes from lifting heavy weights or injuries or dislocation of the shoulder, and causes paralysis of the serratus magnus muscle. The edge of the scapula, to which the serratus is attached, will be winged or prominent, and there will be inability to lift the arm more than to a horizontal position. Disturbances of sensation are sometimes present.

Paralysis of the Musculocutaneous Nerves; Biceps and Brachialis Anticus Paralysis.—Isolated paralysis of this nerve is

rare. The forearm when in supination cannot be flexed, and the supinator action of the biceps, which is exerted when the biceps is contracted, is also absent.

Musculospiral Palsy.—This nerve is very frequently injured or diseased because of its exposed position around the humerus. Paralysis generally comes on after a debauch, the patient while intoxicated lying on his arm, this causing pressure. It is sometimes called Saturday night palsy. The musculospiral nerve supplies the triceps, anconeus, supinator longus, the extensor carpi radialis longior, and all of the extensor muscles of the hand. There is wrist-drop with inability to extend the fingers or the hand upon the wrist, and because of the paralysis of the extensor muscles flexion of the fingers will be imperfect. There will also be inability to pronate the forearm, and sometimes failure to extend the forearm on the arm. Sensory disturbances are not the rule, but they are sometimes found, especially over the radial side of the forearm and hand. Musculospiral palsy often occurs as a result of lead-poisoning, the supinator longus always escaping.

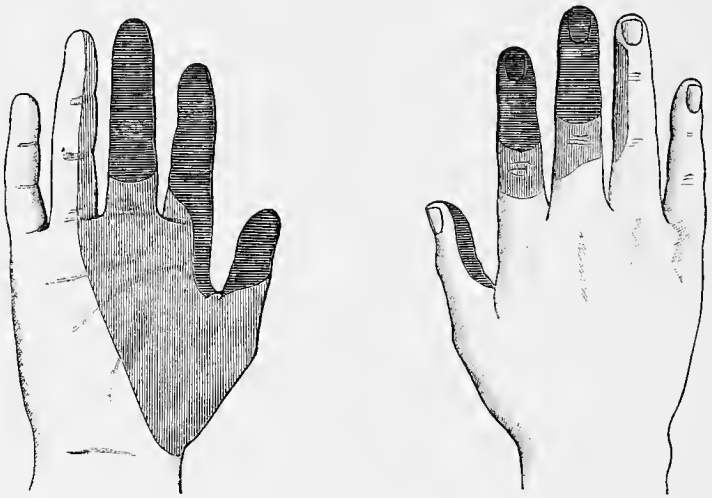


FIG. 425.—SHOWING AREAS OF SENSORY LOSS IN INJURIES OF THE MEDIAN NERVE (Bowlby).

Median Nerve Palsy.—Median nerve paralysis is generally due to injury. It supplies all the flexors of the fingers, the flexor carpi radialis, and the pronator radii teres. The disturbances in motion will consist in inability to pronate the forearm, to flex the hand to the radial side, the fingers cannot be flexed, and adduction of the thumb is lost. Sensory disturbances are uncommon, but when present are limited to the radial side, as shown in Fig. 425.

Ulnar Palsy.—This is usually produced by direct injury to the nerve. It supplies the flexor carpi ulnaris, the ulnar half of the flexor profundus digitorum, and the muscles of the hypothenar eminence, the interossei, the inner three lumbricales, the adductor transverse pollicis, and the flexor brevis pollicis. In ulnar paralysis there is disturbance of flexion of the hand and of the last three fingers, and inability to flex the proximal and extend the terminal phalanges of the fingers. This is especially marked in the last two fingers, and there is also some weakness in adduction of the thumb, this dis-

turbance causing the so-called "claw hand," it being more marked late, when there is atrophy in the involved muscles. Sensory disturbances are not frequent, and when present are limited to the flexor and extensor surfaces of the last two or three fingers (Fig. 426).

Paralysis of the Diaphragm.—Isolated paralysis is very rare. It is supplied by the phrenic nerve, which is derived from the third, fourth, and fifth cervical segments, and usually results from lesions of the vertebra or direct injury. Unilateral lesions of the phrenic nerve may not cause paralysis, but a total paralysis of the diaphragm can be detected by the inaction of the upper part of the abdomen during respiration.

DISEASES OF THE LUMBAR AND SACRAL PLEXUSES.

Isolated paralyses of the nerves of the lower limbs are rare, and are usually the result of traumatism. In such paralyses it is always necessary to exclude muscular dystrophies, anterior poliomyelitis, multiple neuritis, pelvic

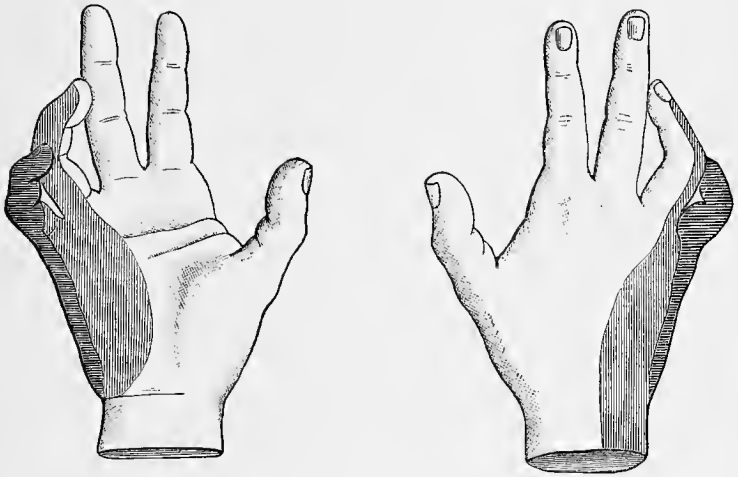


Fig. 426.—Showing Sensory Loss and Ordinary Position in Injuries of the Ulnar Nerve (Bowlby).

tumors and abscesses, and also lesions of the lowest portion of the spinal cord or of the cauda equina.

Paralysis of the anterior crural nerve occurs mostly after injuries. It innervates the iliopsoas and the extensor quadriceps. There will be inability to flex the thigh on the trunk, and if the thigh is raised passively, the leg cannot be extended. The patellar jerk will be absent, walking and standing on the leg will become almost impossible, and raising of the trunk on the thigh from a recumbent position will be impaired. Disturbances of sensation are uncommon, but are sometimes found in the inner part of the thigh and leg.

Paralysis of the obturator nerve sometimes occurs, and interferes with adduction of the thigh, it being impossible to cross one leg over the other. Rotation of the thigh outward is also interfered with.

Isolated *paralyses of the gluteal nerves* are uncommon, and usually occur in conjunction with muscular dystrophies. These nerves supply the gluteal

muscles, and paralysis of them causes an inability to extend the thigh on the pelvis, interfering with going up-stairs or with rising from a sitting posture.

Sciatic Paralysis.—This may come on at the end of a sciatic neuritis or sciatica, or may result from tumors of the pelvis, causing pressure on the nerve, or from direct injuries. The sciatic nerve supplies the muscles of the back of the thigh, a lesion of this part causing inability to flex the leg backward on the thigh, due to paralysis of the semimembranosus and semitendinosus. Walking, however, will be possible because of the action of the quadriceps. Besides, the sciatic nerve supplies the muscles of the leg below the knee, it dividing into the external and internal popliteal nerves.

Paralysis of the external popliteal nerve is common because of its exposed position, it winding around the head of the fibula, where it is liable to direct injury. It divides into the peroneal and anterior tibial branches, which supply the extensors of the toes, paralysis causing foot-drop and inability to abduct the foot. When walking, the patient will lift his knees high from the ground, causing the so-called steppage gait. Disturbance of sensation sometimes occurs in the outer border of the leg and foot.

Paralysis of the internal popliteal nerve is uncommon. It supplies the muscles of the back of the leg, and there will be inability to flex or stand on the toes, the plantar reflex will be absent, as will also the Achilles jerk.

Meralgia Paresthetica.—By this is understood a paresthesia, which is described as a crawling or tingling feeling, usually in the distribution of the external cutaneous nerve of one thigh or of the anterior crural. As a rule, there is no accompanying tenderness to pressure, but there may be some disturbance of sensation. It is usually the result of pressure upon the involved nerves by either a corset or a truss.

TUMORS OF THE NERVES.

These are of rare occurrence and are generally fibromata, but they may be sarcoma, angioma, or any of the other usual forms. The tumor may develop within or upon a nerve-sheath. The amputation neuroma is the best example of a true nerve tumor, and it is possible that pure neuromata do not exist.

Fibroma may grow upon one nerve, or may rarely involve all the nerves of the body, even the cranial. This is called Recklinghausen's disease.

The symptoms will, of course, depend upon the particular nerve involved, and will be those of a neuritis. Besides, there will be present the physical evidences of the growth.

MULTIPLE NEURITIS.

Definition.—An inflammation of many peripheral nerves.

The pathology of neuritis has already been discussed. It must be understood that there are various grades of the disease, and that in the mild forms the pathologic changes are not marked. If the disease is severe, especially in the alcoholic form, there are, in addition, alterations in the cells of the anterior horns and medulla and pons. The changes in the nerve-cells consist of a displacement of the nucleus to the periphery and an alteration in the chromatin substance. Diffuse and degenerate inflammations with hemorrhages, especially in the region of the gray matter of the third ventricle

and the aqueduct of Sylvius, are sometimes found. Degeneration is often found in the anterior and posterior roots, the cranial nerves, and the anterior and posterior horns.

Predisposing and Exciting Factors.—Multiple neuritis is produced by a variety of causes, chronic alcoholism being the most common. Toxic disturbances, such as are produced by the various infectious diseases and the different metallic poisons, chronic diseases, as tuberculosis, malaria, beriberi, leprosy, and senility, may produce polyneuritis. It may come on without any apparent cause, or follow a cold, probably being in these instances, infectious in origin.

Symptoms.—Certain general symptoms are common to all forms of multiple neuritis, and these may be divided into sensory, motor, vasomotor, and trophic, their severity depending upon the extent of the pathologic process.

Sensory Symptoms.—These nearly always come on first, the patient complaining of a sense of numbness, pin- and needle-like sensation, or a crawling or dead feeling in the lower or the upper limbs. These increase until finally the pains become sharp and shooting, accompanied by an increasing tenderness of the muscles and nerves, with pain on pressure, sometimes becoming so acute that the slightest irritation or jar of the body will cause excruciating pain, and it is necessary to protect the patient from the surrounding bedclothes. Ordinarily these acute hyperesthetic symptoms last for a number of weeks, and then gradually subside, leaving for some time considerable tenderness to pressure over the nerve-trunks and muscles. Disturbances of sensation are common, but can only be demonstrated after the hyperesthetic stage, when it will be found that the areas of anesthesia involve part or all of a limb, their extent being often stocking- or glove-like. As the disease subsides the sensory symptoms become less and less, and finally disappear, although in the chronic forms of neuritis pain on pressure over the nerve-trunks with some sensory disturbances can always be found.

Motor Symptoms.—The weakness may come on slowly, the patient experiencing a gradual diminution of power in the lower or upper limbs, or, what is not at all infrequent, there is sudden inability to walk or to use the upper limbs. The motor symptoms usually come on coincidentally with the sensory or may precede them. The paralysis nearly always affects predominantly certain nerves—in the upper limb the musculospiral, and to a less extent the ulnar and radial; and in the lower, the peroneal and anterior tibial, this causing wrist- and foot-drop respectively. The muscles are flaccid and both the skin and tendon reflexes are rapidly lost. In the acute stage it is inadvisable to take the electric reactions, but as soon as it subsides reactions of degeneration can easily be demonstrated. Atrophy of the muscles comes on rapidly, and contractures may develop. The gait is typically steppage, the patient lifting his knees high from the ground because of the foot-drop, the toes coming down first.

Vasomotor and trophic functions are very commonly affected, the skin becoming dry and glossy, the hair may drop out, the nails become shiny and brittle, and in the chronic stages the limbs are cold. Bladder and rectal disturbances hardly ever occur.

In the so-called idiopathic or toxic form, which appears without any apparent cause, there may be a rise of temperature with febrile symptoms and a general feeling of malaise.

The prognosis and duration in most cases of multiple neuritis depend

largely upon their etiology. Most idiopathic and infectious forms get well, the duration of the disease being from one to three months.

ALCOHOLIC MULTIPLE NEURITIS.

The symptoms may come on during an alcoholic debauch, but in most cases they appear after prolonged alcoholism. Women are especially prone.

The symptoms are similar to those described above, and consist in a rapid sensory and motor involvement with acute pains, wrist- and toe-drop,

Fig. 427.—Bilateral Wrist-drop in Acute Alcoholic Multiple Neuritis.

and loss of reflexes. Disturbances in the cranial nerves are not at all uncommon, for we have in alcoholic neuritis not only involvement of the peripheral, but a general toxic change in the whole nervous system. Disturbances in sight sometimes occur and there may be a central scotoma,

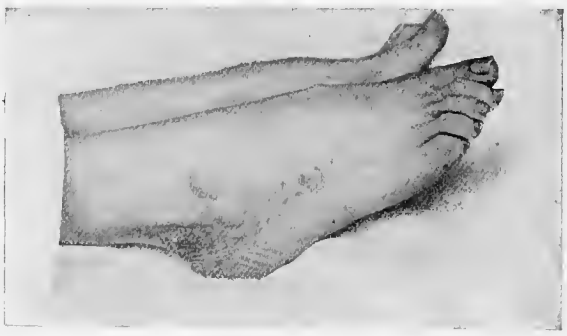


Fig. 428.—Bilateral Toe-drop in Acute Alcoholic Multiple Neuritis.

especially for colors. Differences in the size of the pupils and slowness in their reactions sometimes occur. Of the cranial nerves, the external rectus and the facial are most commonly paralyzed, and there may sometimes also be involvement of the oculomotor and trigeminus. The cranial palsies nearly always occur in the height of the disease, and as a rule only last for a short time, and are always indicative of serious degenerative changes in their respective nuclei.

Sometimes mental symptoms occur, these coming on at the height of the disease, and consist of loss or confusion of memory, especially for recent events, and, in addition, peculiar illusions and hallucinations. This is sometimes called Korsakoff's psychosis.

The course of the disease is rapid, and within a week or ten days the symptoms are at their height, where they may remain for two or three weeks and then gradually subside, leaving the patient with pain and tenderness over the nerve-trunks and muscles, and wrist- and toe-drop. The sensory symptoms are the first to leave, the palsies remaining. In most uncomplicated cases, that is, in which there is no involvement of the cranial nerves, the prognosis is good.

PSEUDO-TABES OR ATAXIC MULTIPLE NEURITIS.

In addition to the symptoms described above, there may develop ataxia of the upper and lower limbs, which may persist even after the acute symptoms have subsided, and it is somewhat difficult to differentiate the disease from locomotor ataxia. We have, however, in the latter, pupillary irregularities, the Argyll-Robertson pupil, girdle sense, bladder and rectal phenomena, and, most important of all, in multiple neuritis atrophy and weakness are prominent and the symptoms have a tendency to become less, the prognosis in most cases being excellent.

LEAD MULTIPLE NEURITIS.

It is well known that lead acts upon the central nervous system, but accurate knowledge of its pathology is lacking. There is no doubt that in lead intoxications the peripheral nerves may be preponderantly diseased and the symptoms of multiple neuritis demonstrated, but it is probable that a careful microscopic examination will show alterations throughout the brain and cord. Workers in lead, type-setters, and others who come in contact with this metal may suffer, although it is possible for nervous symptoms to develop only after mild exposure.

Symptoms.—These are usually preceded by lead colic. The poison seems to have a peculiar affinity for the posterior interosseus, causing paralysis of the extensors of the hand and fingers, while the supinator longus and triceps muscles are usually spared. The nerves of the lower extremity are not, as a rule, involved, but if they are, the peroneal nerve is usually diseased, the tibialis anticus nearly always escaping. Sensation is not often disturbed. Ataxia hardly ever occurs. The muscles are atrophic and reactions of degeneration are soon obtained. A blue line on the gums is an aid to the diagnosis.

The duration of the disease is long and the symptoms are of slow onset. The prognosis in uncomplicated cases is fairly good, provided the patient does not return to the cause of the intoxication.

Lead Encephalopathy.—Sometimes in the course of lead intoxications, accompanying the symptoms of multiple neuritis or without them, there may occur grave cerebral symptoms, such as delirium, coma, convulsions, epileptic seizures, hemorrhages, and transient hemiplegia. There may also be paralysis of the cranial nerves, especially of the third, fourth, and sixth, either alone or in combination. Optic neuritis or atrophy may occur as well as involvement of the vocal cords and the laryngeal muscles.

Pathologically in these cases there are found diffuse areas of inflammation in different portions of the brain and spinal cord. The prognosis is almost always grave.

ARSENICAL NEURITIS.

Arsenic is a frequent cause of polyneuritis, and this fact should be remembered when, as is often the case, arsenic is given in increasing drop doses. The symptoms are similar to those described under the general form. They come on, as a rule, very slowly, and are mild, hardly ever proceeding past the inflammatory stage with tenderness, pains, and wrist- and toe-drop. There are, in addition, the gastro-intestinal symptoms of the poison. The prognosis is good, especially if the cause is early recognized.

POLYNEURITIS DUE TO OTHER METALLIC POISONS.

Mercury, copper, phosphorus, and *carbon disulphid* and *monoxid* or illuminating gas have also been known to produce multiple neuritis, but these instances are rare.

The form of polyneuritis due to *carbon disulphid* is extremely uncommon, and little is known of its pathology, as observations are lacking. Workers in vulcanized rubber are especially prone to this disease. Mental excitation or depression with hysterical manifestations precede the neuritic phenomena, which are, as a rule, of the ataxic form, and resemble greatly the alcoholic form of neuritis. Hysterical symptoms are so common that some authors, as Marie, insist that the hysterical manifestations are among the most prominent symptoms. Ocular phenomena are common, and consist, as a rule, of alterations in the visual fields, especially for colors. Amblyopia is fairly constant. Scotoma, either large or small, is found in some cases. Pupillary rigidity and even nystagmus have been recorded.

It is probable that there is here not a pure multiple neuritis, but a toxic process which so influences the central nervous system as to produce the various mental, ocular, and neuritic symptoms. It must be acknowledged that most of the various manifestations shown in this disease are hysterical in nature; but why should a previously healthy individual who is poisoned by carbon disulphid be hysterical only so long as the influence of the poison lasts?

The action of toxins, whether generated within or without the body, upon the brain and spinal cord is becoming better recognized. It is more than probable that their influence is not selective but general, and that we have alterations not only in the peripheral nerves, but also in the central nervous system. Such is the case, for instance, in lead or alcoholic poisons and in uremia.

DIPHTHERITIC PARALYSIS.

Approximately about one-quarter of the total number of cases of diphtheria are followed by paralysis. It is more liable to follow a severe attack, although paralysis has been known to follow a simple sore throat or diphtheritic inflammation elsewhere in the body. The older the person, the greater the tendency.

As a rule, the paralysis does not appear until the diphtheria has entirely disappeared, in the third or fourth week and sometimes later, although in rare cases it may occur in its height. The symptoms of the polyneuritis are generally mild, and consist only in some pain on pressure over the nerve-trunks, rarely sensory disturbances, and the paralytic symptoms, as a rule, are not very marked.

Paralysis of the palate is the most frequent and early symptom. It

can be recognized by the nasal voice and difficulty in eating, regurgitation of food through the nose being common. Coincident with this or soon after, paralysis of the ciliary muscles appears, with consequent loss of accommodation and impairment of vision for near objects. The palatal and ciliary symptoms, as a rule, do not last more than a few weeks, and then gradually disappear. Occasionally there is temporary impairment of some of the ocular nerves, and more rarely there may be interference with the functions of the vagi, hypoglossus, and facial nerves.

BERIBERI OR KAKKÉ NEURITIS.

This disease is rare in this country, although it is prevalent in the seacoast cities of our southern States. It is quite common in tropical countries. The etiology of beriberi is still in doubt, but it is probably caused by soil infection, and not by a rice diet, although this may be a contributory cause. There are three principal forms—the trophic, dropsical, and mixed, these probably being different stages of the disease. Besides the ordinary symptoms of multiple neuritis, there is great disability. Ataxia is almost always present. Dropsical effusions in nearly all of the serous cavities may be present.

FIG. 429.—BERIBERI (Herzog, in "Philippine Journal of Science").

MULTIPLE NEURITIS DUE TO OTHER CAUSES.

Infectious diseases such as erysipelas, typhoid fever, pneumonia, measles, scarlet fever, gonorrhea, influenza, rheumatism, malaria, and more rarely leprosy, tuberculosis, syphilis, carcinomatous and diabetic toxins, may produce multiple neuritis, but in most of these instances the neuritic symptoms are very mild and slow in onset. Sometimes in old age there is a form of senile polyneuritis which is characterized by a slow onset, absence of sensory disturbances and of any apparent cause. As a rule, there are severe arterial changes, and the neuritis is probably due to the lessened blood-supply.

NEURALGIA.

Definition.—A sensory disturbance characterized by pain of a sharp, shooting character, coming on spasmodically and always in the same distribution. Symptomatically it differs from a neuritis in the fact that there

are present only sensory symptoms, the pain is not continuous, and there is no pain on pressure over the nerve-trunks between the attacks.

Pathologically, degenerations have been found in the peripheral nerves, sometimes changes in the sensory ganglia of the posterior roots, and, as in trigeminal neuralgia, in the Gasserian ganglia. Again, no changes have been found at all. Neuralgia is really a symptom of sensory irritation, and in some cases, as in sciatica, it is difficult to differentiate between it and a true neuritis, but because of the characteristics of the pain this term is generally applied to the functional condition.

Predisposing and Exciting Factors.—Neuralgia may appear without any apparent cause, when it is called idiopathic. It may be due to such general constitutional diseases as malaria, rheumatism, gout, anemia, to local irritations of a nerve resulting from pressure, as from a growth, or it may follow grippe and various metallic intoxications. It occurs mostly in adults and rarely in old age.

Symptoms.—Neuralgia, no matter of what part, nearly always comes on slowly, and there may be at first only mild feelings of paresthesia, to be soon followed by pains, which become more intense, until finally they are typically sharp and shooting and come on spasmodically. They may last from a moment to a few seconds or longer. Between the attacks there may be a sensation of fullness or a dull ache. It is characteristic of neuralgia that the pains come on intermittently, and that they are nearly always in the same distribution, although in the very beginning they may only involve a part of a nerve, as in tic douloureux. Attacks of pain are brought about without any apparent cause.

During the attack the skin distribution is hypersensitive, and the exit points of the nerve are painful to pressure. Sometimes they are also painful during the intermission. Disturbances of sensation are not common. There are, as a rule, no accompanying motor phenomena except those which are superimposed by the pain and are of reflex character.

Disturbances of vasomotor and trophic functions are common, and there may be disturbances of sweat secretion, dropping out of the hair and sometimes even a change of color, the hair becoming grayish after an attack. The skin may also become glossy, and ulcers rarely form, especially in trigeminal neuralgia, in which when the ophthalmic division is diseased, a trophic corneal ulcer sometimes occurs. Herpetic eruptions are common, especially in the intercostal form, and are usually in the distribution of the involved nerve.

The course of the disease is frequently chronic, and unless it is promptly treated and the causes eliminated, may last for years. The special forms of neuralgia will now be discussed.

Occipital Neuralgia.—In this type the upper or the first four nerves of the cervical plexus are involved, and the pain is distributed to the back of the head and neck as far as the occiput, and at times as far as the parietal region. The pains are usually bilateral, and the points of tenderness are generally in the base of the occipital bones posterior to the mastoid process. Besides the usual causes, caries or tumor of the vertebra should be considered. It sometimes occurs in association with disease of the fifth nerve and torticollis.

Brachial Neuralgia.—This usually appears in men without any apparent cause, and the pain may involve the whole brachial plexus, or be limited to any one of its subdivisions, especially the ulnar and radial.

Sometimes in association there may be pain on the side of the neck. Because of the pains the arm is not used, and there may develop some atrophy.

Intercostal Neuralgia.—This usually involves the thoracic nerves between the fifth and ninth inclusive. Both sides may be attacked, but the disease is usually unilateral. The pains are very severe, and are so sharp and agonizing that the patient fixes his trunk, will not move, and coughing, sneezing, and even respiratory movements will be inhibited. Herpetic eruptions are especially common in this form (herpes zoster), and may come on with the pains or independently of them. Besides the usual causes, disease of the vertebra pressing upon the posterior ganglia or roots should always be suspected.

Sciatica.—This form more nearly approaches neuritis than the other types of neuralgia. The onset is usually slow, and consists of numbness or pains in the back of the thigh and calf, which gradually increase until finally there are present typical sharp, shooting pains which start in the buttock and gluteal region and extend along the back of the thigh to the hollow of the knee, and then to the outer part of the leg and foot or to the back part of the calf and leg to the ankle. Besides the exacerbations, pain of a constant, dull aching character is nearly always present, and is increased by movement of the leg. There is also pain on pressure over the whole nerve-trunk, along the back of the thigh, calf, and especially over the sciatic notch. If the leg is extended on the thigh and the thigh flexed on the abdomen, causing stretching of the sciatic nerve, there will be pain over the sciatic notch. Disturbances of sensation are rarely present, but falling out of the hair and a glossy condition of the skin are common. The knee jerks are not altered, but the Achilles jerk is nearly always either diminished or lost. Sometimes because of the effort of the patient to save his leg there results a characteristic deviation of the trunk to the other side, with a lumbolateral scoliosis. The course of the disease is nearly always prolonged, and if improvement is not obtained within a few weeks, it has a tendency to become chronic. Paralysis of movement does not occur, but there may result, especially late in the disease, some atrophy of the muscles of the back of the thigh and leg. Bilateral sciatica is nearly always indicative of a tumor in the pelvis (Fig. 430).

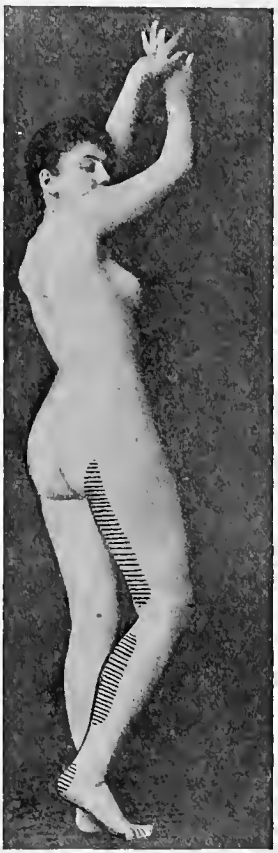


Fig. 430.—Area of Tenderness and Pain in Sciatica and Typical Position of Lower Limb.

Unusual Forms of Neuralgia.—*Mastodynia*, neuralgia of the mammary gland, nearly always occurs in adult women, and is a form of intercostal neuralgia limited to the breasts, which are generally sensitive to touch.

Neuralgia in the region of the *lumbar* and *sacral plexuses* occurs, this causing typical pains in the buttocks, genital organs, rectal region, and a peculiar form which is called *coccygodynia*, which occurs in women, and in which the pain is localized to the coccygeal region, it being increased by walking, sitting, or defecation. Sometimes the neuralgia is limited to the insertion of the Achilles tendon, *achillodynia*, to the heel, *tarsalgia*, or to the *metatarsal bones*.

VASOMOTOR AND TROPHIC DISEASES.

Our knowledge of the vasomotor system is vague. There are supposed to be vasoconstrictor and vasodilator fibers, which no doubt are transmitted by the peripheral and sympathetic nerves to the spinal cord, and then to the brain, this presumption being based upon the fact that lesions in any of these parts cause what are called vasomotor and trophic phenomena. Again, there are diseases which have purely so-called vasomotor or trophic symptoms, or both. These have been described by many authors, and have been given various names, being called after either their leading symptoms or the men who described them, and because of this, confusion exists as to their classification. As a matter of fact, nearly all of these diseases have a common basis, their symptomatology depending upon the method of onset and preponderance of certain symptoms.

By *vasomotor phenomena* are understood an increase, decrease, or loss of the blood-supply, this causing either redness, paleness, or gangrene, and disturbance in glandular secretions, which may be increased, decreased, or perverted. Associated with the above there may be disturbance of sensation, this consisting in either irritative or destructive phenomena, such as paresthesia, described as crawling, pin-and-needle, numb, or a dead feeling, or of severe pain and a loss of sensation which may be partial or total.

Under *trophic disturbances* are understood alterations in the nutrition, structure, or growth of the hair, nails, skin, and the underlying soft and deeper tissues and bones.

In this classification of vasomotor and trophic diseases it must, of course, be understood that the phenomena described under these headings may appear alone or in combination, and that the symptoms of one or both may be associated with disease of the brain, spinal cord, and peripheral nerves.

Etiology and Pathology.—In a word, the causes which lead to vasomotor and trophic diseases are not known. There is nearly always a neuropathic disposition. Pathologically there may be found, in such diseases as erythromelalgia and Raynaud's syndrome, an alteration in the peripheral nerves and vessels and changes in the blood. In the so-called trophic diseases, as acromegaly and progressive facial hemiatrophy, there are, of course, true hypertrophy and atrophy of the tissues.

VASOMOTOR DISEASES.

Under this heading can be described many diseases, but it is probable that they all have an interrelation, and that their differences consist in the onset, character, and preponderance of certain symptoms. The first classification can be made of those in which the principal symptoms are those of paresthesia, with or without trophic changes.

Acroparesthesia.—By this is understood a diffuse sensation of numb-

ness or of a dead feeling in any or all of the upper and lower limbs, especially in the hands and feet. At first it is associated with vasomotor changes, and it is probable that, when occurring alone, it is the early manifestation of a subsequent vasomotor disease, such as chilblain, acrocyanosis, erythromelalgia, angioneurotic edema, or Raynaud's disease.

Chilblain.—This usually comes on after warming of the feet when they have first been either chilled or frozen. There is usually a sensation of pins and needles, associated with a sense of numbness, and the feet feel cold and are so to touch. Often associated with this the skin is pale or red, and there may be ulcerations. It may be the beginning of an acrocyanosis or Raynaud's disease, but, as a rule, occurs independently.

Acrocyanosis.—The leading characteristic of this is a blueness of the extremities, associated with numbness or a pin-and-needle-like sensation. These symptoms may be the only manifestations of the disease, or may be the beginning of Raynaud's syndrome, or, as sometimes happens, after the blueness or numbness disappears the skin becomes profusely red and then white. Rarely there are disturbances of sensation in the cyanotic parts.

Erythromelalgia.—By this is meant pain and redness of the terminal parts of the upper or lower limbs, the feet being more frequently affected. The redness, as a rule, comes on gradually or in paroxysms, and is associated with more or less numbness and pain, and when well marked the limb is generally swollen, red, and painful to pressure. The disease is chronic, and may terminate in gangrene of the toes, terminal part of the foot, or fingers.

Raynaud's Disease; Symmetrical Gangrene.—The disease, as a rule, is slow in onset, and is characterized at first by a sense of crawling or numbness in the fingers, toes, or both, with periodic paleness and coldness which may last from a few minutes to an hour or longer. Very soon there will be in association a bluish condition, which may be succeeded by redness, the fingers and toes feeling numb or painful. As this continues there will gradually develop gangrene of the toes or fingers, usually in symmetrical parts. Gangrene may sometimes occur in the ears, nose, and lips, or in different portions of the upper and lower extremities.

ANGIONEUROTIC DISEASES.

In this group are included those symptom-complexes in which there is a sudden swelling of a part, the result of serous or hemorrhagic effusion from the blood-vessels.

Angioneurotic Edema.—This is characterized by a sudden swelling, coming on either acutely or in a few hours, in the forehead, face, lips, tongue, larynx, or genital organs—in fact, in any portion of the body. The swelling is round, circumscribed, does not pit on pressure, and is not painful. The superimposed skin is white, pinkish, and irritable, and there may sometimes be in association urticarious eruptions. It usually subsides in the course of a few hours. The attacks, as a rule, are not dangerous, except when they occur in the pharynx and larynx, when they may cause interference with breathing. Sometimes it occurs in the intestines or stomach, when there will be in association colicky pains with either diarrhea or vomiting and tenderness to pressure.

Urticaria and Purpura.—It is probable that the changes in the skin and other symptoms which occur in these diseases are similar in etiology to angioneurotic edema, the only difference being in their manifestations. Very

often the three conditions may be present in the same person. Purpural eruptions may be of various intensity, and sometimes are associated with grave constitutional symptoms. They may consist only in a curious pinkish or purplish mottling of the skin of both lower and upper limbs, this being increased when the limbs are held downward and decreased when held up.

Disturbance of Sweat Secretions.—This may consist in excessive sweating, such as occurs sometimes in the palms of one or both hands or of both feet. In association with this there may be a curious overgrowth of the nails with an exfoliation, and sometimes even disturbance in the nutrition of the hands and feet. Disturbances in sweating sometimes occur on one side of the body, or may be referred to one limb or the face, and are generally associated with hysteria. At times there may be a curious odor of the sweat secretion; it may be bloody or of different colors. This, of course, is rare. Sweating may be entirely absent.

TROPHIC DISEASES.

Under this heading will be discussed those diseases in which there occurs an alteration in the nutrition, structure, or growth of part or all of the body.

Scleroderma.—This is a peculiar disease characterized by either a general or local disturbance of the skin and some of the underlying tissues. It may be limited to the face, neck, upper limbs, genital organs, or to certain other limited portions of the skin, when it is called *morphea*. It is characterized by a peculiar hardening and contraction of the skin, which sometimes becomes discolored, and there may rarely be eruptions over the involved parts. The skin is hard to the touch, cannot be pinched, and does not pit to pressure. It produces deformities, and when over the face it will cause a mask-like condition; when in the fingers, a peculiar deformity and contraction; and if limited to the chest, may inhibit respiration. Atrophy of the parts may follow. Its cause is unknown. It is chronic and recovery very rarely ensues.

Myxedema.—Pathologic Definition.—A nutritional disorder, associated with atrophy and loss of function of the thyroid gland. There is a myxedematous infiltration of the subcutaneous tissues and a cretinoid cachexia.

Clinical Varieties.—(1) True myxedema; (2) cretinism (the absence of thyroid function-congenital, or lost during childhood); (3) operative myxedema, due to total removal of the thyroid glands.

Nature of Myxedema of Adults, and Etiology.—Atrophy of the thyroid is usually present, and the gland may either be changed into a small fibrous mass or be absent. The therapeutic test of improvement under the administration of thyroid extract sustains this view. The fact that in a good many cases of myxedema a considerable portion of the thyroid gland is unaltered and partly capable of functionating arouses a suspicion that the hypophysis may share in the production of myxedema. Myxedema may be secondary to exophthalmic goiter, but it is then, as in the case of simple acute goiter, only a transient condition. Women are much more frequently affected than men. The disease may attack several members of a family, and hereditary transmission through the mother is probable. Sisters may suffer, one from myxedema and the other from exophthalmic goiter. Pregnancy may rarely cause a disappearance of the myxedematous symptoms, but these symptoms may reappear after delivery.

Symptoms.—There is retardation of psycomotor action. Mental perception and thought are slow, and the memory, while retentive, is also slow to respond. Not infrequently there may be irritability, and hebetude alternating with sudden periods of excitability. The patient is subject to delusions and hallucinations; or the apathy may pass into a melancholia, ending finally in dementia. Among the minor or accessory features may be abnormal subjective sensations, as taste and smell, and occipital headache. Hemorrhages from the nose, gums, and bowels sometimes occur.

Physical Signs.—The face appears to be swollen, rounded, and the features somewhat distorted and expressionless. The skin and mucous membrane displays a peculiar pallor, or the so-called cretinoid cachexia. The fingers are thick and clubbed, and their cutaneous covering rough and deeply wrinkled at certain portions, while the articular surfaces of the skin may be somewhat elevated. The hair is thin over the scalp and has a rough and lusterless appearance. In extreme cases pubic and axillary hair may be absent. The mucous membranes are also thickened, consequently the tongue, lips, and nose are appreciably enlarged, and the teeth may be loosened. The feet and lower extremities present a condition quite similar to that of the hands. The movements are slow and the gait somewhat uncertain, and there is often disturbed co-ordination.

Palpation.—The hair feels rough and lifeless. The skin is somewhat roughened, and, while appreciably thickened, does not pit upon pressure. Late during the course of myxedema the signs of peritoneal fluid are occasionally observed. Ordinarily it is impossible to palpate the thyroid gland, and this may be in part due to atrophy of the organ on the one hand, and to thickened mycedematous tissue of the neck on the other.

Percussion.—This physical method is of but limited service, except for determining the size of the heart (dilatation) and the presence of abdominal fluid, both of which features are seen late during the disease.

Auscultation.—The speech is slow, somewhat drawn, and accentuation impaired so that the patient's voice is in monotones, to which are attached a peculiar nasal element.

Laboratory Diagnosis.—The quantity of nitrogen excreted through the urine is below that of the normal. In some cases the urine may be found to contain albumin and sugar.

Cretinism.—This is allied very closely to myxedema, inasmuch as it is due to the same cause—congenital maldevelopment or atrophy of the thyroid gland. It is endemic in certain localities, as Switzerland, but may sometimes be found sporadically. The symptoms are recognized early in infancy. The growth is stunted, the figure small, walking is delayed, and the bones are usually poorly formed. The facial appearance is typical, consisting in a retracted nose, large lips and mouth, and lolling and enlarged tongue with some dribbling of saliva. The face is large, the lower jaw and brow are prominent, and the eyes are small. The intellect is usually impaired and talking is interfered with. If the disease is marked, there may be an anteroposterior curvature of the spine with protrusion of the abdomen. The skin is usually waxy and pale and the hair brittle (Figs. 431 and 432).

Amaurotic Family Idiocy.—A rare disease, first described by Sachs, characterized by mental impairment, observed during the first months of life and leading to absolute idiocy, paralysis or paresis of the greater part of the body, which may be either flaccid or spastic, the reflexes being

either normal, increased, or diminished, with diminution of vision terminating in absolute blindness; the latter is typical of the disease and consists pathologically of a cherry-red spot in the region of the macula lutea, and later in atrophy of the optic nerve. The disease terminates fatally, as a rule, before the age of two years. The condition is first noticed from about the third to the sixth month, the first symptoms being those of general apathy, followed by disturbance of vision which rapidly leads to blindness. It is a family disease, and nearly all the reported cases have occurred in the Hebrew race. The etiology of the disease is still obscure.

FIG. 431.—CRETIN BORN IN PHILADELPHIA OF ITALIAN PARENTS.
Five and one-half years old. Height 22½ inches.

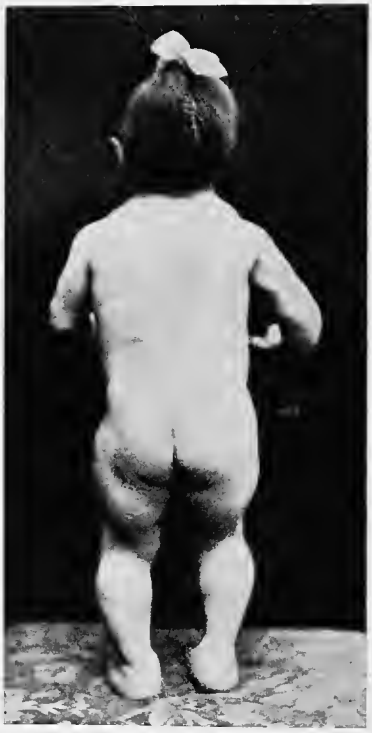


FIG. 432.—POSTERIOR VIEW OF CASE SHOWN IN FIG. 431.

Adiposis Dolorosa.—A disease first described by Dercum, appearing in adult life and characterized by gradual fatty enlargements of various portions of the body, associated with some pain and tenderness. There is usually great muscular weakness and a curious mental disturbance which is generally associated with a neuropathic disposition. The cause of the disease is unknown.

EXOPHTHALMIC GOITER (GRAVES' DISEASE; BASÉDOW'S DISEASE).

A disease characterized by protrusion of one or both eyeballs, enlargement of the thyroid gland, palpitation, and a general neurotic condition. It is

the result either of excessive or abnormal secretions of the thyroid gland. It is more common in women and generally appears in early adult life.

Symptoms.—It generally begins with nervousness, the patient becoming easily excitable, tired, complaining of headache, tremor of the limbs, and palpitation. These symptoms are progressive, and are associated with an increase of heart action, the pulse-rate sometimes being from 150 to 200 per minute. There may be a thrill, which can be detected over the vessels of the neck. As the disease progresses there may be involuntary evacuations of the bowel, disturbances of sweat secretion, and insomnia, with a gradual increase in the symptoms already mentioned, and anemia.

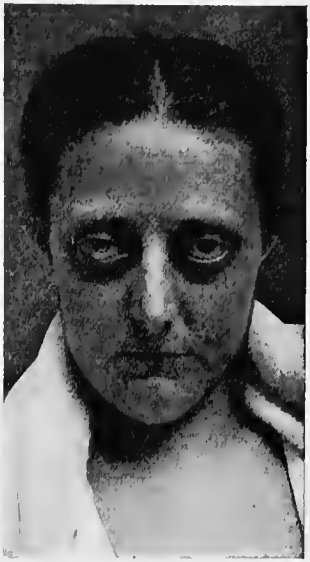


FIG. 433.—EXOPHTHALMIC GOITER, PHOTOGRAPHED AT THE TIME WHEN GOITER WAS NOT MARKEDLY ENLARGED.

Coincidentally there develops a gradual enlargement or protrusion of one, generally of both eyeballs, which is easily detected. When the eyes are made to follow a finger from above downward, there is a lagging of the upper lid (von Graefe's sign), and when the patient first attempts to look at the finger there is usually a spasmodic contraction of the upper lid (Kocher-Boston sign). Because of the protrusion, the palpebral fissure is widened (Stellwag's sign), and there is often failure of convergence (Möbius' sign). Vision is usually not interfered with (Figs. 434 and 435).

1 2

FIG. 434.—METHOD OF OBTAINING BOSTON-KOCHER SIGN, WHICH CONSISTS IN A SPASMODIC CONTRACTION OF THE UPPER LID WHEN FIRST ATTEMPTING TO LOOK AT A FINGER. NOS. 1 AND 2.

FIG. 435.—METHOD OF OBTAINING VON GRAEFE'S SIGN, WHICH CONSISTS IN LAGGING OF THE UPPER LID WHEN THE FINGER IS FOLLOWED FROM ABOVE DOWNWARD.

Enlargement of the neck gradually develops, and, as a rule, is asymmetrical, involving one gland more than the other, and is easily detected.

Summary of Diagnosis.—A young adult with gradually increasing protrusion of both eyeballs and lagging of the upper lid on looking downward; widening of the palpebral fissure; spasmodic contraction of the upper lids on fixing and occasionally failure of convergence; enlargement of the thyroid glands with a general nervousness, consisting in palpitation, rapidity of the pulse-rate, a thrill to be detected over the vessels of the neck; occasional diarrhea, tremor of the limbs, and general increase of reflexes.

Differential Diagnosis.—There should be no difficulty in diagnosticating this disease when the three principal symptoms of enlargement of the neck, protrusion of the eyeballs, tremor, and general nervousness are considered.

Fig. 436.—Method of Obtaining Möbius' Sign, Consisting in Failure of Convergence.

Prognosis and Duration.—The course of the disease is chronic. Sometimes, however, operative interference causes a cure.

ACROMEGALY.

A rare disease, characterized by enlargement of the soft and bony parts, principally of the hands, feet, and face. In quite a number of cases it has been found in association with disease of the pituitary body, but this is not constant. When such is the case, there will be disturbances of vision, as bitemporal hemianopsia, diminution of sight, with either optic atrophy or choked disc, and rarely the general symptoms of brain tumor, as headache, nausea, and vomiting. The disease usually appears in young adults, and is characterized by gradual enlargement of the hands, feet, and face, the hypertrophy involving all parts equally. The fingers and toes become broad and thick, the hands and feet enormously large, the face becomes hypertrophied, involving especially the lower jaw, nose, lips, and supraorbital ridges, the eyes may be prominent, the teeth are widely separated, the tongue is enlarged, and the skull, especially in the frontal and occipital portions, also

hypertrophies. As the disease progresses the terminal parts of the long bones also become hypertrophied, and there develops an anteroposterior curvature of the spine and enlargement of the bones of the chest. The

FIG. 437.—ACROMEGALY IN FEMALE CHILD FIVE YEARS OF AGE.

genital organs may take part in the hypertrophy. The disease is progressive, of long duration, and is usually associated with physical weakness and dullness of intellect (Figs. 437, 438, 439).

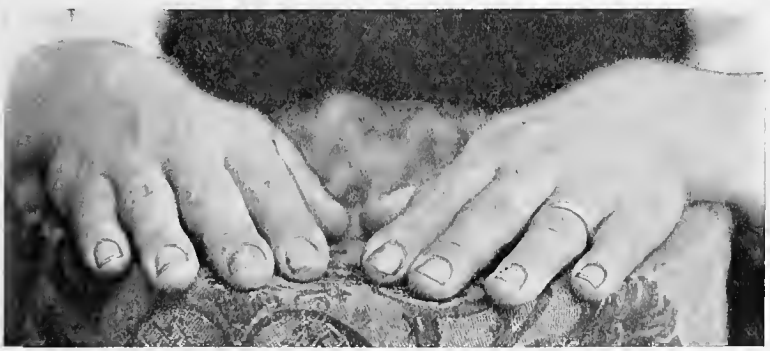


FIG. 438.—ENLARGEMENT OF THE HANDS IN ACROMEGALY.

The enlargement of the bones may be of two kinds. When beginning in early life before the bone has reached its normal development, the hyper-

trophy is in the longitudinal direction, causing what is known as *gigantism.* When occurring later, the tissues enlarge in width and produce acromegaly.

There should be no difficulty in diagnosing acromegaly. Sometimes, however, there may be an enlargement of the fingers and toes, and only rarely of the cranial bones in association with chronic pulmonary disease. This is known as *hypertrophic osteoarthropathy.* Occasionally there may be enlargement only of the bones of the skull, especially of the upper portion. This is known as *hyperostosis cranii.*

PROGRESSIVE FACIAL HEMIATROPHY.

This is a rare disease, coming on, as a rule, in early adult life and characterized by progressive atrophy of one-half of the face. It involves equally the skin, underlying tissues, and bones, and sometimes one-half of the palate

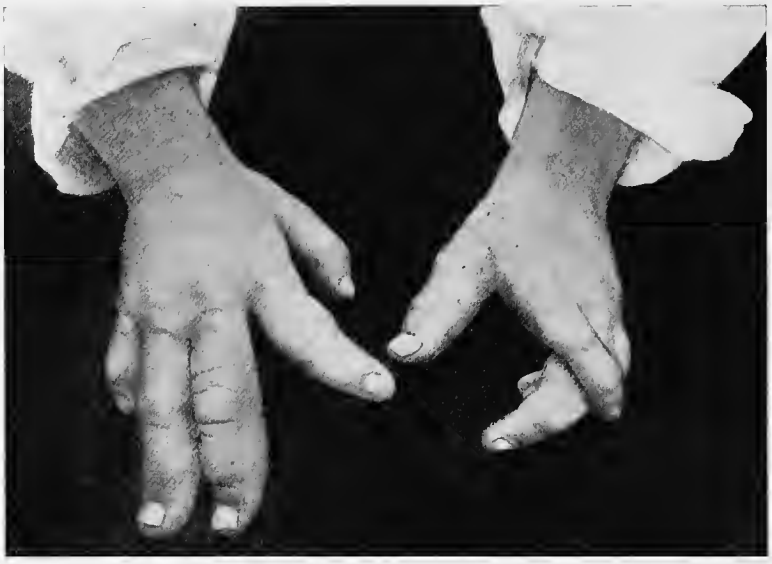


FIG. 439.—HANDS IN A CASE OF ACROMEGALY SHOWN IN FIG. 437.

and tongue. It is of slow onset, the skin generally puckers, becomes dry, and, as the face becomes smaller, there will develop a groove or ridge in the middle of the brow. Disturbances in the growth of the hair and of sweat secretions are common. It is supposed to be a disease of the fifth nerve, but it probably involves more, inasmuch as its distribution is greater. Rarely there may be an involvement of the muscles of the neck and shoulder. Progressive facial hemihypertrophy is rarely seen.

ARTHRITIS DEFORMANS (RHEUMATOID ARTHRITIS).

Definition.—A disease of the joints characterized by alterations in the structure of the cartilage, synovial membranes, and the articular ends of the bones, with bony deposits causing stiffening and deformity of most of the joints of the body.

The pathologic processes in this disease have become better recognized since the discovery of the *x*-ray. There are usually at first changes in the cartilage and synovial membranes, with proliferation of tissue, the changes in the cartilage being most marked, sometimes disappearing entirely. Gradually there appear bony or osteophytic deposits in the membranes, articular surfaces of bones, and especially in the ligaments, and the joint becomes en-

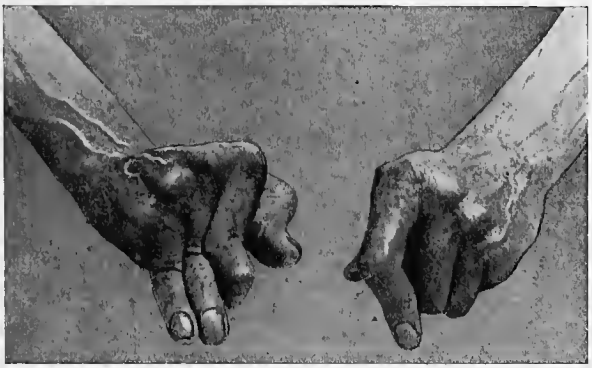


Fig. 440.—Dorsal View of Hands in a Case of Arthritis Deformans.

larged and motion limited. Sometimes there is great atrophy and erosion in the ends of the long bones and they become very friable. This occurs especially in old persons. The pathologic process is slow and may involve one joint, a number of joints, or every joint in the body.

Predisposing and Exciting Factors.—Joint diseases in parents predispose toward this affection. At one time it was thought that rheuma-

Fig. 441.—Palmar Surface of Hands in a Case of Arthritis Deformans.

tism or gout was largely responsible, but such history is only obtained in a few cases, and it is probable that neither is the exciting cause. Autointoxication and chronic infections, as gonorrhea, have been thought to be potent factors. Changes in the nervous system have not been definitely demonstrated. The disease is more prevalent in women, and usually appears between the thirtieth and fortieth year.

Symptoms.—These will necessarily depend upon the number and particular joints diseased and mode of onset. In the usual adult type the involvement of the joints, as a rule, is very slow, although occasionally there may be a rapid onset, with some fever and enlargement of the joints, which are often tender and very painful, as in rheumatism. After the acute symptoms have subsided, the further progress of the disease is slow. In the usual chronic type the metacarpo-phalangeal joints are nearly always the first to be involved, becoming gradually painful and stiff, the pains very often resembling those of a neuritis. This is followed by a gradual symmetrical involvement of the wrist-, elbow-, and then the shoulder-joints. In the lower limbs the knee- and hip-joints are preponderantly diseased, the ankles and joints of the feet and toes usually escaping for some time, although occasionally every joint in the body may be affected. It can readily be understood, then, that the symptoms will vary greatly, for it often happens that only one or two joints may be diseased, or the process is so slow that the diagnosis is for a long time made difficult unless *x*-ray examination is made, when the characteristic joint changes are found. This most frequently occurs when the shoulder- or hip-joints are alone diseased, or in those cases in which these joints are first involved, the pains very often being of such intensity that a false diagnosis of neuritis is made (Figs. 440, 441, 442).

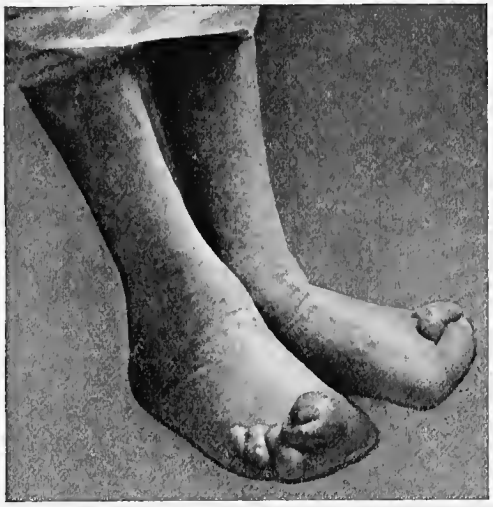


Fig. 442.—Distortion of the Toes with Deformity of the Foot in Arthritis Deformans.

Soon after the onset of the stiffness and pains the joints become enlarged and tender, and characteristic deformities develop. In the metacarpo-phalangeal articulations these are characteristic, and consist in a nodosity of the joints with a deviation of the fingers toward the ulnar side, the forearms, as a rule, being pronated and the elbows flexed. When the shoulder and spine become involved, the head is bent forward, no movement being possible, and in the lower limbs the legs are flexed on the thigh and the thigh on the abdomen. Because of the deformity and contractures there is great muscular wasting, the skin may be glossy and the hair brittle. Crepitation in the joints is early detected. The reflexes may be increased at first, but soon are lost.

Heberden's Nodes.—Very often the disease limits itself to enlargement of the sides of the terminal phalanges of the fingers, consisting only in round, knob-like deposits. They are often swollen, tender, and painful, especially in the early stages, and more rarely after dietary indiscretion. The disease is chronic, of long duration, and the other joints escape. It is more common in women.

Senile Form (Morbus Coxæ Senilis).—Very often in old persons, especially in men, there may be an involvement only of one joint, usually

of the hip, and more rarely of the hip and shoulder, the bones becoming atrophic and brittle. There is nearly always considerable deformity, inability to walk, and great atrophy.

Vertebral Form (Spondylose Rhizomelique).—A disease characterized by progressive stiffness and ankylosis of the spinal column, shoulders, hips, and more rarely of the spinoclavicular, knee, and other joints of the extremities. The cause is unknown. The pathology consists in a rarefaction of the osseous tissue and an ossification of the ligamentous structures of the joints. It is of slow onset, occurs in adults, and is usually preceded by considerable pain, of a sharp, shooting, or numb character, which is followed by gradual stiffening of the back, shoulder, neck, and upper and lower limbs, and finally no movements are possible in any part of the body, those of the hands and feet being retained the longest. Sometimes there may be a preponderance of the stiffness in the upper spine, shoulder, neck, and arms (von Bechterew type), or of the lower spine, hip, and lower limbs (Strümpell-Marie type) (Fig. 443).

Laboratory Diagnosis.—There is an appreciable reduction in the hydrochloric acid of the gastric fluid.

OSTEOMALACIA.

Definition.—A bone disease characterized by gradual softening or decalcification of its structure, due to disappearance of its earthy salts. It usually comes on in adult life, especially in women, and has a direct re-

Fig. 443.—Spondylose Rhizomélique.
Complete rigidity of all joints with the exception of the elbow, wrist, and fingers.

lation with the child-bearing period, usually coming on during or after pregnancy. The shafts of the long bones are principally diseased, becoming soft, friable, and decalcified, and because of this the Haversian canals become larger than normal. The bones gradually become pliable, and it is possible when the disease is marked to twist and bend the bones at will. The bones of the head nearly always escape, although the teeth may be carious. There is usually an excess of excretion of calcium salts in the urine.

Pain of a deep-seated character is first complained of, this becoming accentuated by pressure over the bones or by movement. With the gradual softening and pliability of the bones certain deformities occur which are characteristic of the disease. The stature becomes smaller, often the height diminishing a number of inches; the sternum becomes prominent because of the giving way of the bones of the sides of the chest, and there may develop a deformity of the spine, the head being held forward somewhat stiffly; but, most characteristic of all, the pelvis becomes deformed, the symphysis becoming very prominent and the sides of the pelvis approximated. This deformity can be easily detected by pelvic examination, and has an

important bearing upon future maternity. Because of the pelvic deformity there will develop a gradual and characteristic hopping or waddling gait, which is further accentuated by the deformities of the long bones of the limbs. Sensory disturbances are not the rule, but very often because of spinal deformity there may be involvement of the posterior roots, and rarely of the spinal cord, with consequent characteristic root pains and disturbance of sensation. More rarely the anterior roots are involved, causing fibrillary tremors and atrophy. The reflexes may at first be increased, but, as a rule,

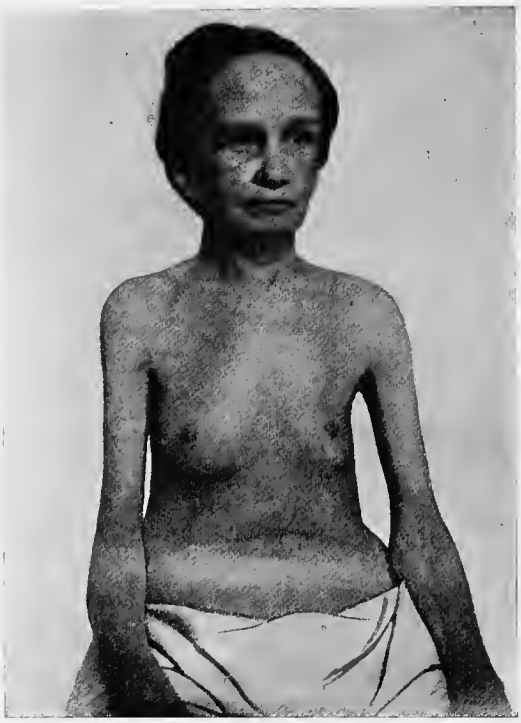


FIG. 444.—CASE OF OSTEITIS DEFORMANS (PAGET'S DISEASE) SEEN IN 1910 AT THE PHILADELPHIA GENERAL HOSPITAL (SERVICE OF DR. B. FRANKLIN STAHL). (Proc. College of Physicians of Philadelphia, 1911.)

become gradually lost. The disease is of long duration, death usually resulting from some other cause.

OSTEITIS DEFORMANS (PAGET'S DISEASE).

A rare disease, usually occurring in males in the latter end of life, and characterized by a gradual irregular thickening of the bones of the head with a softening of the long bones, especially of the lower limbs. Very little is known of its etiology, as few cases have been studied. The changes in the bones are those of a rarefying osteitis with some formation of new tissue. It usually comes on in adults past the fiftieth year, causing enlargement of the bones of the head with characteristic triangular deformity, the base being upward. This may be the only manifestation of the disease, when it is called *hyperostosis cranii*, but, as a rule, there is in association a softening and pli-

ability of the shafts of the long bones. Associated with this there is great muscular weakness. The disease is of long duration, death generally being caused by some intercurrent disease.

TETANUS (Lockjaw; Trismus).

Definition.—An infectious disease characterized by stiffness and tonic spasms, usually beginning in the muscles of the jaw, and finally involving all parts of the body, generally terminating in death. The specific bacillus has been isolated and is definitely known. The infection is carried

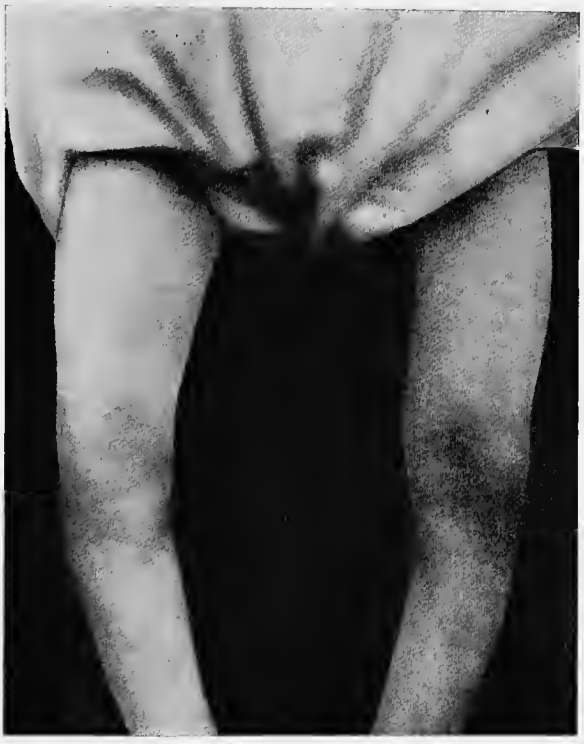


Fig. 445.—Case of Osteitis Deformans (Paget's Disease) seen in 1910 at the Philadelphia General Hospital (Service of Dr. B. Franklin Stahl). (Proc. College of Physicians of Philadelphia, 1911.)

by the soil, and is usually transmitted by wounds to the hands and feet, and sometimes after injury by rusty nails or firecrackers. The point of entrance may be not at all apparent, or may take some time to heal.

Symptoms.—The period of incubation varies. As a rule, the first symptoms appear within a week or ten days, and are usually manifested by a gradual feeling of weakness, malaise, headache, chilliness, and sometimes rise of temperature. Gradually there develops a stiffness of the muscles of the jaw and neck, with an increased difficulty in movement and interference with eating and talking. The muscles of the zygomatic arch usually become involved early, their stiffness and retraction producing the characteristic "sardonic grin."

The progress of the disease from the appearance of the stiffness of the jaw is rapid, and there will gradually develop rigidity of the head and limbs. The back will be arched in the position of opisthotonos or rarely laterally. The lower limbs are usually first involved, and are extended, and the upper limbs flexed. The jaw becomes so rigid that it is impossible for the patient to protrude the tongue or to eat. Spasms of a tonic character make their appearance early, and as the disease progresses involve the whole body, and may be brought on by the slightest cause, as jarring of the bed or noises. They are sometimes so violent that the patient will be shaken from the bed to the floor. Later the spasms may be continuous. The mind is not involved, and is clear to the end.

In those cases in which the symptoms appear within six or seven days the prognosis is invariably fatal, and it is a rule that the longer the onset, the better the prognosis; and in those cases in which the symptoms do not appear until after the twelfth day the prognosis is fairly favorable. There should be no difficulty in diagnosticating this disease.

Cephalic or Head Tetanus.—In this form the infection is usually in the head or face and the onset is rapid. Besides the stiffness and rigidity of the head and neck and retraction of the zygomatic muscles, there is nearly always a paralysis of both facial nerves, and more rarely of some of the ocular muscles. Difficulty in talking and swallowing appears early. Sometimes there is also retraction of the limbs and rigidity of the back. The prognosis is nearly always fatal.

TETANY.

Definition.—A peculiar form of rigidity and spasm of the terminal portions of the upper and lower limbs, characterized by irritability of the nerve-trunks, resulting from some form of intoxication.

The disease is very common abroad, especially in Berlin and Vienna, but in this country it is of rare occurrence, although a number of epidemics have been reported. As a rule, it is ascribed to some form of autointoxication resulting from disturbance of the gastro-intestinal system, but can be produced by removal of the parathyroid glands. It probably is the result of increased elimination of calcium salts, which can be detected by careful metabolic examination. It usually occurs in children, especially in those in whom there is general malnutrition, and is very rare in adults after the twentieth year.

Symptoms.—There may be present the general symptoms of gastro-intestinal disturbance, and the first distinctive symptoms are those of numbness or pain in the fingers, hands, or feet, gradually followed by an increasing stiffness and a curious and characteristic spasm of these parts, which consists in the fingers being extended and approximated in a cone-like manner, sometimes the thumb being in the palms; the wrists are generally flexed, the elbows drawn in toward the side of the chest, and the forearm pronated. In the lower limbs the legs are extended and the feet turned inward, simulating the position in equinovarus. This characteristic position is the result of a spasm which may last from a few minutes to an hour or longer, and may involve both the lower and the upper limbs, but generally only the upper. When the disease is very marked, the spasm may involve the muscles of the chest and diaphragm or larynx, causing interference with breathing, but this is rare. The sphincters are rarely affected.

It is characteristic of these spasms that they may be brought on by any

form of excitation, such as pressure over the brachial arteries or the nerve-trunks of the arm (Trousseau's symptom); by stimulation, especially with the galvanic current, which will produce increased excitability (Erb's sign), and tapping any nerve—as, for instance, the facial on the side of the face—will produce a spasm of the muscles in its distribution (Chvostek's sign). All or these symptoms are only indications of the general irritability of the nerves and muscles, and are given here not as specific symptoms, but because of the fact that they are so frequently described in conjunction with this disease.

The prognosis is nearly always good, the disease lasting from a few weeks to a month or longer, the spasms gradually subsiding. Treatment, of course, has an influence upon the length of the disease.

Spasms, Tics, and Motor Neuroses.

Until recently no differentiation was made between spasm and tic. Chiefly under the influence of the French schools, attempts have been made to distinguish between these, and by tic is meant a movement or movements which are more or less under the control of the will, and result from some emotional or functional basis duplicating or resembling voluntary movements. In whatever part the tic takes place the muscular action is complete, as, for instance, in facial tic the contraction is in the whole facial distribution, its occurrence not interfering with the use of the same musculature for other purposes, as eating and talking. On the contrary, by spasm is meant a movement which is not at all under the control of the will and which cannot be voluntarily duplicated. The contraction may involve part or all of a functionally acting group of muscles, and interferes with other functional uses; as, for instance, in a facial spasm the contraction may be limited to a part or involve all of the facial distribution, the movement does not resemble a voluntary action, and its occurrence interferes with eating and talking. As a matter of fact, while such a theoretic and clinical distinction can be made between facial spasm and tic, it cannot be made in so far as most of the other so-called spasms and tics are concerned. Again, it is of no practical importance to differentiate between them, for in neither has there been established a definite etiology, in both the causes are mainly functional, the course of the disease is identical and the prognosis the same.

Under the general heading of spasms and tics will be discussed all of the different forms of spasms, contractions, or movements which occur in any portion of the body, although it is customary to treat most of these separately, as if they were distinct entities.

Predisposing and Exciting Factors.—The causes of most spasms, tics, and motor neuroses are not known, and therefore a functional basis is ascribed. As a matter of fact, with the exception of those rare instances due to an organic basis, such as facial spasm resulting from a growth on the seventh nerve, or torticollis from direct irritation of the spinal accessory, there are nearly always two principal causes which enter into the etiology. Given a perfectly normal individual, one whose heredity is good and in whom all bodily functions are normal, there is no reason to expect the development of any form of spasm, tic, or in fact any so-called functional disease, even if he be placed under severe mental strain, shock, or any cause which produces general malnutrition. If, on the other hand, an individual has a neurotic heredity and has inherited a weakened nervous system, or one which has lessened resistance, under the same conditions there would

probably develop some form of neurosis, the particular type depending upon the previous history of the patient and the lessened resistance or vulnerability of certain functional activities. In such a person if there should happen to be a diminution of tone in the functional activities of the seventh nerve, there might develop facial tic or spasm; if in the distribution of the spinal accessory, torticollis; if in the functional movements concerning the upper limbs, writer's or any other form of spasm; if in the lower limbs, cramps, etc. It is also probable that in the development of a particular form of neurosis mental impressions play an important part, as, for instance, in a child in whom imitation of certain facial movements will be succeeded by so-called habit spasm, or, because of fright, religious or other emotional causes, there may develop a certain form of jumping or other movements.

Symptoms.—There are certain symptoms which are common to all spasms, tics, and motor neuroses. In all, excluding the cases in which there is an organic basis, there is a functional or mental element, manifested in many ways. It may be in the impressionability or so-called neurotic tendency, which will be discussed under the general neuroses. So far as the character of the movements is concerned, they resemble each other in the fact that they are influenced by the emotional condition of the patient, or by any form of excitation. Most of the movements are quick, intermittent, have a tendency to become chronic, and cease during sleep. Besides the specific symptoms of the spasm there may be increase of reflexes and the symptoms of a neurosis, such as pains and tenderness in the back, headache in the back of the head, occasionally dizziness, insomnia, loss of appetite, and sometimes disturbances of sensation, such as hemihypesthesia or monohypesthesia.

Spasms, Tics, and Motor Neuroses in the Distribution of the Fifth Nerve.—These are rare, and, as a rule, are the result of organic involvement of the fifth nerve, or may be a part of tetanus, epilepsy, or tetany. Occasionally, however, there may be, in hysteria, unilateral or bilateral spasms of the masseters, causing trismus or lockjaw. When both pterygoids are involved, the jaw is opened in the median line; or if only one, to the opposite side. In so-called motor neuroses in the distribution of the fifth nerve there may be either spasmodic or continuous movements of the jaw resembling chewing. It is generally in association with movement of the tongue and face, interfering with talking and swallowing, and often there is dribbling of saliva. There may be in association a weakness or laxness of the ligaments of the jaw.

Spasms, Tics, and Motor Neuroses in the Face.—These may involve the whole or part of the distribution of the facial nerve, and may be in association with movements of the eyelids on one or both sides, and often of the eyeballs. Sometimes there are also movements of the tongue and masseter muscles, as already described.

In so-called facial spasm the movements may start in part of the facial distribution, as, for instance, in the upper, and in the course of time involve all, or they may start in the whole at once. The movements resemble the contraction obtained by means of faradic electric excitation of the nerve, and cannot be controlled by the will or duplicated. They may be momentary, or last several minutes, and during the spasm talking will be impossible. After it is over there are generally fibrillary tremors in the facial distribution.

In so-called *facial tic* the movements can, in most instances, be partially controlled by the will. They resemble volitional movements and do not

interfere with talking. Very often there may be in old facial paralysis tic of the whole, but especially of the lower part, of the face.

Sometimes in association with facial tic, or independently, there are movements of one, but generally of both eyelids and orbicularis muscles. This is called *blepharospasm.* As a rule, they occur in children, come on gradually, and consist in a tonic or clonic spasm of one or both lids and eyebrows. In exaggerated cases there may be, in association, movement of the eyeballs, elevation of the nasolabial folds, or sniffing or sucking-like movements. This is sometimes called *habit spasm,* or *habit chorea.*

Spasms in the Muscles of the Tongue, Palate, Pharynx, and Larynx.—All of these conditions are rare, but occasionally there may occur in hysteria isolated spasm of the tongue. Rarely in association with lingual spasm there is involvement of the pharyngeal muscles, producing swallowing movements, and, as a rule, movements of the jaw. Spasm in the laryngeal distribution sometimes occurs, but is generally hysterical or part of chorea or tetany, or it may be in association with spasm of the diaphragm.

Spasm in the Respiratory Muscles.—This involves the diaphragm, and may be tonic or clonic. When tonic, the lower part of the chest and epigastrium become prominent, there is pain over the region of the diaphragm, and breathing is painful, rapid, and performed entirely by the upper respiratory muscles. If continued, it may cause death. When intermittent or clonic, it causes so-called *hiccough* or *singultus.* This is sometimes due to an irritation of the phrenic nerve, or may result from many different causes, such as gastro-intestinal or general constitutional disturbances, but in many cases it is purely hysterical. The course and prognosis depend upon the cause. In most instances it subsides in the course of a few hours, but may last days, especially when functional in character.

Spasm in the Distribution of the Muscles of the Neck; Torticollis; Wry-neck.—Inasmuch as the spinal accessory nerve supplies the sternomastoid and trapezius muscles, an irritation of it, such as results from pressure, will cause spasm in its distribution, or torticollis. In most instances, however, the cause is not known. It may come on suddenly as the result of a fright, but usually the onset is gradual, the spasm growing more and more severe, the particular kind depending upon the muscles affected. If the sternomastoid alone is involved, the head is turned to the opposite side, the chin pointing a little upward; if the trapezius, the head is retracted on the same side to the shoulder, the chin pointing upward; if both the sternomastoid and the trapezius, the head is turned to the opposite side, backward, and the chin higher than when either are alone diseased. Very often in association with the sternomastoid and trapezius muscles the rotators of the neck, muscles of the shoulder, the erector capitis and splenius muscles of one or both sides take part in the spasm, and the movements are very complicated. When the rotators alone are involved, the head is turned toward the same side, the chin being on a straight line; when the splenius, the head is retracted, the chin upward, differing from the action of the trapezius in the fact that in the latter the head is retracted toward the shoulder. When both sternomastoids are affected, the head will be drawn forward, and if the movements are clonic, there will result so-called nodding or salutatory spasms, which are especially common in children. The spasms may be either tonic, when it is difficult to return the head to its original position, or clonic, the movements being intermittent. Ordinary stiff-neck or rheumatic torticollis hardly enters into the discussion. The course of the disease, as a rule, is long and the prognosis not very good.

It is best in those cases in which treatment is instituted early, and in which absolute control of the patient can be obtained.

Spasm in the Distribution of the Upper Limbs.—These are rare, and may partake of many different forms, and, as a rule, are of functional origin. The spasms may be limited to the fingers, or may involve the whole upper limb, and may sometimes be in association with spasm of the leg and face of the same side. They must be distinguished from hemiathetosis or hemichoreic movements following hemiplegia. The movements of the fingers may be irregular, but, as a rule, are rotatory, and rarely quick and lightning-like.

Occupation Neurosis; Writer's Cramp.—This generally occurs in those persons who are occupied constantly in the performance of certain rhythmic movements, as writing, typewriting, playing of an instrument, like a piano or violin, or, in fact, in any movement in which there are in association many muscular contractions. It comes on, as a rule, slowly, and is first manifested by a tired or aching feeling, and occasionally tenderness over the nerve-trunks. This fatigue may last for some time, and then it is noticed that writing is not as free as before, and gradually there develops a tremor which interferes with writing, or a spasm of the muscles, which may be either tonic or intermittent. In writing the pen may suddenly be dug into the paper or fly forward, or the whole hand may be in a tonic spasm until writing becomes absolutely impossible. Occasionally there is weakness in the muscles, but this is unusual. The spasms come on only when the muscles are used for the particular movements concerned, and can be used for any other purpose. The prognosis, as a rule, is unfavorable.

Spasm in the Distribution of the Lower Limbs.—These are generally of an organic basis, but occasionally there may develop irregular movements of the lower limbs similar to those described in the upper. Rarely there may be an irregular, symmetrical, spasmodic contraction of the muscles of the thighs and toes, causing sudden jumping or lifting movements. These occur especially under the influence of religious emotions, are common in Oriental countries, and the people presenting this condition are known as "jumpers." Occasionally the spasm may be limited to the muscles of the calves, *cramps*, and may occur after exertion or independently.

Spasm in the Distribution of the Muscles of the Shoulder, Chest, and Abdomen.—These do not occur very often, but occasionally there are present spasms in the muscles of the shoulder in association with contraction of the pectoralis muscles, and rarely there may be unilateral or bilateral spasms of the muscles of the abdomen, causing an approximation of the shoulders and thighs, or bending movements.

Tic Convulsif, or Maladie des Tics.—So far we have discussed those spasms, tics, and motor neuroses which involve a part or all of a limb or body. Under the above headings, however, are understood those spasms which involve most of the movements of the body. It is rather difficult to describe this form, because the movements vary so greatly. As a rule, there are grimacing spasms in the face, associated with sniffing of the nose, sucking or blowing movements of the mouth, with sudden ejaculation, or repetition of certain words, often obscene, known as *coprolalia* and *echolalia* respectively. Associated with this there may be movements of the upper and lower limbs, such as sudden lifting up of the shoulder or arm or the taking of a few steps, then a retraction, or there may be, in association, hysterical contractures in the lower limbs. There are always found many hysterical stigmata.

Paramyoclonus Multiplex.—Under the term paramyoclonus multiplex is understood a form of bilateral, regular, more or less rhythmic clonic contractions of groups of muscles, generally of the trunk and proximal parts of the limbs, and rarely involving most of the body. The cause of the disease is unknown, but it is probably functional in nature. The movements resemble to some extent those of generalized spasm or tic, but differ in their regularity, being bilateral and clonic.

It generally occurs in adults and the onset is slow. A well-marked case consists in a clonic spasm of symmetrical groups of muscles, for instance, of the quadriceps or shoulder and upper arm groups, the muscles standing out prominently as if held, the spasm lasting several minutes or longer. The spasm consists in a series of clonic contractions coming on one after another, sometimes the number of contractions being from fifty to one hundred and fifty to the minute. Besides the spasms there are fasicular contractions. Sometimes they are of such severity and extent that they involve the muscles of the chest, abdomen, and limbs, and cause irregular movements of the body and extremities. This is exceptional. There are, as a rule, no associated symptoms of motor palsy or sensory disturbance, but the reflexes are generally increased. The course of the disease is chronic, of long duration, but occasionally cure is obtained.

Sometimes in association with this there may be epileptic convulsions. This has been called the *Unverricht's type of myoclonus, or myoclonus epilepsy.*

If the myoclonus is limited to one-half of the body, the movements resembling those produced by the stimulation of an electric current, they are sometimes called *electric chorea, Henoch's chorea, or Dubini's disease.* These, however, are bad terms.

Myokimia.—By this is understood a well-defined, irregular, fasicular muscular contraction, involving most of the muscles of the body. The contractions are wave-like and more or less constant. They differ from paramyoclonus multiplex in the fact that they are not symmetrical or clonic, and are constant.

Course and Prognosis.—Most spasms, tics, and motor neuroses are difficult to cure, and the prognosis should always be guarded. So much depends upon the kind of spasm, the elimination of the cause, and the control of the individual.

CHOREA

(ST. VITUS' DANCE; CHOREA MINOR; SYDENHAM'S CHOREA).

Definition.—A disease occurring principally in childhood, characterized by irregular, unpurposeful movements of any portion of the body, and in which the prognosis is almost invariably good.

The disease is supposed to be of infectious origin, although the specific bacillus has not been isolated. Pathologically, so-called choreic amyloid corpuscles have been found in the nervous system, but are not constant. In about 20 per cent. of the cases it is in association with rheumatism. It generally comes on in children, and especially in the spring of the year, is less frequent in the winter months, and is more common in girls. There is nearly always some malnutrition and a general anemia, manifested by blood-changes, and sometimes a hemic murmur, which is best heard at systole, although there may be an organic murmur, caused by endocarditis. It is probable that the disease is the result of a general lowering of tone, associated with a certain inherited or acquired neurotic disposition, for it generally occurs in school-children, who are more or less overworked

in the spring of the year, and only in those in whom there is a neurotic tendency.

Symptoms.—These are first manifested by a growing restlessness, and a peevish, irritable condition of the child, associated with more or less gastro-intestinal disturbance, loss of appetite, and insomnia. Gradually there will be noticed irregular, unpurposeful movements, generally of the hands or face. The child begins to make grimaces, pucker the lips, elevate the brow, move the head or arms, extend the legs, twitch the shoulder, and is generally restless. The movements are increased by attention and excitement, cannot be controlled by the will, and generally cease during sleep, although in grave cases they may be present even then. In mild cases the irregular movements may be limited only to the face and arms, but when more severe there may be interference with the respiratory movements, and even with talking and sometimes eating because of the involvement of the muscular apparatus concerned. The course of an ordinary case is from two to three months, the movements gradually ceasing, but in the more severe cases it may last for a long time. One attack nearly always predisposes toward another, and it is not at all uncommon for a child to have successive attacks for three or four years.

As a rule, there is no motor weakness, but sometimes the movements may not only be limited to one-half of the body, but there may be distinct weakness of the arm and leg. This is known as the hemiparalytic form of chorea. Sometimes the limbs may be weak on both sides. The reflexes are not altered, but occasionally on tapping, for instance, the patellar tendon, there may be an irregular jerking propulsion of the leg instead of the usual response. Sensation is hardly ever altered, and there is never involvement of the bladder and rectum, although it is not at all uncommon for children to have an enuresis during the course of the disease.

Sometimes chorea occurs in adults, but the form does not differ from that in the child. In rare cases the choreic movements are present from childhood, but in these instances it is probable that there is a cortical condition rather than one of pure chorea.

Sometimes choreiform movements may complicate pregnancy, especially in primipara, *chorea gravidarum*. It comes on in the first half of pregnancy, as a rule, and in nearly all cases there has been a previous history of such disease. The prognosis in these cases is not so good, and sometimes it is necessary to produce abortion.

Summary of Diagnosis.—A school-child, especially in spring, becomes peevish, irritable, cannot sleep, is constipated, and begins to have irregular movements of the arms or face, which consist in a purposeless grimacing and twitching of the shoulder or any portion of the limbs, this being increased by excitement and not controlled by the will. It usually stops during sleep. Associated with this there is nearly always malnutrition, with some anemia. There should be no difficulty in diagnosticating choreiform movements from any other.

Clinical Course and Complications.—The course of the disease is usually regular, ordinary cases not lasting longer than two or three months. Treatment, of course, has a direct relation to the length of the disease. In the more complicated cases, and especially in *chorea gravidarum* or chorea of pregnancy, the prognosis is not so good, and occasionally death results. Occasionally in this type there may arise grave mental symptoms, which occasionally are permanent.

HUNTINGTON'S CHOREA.

A hereditary family disease, characterized by irregular choreic movements, generally beginning between the thirty-fourth and fiftieth year, terminating nearly always in mental impairment. The disease was first described by Dr. Huntington, of Long Island. It usually runs in families, and it is characteristic that it seldom skips a generation, and if it does, the disease ends in that particular family. There may be no symptoms in the early life of the individual, but there may be general indications of an inherited neurotic disposition. About the age of thirty-five the choreic movements begin, and resemble very much those of chorea minor, generally involving the face and upper limbs, and interfering considerably with walking. These persist and become worse. Soon after the appearance of the movements there develops a gradual mental deterioration, which in a few years terminates in total mental impairment. The prognosis is invariably bad.

PARALYSIS AGITANS (PARKINSON'S DISEASE).

Definition.—A disease of the latter end of life, characterized by rigidity in the muscles of the whole body, with slowness of all voluntary movements and a characteristic rotatory tremor which nearly always improves by effort.

It occurs mostly in men, usually about the age of forty, although rarely it may occur in young persons. Its pathology is not definitely known. Microscopic examinations of the brain and cord show the usual changes of senility, the muscles show atrophy, and occasionally disease of the parathyroids has been found. The disease is sometimes hereditary and may occur in families. Rare instances are reported in which sudden fright has been followed by paralysis agitans.

Symptoms.—The disease, as a rule, comes on slowly, and is usually first manifested by an increasing rigidity and tremor in one limb, which finally involves the whole body. It usually starts in the right upper limb, to be followed in the course of time by the right lower, and then the left limbs. Occasionally it may be limited to the limbs on one side for a long time. In a well-marked case the attitude is typical, and the diagnosis can be made from it alone. The patient stands with his knees bent, feet close together, the body bent forward, head flexed on the neck, the back is held rigidly, the arms held by the side, the elbows touching the chest, the hands in pronation and the fingers in a typical pill-rolling attitude. The face gradually loses its expression, the wrinkles are smoothed out, and laughing and crying are rarely observed. In fact, all the emotions are subdued. Talking is slow and the voice becomes low and monotonous. Occasionally there is dribbling of saliva. All movements become slow, as is shown by the slowness in rising or in movement of any of the limbs. It is, of course, to be understood that the rigidity develops gradually, and it may take years before the typical attitude described is obtained. Sometimes the rigidity becomes so extreme that there is great resemblance to rheumatoid arthritis.

Coincident with the rigidity and slowness in movement there develops the typical tremor of the disease. In a well-marked case the attitude and movements of the fingers are those of pill-rolling, the tremor involving every portion of the body. It is characteristic of this tremor that it becomes

momentarily better on effort, differing from the intention type of multiple sclerosis. It also becomes less when the patient is quiet or when his attention is attracted, although occasionally it may be intention in type. It usually stops during sleep. It is to be remembered that paralysis agitans may occur without any tremor, and that the diagnosis may be made upon the characteristic attitude and rigidity.

With the rigidity, attitude, and tremor, there develops late in the disease a certain peculiar form of gait. Because of the attitude the patient's center of gravity is brought forward, and in attempting to walk, to regain his center of gravity, which is always in front, the patient will have a tendency to gradually bend over, causing his steps to become shorter and more rapid, until he either falls on his face or with an effort straightens himself up, the falling gait being again repeated. This is called festination, and when not present can sometimes be brought out by giving the patient a sudden push forward. Very often instead of this festinating gait there may be a tendency to walk backward or to one side (Plates XXV and XXVI).

The course of the disease is chronic and the rigidity and tremor increase until finally the patient becomes bedridden. The reflexes are at first increased, but later may become lost or diminished on account of the rigidity. There may develop muscular wasting, and the electric excitability of the muscles may become diminished.

One of the most distressing features of the disease is the excessive flashes of heat, warmth, or chilliness, these coming on either periodically or constantly, the patient sometimes complaining more of this than anything else. The mentality becomes gradually dulled with the progress of the disease.

Summary of Diagnosis.—An adult, generally after the fortieth year, develops an increasing muscular rigidity of the whole body, with slowness in movement, lack of expression in the face, tremor of a rotatory type, and festinating gait. This picture is characteristic, and there should be no difficulty in making a diagnosis. Occasionally, however, the disease may be unilateral for a long time, or there may not be present tremors, when the disease should be diagnosticated from the rigidity and attitude.

SENILE TREMOR.

Occasionally there develops a tremor in the head or limbs, which in nearly all cases is fine and is to-and-fro in character. It generally comes on in old persons, is sometimes hereditary, and is distinguished from paralysis agitans by absence of rigidity and characteristic attitude.

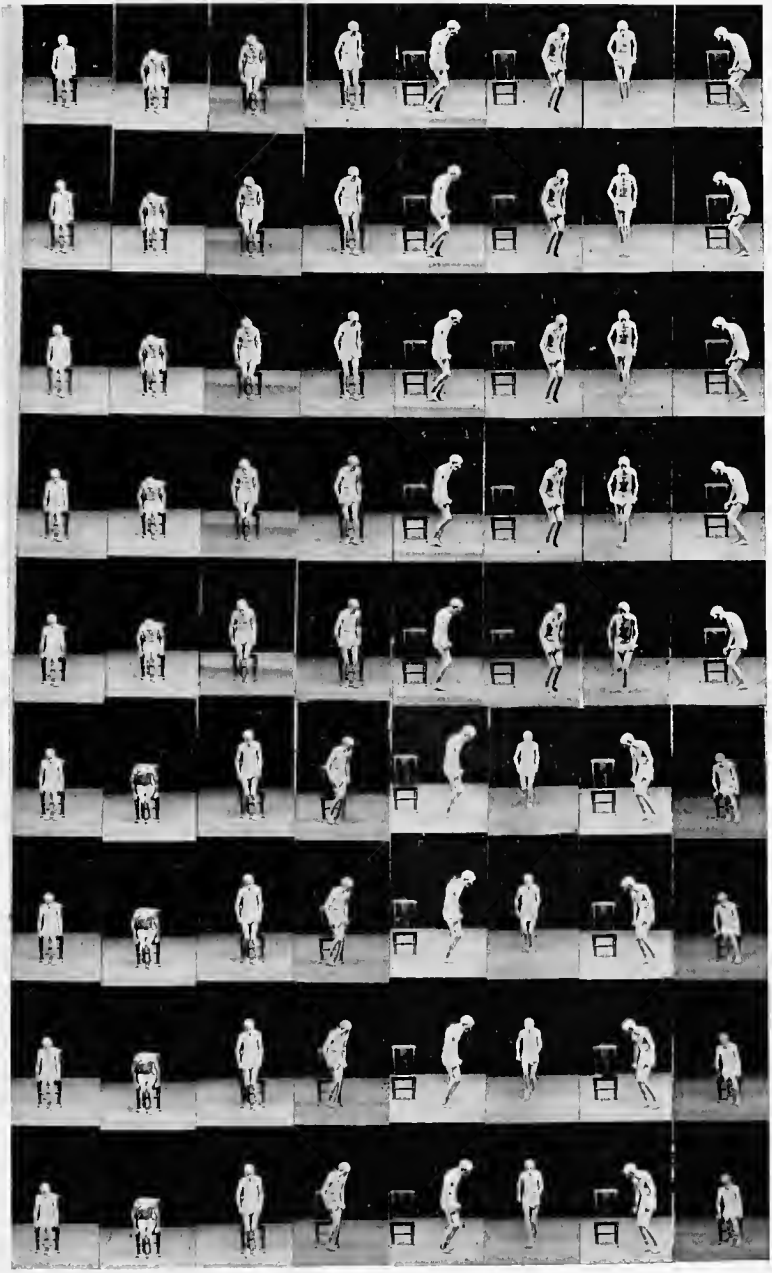
MYOTONIA CONGENITA (THOMSEN'S DISEASE).

A family disease, generally occurring in the male, coming on at infancy and characterized by a tetanic like contraction of the voluntary muscles when they are first put to use.

The disease is rare and its cause unknown. Microscopic examinations of the nervous system have been negative, although there have been found changes in the muscles, consisting in an increase in the size of the fibers and nuclei.

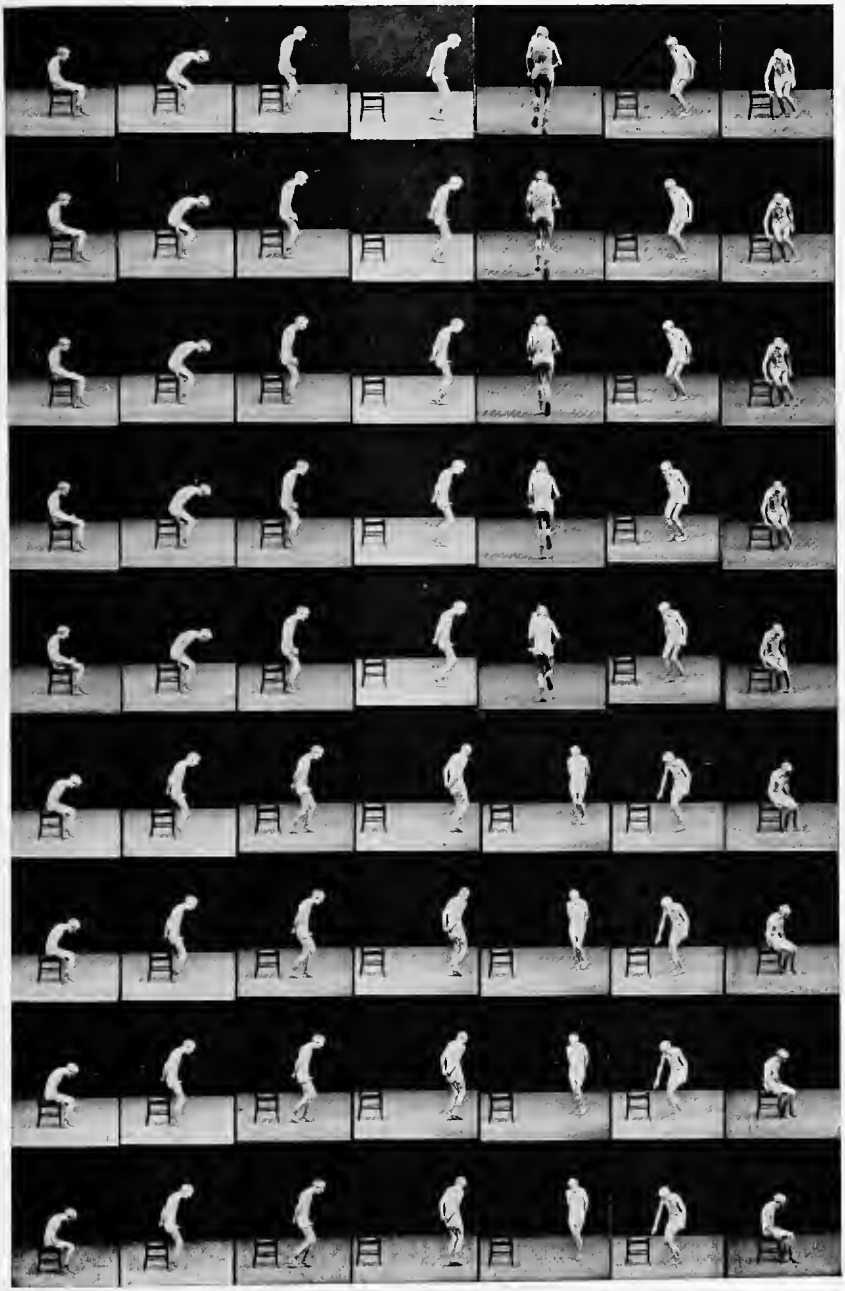
Symptoms.—The symptoms are present from birth, and because of the contraction of the voluntary muscles there will be an overdevelopment, which, at first, especially in young adults, may resemble a muscular hyper-

PLATE XXV



Moving Picture of Attitude and Gait in Paralysis Agitans. (Courtesy of Mr. Sigmund Lubin of Philadelphia, Pa.)

PLATE XXVI



Moving Picture of Attitude and Gait in Paralysis Agitans. (Courtesy of Mr. Sigmund Lubin of Philadelphia, Pa.)

trophy. The tetanic like spasms or contractions are apparent when the patient arises or attempts to walk after resting, the movements being stiff, slow, and only made with difficulty. Gradually, however, the spasm diminishes, until finally the patient walks with comparative ease, only to have the spasm reappear on the next effort, after another rest. There are no other motor and no sensory symptoms, the reflexes may be exaggerated, but there is nearly always an increased and heightened contraction to a constant galvanic or a rapidly interrupted faradic current—the so-called myotonic reaction. The lower limbs are preponderantly involved, but in a well-marked case the upper also take part in the spasm. The disease is of long duration, the symptoms continuing until the death of the patient.

AMYOTONIA CONGENITA (OPPENHEIM'S DISEASE).

"A condition of extreme flaccidity of the muscles, associated with an entire loss of deep reflexes, most marked at the time of birth, and always showing a tendency to slow and progressing amelioration. There is great weakness, but no absolute paralysis of any muscle. The limbs are most affected, the face is almost always exempt. The muscles are small and soft, but there is no local muscular wasting. Contractures are prone to occur in the course of time. The faradic excitability in the muscles is lowered, and strong faradic stimuli are borne without complaint. No other symptoms indicative of lesions of the nervous system occur." (Oppenheim.)

There have been so few autopsies that the etiology has not been made very clear. It is probably the result of congenital changes in the muscular system, the condition being prenatal. The symptoms are noticed either directly or soon after birth. The affection is strictly symmetrical, and may involve all the muscles, but, as a rule, the muscles of mastication and deglutition escape. The lower extremities are most often involved, next the upper, and then the trunk and face. The muscles are completely toneless, small and soft to touch, and a striking peculiarity is the impossibility of distinguishing by touch between the skin and underlying structures. There is no wasting. The lack of tone is freely demonstrated by the fact that it is possible to place the limbs in any position. The loss of power is hardly ever complete, although this varies in different cases. Contractures sometimes develop in the latter end of the disease. It is necessary to have an increased faradic current to obtain a reaction, and while this condition is present in most of the muscles, it is best demonstrated in those which are most affected. The deep reflexes are lost, but the superficial are normal. The sphincters are never involved.

The course of the disease is slow and there is a tendency to spontaneous improvement. This is shown by the increasing tonicity of the muscles and the fact that the reflexes return.

NEURASTHENIA.

Definition.—A term given to a combination of nervous and physical symptoms in which the general characteristics are irritability, abnormal sensitiveness, mental depression, and physical weakness.

Predisposing and Exciting Factors.—A neuropathic heredity, whether it be some form of nervous or mental disease in the parents, or any disease of the individual which would have a tendency to cause a

diminished resistance in the nervous system, are important factors in the production of neurasthenia; for it is well known that in a number of persons under the same mental and physical strain, only a certain few will develop so-called neurasthenia. While it is probable that a congenital weakness or lessened resistance are the prime causal factors, it is possible for the disease to develop in one in whom these conditions are not present. It is probable that the various symptoms which are described under the term neurasthenia result primarily from a lack of proper mental appreciation and ideation, for in the development of a neurasthenic, while it is probable that there may have to be a fertile soil, the mental symptoms always predominate and the physical are the result of these. It is a well-known fact that neurasthenia is much more common in well educated persons, especially among professional men and society women, and although this symptom-complex is present in persons not well educated, the symptoms are necessarily few and not so well defined.

It has been estimated that neurasthenia is much more common in men than in women, although this is doubtful. It may develop at any age, but is a disease essentially of adults. The exciting causes are many, but worry, especially financial, is the predominating cause. Prolonged mental work with lessened bodily activities; injuries, especially those produced in railroad accidents; and then the countless causes which may be referred to almost any part of the body, such as eye-strain, disturbance of the nose, throat, ear, the various internal organs, and sexual functions are frequent factors. In fact, neurasthenia has been described as resulting from any and every cause. Even in perfectly normal individuals who develop, for instance, such a disease as pneumonia and typhoid, or possibly a slight surgical contusion, there may develop for the time being so-called neurasthenia.

Symptoms.—These are rather difficult to describe, because they vary so greatly in different persons, for while there may be a general resemblance, no one case has the exact symptoms of another, and the preponderance of certain symptoms will depend upon the education, station in life, occupation, previous health, resistance, and the immediate exciting cause. It is possible for a neurasthenic to have very few symptoms which he himself recognizes. Then, again, there may be many. Perhaps the most important are the mental, for the other symptoms are dependent upon them.

Mental Symptoms.—Their development will depend largely upon the immediate cause of the disease. If resulting from injury, they will be manifested promptly. If resulting from the usual cause of worry and overwork, they will be gradual in their development, and will be generally first manifested by an increased worry over whatever is occupying the patient's mind at the time, and inability to clearly comprehend and appreciate external conditions. Such a person will become depressed, will be unable to see any method of getting out of the trouble he is in, will assume a pessimistic attitude, everything will go wrong, and nothing of a cheerful nature will appear in his horizon. He will become easily annoyed, ordinary things which would not have bothered him before will irritate him, the slightest noise or whatever may occur will distract his attention, he will be unable to concentrate, and because of this will usually complain of loss of memory. Generally such a person, if he is addicted to smoking or drinking, will increase his habits in this direction, and if in women they will drink more tea and coffee to brace themselves up. Very soon they will be unable to sleep, and the harder they try, the less they succeed, and will

rise in the morning more tired, irritable, fretful, and discontented with themselves.

If this continues such a patient will constantly think of his own troubles, until finally these will be the only source of his thoughts, to the exclusion of everything else. If he should happen to have some malady, this will be exaggerated. If a physician, he will probably develop what occupies him most, and will have a special dread for locomotor ataxia and general paresis; if a medical student, generally heart disease; if a nurse, tuberculosis; if in a layman, they are not so marked and are generally of a diffuse character; but the specific mental symptoms are nearly always dependent upon some form of mental suggestion, based upon a preconceived knowledge or the suggestion of others. If, for instance, a patient should have a history of carcinoma in the family, he will have a dread of that; or if insanity, may fear that he will become insane. Again, patients may develop a curious form of predominating ideas or obsessions; for instance, some will not walk under a ladder, or when going to a theater or church will have a dread of fainting or having a spasm; others will have a fear that the chandelier will fall down or the house will catch afire. Again, other persons may have a dread of walking on certain sides of the street or riding backward, or perchance when going to sleep, if they do not think they say their prayers with the proper amount of devotion, may repeat a number of times until satisfied.

If the condition continues, there will develop in the patient the so-called neurasthenic habit, which, after it becomes well established, is difficult to lose. In a well-marked case the patient may cease the occupation previously engaged in and do nothing besides sit all day, and if given an opportunity will constantly talk of herself; and very often when consulting a physician, for fear that she will not remember all of her symptoms, will have long written descriptions of them. Under proper treatment the neurasthenic mental condition may become largely alleviated, but nearly always there will remain a neurasthenic tendency. Of course, it is to be understood that the above symptoms are those of the gravest sort of case, and that in the mild form there may only be few manifestations.

Physical Symptoms.—These will develop in conjunction with the mental, and are manifested in many forms. They may be divided into general, motor, sensory, and special.

General.—There is nearly always a diminution of the different bodily functions. The patient will nearly always become constipated. The urine may become scanty and concentrated, principally because the neurasthenic very rarely drinks water. Indigestion is commonly complained of and there is a well-known nervous type. Appetite is generally poor, although sometimes the patient may eat voraciously and unusual things, this being especially so in women. There is usually a bad taste in the mouth and there may be flatulence. Often there may be serous and copious evacuations immediately after eating or before the performance of certain mental work, like giving a lecture. Coughing sometimes develops without a respiratory cause and may become distressing. Pulsation of the vessels, especially palpitation, is very common, and often patients will be prevented from sleeping on the left side because of the beating of the heart or of the pulsation of the vessels of the head or limbs. Insomnia develops in most cases very early, and the patient usually complains of not being able to sleep at all and of feeling much more tired in the morning than on retiring. Often such patients may drop asleep after meals or without any apparent cause. Urine examination may sometimes demonstrate an increased

amount of urates and indican; and blood examination a diminished amount of hemoglobin and of red and white cells.

Motor.—These develop gradually, and in a well-marked case any muscular effort will be followed by great exhaustion, so that some patients are confined to bed. The predominating motor symptom is fatigue. The grip of the hands, as a rule, will be poor, as well as the resistance against movement. Fasicular and sometimes fibrillary tremors in the limbs and muscles of the body, but especially the face, are very common, and when shutting the eyes there is nearly always fluttering of the lids. The reflexes, as a rule, are increased, and rarely, when ankle clonus is attempted, there may be one or two abortive movements.

Sensory Symptoms.—These are very common, and, as a rule, are manifested early. The patient usually complains of headache, this being nearly always "at the base of the brain," the occipital region, top or front of the head, and is nearly always described as a pressure sensation or as if a weight were pressing down. Sometimes it is described as a tight band or a "rush of blood to the head." Dizziness is often complained of, and is usually described as a swimming sensation, and is especially present when suddenly rising from a sitting posture. Pain along the spine is very common, and it is only rarely that it is not possible to demonstrate a point of tenderness somewhere along the back, generally in the mid or lower spine, this being described nearly always as a pressing or drawing sensation. Sometimes a drawing or pressure sensation is described in the front of the neck or in the throat. Pain on pressure can also sometimes be demonstrated over the ovarian and inframammary regions in women and inguinal areas in men. Instead of pains it is very common for the patient to complain of burning, itching, numb, or pin-and-needle-like sensations, in various portions of the body, as, for instance, in the limbs, face, and most commonly in the genital region.

Special Symptoms.—These depend entirely upon the particular organ involved. Ocular disturbances are perhaps present more frequently than any other, and may be diversely manifested. A patient when reading may have the letters swim together, or have the lines blur, and because of this may be forced to limit his reading. Often dark spots or curious linear and various shaped specks or lines may appear in the visual fields, but it is a fact that they appear only when the patient thinks of them or when something worries him. There may also be a contraction of the visual fields, but this, as a rule, is for white and not for colors; and often there may be a curious rapid alteration in the size of the pupils—so-called hippus. Sometimes there may be photophobia or dread of light.

In association with the ocular phenomena, or independently of them, there may be disturbance of taste, smell, or hearing. The patients may complain of peculiar taste or odors or of a hissing, buzzing noise in one or both ears. This, however, is not very common.

Sexual Neurasthenia.—This generally occurs in young boys or adults, but is sometimes present in girls. It generally occurs in those who are or have been addicted to self-abuse, but may come on independently. As a rule, the symptoms develop in early adult life. If, for instance, a neurasthenic condition should develop through extraneous causes, such as mental or other worry, and if there should have been in the previous history of such an individual any sexual factor, this will usually be ascribed as the cause. This is unfortunately furthered by the many advertisements of the so-called specialists who gain a livelihood by the furtherance of such doctrines.

The symptoms are various. They may be manifested by impotence, lessened desire, premature ejaculation, frequent nocturnal emissions, or constant seminal discharge. Very often in conjunction with these symptoms or without them there may be itching sensations in the genital organs.

Summary of Diagnosis.—A gradually developing functional nervous exhaustion whose chief characteristic is an abnormal tendency to mental and physical fatigue, to worry, an exaggeration of symptoms to the detriment of the person concerned, with headache, numbness, pain, insomnia, loss of appetite, indigestion, intestinal difficulty, circulatory disturbance, ocular weakness as shown by the early fatigue in reading, occasionally specks before the eyes, and sexual symptoms.

Differential Diagnosis.—There should be no difficulty in diagnosing this disease by the many symptoms given. It must be remembered, however, that in general paresis the early manifestations may be those of neurasthenia, but in the former there are always pupillary irregularities with diminution in their reactions, disturbance of reflexes, and a general happy, expansive, optimistic mental attitude, which is so different from the selfish depressed mentality of the neurasthenic.

Clinical Course and Complications.—The course of a neurasthenic condition varies according to the severity of the attack, the predisposition of the individual, and the promptness with which treatment is instituted. Mild cases nearly always get well, provided conditions are favorable, but after a neurasthenic habit is once established for a number of years, it is probable that it will become chronic, and although the patient may get well, the slightest untoward influence may bring on a renewal of some of the symptoms. Sometimes there may develop a hypochondriacal condition, but this is not the rule.

HYSTERIA.

Definition.—It is almost impossible to give a brief and accurate definition of hysteria because so many symptoms are included under it. It is, however, an altered mental condition resulting from inhibition of mental processes in which the normal relation and appreciation of thoughts, ideas, ordinary occurrences of daily life and of the bodily functions are distorted and falsely appreciated.

It is a distinct disease and a grave one, and should not be spoken of and considered lightly—a habit which is only too prevalent, not only among the laity, but among medical men. The reason for this is because its principal symptom is suggestibility, and that many of the symptoms can be alleviated by persuasion.

Predisposing and Exciting Factors.—As in all neuroses, especially is it true in hysteria, that a neuropathic tendency dependent upon such heredity is an important predisposing factor. In the majority of cases there is a history of "nervousness" in early life, and of such diseases as chorea, tic, or of a more or less unstable physical and mental childhood. Again, in others there is no appreciable cause. Sudden fright is perhaps the most frequent exciting cause, and this is especially true of railroad and other accidents, in which it is not so much the physical injury as the mental impression that is the important exciting factor. It is also noticeable that the character of the symptoms is largely dependent upon the exciting cause; for in a person, for instance, who has been injured in the back, the symptoms will be preponderantly present in that area; while in a fright,

in which one sees another hurt in the knee, there may develop hysterical contracture in that part. Emotional disturbances of any form, sexual errors, and religious excitement are frequent causes. Imitation plays an important rôle, for often whole communities may become hysterical. It must also be remembered that functional symptoms often accompany or complicate organic diseases, as in early multiple sclerosis, and as complications of various infectious diseases they are not infrequent. Not every patient, however, will develop hysteria due to fright or other causes, and it may be necessary to have a tendency to the disease which may consist in an altered physical and mental condition or an unstable nervous system.

It is much more common in women than in men, although serious cases are seen in the male. It usually occurs in young adults, generally about the twentieth year, but is not at all infrequent in children. It is peculiarly prevalent among the Slavs and the Jewish race, who have a tendency to so-called functional disorders.

Symptoms.—It is difficult to describe the symptoms of hysteria, because they vary greatly, for one case hardly ever has the identical symptoms of another. Again, we hardly ever see in this country the grave forms which are so common in Europe, and especially in France. All hysterical symptoms, however, have a certain general resemblance: first, they are dependent upon a "functional" basis, because they appear and disappear and leave no trace; second, their suggestibility, in the sense that most of the symptoms can be suggested; third, they may be made to disappear by persuasion; fourth, there are certain symptoms known as "stigmata" which are present in nearly all cases, these being principally of a sensory nature; and, lastly, in all cases, whether the symptoms are mild or severe, there may occur convulsions or spasms, which, while they differ greatly in their form, have a certain general resemblance to each other. For the purpose of facility in description, and not because the symptoms appear in this manner, division will be made into mental, physical, sensory, motor, and special.

Mental.—Hysterical patients generally describe themselves as "nervous," and are highly impressionable, emotional, irritable, sometimes irrational, and are given to extremes of passion. In a well-marked case the patient's own condition is the principal burden of thought, and all occurrences will be made to apply to themselves. They generally complain of loss of memory and inability to concentrate, although it is possible sometimes for them to do highly creditable mental work. The more severe mental symptoms, such as those which occur in hysterical spasms, will be described under that head.

Physical and Visceral Symptoms.—There is, as a rule, lessening of the bodily activities, but sometimes the patient may apparently be in perfect physical health and still have the gravest form of hysteria. There may be diminution in the amount of hemoglobin, and the quantity of urine may be increased or diminished and there may be frequent urination. Loss of appetite and indigestion are very common, and constipation is a constant fault. Not infrequently there may be involuntary evacuations from the bowel, and sometimes there may be excreted casts of the intestinal wall. Borborygmi, or rumbling of the bowels, and sometimes so-called phantom tumors of the abdomen, resulting from localized gaseous swelling of the intestines, may be present. Flatulence and gaseous eructations are complained of, and there may sometimes be excessive vomiting, with or without nausea. The heart action, as a rule, is not disturbed, but palpitation is not uncommon, and is usually associated with pain over the precordial

area, and often there may be pseudo-angina pectoris. The pulse-rate may be altered and rapid, but, as a rule, its rate is not disturbed. Hysterical cough is quite common, and hiccough is frequent and may last for days. The respiratory rate is, as a rule, not disturbed, but there may be all sorts of alterations, consisting in repeated sighing, sobbing, sneezing, laughing, or crying. Aphonia or loss of voice is frequent, and may come on suddenly, the patient not being able to talk at all or only in a whisper. The temperature, as a rule, is not altered, but elevation, even as far as 105° F., has been reported, but its occurrence is doubtful. Vasomotor and trophic disturbances may occur, and consist in flushing of the skin, excessive or perverted sweating, and, rarely, skin eruptions of various sorts.

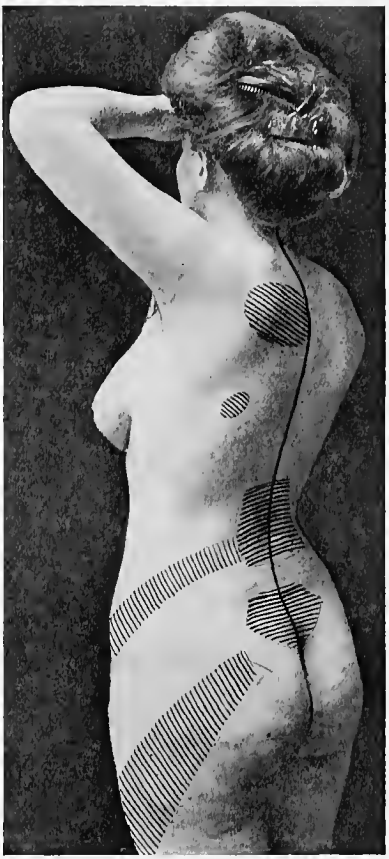


Fig. 446.—Areas of Pain, Tenderness, and Anesthesia in Hysteria.

Sensory Symptoms.—Headache is common, especially in the back or top of the head, and is usually described as a boring, aching pain, and sometimes as if a nail were driven into the skull, or as a tight band or a drawing sensation. There is nearly always pain and some point of tenderness in the spine, especially in the middle and lower portions (Fig. 446). Pain on pressure is almost constant over both ovarian and inframammary regions in women and the inguinal areas in men. Because of the frequency of these hypersensitive areas over the back, ovarian, inguinal, and inframammary areas, they are commonly known as the sensory *stigmata of hysteria* (Fig. 447). Often pressure over one of these so-called hysterogenic areas will produce or stop a hysterical spasm. Pains of a diffuse character may be present anywhere,—in the eye, ear, nose, etc.,—and are especially common in the throat, where they are described as drawing or band-like, and sometimes as a ball—so-called "globus hystericus." In fact, there is hardly a place in the body where pains may not be present. Numbness, tingling, pin-and-needle, or dead-like sensations are often complained of in the limbs, body, and frequently in the rectal and genital organs.

Diminution or loss of sensation is present in nearly all cases of hysteria. It is characteristic of these and all other sensory symptoms that they may vary from day to day or in successive examinations, either because of suggestion or other cause, and that the patient may be not at all aware of their presence until an examination is made. Because of this it is thought by some neurologists that sensory symptoms are nearly always the result of suggestion by the examiner. Hemianesthesia is quite common and its

form is characteristic. It is limited entirely to one-half the body, and the moment parts past the median line are approached recognition is prompt. It nearly always involves all forms of sensation, that is, touch, pain, temperature, and often vibratory and electric stimulation, and is sometimes associated with loss of half-vision on the same side. It can be differentiated from organic hemianesthesia by the fact that the latter is never limited by the median line, but nearly always projects over; that it is rarely complete for touch, pain, and temperature, and the loss of sensation is always more marked in the peripheral than in the central parts of the limb (Fig. 449). Again, it is nearly always in association with some motor symptom, and, most important of all, it is permanent and cannot be modified by suggestion. Hysterical anesthesia nearly always involves all forms, sometimes only pain and temperature sensations, and hardly ever touch alone. Over the anesthetic areas there may be vasomotor disturbance, demonstrated by the fact that pricking by a pin will not cause immediate flow of blood. In conjunction with loss of sensation there may be loss or disturbance of muscle sense or of some of the special senses, as sight, hearing, taste, and smell.

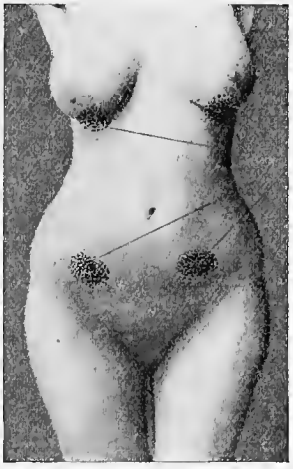


Fig. 447.—Hysterogenic Zone in Hysteria.

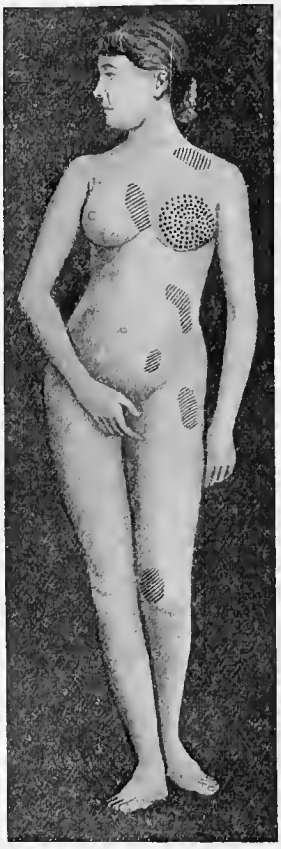


Fig. 448.—Location of Pain and Tenderness in Hysteria.

Motor Symptoms.—These vary greatly, and may be of either an irritative or a paralytic nature. *Tremors* are common, and may consist only of a fluttering of the eyelids, of twitching or fasicular movements of the muscles of the face, or of violent movements of a limb. They may be present for years, and usually cease during sleep. It is characteristic that their rate and character may alter as the result of suggestion, and when the patient's attention is attracted elsewhere, may cease for a time.

The tendon and superficial *reflexes*, as a rule, are exaggerated. It has been a mooted question as to whether ankle clonus can occur in hysteria, but its presence in rare cases is undoubted. It has a distinct character which renders its recognition easy. As a rule, when obtaining ankle clonus in an organic disease it is best to bend the leg on the thigh, and sometimes a true ankle clonus can be obtained only when this is done. On the other hand, hysterical ankle clonus can be obtained when the leg is fully extended on the thigh and the foot is suddenly bent forward. In organic cases the movements are rhythmic, the rate never varying; its intensity gradually becomes exhausted, while in hysteria the movements may be irregularly rapid or slow, and can be kept up sometimes indefinitely, and de-

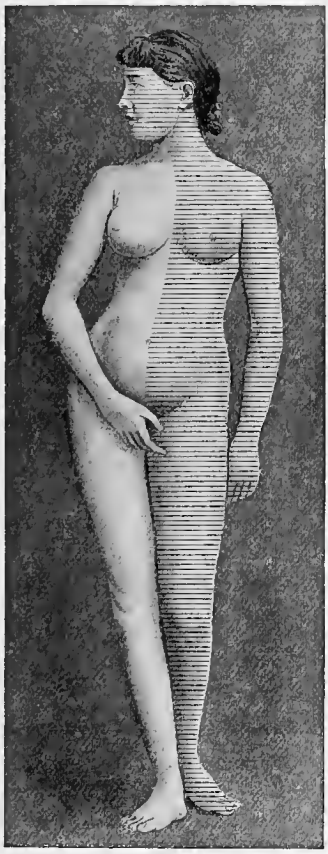


Fig. 449.—Hysterical Hemianesthesia, showing the Absolute Limitation to the Median Line.

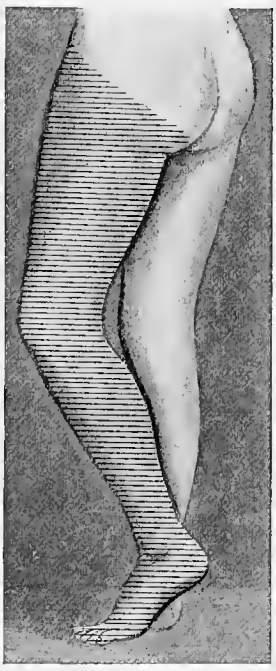


Fig. 450.—Hysterical Anesthesia of Lower Limbs.

pend entirely upon the mental condition of the individual. (Personally I have observed this in a number of cases, and have at one time obtained ankle clonus in a hysterical patient for over ten minutes. I have also obtained patellar clonus in hysteria, its character being similar to the hysterical ankle clonus.)

There are certain reflexes which are very often absent in hysteria, which are of diagnostic value. Among these is the pharyngeal reflex, in which it is possible to irritate the pharynx without producing gagging. Again,

when irritating the nasal mucous membrane there is, as a rule, a flow of tears, while in hysteria this may be absent. Sometimes, also, irritation of the cornea does not produce winking. This is nearly always associated with anesthesia of the cornea. It is also sometimes possible to pass a stomach-

Fig. 451.—Tonic Phase, the Tongue Rolling from One Angle of the Mouth to the Other (Richer).

tube in hysterical patients without the slightest resistance, due to the anesthesia of the parts concerned.

Hysterical paralysis is quite common, and, like anesthesia, varies greatly. It usually comes on suddenly, and may involve one-half of the body, so-

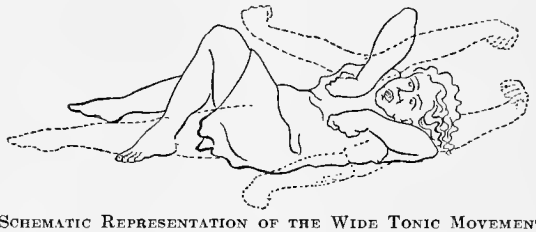


Fig. 452.—Schematic Representation of the Wide Tonic Movements (Richer).

called hysterical hemiplegia. It can be distinguished from an organic lesion by the fact that the lower part of the face is hardly ever involved, and that there is either complete flaccidity or exaggerated tonicity in the

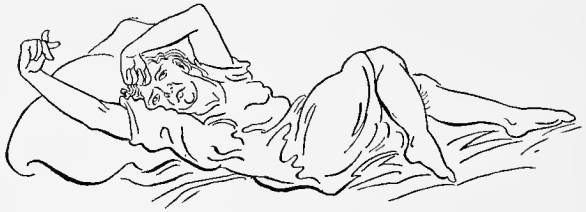


Fig. 453.—Tonic Phase, Circumduction Movements of Upper Members (Richer).

paralyzed limbs, and, most important of all, the Babinski reflex can never be demonstrated. Again, in walking there is not the typical hemiplegic gait, the leg being dragged instead of swung around, as in organic hemiplegia.

Hysterical hemiplegia is hardly as common, however, as paraplegia, which occurs especially after railroad injuries. There may be paralysis of both upper and lower limbs or of one limb. Weakness of the lower limbs, with giving way of the limbs when walking, is sometimes called *astasia abasia.* Hysterical paralysis may be recognized by the suddenness of the onset, the extreme exaggeration or flaccidity of tone, the absence of the Babinski

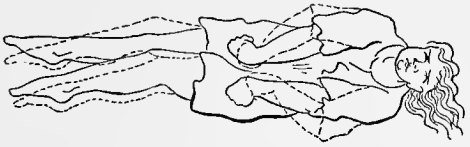


Fig. 454.—Clonic Phase, Schematic Representation of Clonic Movements (Richer).

reflex, and in association there are nearly always hysterical sensory stigmata, and, most important of all, the paralyses may be altered or relieved by suggestion. Paralysis of an eyelid, so-called spastic lid paralysis, is rarely seen, and ocular paralyses are very uncommon. Involvement of the vocal cords has already been mentioned.

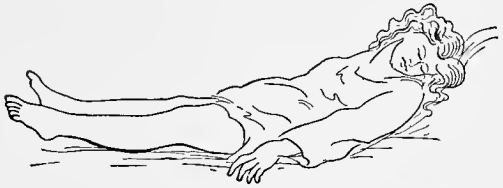


Fig. 455.—Phase of Resolution (Richer).

Hysterical contractures are common and may develop with or without paralysis. The form of the contracture differs from the organic variety and may assume any shape. For instance, in organic hemiplegia the contracture in the upper limbs is greater in the flexor, and in the lower in the extensor, distribution. In hysteria this may be reversed. Again, in the

Fig. 456.—Phase of Resolution, Retaining Partial Contractures (Richer).

functional condition the contracture may come on without paralysis, and the distortion may be extreme. It may cease during sleep and is usually absent during ether narcosis.

Convulsions may appear in any hysterical patient, but they are not as common in this country as in continental Europe. The attacks usually

have certain recognized stages. They may come on at any time, either suddenly or there may be a so-called prodromal period, which may last for a day or longer, in which the patient becomes irritable, depressed, emotional,

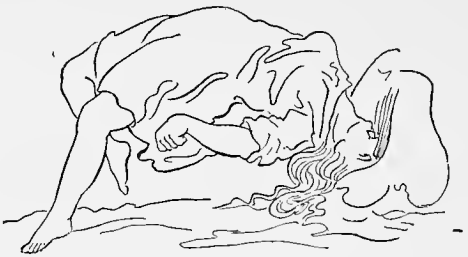


Fig. 457.—Posterior Arc de Cercle (Richer).

or somnolent, and sometimes maniacal. There is usually a so-called hysterical aura, which consists of a sensation of a ball rising from the stomach to the throat, and is known as globus hystericus. This is succeeded by the epileptoid stage, which hardly ever lasts more than a few minutes. The

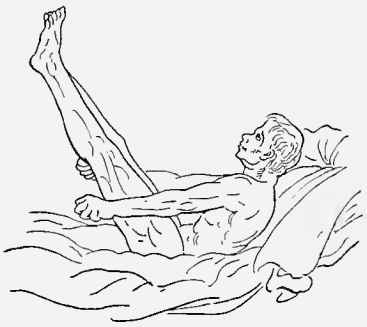


Fig. 458.—Anterior Arc de Cercle (Knobloch).

movements are characteristic, the patient usually throwing the limbs in a wild, irregular manner, the back is usually rigid and arched, and while there may be at first a tonic movement, which is succeeded by a clonic, this is not constant. It is then succeeded by the third or so-called emotional

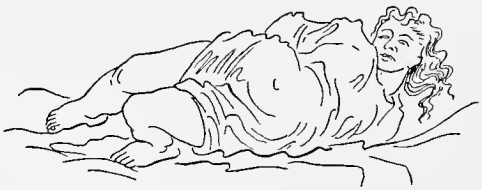


Fig. 459.—Lateral Arc de Cercle (Richer).

or passionate stage, in which the patient assumes different attitudes, depending upon whatever hallucinations may happen to possess him, and may consist either in expression of wild exhilaration, joy, anger, or passion.

This may last for several hours or longer, and then is succeeded by the last stage, during which the patient generally quiets down and passes into a deep sleep or may have various hallucinations or deliriums. These attacks can be differentiated from epilepsy by the fact that there is no epileptic cry, there is hardly ever frothing at the mouth or passing of urine, and while

Fig. 460.—Passional Attitude of Struggling with an Assailant (Richer).

Fig. 461.—Passional Attitude of Solicitation (Richer).

there may be clouding of consciousness, there never is absolute loss of memory and there is not the typical tonic and clonic succession of movements which is so characteristic of the organic disease. This is *hysteria major*.

Instead of the typical succession described above there may be only a

Fig. 462.—Zoöpsia (Richer).

Fig. 463.—Delirium of the Fourth Period (Richer).

so-called mild attack, or *hysteria minor*, in which the patient is suddenly launched into any of the stages described or may become cataleptic, in which there is both physical and mental inertia, and in which it is possible to bend the limbs in any direction, they remaining in the position in which they are placed; or he may pass into a deeper mental stupor, lethargy, or

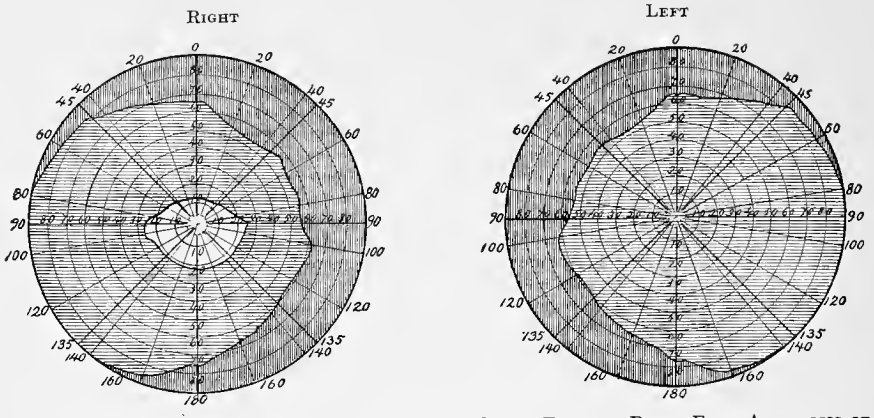


Fig. 464.—Hysterical Concentric Contraction of Visual Field of Right Eye; Amaurosis of Left Eye (Tourette).

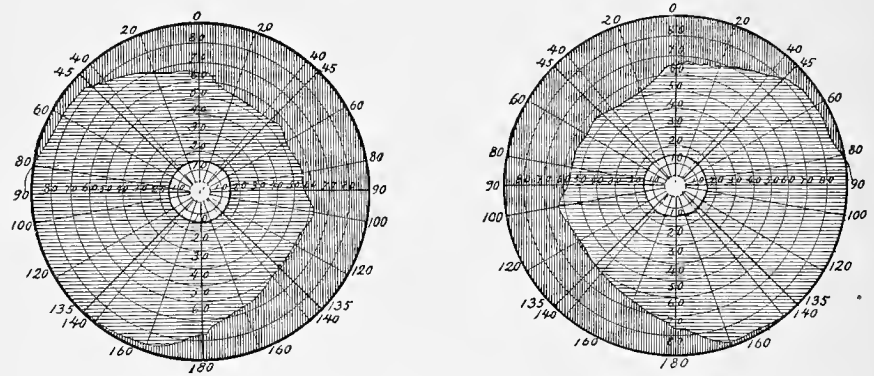


Fig. 465.—Hysterical Bilateral Concentric Contraction of Visual Fields (Tourette).

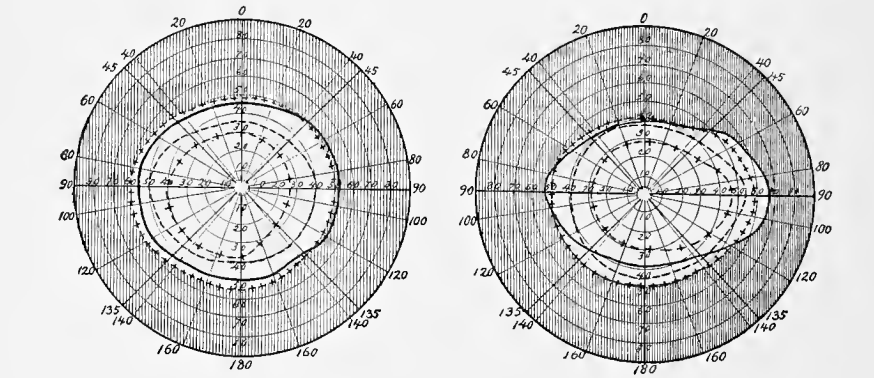


Fig. 466.—Concentric Retraction of Visual Fields for Colors Usually Found in Hysteria (Souques).

Red field inclosed thus: + + +; white field, ——; blue field, — — — —; green field, +— + — +.

trance. Rarely there may be so-called automatic ambulatory automatism, in which the patient wanders for days at a time, performs apparently normal acts, and has only a faint recollection of what has happened in the interim, or there may be disturbance of sleep or somnambulism.

The usual hysterical spasm, however, seen in this country is different from that described. It usually comes on quite suddenly, with an emotional outburst, or may be brought about by pressure on any of the hysterogenic areas and may be stopped in a similar manner, the patient falling to the ground but never hurting herself, and nearly always in the presence of others whose sympathy she desires to arouse. Usually the patient assumes a rigid attitude, the back is arched and rigid, and the limbs are thrown about in a wild, irregular manner, the whole lasting from a few minutes to an hour or longer. There is hardly ever loss of consciousness, the patient being nearly always able to describe what has happened during the spasm.

Special Symptoms.—Under this head will be discussed those phenomena which are concerned principally with the special senses, for many of the so-called special symptoms have been described under the physical.

Ocular Symptoms.—These are quite common and vary greatly. They may consist only in a photophobia or pain in the eye, flashes of light in the visual fields, and total loss of vision in one or both eyes. Amblyopia, however, is not very common, and its occurrence is somewhat doubtful. Hemianopsia has also been reported in hysteria, but it is probable that its occurrence is also somewhat doubtful. The most common ocular manifestation of hysteria, however, is the concentric, regular, or irregular contraction of the visual fields. This is usually for form and color, but either may be present alone. Quite commonly there is reversion of the color fields, or there may be a loss of vision for certain colors or a distortion to one color only. There may be loss of the central field of vision, the peripheral being intact, or a so-called tubular field in which the patient is able to see only in a certain limited area for both near and far points. Still more rarely there may be enlargement of the visual fields (Figs. 464–466).

Loss of *smell* or *taste* or perversion of these functions is quite common. It may be present only on one side, but it is generally bilateral. There may also be buzzing, hissing noises in the ear or loss of hearing, but nerve tests will always demonstrate an intact auditory nerve.

Summary of Diagnosis.—A peculiar mental and physical condition, characterized by suggestibility of symptoms which may be of any character. The patient is usually a young adult who is emotional, irritable, and one who constantly complains and thinks of herself, and perverts everything which may occur as having something to do with her own condition. There may be headache, backache, pains in various portions of the limbs, numbness or pin-and-needle-like sensations, hemianesthesia or anesthesia anywhere, points of tenderness in the back, ovarian, and mammary region, increase of reflexes, paralyses of various sorts, contractures, tremors, disturbance of vision, smell, and taste, and convulsive attacks which may assume almost any character. The most important point of all is the suggestibility of all the symptoms, their variance from day to day, and the fact that any or all may be removed by persuasion.

Clinical Course and Complications.—The course of the disease varies greatly, and depends upon its intensity. In most cases the symptoms can be alleviated to a large degree, but in a well-marked case, after the symptoms have been well established, it is rather difficult to effect

a permanent cure. Sometimes the symptoms progress to such an extent that the patient becomes bedridden.

TRAUMATIC NEUROSES.

Under this head will be discussed those functional nervous disorders which result from injury. Most patients present the symptoms of both neurasthenia and hysteria, and therefore it is inadvisable to discuss them under a separate classification. The degree of the injury in many cases is no criterion of the symptoms that may develop, for very often the slightest trauma may produce the severest neurosis. In some cases it is not so much the injury as the accompanying mental fright which produces the symptoms, and often fright alone is the sole causal factor. It is a matter of common knowledge that not every person who is hurt develops a neurosis, and its occurrence is somewhat dependent upon the health of the individual at the time of the injury and the presence of a neuropathic tendency.

Another factor which enters into the promulgation of the symptoms is the fact that most of these cases become sources of litigation. Corporations or those who are responsible for the injury nearly always assume the attitude that there is not much the matter with the patient, and create in the mind of the litigant a resentful attitude—a factor which does not tend to alleviate the symptoms. On the other hand, very often, for the sake of money, the patient will consciously or unconsciously exaggerate the symptoms, seeing in each a possible source of income. This is furthered by the constant examination of different physicians and lawyers. It is therefore best for the patient, from the medical standpoint, to settle the case promptly. It must be remembered, however, that while it is the rule that most neurotic symptoms are increased during the course of litigation and tend to diminish after its disposition, there are exceptions, and there may be present the severest form of neuroses when there is no question whatever of litigation.

Symptoms.—These vary largely, and depend upon the previous history of the patient and the nature and severity of the accident. As has already been mentioned, the severest form of neuroses may develop from the mildest form of injury. Again, it must be remembered that so-called neuroses are associated nearly always with actual injuries to the brain, the spinal cord, or the muscular structures, and that it is necessary in forming an opinion to carefully exclude an organic basis. In most instances the symptoms are those of both hysteria and neurasthenia, and do not differ from those which have been described under the separate headings. It is only necessary to add that the symptoms come on promptly, and that their specific nature will depend somewhat upon the form of the injury. For instance, a patient who is hurt in the back may develop so-called traumatic lumbago, in which the pain may become so excessive that movement of the trunk and limbs will be almost impossible. This may last for years, and is sometimes dependent upon an actual change in some of the ligamentous and muscular structures about the vertebra. Tremor is common and may be violent. Often in association with tremor of the lower limbs there is produced a pseudo-patellar and ankle clonus, especially when there is an accompanying hysterical paralysis. Paralysis of both lower limbs is probably more common than any other form, especially when there has been an injury to the lower part of the back, and there should be no difficulty in recognizing its hysterical character.

In the ordinary cases of injury, however, in which the patient is only jarred up slightly, there will usually be excessive nervousness, irritability, emotionalism, loss of sleep, anorexia, loss of memory, inability to concentrate or do work, and muscular fatigue. Examination will nearly always demonstrate a hemihypesthesia or anesthesia with increase of reflexes, and the usual hysterogenic zones of hysteria with tremor; and if there happens to be contusion or injury of a certain part, great tenderness of this area.

Clinical Course and Complications.—This, of course, depends upon the nature and severity of the injury and the influence of litigation. While, as has already been emphasized, most patients improve after their case is settled, there are very frequent exceptions to this rule.

GENERAL PARESIS.

Definition.—This disease is known under various terms, such as general paralysis of the insane, paresis, paralytic dementia, and "softening of the brain." It is a disease of the brain characterized by progressive diminution of the mental faculties, terminating in total dementia and accompanied by progressive paralysis of different portions of the body.

Pathologically there is found a diminution in the size of the brain, with lessening of its weight, and atrophy or shrinking of the convolutions, especially in the frontal and to a less extent in the motor and sensory portions, with a widening of the intermediate fissures and thickening of the pia-arachnoid, often with adhesions to the underlying cortex. Microscopically there is a marked degeneration of the cortical cells, with infiltration of round cells, destruction of the nerve tissue, and thickening of the vessels, which is present in nearly all parts of the brain. Similar changes are often found in the spinal cord. Sometimes in the so-called tabetic form of paresis the disease involves equally the brain and cord, and for a long time the spinal cord symptoms predominate, but pathologically the process attacks equally both structures.

Predisposing and Exciting Factors.—A previous history of insanity or paresis in the parents or some neuropathic disease is found in about one-third of the cases. No better evidence of the importance of heredity in the production of this disease can be had than the fact that there is a type of paresis appearing in infants, known as the juvenile form. There is obtained in about 60 per cent. of the cases a previous history of syphilis, and it is the opinion of many that almost every case is due to that disease. However that may be, it is a fact that in certain countries in which syphilis is common, as in China, paresis is rare; so, besides the occurrence of syphilis, which is all-important, there must be some predisposition for the disease. Overwork, mental worry, alcoholic and other excesses are often given as causes.

Symptoms.—The disease occurs more often in men, and usually manifests itself about the thirty-fifth year. The symptoms are generally slow in onset, although rarely they may be ushered in with an epileptoid or hemiplegic attack. In the ordinary case there are present for many years such symptoms, which are usually recognized as neurasthenic, and it is important, therefore, to examine every neurasthenic for the physical symptoms of paresis. There is in most cases a slow change in disposition, and a person who had previously been living a moral life may now begin to live loosely and indulge in sexual, alcoholic, and other excesses. His habits

gradually change, and he becomes more or less irritable, forgetful, and somewhat emotional. He may complain of headache, dizziness, and may have an indefinite sensation that there is something the matter with him, and may often have fits of depression; but, as a rule, most paretics, in contradistinction to neurasthenics, do not dwell very much upon their own symptoms. This preliminary stage may last for a year or longer, constantly increases, and the patient becomes more or less irresponsible, does not attend to his business, and begins now to have certain physical symptoms.

These generally consist in a tremor in the hands, producing tremulous writing. Tremor is especially manifest in the muscles of the face, and can be demonstrated when closing the eyes or showing the teeth, being at first fine and becoming more marked as the muscular action is continued. The tremor not only involves the facial muscles, but also those which are concerned with speech, and gradually it is noticed that the patient has difficulty in pronunciation, and such test words as "truly rural," "fibrillary," "February," and "perspicacity" are slurred over and pronounced with difficulty. The pupils become irregular and their reactions to light and movement become gradually diminished and finally are lost, and there may be the so-called Argyll-Robertson pupil. Temporary ocular palsies are not at all uncommon, the patient complaining of seeing double for two or three days or longer at a time, this disappearing and reappearing and sometimes becoming permanent. The reflexes generally become increased and irregular, and it is not at all infrequent to find one knee jerk more prompt than the other or lost. Ataxia sometimes is present in both the upper and lower limbs, and hearing is often interfered with, and there may occasionally be dizziness.

Coincident with these physical stigmata, the mental symptoms develop. These generally consist in a growing irresponsibility of the patient, who now has well-marked loss of memory for current and past events, may have no idea of time or space, and the whole mental attitude is one of general good nature, cheerfulness, and irresponsibility, the patient on questioning always saying that he is well, is not sick, and there is not much the matter with him. Delusions may develop very early, and are expansive in type, the patient in a well-marked case claiming that he or she may be the richest person in the world, may possess untold strength, may be a deity, or may have any other idea of grandeur. These patients become very extravagant, buy things without any need for them, and will give away anything they possess and many things they do not. As the disease progresses dementia gradually supervenes, until finally the patient is a hopeless dement. Coincidently the physical symptoms increase, the tremor becomes very marked, and the patient will become bedridden. They usually die from an apoplectic attack or uremia.

Differential Diagnosis.—This disease is considered here principally because in its early forms it may resemble neurasthenia. There is this difference, however, that while in paresis the patient will complain of indefinite headache, pains, insomnia, and all sorts of trouble, he will not, as a rule, discuss these as does the neurasthenic, and generally assumes an optimistic attitude. There may be in both a tremor of the facial muscles. In fact, in neurasthenia this is quite common, but in paresis the tremor may interfere with speech, which is not the rule in neurasthenia. In paresis there gradually develops such physical symptoms as irregularity of the pupil with diminution of the light reflexes, and either increased or diminished tendon reflexes. In neurasthenia these are not present. Lastly, in paresis there will gradually develop extensive delusions, which are never present

in neurasthenia, and lumbar puncture will demonstrate the usual changes found in syphilitic disease, and the Wassermann reaction.

MIGRAINE (SICK HEADCHE; HEMICRANIA).

Definition.—A disease characterized by paroxysmal attacks of headache, usually preceded by sensory irritation, especially ocular, and followed by nausea and vomiting.

Pathologically no cause has been found. There are two theories: one, that it is the result of autointoxication, especially gastro-intestinal; the other, and the more probable, that it is a cortical disease similar in type to epilepsy.

Heredity plays an important rôle in the development of the disease, and most persons who suffer from it have a history of some organic or functional nervous disease in the parents. The symptoms nearly always develop either before the tenth year or at the time of puberty, and very rarely after the twenty-fifth year, and are such as would be obtained from cortical irritation. Taking these facts into consideration, it is probable that the disease is not an autointoxication in the sense that it is not an acquired disease, but that those who are migrainous have a congenital tendency for it, and that extraneous causes, such as gastro-intestinal or other forms of intoxication or irritation, bring on the attacks.

Symptoms.—The disease is slightly more common in the female sex. The development of the attacks is, as a rule, gradual. In the history of most patients they come on either before or at the time of puberty, and are characterized as sick headaches, and, as a rule, are not at first of great intensity. Gradually the attacks attain the typical characteristics of the disease. They may come on periodically, especially in women at the time of the monthly period, or may be brought about by different forms of irritation, such as mental worry, eye-strain, gastro-intestinal disturbances, or, in fact, any cause may bring on an attack.

The character and frequency of the attacks of course varies. As a rule, patients are aware of the onset, and there may be either a sense of exhilaration and well-being from a few hours to a day, or the patient may feel depressed, irritable, and sleepy. Occasionally there are no prodromal symptoms, the patient waking up in the morning with headache. In about one-third of the attacks there is a preliminary sense of irritation, which is nearly always ocular. This may vary from a sense of pain in the eye, photophobia, occasional flashes of light in the whole or part of the field to the zig-zag, round, or various shaped bright colored lights which are so characteristic of the attacks. These ocular irritative symptoms may be manifested in the whole or part of the visual fields, corresponding to a hemianopic distribution, and are sometimes followed by a typical homonymous hemianopsia which may last for several hours. Shutting the eyes does not stop them. They may last from a few to ten or fifteen minutes, and are succeeded by the headache.

Pain in the head may be first localized to one spot, generally the temple, and then rapidly spreads over one side of the head, and rarely to both. The headache is nearly always localized to the temporal area, but may be in the frontal or occipital regions. The scalp is generally tender to pressure, although there is no pain over the exit points of the fifth nerve. The headache may last from one to twenty-four or more hours, but the ordinary pain lasts only for two or three hours. During its height the patient feels weak, exhausted, and prefers to be in a darkened room, because irritation or movement of

any sort will increase the pain, although sometimes pressure over the head will cause a sense of relief.

Generally at the height of the headache nausea develops, the patient feeling sick in the stomach, with a tendency to vomit at first the contents of the stomach and then bile, the retching sometimes being distressing. Generally after the nausea and vomiting the headache is relieved, and the patient, after sleeping for some time, generally wakes up in a fairly good condition.

Sometimes instead of the usual ocular irritative phenomena there may be a tingling or numbness on one side of the face, neck, or upper or lower limb; in fact, it may resemble closely the so-called sensory central pains occurring as a result of parietal or other sensory lesions. More rarely there may be motor symptoms, such as drooping of one upper lid with diplopia and diminution of vision, sometimes called *ophthalmic migraine*, or temporary weakness of an upper or lower limb, and, more rarely still, a temporary sensory or motor aphasia, all these symptoms indicating a possible cortical origin.

Rarely mental symptoms complicate the attacks. Because of the chronicity of the disease these patients acquire a so-called migrainous habit in which they learn to do or not to do certain things which have an influence upon the frequency of their attacks. Again, there may sometimes be, preceding or during an attack or following it, confusion, loss of memory, and a mental irritability, this varying greatly in different cases.

In a fair percentage of patients vasomotor symptoms may precede the headache or may accompany the sensory irritative phenomena. This may consist in either a unilateral or bilateral pallor of the skin, especially of the face and rarely of the extremities, followed by profuse sweating, or there is a flushing of the skin with marked throbbing of the vessels and rarely dilatation of the pupil. These vasomotor symptoms may last from a few minutes to an hour or longer.

Summary of Diagnosis.—Paroxysmal headache, usually preceded by sensory irritative phenomena, such as flashes of light, tingling of one side of the face or limbs, lasting from ten to twenty minutes, succeeded by headache generally starting in the temporal area and involving one or both sides of the head, which may last from one to twenty-four hours. At the height of the headache nausea, and vomiting, this usually relieving the headache. This is followed by sleep, the patient generally waking up in fairly good condition.

Clinical Course and Complications.—Most patients with an established migrainous habit have a tendency to recurring attacks, although in women after the menopause and in men after the fiftieth year there is a tendency to cessation. The occurrence of the disease has no influence upon the length of life. Treatment, of course, has a direct bearing upon the length and course of the disease.

PELLAGRA.

Definition.—A disease characterized by gastro-intestinal and cerebrospinal irritation, with skin eruptions on the exposed parts of the body, especially over the extensor surfaces of the hands, accompanied by prostration.

Contributing and Exciting Factors.—Up to 1901 it was thought to be endemic to Italy and the countries surrounding the Mediterranean Sea and confined to people who subsisted on corn. Since 1901 many cases have

been described in the United States, and especially in the south, particularly in the asylums for the insane, but cases have been found in the north, and I have seen one in Philadelphia (March, 1910), this probably being the first to be observed in Pennsylvania. Sporadic cases were described in the United States previous to 1901.

It has been thought that the disease was of a toxic nature, due to the eating of maize, whether or not decomposed. It is probable, however, that the corn theory will have to be discarded, because authentic cases have been described, as in my case, in which there has been no suggestion of the eating of corn. At the present time no explanation for the disease can be offered, other than that it is of a toxic nature, the cause not being definitely known.

Symptoms.—The onset varies considerably, but inasmuch as in a well-established case there are present gastro-intestinal, skin, and cerebrospinal symptoms, these will be described separately, although it is to be understood that they may appear at the same time.

Gastro-intestinal Symptoms.—These usually consist in a stomatitis, gastric distress and pains, vomiting, and especially diarrhea. They may come on very early in the disease and be very intense, and then again chronic. The stools may have mucus or blood. Ulcers in the mouth are very common.

Dermatitis.—Skin symptoms, as a rule, come on in the early spring, and are generally manifest over the exposed parts of the body, especially over the extensor surface of the hands and forearm as far as the elbow. They are less common over the face, and rarely present over the dorsum of the feet and chest. They are usually ushered in by blebs containing gelatinous fluid, which burst and form a crust, underneath which a raw surface appears. The eruption may be dry or wet and has a tendency to fade gradually and disappear and, as a rule, reappear the following spring. Instead of the eruptions there may be only an erythematous condition of the skin of varying intensity.

Nervous Symptoms.—These vary. It is not at all uncommon for pellagra to be ushered in with general neurasthenic symptoms. There may be fibrillary and coarse tremors in various parts of the body, weakness of the limbs, and generally increase of reflexes. The mental symptoms vary; the patient may be depressed, refusing to eat and drink, and again may be noisy and maniacal. As a rule, there is present a dull apathetic condition. Chronic delirium or pellagrous mania has been described. In Italian countries pellagrous insanity is quite common, and may be of different kinds.

General Symptoms.—Fever is uncommon. Examination of the blood has shown a diminished amount of hemoglobin, and often changes which are common in pernicious anemia. Serum diagnosis so far has been negative.

Summary of Diagnosis.—A disease occurring mostly in damp or tropical countries and among people who eat corn, the symptoms consisting of eruptions on the extensor surface of the hand and exposed parts of the body, accompanied by gastro-intestinal irritation and cerebrospinal symptoms.

Differential Diagnosis.—There should be no difficulty in diagnosing this disease, the presence of the skin eruption over the extensor surface of the hands and exposed parts of the body being the most important diagnostic symptom.

Clinical Course and Complications.—The disease has a tendency to become chronic, and recurrences are frequent. The mortality is variously estimated in different countries, and may be as high as 50 per cent., but in the great majority of cases recovery can be hoped for.

INDEX.

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