PRACTICAL POINTS
ON THE
DIAGNOSIS AND TREATMENT
OF HEART DISEASE

BROCKBANK

THE DIAGNOSIS AND TREATMENT OF HEART DISEASE

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PRACTICAL POINTS FOR STUDENTS AND PRACTITIONERS

BY

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WITH ILLUSTRATIONS

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PREFACE TO SECOND EDITION

The demand for a second edition of this small work has enabled me to alter and add to it considerably. I hope it will prove even more useful now to students and practitioners than it did in its original form. It could have been improved further had not National affairs distracted attention from teaching matters.

E. M. B.

PREFACE TO FIRST EDITION

(Entitled "Heart Sounds and Murmurs, Their Causation and Differentiation.")

In this small work I have attempted to put simply, clearly and in convenient pocket-book form for clinical reference the elements of cardiac auscultation for the use of students. I have repeated myself a good deal with the object of making an argument or explanation as complete as possible in the one place and thereby of avoiding turning backwards and forwards.

E. M. B.

Manchester, 1911.

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PRACTICAL POINTS IN THE DIAGNOSIS AND TREATMENT OF HEART DISEASE

I

PRELIMINARY CONSIDERATIONS

Anatomy and Physiology.

Anatomy.—The heart is a conical-shaped organ with the great vessels attached to its base; it is about the size of its owner's clenched fist, and lies obliquely in the thorax, inclined from above downwards, forwards, and to the left, with one-third (the base) to the right and two-thirds to the left of the mid-sternal line.

The anterior aspect of the heart is formed almost entirely by the right ventricle, right auricle, and infundibulum or conus arteriosus; only a small part of it, namely, the apex and about half an inch along its left margin, is formed by the left ventricle. The tip of the left auricle just comes to the surface behind the second interspace. It is covered by lung all over except in the mid-sternal line, and a triangular space formed by the mid-sternal line, a line from the fourth chondrosternal articulation to the junction of the fifth costal cartilage with its rib, and the line of liver dulness, the so-called area of superficial cardiac dulness (see p. 26).

Surface anatomy.—The upper limit is behind the second costal cartilage. Its right side extends half an inch beyond the right edge of sternum. Its apex or its limit to the left is in the fifth intercostal space two and a half inches beyond the left edge of the sternum; in adult males this is about one inch below and three-quarters of an inch to the sternal side of the nipple. Its lower limit is about the sixth rib, where the heart lies in con-

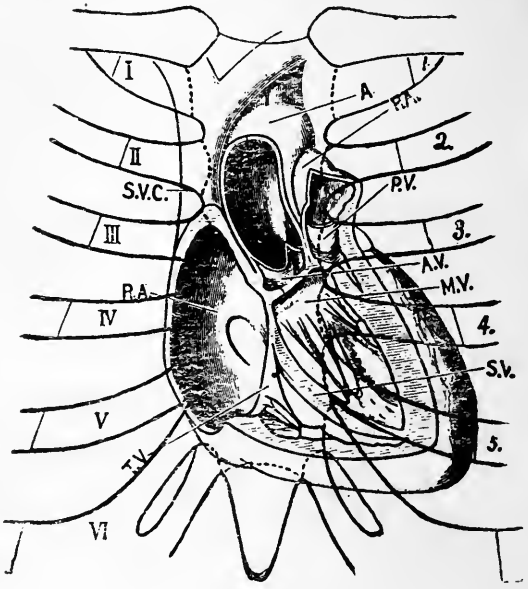

FIG. 1.

A., Aorta; *A.V.*, aortic valve; *M.*, mitral valve; *P.A.*, pulmonary artery; *P.V.*, pulmonary valve; *R.A.*, right auricle; *S.V.*, septum ventriculorum; *S.V.C.*, superior vena cava; *T.V.*, tricuspid valve. (Cunningham.)

tact with the diaphragm. In children the heart may be rather higher and its apex farther out in the chest.

N.B.—*The nipple in men*, normally situated *about* 4 *inches from the mid-sternal line*, may be nearer than this to the sternum if the chest is not well developed. An apex-beat outside the vertical nipple line, therefore, need not necessarily indicate disease. In children the

apex-beat may be farther out and up in relation to the nipple than it is in man. The nipple in women is useless as a point which has definite position.

The surface lines or landmarks which are of use in the examination and note-taking of thoracic affections are :

The *mid-sternal line* down the middle of the sternum.

The *lateral sternal lines* down the right and left edges of the sternum.

The *parasternal lines* midway between the lateral sternal lines and

The *mid-clavicular lines,* which are lines drawn perpendicularly from the middle of the collar-bones or midway between the middle of the suprasternal notch and the top of the acromion.

The *nipple line* in the normal adult male is practically the same as the mid-clavicular line, the nipple being situated about four inches from the mid-sternal line.

The *axillary lines,* anterior, mid, and posterior, running perpendicularly downwards through the anterior, mid, and posterior regions of the axilla.

The *scapular lines,* through the angles of the scapulæ.

Nervous mechanism of the heart-beat.—The stimulation of the heart for its beating is carried on by means of a special form of tissue, partly nervous and partly heart muscle in structure, which occurs in aggregations or nodes, and connecting or end fibres.

(1) The *sino-auricular node* is in the subepicardial tissue at the junction of the superior vena cava and the right auricular appendix. This node is the most highly developed aggregation of the neuro-muscular tissue, and it initiates the stimulus and sets the pace and rhythm of

the heart. It is believed to receive fibres from the vagus and sympathetic nerves. From it the stimulus passes, in some way not fully understood, through

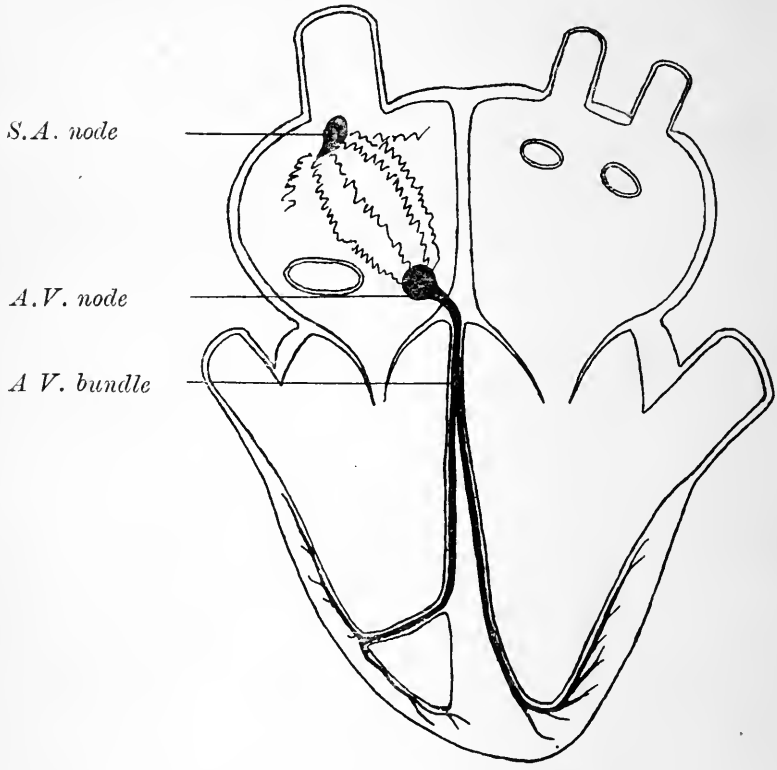

FIG. 2.—THE CARDIAC NERVOUS MECHANISM.

S.A. node, Sino-auricular node; *A.V. node*, auriculo-ventricular node (Tawara) *A.V. bundle*, auriculo-ventricular bundle. The connection between the S.A. node and the A.V. node is indefinite. It may be through the contracting auricular muscle.

(2) The auricular walls to the *auriculo-ventricular node*, which lies in the right side of the auricular septum. From this *a.v.* node the stimulus is carried by

(3) *The auriculo-ventricular bundle* (*a.v.* bundle) to the ventricles. This bundle divides in the interventricular

septum into two branches, which ultimately spread out in the subendocardial tissue of right and left ventricles as Purkinje's fibres. The papillary muscles are said to be the first part of the ventricles to receive the stimulus to contract.

It is not known how the stimulus from the sino-auricular node traverses the auricles. It is probably carried by the muscle fibres, regular and complete contraction of which is essential for the regular contraction of the ventricles. At any rate, irregular and incomplete contraction of the auricles results in irregular contraction of the ventricles. In a marked condition of auricular disease, in which the wall is dilated, and in which the muscle is in a state of over-excitability and is making constant incomplete attempts to contract (see Auricular fibrillation and Flutter), there is an extreme degree of irregularity in the ventricular rhythm.

Whilst the stimulus for the heart beat arises normally in the sino auricular node, and is rhythmic or regular in origin, in abnormal conditions, functional or organic, it may arise in other parts of the nervous mechanism, or be arrhythmic or irregular in origin. Thus it not uncommonly starts in the auricle or ventricle causing the condition known as premature systole (*q.v.*), or premature stimuli may leave the sino-auricular node with the same result.

If the nervous tissue of the heart be changed structurally by disease, or become unduly irritable, irregularity in the action of the heart results, giving rise to conditions (*q.v.*) known as premature systole, heart-block, auricular fibrillation, and paroxysmal tachycardia.

II

ROUTINE EXAMINATION

What Patients with Heart Disease complain of.

Patients suffering from the worst forms of heart disease may feel perfectly well, and have no complaint of any sort. Their cardiac weakness may only be discovered during the course of an examination for life insurance, or for some illness not connected with the heart. As a rule, however, they do complain of one or more subjective symptoms, chiefly shortness of breath, palpitation, and pain.

1. **Shortness of breath**, especially after only slight exertion, as on going up a flight of household stairs or up a short hill. In more severe conditions the patient may be awakened from his sleep with a sensation of breathlessness and tumultuous action of the heart—a very distressing condition.

2. **Palpitation**, or unusual action of the heart, which attracts its subject's attention. The quick action with a "throbbing" sensation in the neck resulting from emotional or muscular overaction is commonly met with, and generally described as palpitation. It may be met with in normal as well as diseased hearts. A "fluttering" or "tumbling" sensation is often felt. This, how-

ever, in its slighter forms may be present when the heart is quite sound, and be due to functional disturbance of the regular stimulation of the heart. In disease, when the heart is enlarged and its action forcible, this symptom is often most distressing, especially when it wakens the patient up out of his sleep with a smothering feeling.

3. **Pain** (1) over the præcordia, made worse by exertion or increased quickness of beat, is met with chiefly in cardiac enlargement due to valvular disease or adherent pericardium. There is much tenderness to the touch in these cases.

Or (2) shooting down the arms, especially the left arm, or in the shoulder or up the neck, in aneurysm or angina pectoris. There is often also a sense of suffocation or of gripping of the heart, with apprehension of impending death, in anginal cases while the pain is present.

N.B.—*Pain in the upper arms, especially in the left arm, or up the back of the neck, may be the first indication of aneurysm of the arch of the aorta.*

The Cause of the Cardiac Pain.—The pain is one form of referred pain. The heart, developmentally, is associated with the first eight dorsal segments and nerves, and painful impressions arising in it are usually referred to the first and second dorsal nerves. The cutaneous distribution of these nerves is the inner aspect of the arm as far down as the elbow, and anginal pains which arise from disease at the base of the ventricles, or first part of the arch of the aorta, are referred to the shoulder and down the arm.

Hæmorrhage from mucous surfaces may be complained of by patients with mitral disease, especially

mitral stenosis. Epistaxis, hæmoptysis, hæmatemesis, and bleeding from the ears, have been present in cases under my care. The heart must therefore always be carefully examined in all cases of mucous membrane hæmorrhages.

Dropsy.—In heart disease dropsy affects the legs in the first place, and is improved by rest in bed. In looking for œdema pressure should be applied deliberately, for about five seconds, over the face of the tibia near the ankle, and also over the sacrum. Slight degrees of it will be overlooked unless this care be taken.

Cough from congestion of the pulmonary mucous membrane is a frequent consequence of disease of the mitral or tricuspid valves.

The Arterial Pulse.

N.B.—The pulse should be examined at the commencement of the investigation of any heart affection.

Much valuable information can be obtained in heart affections by an intelligent feeling of the radial pulse with the fingers. The sphygmograph is an interesting instrument, and gives instructive information when you know the kind of tracing you ought to get and the proper way to get it. But even to one experienced in its use it gives but little information which escapes the practised fingers.

The best way to feel the radial pulse is to use two hands for the purpose. One hand is to steady the patient's wrist, by holding his hand so as to allow the delicate application of three fingers of the other hand to the artery, as it runs over the end of the radius. The thumb, to further steady matters, should be placed behind the patient's wrist. All three fingers should be

placed closely together over the end of the radius, so that the artery can be pressed against it, and not against the less resisting soft tissues. The index finger should be applied firmly as close to the base of the metacarpal bone of the thumb as possible to obliterate the junction of the radial artery with the palmar arch, and prevent any wave coming round that way. The ring finger is used to tell when the pulse in the artery is beating or not, and the fourth finger to apply pressure over the artery and estimate the amount of force necessary to obliterate the pulse in the radial artery. In this way changes in the tension of the bloodvessel can be very accurately estimated with a little practice and knowledge of the normal condition of affairs.

Another useful way of feeling the pulse is to grip the lower end of the radius between the forefinger and thumb of one hand, with the finger applied firmly over the artery close up to the base of the metacarpal bone of the thumb to cut off the palmar arch anastomosis pulse, and then to apply the first and second fingers of the other hand to the radial artery as before. In either method all three fingers must occupy as little space as possible, so that they can manipulate the artery as it runs in front over the end of the bone.

It is sometimes instructive to feel the pulse by gripping the wrist with the hand in such a way that the palm of the observer will lie over both radial and ulnar arteries and feel their pulsation, at the same time elevating the patient's forearm so that there is a considerable stretch of perpendicular artery. In this way the water-hammer or Corrigan's pulse is best developed, both arteries slapping their pulse wave against the palm of the hand, and its simulation by a low tension pulse of cardiac dilatation reduced to a minimum.

No apology need be made for giving such details of good methods of feeling the pulse, for by their adoption an accurate estimate of changes in the pulse tension may be more easily made, and variations from the normal in other ways more easily recognized than if less deliberate methods be adopted.

Congenital abnormalities in the anatomy of the radial arteries may occur, and must be allowed for. Thus the radial artery may be of smaller size than normal in one or both wrists, and make pulse-feeling rather difficult.

Comparison of the two radial arteries.—*Inequality in the pulse wave* of the two radial arteries may occur, and be due to—

1. Congenital causes.
2. Pressure on the artery somewhere on the proximal side of the wrist (with a thoracic aneurysm or new growth).
3. Changes in the wall of the artery (atheroma).
4. Obstruction in the lumen of the vessel (embolism or thrombosis).

The points to be noticed in feeling the radial pulse are, the rate, volume, regularity, tension, condition of the vessel wall, aud the comparison between the two radial pulses.

Pulse-rate. — In counting a pulse-rate, it is to be remembered that the result does not always indicate the rate the heart is beating at. It generally does, but in one form of heart disease—namely, auricular fibrillation—in which the rhythm and force of the ventricular systole is extremely irregular and uneven,

many of the ventricular beats, which are considerably increased in number, are so feeble that they do not transmit a pulse wave to the radial artery. Therefore, when the radial pulse is irregular in rhythm and size of wave, and increased in frequency above the normal, the stethoscope should be applied to the heart whilst the pulse is being counted.

A quick pulse in heart disease practically always means either

1. *Some debility of the heart muscle*—that is, inability to do the extra work thrown on it by the abnormal condition by its usual number of beats. It often means some giving way of the heart muscle, that is, cardiac muscle failure, but not necessarily so; or

2. *An abnormal excitability of the nervous mechanism* which causes an abnormally increased number of stimuli sent to the ventricles.

These points will be referred to again under the headings tachycardia and irregular pulse.

An abnormally slow pulse in heart disease—that is, a rate below 50, especially one below 30—frequently means some pathological change in the auriculo-ventricular (*a.-v.*) bundle, which impedes the conduction of stimuli from the auricular nervous mechanism. (See Bradycardia.) In this case the auricles will be contracting more frequently than the ventricles.

A certain but small number of persons have a heart-rate below 50 with auricles contracting as slowly as the ventricles.

The volume of a pulse, that is, whether it is large and full or small and empty, depends chiefly on the amount of blood propelled by the ventricle into the aorta at each systole.

Small and empty pulses.—*In mitral disease* the pulse is often small and empty, because in stenosis less than the normal amount of blood enters the left ventricle during its diastole, and in incompetence some regurgitates into the auricle. In either case an abnormally small amount passes from the ventricle into the aorta with each beat. Furthermore, when the ventricles in mitral disease are contracting with increased rapidity, irregularity in rhythm, and inequality in force, only a considerably reduced amount of blood has time to pass into the left ventricle for it to contract on.

With marked *aortic stenosis* the pulse is a small one, and there is delay in its transmission to the wrist.

A large and full pulse is met with when there is an abnormal amount of blood expelled from the ventricle into the aorta, and this is met with in cases of dilatation of the left ventricle with hypertrophy. In dilatation without adequate compensation the pulse often has a soon-full, soon-empty, or slapping character, which may simulate very closely a Corrigan's or water-hammer pulse (*q.v.*).

Regularity.—Normally the heart, and therefore the pulse-beats, follow each other regularly in rhythm and uniformly in character. It is very common, however, to meet with irregularity in rhythm and varying character of heart-beat and pulse-wave, not only when the heart is diseased, but also when it is quite sound.

In healthy children the heart may be irregular, its rate varying with inspiration and expiration. A similar irregularity in rhythm is often met with in nervous people.

The commonest form of irregularity in adult life is the so-called missed beat, in which a heart-beat and a

pulse-wave apparently drop out. What happens is that the heart gives a premature and weak contraction, which does not create a pulse-wave. (See Premature or extra systole.) This frequently occurs in normal hearts as the result of digestive disturbances.

The other extreme is met with in certain forms of heart disease when scarcely two successive beats of the heart and pulse are regular in rate or uniform in character. The beats occur in a most irregular way (delirium cordis), and with more or less greatly increased rapidity. Sometimes the pulse-rate is uncountably fast. (See Intermittent pulse and Auricular fibrillation.) Between these two extremes lie varying degrees of irregularity.

Tension.—***Increased tension*** is an important indication of pathological processes taking place in the system, the most important of which are arteriosclerosis and interstitial nephritis. It may be the first physical sign of these diseases, and when it is present the urine must be examined carefully by boiling and acidulation—the most delicate test for albumen. The specific gravity of the urine will probably be low, and often only a very faint trace of albumen is found, or none at all.

Nocturnal polyuria.—There will, however, generally be obtained a history of nocturnal polyuria as well—that is, the necessity of getting up regularly at night two or three times to pass a considerable amount of urine. In making this point, it must be ascertained how much fluid is drunk late at night, for many men take half a pint at least of nightcap fluid, such as whisky and water, hot milk, or plain hydropathic hot water; but this will rouse them only once in the night as a rule, and generally early in the morning. Further, the increased frequency of micturition in small amounts caused

by an irritating prostate must not be confused with high tension polyuria.

Increased tension may be caused by absorption of toxins from the alimentary canal without there being any primary interstitial nephritis. This condition may last for years, with its attendant discomforts.

In mitral stenosis the pulse, though small, is often one of considerable high tension.

The distinction between *hardness of the wall* of the artery and *increase of arterial tension* must be borne in mind. Either may be present without the other, or both may be present together.

Abnormally low tension is rather more difficult to make out. It is present in debilitated states in children who are subject to fainting attacks with no definite ascertainable cause.

The condition of the wall of the artery must be made out by rolling it under the fingers against the end of the radius. When this is done, a normal artery can scarcely be felt at all after the pulse-wave has been obliterated, whilst a diseased one feels more or less hard, like a piece of string when there is fibroid degeneration of the middle coat, or like a hard calcareous tube (pipe-stem artery) with marked atheromatous degeneration. Movements of the fingers in the long axis of the end of the radius will reveal any annular deposits of calcareous material.

Venous Pulse.

The venous pulse affords valuable information in heart disease, especially of the condition of the *right side of the heart*. The only vein in which it can be observed is the jugular vein, between the thoracic attachments of

the sterno-mastoid muscle. When there is obstruction to the flow of blood from the right auricle into the ventricle, the jugular veins are distended, and stand out

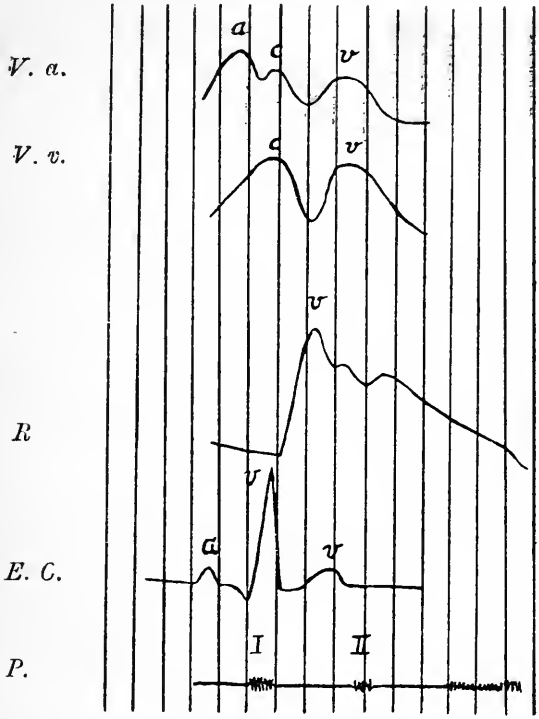

FIG. 3.

V. a., Auricular form of venous pulse
V. v., Ventricular form of venous pulse.
R., Radial pulse.
E. C., Electrocardiogram.
P., Phonoscope tracing showing heart sounds and murmur
a., Auricular wave; *v.*, ventricular wave; *c.*, carotid waves.
I. and II., first and second heart sounds. ·1 second intervals of time.

prominently, but often there is no visible distension, let alone pulsation, of the veins.

With obstruction to the large veins in the chest there will be distension of the veins in the neck but no pulsation.

The venous pulse cannot be felt by the finger like the arterial pulse, but requires a special recording apparatus for its investigation. For the use of this *clinical polygraph* considerable experience and practice is necessary. Its application is consequently limited to hospital practice or to the work of a few specialists. Whilst it gives information which helps in the understanding of the conditions present in heart disease, especially concerning the condition of the auricles and the relative number of auricular and ventricular contractions, it does not as often afford much assistance in treatment beyond that gained by other and more manifest evidence of cardiac muscle failure.

Jugular pulse waves.—The common form of jugular pulse tracing is known as the auricular form, and shows three waves. The first, or the auricular (*a.*) wave, is caused by auricular systole; the next and smaller wave is called the carotid (*c.*) wave, and is probably caused by the pulse-wave in the carotid artery being transmitted through the jugular vein to the surface; and the last, or ventricular (*v.*) wave, is caused by the rebound of blood in the filling auricle against the closed tricuspid valve (Fig. 3, *V. a*).

In the other form of venous pulse, known as the ventricular venous pulse, there is no *a.* wave, the *v.* wave predominating. In this case there is no contraction of the auricle, this organ being paralyzed and acting merely as a reservoir (Fig. 3, *V. v*).

The jugular pulse, then, gives information as to what is going on in the right side of the heart chiefly. It is recorded synchronously on paper by means of a polygraph with the radial pulse, or occasionally with the apex-beat, and for its interpretation some time standards

and a pair of compasses are necessary. The time standards are the following :

The radial pulse follows one-tenth of a second, and the carotid pulse one-twentieth of a second after the onset of ventricular systole, which in its turn is preceded by auricular systole by one-fifth of a second. Before the tracings are taken, the two recording pens are moved up and down across the paper to mark the relative positions of the venous and arterial pulses at the same moment of time, and to give the "ordinate" for accurate measurement. A time-recorder constantly marks intervals of one-fifth (two-tenths) of a second. When a tracing is completed, the distance between the ordinate for the artery tracing and the beginning of the upstroke of its pulse-wave is measured accurately with the compasses. The stretch between the points of the compasses is then decreased by a distance exactly equal to a one-tenth of a second interval on the time-marker. The compasses are now applied to the venous pulse, one limb being placed accurately on its ordinate, and the other on the tracing, where it will fall exactly where the carotid wave ought to be developing. This spot is then marked as the rise of the *c.* wave. Each successive *c.* wave is marked out by resetting the compasses to a space equal to the distance between the commencement of the upstroke of the arterial pulse tracings, and measuring successive complete pulse-waves along the venous tracing from the originally marked *a.* wave, marking each *a.* wave as before. The auricular wave ought to begin two-tenths of a second before the *c.* wave.

General Inspection in Cases of Heart Disease.

The general inspection should always be carried out with the patient facing a good natural daylight, as

artificial light destroys the delicate changes in shades of colour, especially the yellow of jaundice, which are such important indications of the health. Whilst patients suffering from heart disease, even in its most serious forms, may look "the picture of health," with a general natural freshness of complexion, there are certain signs in the face and general aspect pointing to cardiac disease which are visible to the eye of a skilled observer.

The general signs are those of shortness of breath on slight exertion, or even without any, inability to breathe in any but the erect position (orthopnœa), œdema, —especially swelling of the legs and dependent parts. Marked throbbing, with short and sudden pulse-waves, in the carotid or other arteries, may be seen, and generally means aortic regurgitation, but a quickly acting dilated heart may cause similar appearances. Distension of the veins of the neck with or without visible pulsation may be present.

In the face, in addition to signs of the general distress, there may be seen, but not always, some special changes.

Mitral stenosis often gives rise to a high colour, localized more or less to the malar eminences of the cheeks, and due to dilated veins (venæ stellatæ). An exactly similar condition is met with in people who have been exposed to the weather, and a somewhat similar condition, and one which often deceives the student, is met with in myxœdema. Here, however, there is a peculiar yellowish, waxy appearance of the rest of the face.

In aortic disease, when it is severe, there is generally

much pallor, from the tendency which incompetence of the valve has to empty the blood vessels rapidly, and often also marked "lines" running from the nose to the outer angle of the mouth.

Capillary pulsation may be seen if looked for carefully over the forehead; but to see this properly it is generally necessary to make a small patch of blush by rubbing the forehead with the back of the finger-nail.

Cyanosis.—When the cardiac muscle, especially that of the right heart, fails, the pulmonary circulation cannot be carried on effectively, with the result that the venous blood is imperfectly oxygenated. It then collects in the systemic circulation, and is seen in the skin and mucous membranes as a bluish discoloration, or cyanosis. Cyanosis is most marked in dependent parts, the lobes of the ears, the fingers and toes, but it is well seen in the face. This form of cyanosis is the result of a deficient force pumping blood through the lungs.

With heart disease of long standing, especially of congenital origin, the fingers and toes are often clubbed as well as purple.

A bluish, ashy grey appearance is, however, often met with in chronic bronchitis and emphysema with the heart working well. This is due to inability of the lungs to expand freely and oxygenate the blood properly.

In congenital heart disease there is generally a very deep red, purple, or bluish aspect. When congenital heart disease is met with in people over puberty, especially over twenty, as it occasionally is, it is almost always due to pulmonary stenosis. Other congenital heart lesions generally terminate in childhood, but I saw one adult of thirty-one who died from the effects of a

congenital septum in the auricle, which simulated mitral stenosis. How he lived as long as he did was a wonder.

Jaundice is also often present. For this to be definitely established the conjunctivæ under the upper eyelids must be examined, the yellow discoloration so often seen in the exposed parts not being due to bile pigments.

Anxiety.—In angina pectoris or severe intrathoracic aneurysm there is often an appearance of extreme anxiety, and, if the pain be present, of great distress.

The attitude is fixed and immobile as though movement of any sort were feared, in marked contrast to the excited movements of hysterical angina.

Inspection of the Neck.

Arterial pulsation.—Pulsation or throbbing of the carotid arteries, varying in degree, will often be seen, a thin neck favouring its visibility. In the majority of cases of slighter degree it means nothing abnormal, merely a favourable position of the arteries for their pulsation to reach the surface. Even when marked it may be due simply to nervous overaction of the heart. With aortic regurgitation it reaches its highest development, when the throbbing of a soon-full, soon-empty vessel is very striking. The frontal artery may also be seen to throb in aortic incompetence, but a visible pulse wave in any exposed artery may occur without any heart disease being present. In dilatation with hypertrophy, the carotid arteries may pulsate in a way very suggestive of an aortic pulse.

The **condition of the veins** must be noted, distension indicating obstruction to the return of blood to the right

auricle, or to its flow through the right side of the heart. The jugular veins are often seen to be very full, and if examined carefully between the attachments of the sterno-mastoid to the clavicle and sternum pulsation in them may be seen and felt. A transmitted impulse from the carotid artery must be discriminated from true venous pulsation.

The direction in which blood flows to fill the veins is important. If a stretch of vein be emptied between two fingers the vein will fill from below upwards if there is tricuspid regurgitation, and from above downwards when auricular distension alone or venous obstruction is present.

Any general swelling of the neck, if present, must be noted, as it is generally a sign of serious intra-thoracic disease or venous obstruction.

Inspection of the Præcordia.

Inspection of the præcordia must never be omitted, as it gives most valuable information in disease. A side or tangential view should be taken if the direct inspection is negative, pulsation being sometimes seen by it, and not otherwise.

The **normal cardiac impulse** may be *invisible*, and negative observation may mean nothing abnormal. The anterior and mid-axillary regions should be most carefully examined, as the impulse is often surprisingly far out in disease.

Diffuse pulsation between the mid-clavicular line and the sternum generally means disease of the right ventricle, and widely spread pulsation, outwards as well, means enlargement (dilatation with hypertrophy) of both ventricles. Auricular pulsation is never seen. Pulsation over the base of the heart generally indicates

aneurysm of the arch of the aorta. The impulse of a forcibly beating normal heart may extend outwards to the nipple line.

Bulging of the præcordia is generally best seen in children, whose chest walls give easily in front of enlarged hearts. When limited to intercostal spaces it often indicates pericardial effusion.

Systolic retraction of the interspaces may be seen; it is often diffuse, and means adherent pericardium or great cardiac enlargement, the contraction of the heart causing negative pressure in the thorax, and consequent retraction of the intercostal spaces.

Palpation.

Palpation is a very valuable method of cardiac diagnosis, and should always be practised. The hand must be warm, and must be placed over the region of the apex-beat below and within the nipple line. As much of the hand as possible should be laid over the præcordia, from the sternum outwards, with the region of the distal interphalangeal joints about where the impulse is expected. As the præcordia are often very tender, a light touch must always be used.

Cardiac impulse.—When the impulse is felt by the hand, the outermost limits of it to the left should be determined by the pad of one finger applied in the intercostal spaces. This gives very accurate information as to the area of heart dulness, which will be further investigated later by percussion. If the impulse cannot be felt in the lying position, let the patient sit up and lean forward, but not to the left; also try the effect of a deep expiration and the holding of the breath afterwards.

N.B.—*The heart often extends surprisingly far into the axilla,* and mistakes in diagnosis result from want of attention to this fact.

An absent cardiac impulse may mean thick chest walls, with too much adipose tissue, an intervening rib, emphysema, feeble heart action, or pericardial effusion.

The *normal apex-beat* has characters which must be learned by experience. It is produced by systolic contraction and elongation of the heart, the elongation being communicated to the chest wall by the apex of the left ventricle. It is limited in area to that of about half a crown, and is moderately forcible and abrupt.

In *disease* it may no longer be an apex-beat from the left ventricle, but becomes a diffuse impulse from the anterior surface of the right ventricle.

A *distinction* should therefore be drawn between the ***apex-beat*** and the ***cardiac impulse.***

In *hypertrophy* of the heart the impulse is slow, heaving, and stronger than normal.

In *dilatation* of disease, or feeble action in debility, it is quicker, feebler, and more slapping, than in health.

In *mitral stenosis* the accentuated first sound can be distinctly felt, and should be a definite indication of what will be heard on auscultation; a thrill may be felt.

In *pulmonary valve lesions* an accentuated second sound may sometimes be felt in the second left intercostal space, and in aneurysm of the aorta the aortic second sound may be felt as a diastolic shock.

Thrills arising within the heart are caused by palpable vibrations set up in the diseased valves of the heart and carried to the surface of the chest. They arise at a rigid and narrowed orifice, with altered curtains,

and may be systolic or diastolic in rhythm. The sensation of liquid being sprayed against the hand, or of a cat purring, is conveyed by them.

Systolic thrill is met with (*a*) at the apex of the heart in regurgitation through a mitral or tricuspid valve made incompetent by stenosis;

(*b*) At the base in obstruction due to an atheromatous aortic valve. A pulmonary thrill is extremely rare.

Diastolic thrill is met with (*a*) at the apex, with blood entering the ventricle through a stenosed mitral or tricuspid valve.

Regurgitation through an incompetent aortic (or pulmonary) valve may cause a thrill over the ventricles.

(*b*) At the base a diastolic thrill is extremely rare.

A *crescendo thrill* is felt with a crescendo murmur. The rhythm of this, as usually taught, is presystolic or auricular systolic—that is, just before the systole of the ventricle. As will be seen later, the rhythm is probably early ventricular systolic.

Pericardial friction may be felt, and simulate a rough thrill, but it has not the character of sprayed liquid, but more that of two rough surfaces rubbing together.

Percussion.

Light percussion gives the best results, and should be cultivated. Changes of resonance can be best appreciated by the sensation of touch aiding that of sound. Apart from this, the præcordia in subjects of heart disease are often exquisitely tender, and most patients, especially in private practice, prefer gentle methods. After locating the outermost limits of the cardiac impulse by palpation, the heart should be percussed in a routine way, and always from resonance to dulness.

The most delicate results are obtained by very light percussion in an intercostal space, thus avoiding the sounding-box of a rib, and by quick contrasts brought about by two strokes in one place followed quickly by two in another not more than half an inch away.

Note the results by measurement from the mid-sternal line, the normal distance in an adult being about three and a half inches from it.

Routine method of percussing out the heart dulness.—

N.B.—It is very important to follow regularly a definite method of percussion in the examination of the heart.

1. *Limit to the left.*—Percuss horizontally along the line of the normal apex-beat from the axilla inwards. If the heart is enlarged or displaced, begin to percuss a couple of inches farther out, and follow along its changed horizontal line.

2. *Limit to the right.*—First locate the liver dulness (about the sixth rib) by percussion downward in right mid-clavicular line. Then take a line one inch above this and percuss inwards towards the sternum. It is very unusual to get any distinct line of dulness to the right of the sternum in health, although the right auricle extends half an inch in this direction, the ribs and sternum acting as a sounding-box. When the right auricle is enlarged or the heart displaced to the right, dulness will be found.

3. *Limit upwards.*—Percuss in a line one inch to the left of the sternum. Dulness will be found about the third rib.

4. The *limit downwards* is the upper limit of the liver and diaphragm against which the heart lies.

Superficial or absolute cardiac dulness is obtained over the small triangular part of the heart which is uncovered by lung—namely, that bounded by the mid-sternal line, a line running from it at the level of the fourth left costal cartilage to the union of the fifth rib with its costal cartilage, with its base line at the level of the diaphragm (sixth rib).

The dulness obtained over this part of the heart with no intervening lung is more marked than that obtained over the deeper-seated part of the heart. Its determination is not important, unless it is to note the encroachment of an emphysematous lung towards the middle line.

Deep cardiac dulness is usually that sought for on percussion of a heart. Its limit is that of the heart in the chest. The more lung tissue in front of the heart, the less absolute is the dulness obtained. With much emphysema it may be impossible to get any dulness at all.

Practical points—***In health.***—There should be no dulness to the *right of the sternum,* to the *left of the left mid-clavicular line,* or *above the third rib* on a line one inch to the left of the sternum in the adult.

In disease.—1. Dulness to the *right of the sternum* means enlarged right auricle or displaced heart. Do not forget the possibility of a congenital development of the heart on the right side.

2. Dulness to the *left of the mid-clavicular line* means dilatation of the left ventricle or great dilatation of the right ventricle, or displacement of the heart to the left.

3. Dulness *below the fifth costal space,* in the region of the nipple line, means hypertrophy of the left ventricle.

In hypertrophy of the right ventricle pulsation of this ventricle can be felt under the costal arch, close to the xiphisternum.

4. Dulness to the *left of the left mid-clavicular line* and *below the fifth interspace* means dilatation and hypertrophy of the left ventricle or of both ventricles.

5. Dulness *above the third rib*, due to cardiac changes, means pericardial effusion. The whole cardiac dulness in this case is pear-shaped, with the base of the pear downwards, and the cardiac impulse, when it is felt, is sometimes well within the outer limits of dulness.

Diminished cardiac dulness means emphysema or pneumothorax.

Extrinsic causes of displacement of the apex-beat.—The whole heart, and consequently the apex-beat, may be displaced by other causes than those which arise within the heart itself or within the pericardium.

The heart may be pushed out of place—

(*a*) By *intrathoracic causes*, such as fluid or air in the pleural cavities, new growths or aneurysm.

(*b*) *Abdominal causes*, ascites, or new growths, which push up the diaphragm. The apex-beat is generally pushed up and out by ascites.

The heart may be pulled out of place by shrinking of the lung, especially in an upward direction, as by phthisis of the upper part of the left lung.

Auscultation.

A wooden stethoscope gives the most delicate results in auscultation, but a binaural, with its flexible rubber tubes, is often more convenient. The use of both may

be cultivated with advantage. Nothing should be allowed to rub against the stethoscope, otherwise very alarming auscultatory phenomena will be heard.

N.B.—*The præcordia are often exquisitely tender in heart disease*, and the stethoscope chest-piece must be applied with this in mind.

Localize the apex-beat. Before using the stethoscope, it is of the utmost importance to find the apex-beat of the heart, or the outermost limit of the heart to the left. Unless this be done, murmurs, especially the crescendo murmur heard only at the apex of the left ventricle as a rule, may be overlooked.

The proper way to use a stethoscope, whether binaural or monaural, and with the patient in or out of bed, is *to place one hand on the patient's shoulder*, and to use the other for moving the stethoscope from place to place. This not only prevents the examiner from adopting the unbusinesslike and objectionable habit of keeping one hand in his pocket, but it also maintains the proper distance-relationship between the examiner and the patient, and facilitates the timing of murmurs by the carotid artery.

III

THE HEART SOUNDS

It is important to bear in mind the anatomy and mechanism of the normal valves in studying the changes in the normal heart sounds which are met with in disease.

THE FIRST SOUND

The first sound is produced by the act of closure of the normal mitral and tricuspid valves (auriculo-ventricular or *a.-v.* valves) by the contraction of the normal ventricles.

No sound is heard whilst the blood is passing through the *a.-v.* valves into the ventricles.

Anatomy of the auriculo-ventricular valves.—

1. The fibrous, inelastic valve curtains, two in the left, or mitral, valve; and three in the right, or tricuspid, valve. From the edges of the curtains run tendinous cords (chordæ tendineæ) to—

2. The musculi papillares, which arise from the muscle of the ventricular wall.

3. Muscular fibres around the orifice of the valves. It used to be taught that these were arranged as circular fibres, but recent observations suggest that in addition to some circularly disposed fibres the spiral muscle

fibres of the ventricle are attached to the fibrous valve ring in such a way that when they contract they help to diminish the valve orifice.

Mechanism of the closure of the auriculo-ventricular valves.—

1. When blood is passing from auricle into ventricle the valve curtains lie against the ventricle wall and offer no impediment to its flow.

2. As the ventricle fills the blood floats the edge of the curtains of the valves into such a position that—

3. The curtains are locked in competent apposition at the very onset of ventricular systole (*v.s.*), when the papillary muscles are said to contract, and before the intraventricular blood-pressure has reached its maximum.

4. At an early phase of *v.s.* the "circular" muscular fibres contract, and so reduce the areas of the orifices of the valves that the curtains, when in apposition, competently close the valve orifices.

5. The force with which the ventricle contracts is determined by the resistance of the arterial circulation, and for this to act with maximum efficiency the valves must be competent. The greater the resistance to its contraction, the more vigorous is the systole of the ventricle, within certain limits.

Causation of the normal first sound.—The first sound of the heart is caused by—

1. *Vibration,* or *tension,* set up in the curtains of the mitral and tricuspid (*a.-v.*) valves by the force of intraventricular blood-pressure which closes the valves. This is the dominant factor in the causation of the first sound.

2. *Muscle vibration,* or *tension,* in the contracting walls of the ventricles.

3. *Arterial tension.* There must be a certain amount of pressure in the arteries to afford that resistance which is necessary for the ventricles to contract with a certain degree of deliberation and force.

There is a considerable degree of *elasticity* in forces 2 and 3.

The valve curtain note is of higher pitch than the muscle note, but the sound cannot be resolved into its component parts by the ear.

No sound is heard whilst blood passes through the normal open valves into the ventricles.

The Mitral sound louder than the Tricuspid sound. —The left side of the heart, though more deeply seated, is the predominant partner in the causation of the first sound, because the blood-pressure which acts in closing and maintaining competent its valves is two or three times greater in the left ventricle and the aorta than in the right ventricle and pulmonary artery. This suggests that the valves of the left side of the heart close with twice or thrice the tension of the valves of the right side.

Location of the apex-beat.—The importance of localizing the apex of the heart as accurately as possible by palpation or percussion, or by both these methods of examination, has been referred to under the section on Auscultation. This point cannot be emphasized too much, for, *unless the stethoscope be placed over the left ventricle, sounds or murmurs produced at the mitral valve may be entirely overlooked.*

When the heart is enlarged and the right ventricle is dilated, the true apex of the heart (the apex of the left ventricle) may not come to the

surface, but by finding the outermost limit of the heart to the left we get as near to it as possible, and reduce possible errors to a minimum.

Characteristics of the first sound.—The normal first sound is dull and deliberate, and is imitated by the sound *ŭbb*, as compared with the higher pitched, shorter sound produced at the semilunar valves, imitated by the sound *ŭp*. To produce this sound, tension in the curtains of the closed *a.-v.* valves and in the contracting muscle of both ventricles, and the blood-pressure in the arteries must be normal

There is some degree of elasticity in the papillary muscles acting on the *a.-v.* valves, like sailors holding the ropes of a flapping sail, which probably prevents sudden, inelastic tension of the curtains, and produces a note of lower pitch than that developed at the rigid inelastic curtains of the semilunar valves, which may, to continue the simile, be compared with a flapping sail fixed to the rigid sides of the ship.

To summarize, then, the first sound is produced by tension of the *a.-v.* valve curtains and of the ventricle muscle of the right and left sides of the heart, and is a *double compound sound*, though it is caused chiefly by the mitral valve and left ventricle.

Time.—It is important to note that the first sound is developed at an *early phase* of *v.s.*, and only occupies the first part of this event.

A first sound can therefore be continued, or *followed, by a murmur produced at a later phase of v.s.* (in a manner to be described later) *through defects at the same valve orifice which allow of regurgitation of blood into the auricle during the completion of v.s.* (systolic murmur).

Distinctness.—The distinctness with which the *normal first sound* of the heart can be heard over the præcordia depends on—

(1) *The thickness of the structures which intervene* between the heart and the stethoscope. Thus, much adipose tissue in the chest wall will diminish the sound, as also will an emphysematous lung pushing forwards over the heart, or pericardial fluid displacing the apex.

(2) The *force of the contracting ventricle.* A forcibly contracting ventricle, as in nervous palpitation, or after exertion, makes the sounds so loud that they can be heard by the subject himself, or by an observer at a short distance from the chest, without the help of any conducting medium.

On the other hand, a *feebly beating* normal heart, as in debility, or syncope, or "fainting," gives rise to a correspondingly feeble first sound.

Modifications of the First Sound.

Departures from the normal condition of the valve curtains, cardiac muscle and blood-pressure which produce the normal first sound, will modify the character of the sound, or produce additional audible signs.

I.—Changes in the valve curtains may modify the sound.—

1. *Weakening or absence of the sound.*—The curtains of the valve may be more or less stiffened by disease so that they can only close slowly or that they cannot close at all. The sound will then be modified, or it will not be heard at all.

2. *Production of murmurs.*—When the curtains are so diseased that they are unable to move and close the

valve, they not only no longer produce a sound, but they may cause the development of a murmur of incompetence. This new sign will be referred to fully later.

3. *Accentuated sounds.*—When thickened by disease, but still able to close more or less competently, the curtains may come together with a snap or markedly accentuated sound. Such an unusually sharp and accentuated first sound is best heard in *a.v.* stenosis.

4. *Perforation of the curtains* through ulceration may lead to the production of a murmur with or without a first sound, according to whether the valve is or is not too much diseased to produce a sound.

II.—**Changes in the muscle of the ventricle** may modify the first sound in two ways:

1. *Thickening*, or *true hypertrophy*, with slower, more deliberate and more forcible contraction of the heart muscle, and a less sudden development of tension in the curtains, as compared with normal, gives rise to a note which is of lower pitch, more lengthy duration, and is dull or muffled, as compared with the normal first sound.

The thickened ventricular wall probably aids further in the production of this dull sound by hindering conduction of the valve tension element of the sound.

2. *Thinning of the wall*, as in *dilatation of the ventricle*, on the other hand, owing to the quicker and less efficient contraction of the heart muscle and more inelastic production of curtain tension, causes a note which is more abrupt and slapping and of higher pitch than the normal sound. The unusually thin walls of the ventricles probably facilitate the hearing of the valve tension element of the sound.

In marked cases of dilatation of the ventricles the sound heard is probably mostly one of valvular tension,

the papillary muscles contributing to this effect by losing their elastic pull on the valve curtains.

3. *Any structural changes* in the walls of the ventricle which (*a*) *diminish the force of contraction*, such as myocarditis, fatty degeneration or infiltration, will diminish the loudness of the first sound; or

(*b*) *Any changes which diminish its elasticity*, such as fibroid degeneration, will accentuate the sound.

III.—**Changes in blood-pressure** also have an effect on the heart sounds. A high pressure, generally associated with some muscle hypertrophy or increased force of heart-beat causes a more vigorous ventricular contraction and a louder sound than a low blood-pressure, which tends to weaken the sounds.

IV.—**An association of two or more** of the above-mentioned factors which modify the first sound of the heart, a condition frequently met with in heart disease, will cause more marked changes in it.

A weak or absent first sound will result from—

1. *Changes in the valve curtains* which interfere with, or do away with altogether, the development of tension in them at the onset of *v.s.* The valve curtains may become so thick and stiff, forming a permanent funnel-shaped passage, that they are immovable, and cannot be closed or thrown into tension. No valve sound is then heard, a *murmur of regurgitation* or *incompetence* taking its place; a murmur *of obstruction* is also generally heard during the passage of blood in the proper direction through the valve.

2. *Feeble action of the heart*, as in debilitated states, after continued fever, or during faintness.

3. *Pericardial effusion,* when there is a fair amount of fluid present.

4. *Diminution of the blood-pressure.*

Accentuation of the first sound.—The term *accentuation* is generally applied to the first sound heard in abnormal conditions, in which, however, the valve curtains are either normal, or not diseased enough to affect their mobility.

1. The tone is clearer, sharper, shorter than normal. Such an accentuated sound is heard with *dilated ventricles,* in which there is a more sudden stretching of the valve curtains owing to the weak, inelastic condition of the papillary muscles. It is also heard when the *blood-pressure is increased.*

2. The dull, prolonged, and thudding note of *ventricular hypertrophy* is much muffled in its conduction to the surface by the thickened ventricular wall.

3. When the thickened, almost cartilaginous valves of *a.-v.* stenosis come together, they produce an abrupt and accentuated sound which is characteristic of this form of valvular disease.

4. When a heart is lying in contact with a stomach distended with air or gas, the sounds may have a peculiar "tympanitic" ring, which disappears when the flatulent distension passes off.

Reduplication of the first sound.—Normally the ventricles contract synchronously, and give rise synchronously to the sounds which blend into what we hear as the single first sound. We often hear, however, the single first sound replaced by two sounds—that is, by reduplication or doubling. An excellent imitation of the single or double first sound can be made by striking on the forehead by two fingers synchronously, and then

with varying interval between the contact of the two fingers with the forehead.

The *distinctness of the double first sound* may vary from a slight degree which has been imitated by the sound *trup* to one in which the two component sounds are quite separate (*ter-up*). These variations can be well imitated with the fingers on the forehead.

The ***explanation*** of a double first sound which is usually given is want of synchronism of ventricular contraction generally due to abnormalities of blood-pressure in the two circulations. If one ventricle has more work to do than in the normal condition of affairs, it is said that it will reach that part of its systolic phase at which the first sound is produced earlier than does the other ventricle, and so produce the double first sound.

This explanation of reduplication of the first sound of the heart is not without objection. To begin with, the blood-pressure in the left ventricle and the aortic circulation is two or three times as great as that in the right ventricle and pulmonary circulation; but, on the other hand, the wall of the left ventricle is correspondingly thicker, and therefore more powerful than that of the right ventricle. If the blood-pressure in one of the circulations is increased, one of two things results:

1. More muscle is developed — compensatory hypertrophy—to deal with this increased pressure, and therefore this ventricle is enabled to start on level terms with the contraction of the other ventricle; or—

2. Dilatation of the ventricle and muscle incapacity results, with dilatation of the valve and consequent regurgitation. This safety-valve action

relieves the increased pressure on the overworked muscle, and again takes away any handicap as between the ventricles.

Physiology teaches us that normally the left side of the heart commences its contraction about one-fiftieth of a second before the right, but this difference is imperceptible to the human ear.

It seems to me that we may have another explanation of the double first sound when it occurs with actual or relative weakness of the papillary muscles of one or both ventricles. The intraventricular blood-pressure is at its highest after the contraction of the papillary muscles, which occurs at the earliest phase of *v.s.;* but whilst the papillary muscles maintain their systolic contraction as long as that of the ventricle persists, in the normal condition of affairs, it seems quite possible in disease of the ventricle muscle, or in abnormal pressure in the ventricles—such as is met with in an increased arterial tension—for the papillary muscles to fail after their initial contraction, but before the systole of the ventricle is completed, and allow the valves to fly back and to be put suddenly into a state of secondary tension by the stretching of the musculi papillares and the chordæ tendineæ. At any rate, it is in conditions which cause increased arterial pressure, and in muscle failure of the ventricle with or without abnormal blood-tension that we get the double first sound.

Clinical significance of double first sounds.—

1. *Functional causes.*—Doubling of the first sound may not have any pathological significance—that is, it may be heard with no organic lesion. It may occur at

the end of full expiration and the beginning of inspiration on deep breathing.

2. *Organic causes.*—A double first sound may be a physical sign of severe disease in which increased arterial tension develops, or in which the heart muscle fails. In this latter case the rhythm of the cardiac cycle is altered, and the first sound follows the second abnormally quickly (see Rhythm of Sounds of the Heart and Bruit de Galop, p. 45).

Doubling of the first sound is *not a special sign* of any form of *valvular disease.*

THE SECOND SOUND

The second sound is produced by the act of closure of the normal aortic and pulmonary valves by the intra-arterial blood-pressure.

No sound is heard whilst blood passes through the normal open semilunar valves into the aorta or pulmonary artery.

Anatomy of the semilunar valves.—The semilunar valves at the aortic and pulmonary orifices consist of fibrous, inelastic valve curtains, three in each valve. These curtains are attached by their bases to the fibrous ring of the valve orifices, which also contains a little elastic tissue.

There is nothing in the semilunar valves to compare with the musculi papillares or the chordæ tendineæ of the *a.-v.* valves.

Mechanism of the closure of the semilunar valves.—

1. Whilst blood is being driven into the aorta and pulmonary artery the curtains of the semilunar valves are closely apposed to the wall of the vessels so as to

offer no resistance to the flow of blood through the valve.

2. The curtains of the valves are locked in competent apposition when the pressure in the large arteries is greater than the driving force of the ventricles.

Causation of the normal second sound.—The second heart sound is caused only by tension, and has no muscle element in it.

No sound is heard whilst blood passes through the normal open valves into the large arteries.

The closure of the semilunar valves is brought about by an elastic force—namely, the contraction of the muscle fibres and the elastic tissue of the walls of the aorta and pulmonary artery. It is important to remember this, because loss of elasticity in the wall of the large arteries through vascular sclerosis modifies the sound.

N.B.—This force, which may be called the systolic contraction of the aorta or pulmonary artery, *is at its maximum in its earliest phase,* and not at a later period, as in the case of ventricular contraction.

> This elastic force gives rise to one act of curtain tension and one sound in each of the two valves. This action may be compared to the single sound which can be made when an inelastic object—*e.g.*, a hammer (the column of blood in the arteries)—is brought suddenly against an inelastic resistance—*e.g.*, an anvil (the valve curtains)—by an elastic force—*e.g.*, the muscles of the arm governed by the will (the elastic structures in the arterial wall). On the other hand, if the inelastic hammer is allowed to fall by its own weight on the inelastic anvil, a double sound of primary and rebound impact will result. The importance of this point

will be seen when we speak of reduplication or doubling of the second sounds.

The normal second sound, then, is a *compound sound*, being caused by the closure of the aortic and pulmonary valves; but as the blood-pressure in the aorta is two or three times as great as that in the pulmonary artery, the aortic valve probably contributes chiefly to its causation. On the other hand, the deeper situation of the aortic valve must be borne in mind.

Any second sound heard over the carotid arteries is aortic in origin.

Characteristics of the second sound.—Normally the second sound has definite characteristics of pitch and duration. It is sharper and shorter than the first sound, being imitated by the sound *ŭp*, as compared with *ūbb* of the first sound, and suggests valve tension only as its causative agent.

Time.—The second sound is developed at the very outset of *v.d.*, and therefore it can be followed by any abnormal sound which may be produced at a defective valve during the remainder of *v.d.* (diastolic murmur).

Distinctness.—The conditions which affect the audibility of the normal first sound—namely, thickness of intervening structure, force of heart-beat—will also affect that of the normal second sound.

Modifications of the Second Sound.

Modifications of the condition of the semilunar valve curtains, of the elasticity of the arterial wall, and of the blood-pressure, will modify the second sound, much as similar changes modify the first sound.

I.—**Changes in the valve Curtains** may modify the sound.—

1. *Weakening or absence of the sound.*—The curtains of the valve may be so stiffened by disease that they cannot close at all or can only close slowly. The sound will then not be produced at all, or it will be modified.

2. *Production of Murmurs.*—When the curtains are so diseased that they are unable to move and close the valve, they not only no longer produce a sound but they may cause the development of a murmur. This new sign will be referred to fully later.

3. *Accentuated sounds.*—With the curtains diseased, but still able to close competently, the sound of closure is more accentuated, and may be followed by a murmur.

4. *Perforation or injury to a curtain* may occur in aortic disease after unusually severe and sudden exertion. Some sound of tension, followed by a murmur of incompetence, is then heard.

II.—**Changes in the wall of the artery.**—

1. *Loss of elasticity* from atheromatous degeneration of the middle coats leads to a more abrupt accentuated sound of tension.

2. When the above change is followed by dilatation of the artery (especially of the aorta), the second sound is often of a peculiar ringing character, which may be the first sign of the condition of the aorta.

III.—**Changes in the blood-pressure** affect the second sound very markedly, accentuating it when it is abnormally high (increased tension), and diminishing it when it is abnormally low.

A **feeble** or **weak second sound** will be heard when

the arterial blood-pressure is abnormally low, and is generally associated with a weak first sound.

The second sound may be *absent* altogether if the valve curtains are so stiffened by disease as to be unable to come together during *v.d.*, or to be thrown into a state of tension sufficient to produce a sound.

Accentuation of the second sound.—*Physiologically*, this occurs at the end of deep inspiration and the beginning of the following expiration, and can be brought on by holding the breath.

In disease it is very common to hear the aortic second sound accentuated when arterial tension is high, especially in Bright's disease, and the pulmonary second sound in congestion of the lesser circulation, especially in mitral regurgitation.

Accentuation of the second sound is often associated with accentuation of the first sound in these conditions.

Ringing Sound.—In one condition in which there is an abnormally large amount of blood just above the valve—aneurysm of the aorta—the second sound has a peculiar accentuated ringing sound, which may be the only auscultatory sign of aneurysm of the first part of the aorta.

Effect of Thickened Curtains on the Sound.—Normally the edges of the cusps of the valves which come into contact on closure of the valve are very fine and uniformly smooth, and no sound of impact is caused from the way in which they are "floated" into apposition before the final locking in competent closure. If the edges of the cusps of the valve be thickened or calcareous whilst the cusps are still capable of coming into competent apposition, an accentuated sound may be produced by the impact of these cusps.

Reduplication of the second sound.—A double second sound is usually said to mean asynchronous closure of the aortic and pulmonary valves, and to be due to a higher blood-pressure in one of the circulations which induces abnormally early closure of the valve of the artery in question.

[A fallacy in this teaching suggests itself at once —namely, that as the pressure in the aorta is normally twice or thrice that in the pulmonary artery, we should get reduplication of the second sound normally.

It is not the amount of pressure in the large arteries, then, that determines the time of closure of the semilunar valves. They close when the pressure in the arteries is greater than that in their corresponding ventricles; so if asynchronism of closure of the semilunar valves is to occur, it must be because one ventricle takes longer to drive blood into its artery than the other, not because there is abnormal pressure in the aorta or pulmonary artery. Thus, a dilated ventricle may be pumping blood into its artery for a longer time than the other ventricle, or a badly filled ventricle (often met with in mitral stenosis) may pump its blood into the aorta sooner than the normal or dilated right ventricle. In both these cases double second sounds would be heard.

It is, however, quite possible for an increase in the blood-pressure of the aorta or pulmonary artery to give rise to a double second sound by causing a rebound of the column of blood which throws the semilunar valves into sound-producing tension, such a rebound being due to a want of elasticity in the

vessel wall which frequently accompanies increased tension. In this case there would be a comparatively inelastic force closing the valve, and with this rebound a double sound would result in the manner described on a previous page (p. 46).]

The **rhythm of the first and second sounds is of very great importance** in the diagnosis of the condition of the heart muscle, and may be referred to here.

In a normal heart beating seventy-five times per minute the whole duration of a cardiac cycle is 0·8 second. Of this time 0·3 is occupied by *v.s.* and 0·5 by *v.d.* As the

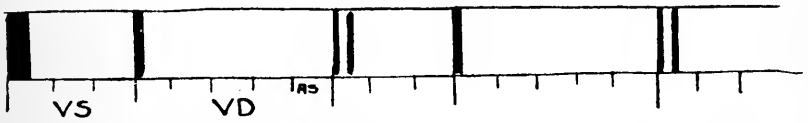

FIG. 4.—RHYTHM OF NORMAL SOUNDS OF A HEART BEATING 75 PER MINUTE.

VS equals three-tenths; VD, five-tenths; auricular systole, one-tenth second. All the figures are thus spaced except Figs. 5 and 6.

first and second sounds occur at the beginning and end respectively of *v.s.*, it follows that there is a longer interval between the second and first than between the first and second sounds in the proportion of 5 to 3. This relative duration of the intervals between the heart sounds is maintained, and can be recognized in hearts

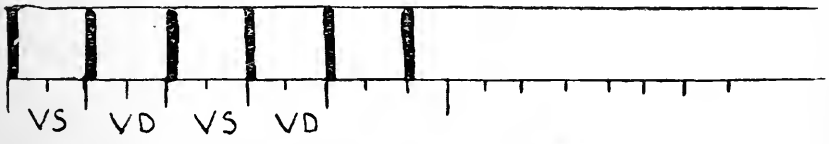

FIG. 5.—TIC-TAC RHYTHM.

Space between first and second and second and first sounds equal. Diastolic rest much shortened.

beating rapidly—120 to 130 per minute. When the

time intervals between the sounds become equal—the so-called "tic-tac" or "embryocardial" rhythm; or when the second sound follows too quickly on the first sound, and there is a proportionately longer pause between the second sound and the following first sound, it is most probable that there is some lesion of the cardiac muscle (see later, p. 47).

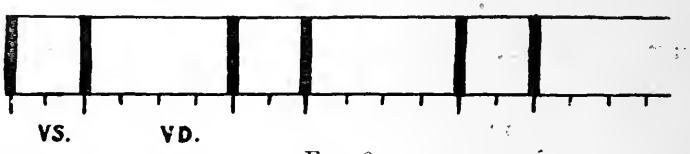

FIG. 6.

Space between second and first sounds too short. Diastole comparatively prolonged.

Cantering rhythm, or Bruit de galop.—In its slighter degrees, reduplication of the first or second sounds of the heart is not readily recognized by the beginner, but familiarity with the peculiar characteristics of the abnormal sound can be obtained by practising, as described previously (p. 36), its imitation by the finger tips on the forehead. The more marked degrees of reduplication are easily recognized, and the only difficulty is to

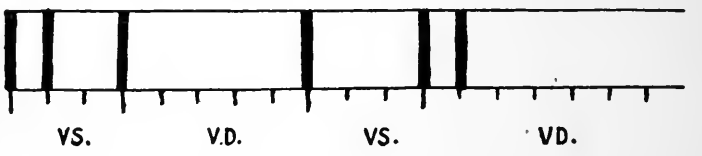

FIG. 7.—BRUIT DE GALOP.

Double first and single second sound, and single first and double second.

determine which of the sounds is doubled—a difficulty which is removed by timing them by the carotid artery. The carotid pulse follows the onset of ventricular systole at an inappreciable interval of only 0·05 second. There-

fore any sounds which are synchronous with the carotid pulse are synchronous with ventricular systole, and any which are not are synchronous with ventricular diastole (see Timing of Murmurs).

When reduplication is marked, and one of the three sounds is accentuated, the rhythm suggests the simile of a cantering or galloping horse; hence the name which is often applied to the condition. It may be phonetically represented by the sounds *lub terup, lub terup,* or *terup tup, terup tup.* Any one of the three sounds may be the most accentuated.

Whatever pathological significance reduplication of the first or second sounds of the heart may have when the sounds maintain their relative distance from each other, the *prognosis is much worse when the first sound,* whether single or double, *follows more closely on the second,* whether single or double, *than it ought to.* This means that the ventricles are not getting their proper amount of diastolic rest, and that we have almost certainly to deal with a *severe affection of the cardiac muscle*—cardiac muscle failure, either temporary or permanent—and effective measures of treatment must be undertaken at once. An invariable symptom in true bruit de galop is dyspnœa on slight exertion.

IV

HEART MURMURS

Hitherto we have been considering conditions in which the valve curtains, even if altered structurally, have been able to meet properly and to competently obliterate the orifice they guard with or without modification of the normal sound produced by the closure of the valve. The valve also has offered no resistance to the passage of blood through it in the normal direction of flow through the heart.

Murmurs.—In disease, however, it frequently happens that the valve is so changed that it either *obstructs* the flow of blood in the normal direction through the heart or allows the blood to *pass back* in the wrong direction (to *regurgitate*), setting up sound-producing vibrations in the blood itself or in the valve curtains, or in both, and giving rise to new or adventitious signs—"murmurs" or "bruits"—instead of, or in addition to, the ordinary sounds, and these are heard over the heart.

Timing of murmurs.—In determining the valve at which a murmur is caused, it is essential to know the phase of the cardiac cycle during which the murmur is produced.

It is of the utmost importance to time all murmurs, for by so doing inexcusable and serious errors in diagnosis

are avoided. It is often impossible to feel any apex-beat, or, when it is possible, we may want to place a stethoscope over it Therefore, the best way to time a murmur is to keep a thumb on the carotid pulse, which follows 0·05 second after the onset of *v.s.*

One-twentieth of a second is inappreciable to the ordinary observer, but the radial pulse is 0·1 second after the carotid pulse, and this is appreciable; for instance, the crescendo murmur, as a rule, is not more than 0·1 second in duration.

We may say again here that any murmur which is

Fig. 8.—Absolute Stenosis of Semilunar Valve through Thickening of Curtains of Valve: Vertical Section.

Fig. 9.—Absolute Stenosis of Mitral Valve through Thickening and Adhesion of Cusps of Valve Curtains: Viewed Transversely.

synchronous with the carotid pulse is *v.s.* in rhythm, and any one not syncronous is *v.d.* in rhythm. The *a.v.* crescendo murmur is an exception to this rule (see p. 79).

Changes in the Valves which lead to the Production of Murmurs.

Broadly speaking, these changes result in the valves becoming obstructive or incompetent.

1. **Valvular obstruction.**—Through thickening of the curtains the lumen of the valve is diminished, causing obstruction to the free passage of blood through it in the normal direction of the circulation (Figs. 8 and 9).

In most of these cases of obstruction there is a greater or less degree of adhesion of the curtains to each other where they arise from the fibrous ring of the valve which correspondingly diminishes the free-way, and causes the condition known as valvular stenosis, and generally, but not always, a murmur of obstruction.

Valvular stenosis may be actual or relative.

Actual stenosis is the term applied when the valve orifice and its fair-way is actually narrowed by disease.

Relative stenosis is the term applied when the *lumen* of the valve *remains normal,* but the *cavity beyond* it,

FIG. 10.—RELATIVE STENOSIS OF SEMILUNAR VALVE, ARTERY BEING DILATED: VALVE ORIFICE NORMAL IN SIZE.

FIG. 11.—DILATATION OF *a.-v.* VALVE RING: NORMAL CURTAINS UNABLE TO MEET ACROSS THE DILATED ORIFICE.

into which the blood passes from the valve, *is abnormally large* or *dilated.* Such a condition is only met with practically in connection with the semilunar valves and the aorta and pulmonary artery beyond them (Fig. 10).

A stenosed valve, actual or relative, *may, but does not always, cause a murmur* during the passage of blood through it *in the normal direction of flow* through the heart.

2. **Valvular incompetence.**—Here the curtains are unable to prevent regurgitation of blood through the valve in the opposite direction to that of the normal

course through the heart, and during this regurgitation a murmur is heard.

Such incompetence may arise in two ways:

(1) *The valve curtains are normal,* but the *orifice is dilated*—that is, the area of the orifice is rendered abnormally great through stretching of the structures which normally limit it. This condition is met with chiefly in stretching of the circular muscular fibres around the orifices of the *a.-v.* valves (Fig. 11).

(2) *The valve curtains are diseased and unable to meet competently* across the orifice of a valve, which, as the result of the disease, is generally of diminished lumen, or actually *stenosed* (see Figs. 8 and 9).

Failure of the circular muscular fibres may be present along with disease of the valve curtains.

Murmurs then can arise through—

(*a*) *Obstruction* to the normal flow of the blood through the heart; or

(*b*) *Incompetence* of a valve to prevent the regurgitation of blood in the reverse direction to the normal flow.

The Physical Causes of Cardiac Murmurs.

There are two chief physical causes of cardiac murmurs —fluid veins and vibration of valve curtains. Changes in the viscosity of the blood also may have some influence.

1. **Fluid veins.**—When a stream of blood is driven through an orifice which is of relatively small area compared with that of the mass of blood into which it passes, sound-producing currents, or eddies of blood, are produced which are more intense when there is a greater disproportion between the moving column and the mass

into which it passes and when the driving force is increased.

Such a method of producing a murmur can be imitated by blowing through the pursed-up lips, the lips remaining rigid.

For the development of a fluid vein it is necessary that some degree of *stenosis*, absolute or relative, should exist.

(*a*) *In Absolute Stenosis* there is an actual narrowing of the valve orifice with the cavity beyond (that is, in relation to the direction of the stream of blood causing the murmur) of normal or abnormal size.

The most typical conditions in which this absolute stenosis is met with are those of mitral and aortic stenosis. In these conditions the valve orifice is more or less narrowed by adhesion of the cusps of the valves, with some thickening of the non-adherent portions of the valves (see Figs. 8, 9, and 12).

Tricuspid and pulmonary stenosis also occur, though not so commonly.

(*b*) *In Relative Stenosis* the valve orifice remains normal in area and the cavity beyond is pathologically dilated. It occurs only at the aortic and pulmonary valves. Theoretically, it might occur at the *a.-v.* valves if the ventricles be much dilated, and so account for rare *a.-v.* diastolic murmurs heard in cardiac dilatation. But in practice relative stenosis is only important at the semilunar valves.

The *fluid vein murmur* is of a smooth, soft, blowing character, and probably not transmitted as widely as when there is valve vibration as well.

Stenosed valves are also often *incompetent as well as obstructive*, when regurgitation of blood through the narrow opening will cause a murmur of fluid-vein origin, and probably also of valvular vibration origin.

It must be remembered that when there is obstruction to the flow of blood through a valve, the cavity of the heart behind the valve (in relation to the normal direction of blood-flow) is often imperfectly emptied, and therefore contains blood for the development of a fluid-vein regurgitant murmur.

It must also be borne in mind that the curtains of a valve which adhere to each other and cause absolute stenosis may be thrown into vibration by the stream of blood and alter the character of the murmur [see next subheading (2)].

2. **Vibration of the valve curtains.**—(1) *Obstruction murmurs* may be produced by the blood throwing the rough, stiffened curtains into sound-producing vibration as it passes over them in its direct passage through the heart.

(2) The *regurgitation of blood* over similarly diseased curtains throws them into vibration and produces a murmur.

The sound-producing vibration of the valve thus caused is analogous to that of a flag fluttering in the wind.

Vibration murmurs are of a rougher, more vibratory character than those produced by a fluid vein.

Not uncommonly *a tag of fibrous tissue* attached to a valve is thrown into rapid vibration by the blood-stream, and produces a musical murmur without causing any symptoms of heart disease; and it may be pointed out here that *the loudness of any cardiac murmur is not necessarily an indication of the severity of the lesion*, the "bark" of a murmur often being worse than its "bite."

N.B.—It is the condition of the cardiac muscle, not of the valve, that determines prognosis.

Thrills.—In marked cases we can feel the vibration murmur as a "thrill," typical instances of which are the thrills met with frequently in mitral and aortic stenosis.

3. **Murmurs due to changes in the blood—Hæmic murmurs.**—In marked cases of anæmia a systolic murmur, usually called a "hæmic murmur," is frequently heard over the heart, with its point of maximum intensity generally in the pulmonary valve region.

It used to be thought that the actual blood change—namely, the diminished number of red corpuscles per c.m., especially if the total blood volume be abnormally great, as in chlorosis—gave rise to the sound. There is, however, no satisfactory proof that endocardial murmurs can arise as a consequence of diminished viscosity. They are probably of valvular origin, and arise in one of two ways:

(1) *Regurgitation through the mitral valve,* which has been rendered incompetent by the circular muscular fibres of the valve partaking in the condition of ventricular weakness not uncommonly met with in severe anæmia; in other words, mitral incompetence through valvular dilatation, the curtains remaining normal. This murmur is best heard at the apex of the heart.

(2) *In relative stenosis* of the pulmonary valve, which is brought about by dilatation of the artery in its first part immediately beyond the valve, the valve orifice remaining normal. Such a condition of dilatation can be made out in the post-mortem room. This murmur is best heard over the region of the pulmonary valve.

N.B.—Murmurs caused in the above-mentioned ways are all diminuendo in character, dying away with the force which produces them.

Changes in the Different Valves which Give Rise to Murmurs.

It will make things clearer if the changes in the different valves which give rise to murmurs be now considered.

1. **Auriculo-ventricular valves**—(*a*) ***Actual stenosis.*** —This results from thickening and adhesion of the valve curtains, which consequently oppose the passage of blood from auricle to ventricle; and if the forces moving the blood, especially that of auricular systole, are strong enough, which is not always the case, a diastolic murmur will be heard. Sometimes vibrations are thus produced

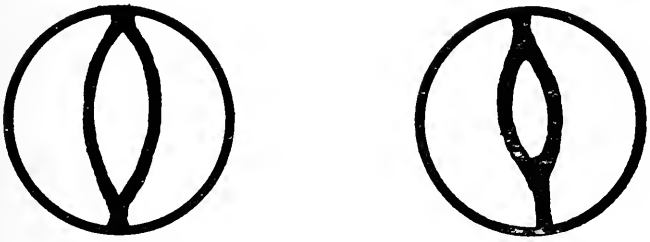

Fig 12.—Two Degrees of Adhesion of Mitral Curtains causing Stenosis.

in the valve curtains, and these can be palpated at the apex as a diastolic thrill.

The degree of stenosis varies, and may be so extreme as to narrow the orifice of the valve from an area admitting three finger-tips to one scarcely admitting the tip of the little finger only—the so-called button-hole orifice (see Figs. 9 and 12).

The musculi papillares and chordæ tendineæ participate more or less in this thickening and rigidity, and may in consequence fix the edges of the valve curtains in a funnel-shaped structure, and prevent the normal movements and co-aptation of the curtains during *v.s.*

The process which causes this sclerosis is generally of a chronic, slowly progressing nature, mostly set up in childhood, and often without symptoms to attract attention.

As a result of these changes the valve, in addition to being obstructive, is very often ***incompetent,*** allows of *regurgitation,* and creates a systolic murmur during *v.s.*

The presence of *vegetations on the valves* without adhesion, which occurs in acute endocarditis, will obstruct the flow of blood from the auricle to the ventricle, and may cause a diastolic murmur. They generally

FIG. 13.—VEGETATIONS ON MITRAL VALVE (CLOSED) WHICH PREVENT COMPETENT CLOSURE, AND WHICH OBSTRUCT THE FLOW OF BLOOD THROUGH THE OPEN VALVE.

There is no adhesion of the curtains and no true stenosis.

make the valve incompetent, and give rise to a systolic murmur of regurgitation.

Confusion of terms.—The terms *obstruction* and *stenosis* (referring here to actual, not relative stenosis) are often rather loosely used as meaning the same condition of a valve. Whilst all forms of absolute stenosis obstruct the flow of blood through the valve, a condition may occur in which, through marked roughening or the presence of vegetations on the curtains without adhesion of the curtains, the normally unhindered flow of blood through a valve may be obstructed and an *a.-v.* diastolic murmur result (see Fig. 13).

The latter condition would be really one of obstruction, but hardly one of absolute stenosis, this term generally implying absolute narrowing of the blood-way through the valve caused by *adhesion of the curtains* or *nodular excrescences* stiffening the valves

(*b*) ***Valvular dilatation.***—Here the deficient factor in the closure of the valve is the inability of the muscular fibres round the valve ring to reduce the orifice of the valve during *v.s.* to an area which the curtains, remaining normal, can competently close. In consequence, a systolic murmur of regurgitation is caused during *v.s.* This condition results from cardiac muscle failure (see Fig. 11).

The musculi papillares may also be unable to contract and hold the edges of the curtains down. In this case the curtains bulge into the auricle and probably become incompetent.

(*a*) *and* (*b*).—As the result of acute endocarditis the valve curtains may become affected with vegetations, which impede the flow of blood from auricle to ventricle and prevent competent closure of the valve during ventricular systole; and at the same time the muscle element of the valve may, as a result of coincident myocarditis, fail and cause enlargement of the ring of the valve. In such a condition a systolic murmur is almost certain to be heard, and possibly a diastolic murmur also.

Cardiac dilatation with no murmur.—It must be noted that cardiac muscle failure, leading to dilatation of the heart, may occur, and yet no systolic murmur of valvular incompetence be heard. In these cases the circular and spiral muscular fibres of the valve ring

must be more resistant to stress and strain than the rest of the ventricular muscle, and are not stretched at all, or only to a slight degree which the valve curtains can make good and so prevent regurgitation. We shall then have no systolic murmur of regurgitation, only an accentuated or reduplicated first sound.

Ulcerative erosion of curtains.—Valvular incompetence may result from ulcerative endocarditis eroding holes through the valve curtains and thus allowing of regurgitation; but such a condition would be impossible to recognize clinically. It would probably occur with changes in the edges of the valve curtains, which would themselves create incompetence.

2. **Semilunar valves.**—The changes which lead to valvular murmurs at the aortic and pulmonary orifices are practically the same as those above described at the *a.-v.* valves, with the difference that there are no musculi papillares and practically no muscular or elastic element in the valve ring. The rhythm of the murmurs is, however, different. Thus, at a stenosed semilunar valve the obstructive murmur is systolic in rhythm and the regurgitant murmur diastolic, whilst at a stenosed auriculo-ventricular orifice the obstructive murmur is diastolic and the regurgitant systolic.

The aortic obstructive murmur, being always caused by the great force of ventricular systole, is much more regularly heard than the mitral obstructive murmur. The obstructive aortic murmur may be accompanied by a *thrill* palpable over the aortic area.

N.B.—A *systolic murmur* at the auriculo-ventricular valves, or a *diastolic murmur* at the semilunar valves, *per se*, only means *incompetence* of these valves. *The*

rhythm of these murmurs gives no information whatever as to the cause of the incompetence—that is, whether this is due to stenosis or dilatation. The means of determining this point will be referred to later.

The Crescendo murmur.—So far we have dealt with murmurs which, however they may be caused, have a common feature—namely, they are *diminuendo* in character, and die away with the force which creates them.

There is, however, another murmur which, instead of dying away, *increases in force* until it is abruptly cut short at its maximum intensity by an accentuated first sound. It is therefore a *crescendo* murmur. In addition to this, it has another essential characteristic—namely, it *rises in pitch* progressively with its increase in vigour, and is at its highest pitch and maximum force when it is abruptly terminated by the accentuated first sound.

This type of murmur is only produced at a stenosed auriculo-ventricular valve, and almost always at the mitral valve. A tricuspid crescendo murmur is very rare. A crescendo murmur, then, has three essential features:

(1) *Increasing force*, (2) *Rising pitch*, and (3) *Abrupt termination by an accentuated first sound at its maximum of force and highest pitch.*

Such a murmur, except the terminal abrupt first sound, can be imitated closely by blowing, with increasing force, through the lips pursed up to produce a low-pitched whistling sound, and closing the lips whilst doing this. In this way the sound represented by *oo-ip* is created. It can also be imitated by blowing through a piece of drainage tubing and obliterating the lumen of the tube with the fingers or teeth whilst doing so. The rising pitch characteristic of the murmur can only be caused

at an orifice which *is being progressively closed during the production of the murmur.*

Causation of the Crescendo murmur.—It is usually taught that this crescendo murmur is caused immediately before the onset of *v.s.* by systole of the auricle, and therefore it is called a *presystolic* or *auricular systolic* murmur. The auricle is supposed to contract with a crescendo force, and to terminate its contraction abruptly at its maximum of effort. During this contraction blood is forced through the stenosed mitral valve and produces the murmur. How the rising pitch

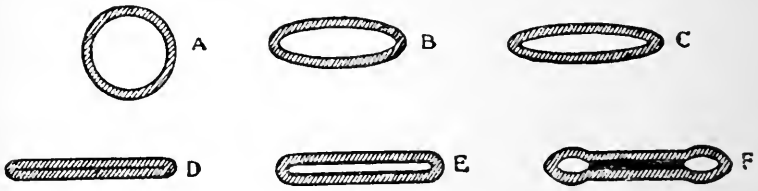

FIG. 14.

A, B, C, D, Progressive stages in the obliteration of the lumen of a tube in which is being produced an imitation crescendo murmur; E, F, condition of the lumen of the tube when a systolic murmur continues the crescendo murmur.

characteristic of the murmur is imparted to it during *a.s.* has never been explained. This, as I have said, must be produced at an orifice which progressively closes whilst a murmur is being produced at it; and how the auricle can at one and the same time keep open and yet progressively close the stenosed valve by driving blood through it has never been explained.

The Crescendo murmur is early ventricular systolic in rhythm. The *a.-v.* crescendo murmur must, in my opinion, be produced between the onset of *v.s.* and the carotid pulse; that is, be *early v.s. or presphygmic in rhythm.*

The accentuated first sound which terminates the crescendo murmur is probably caused by the forcible coaptation of the thickened margins of the valve, and must be produced at a later phase of *v.s.* than the normal first sound. This view of the early ventricular or presphygmic rhythm of the *a.-v.* crescendo murmur, and the lateness of the first sound which terminates it, has been supported by independent observers with evidence obtained by accurate timing of the events of the cardiac cycle in mitral stenosis with instruments recording synchronously tracings of the carotid pulse and of the heart sounds.

Semilunar valve crescendo murmur.—Theoretically, a crescendo murmur may be caused at a diseased semilunar valve, with curtains which are not as mobile as the normal ones, but require some greater force than the cessation of the *vis a tergo* of ventricular systole to close them. This might be developed at a later phase of ventricular diastole than that at which the normal semilunar valves close, by "systole" of the artery, and whilst the valves were being closed blood would regurgitate through their gradually narrowing orifice and create a murmur of rising pitch. It is very probable that the short aortic diastolic "whiffs" are caused in this way.

Murmurs accompanying heart sounds produced at the same phase of the ventricular cycle.—Sometimes a murmur is accompanied by a heart sound produced during the same phase of the ventricular cycle. Why this should be so is not always clear.

It is easy to understand how a first sound produced at the *a.-v.* valves can accompany a systolic murmur produced at the semilunar valves, or if the *a.-v.* valves give

rise to a diastolic murmur, and the semilunar valves are normal, a second sound will be heard with the murmur.

Difficulties arise, however, when both the sound and murmur are produced at one of the pairs of *a.-v.* or semilunar valves. Such an occurrence may be due—

1. To the mitral (or the aortic) valve being so affected as to create a murmur of regurgitation but no sound, the tricuspid (or the pulmonary) valve curtains respectively remaining normal and producing an audible sound; or *vice versa* to the tricuspid (or pulmonary) valve allowing regurgitation, the mitral (or aortic) valve remaining normal and producing a sound which is audible through the murmur.

2. To the curtains of one affected valve being in such a condition that they can close with a sound and yet not be sufficiently competent to prevent some regurgitation and the development of a murmur after the sound. This condition may be met with in incompetence from stenosis or dilatation. When the murmur and sound arise at the same valve during the same phase of the ventricular cycle the murmur distinctly follows the sound. There is one exception to this, namely, the crescendo murmur which precedes the first sound, both murmur and sound being caused by *v.s.* (p. 60).

Absence of sounds with apparently normal right heart valves.—It not uncommonly happens that where there is apparently disease of only the mitral or the aortic valves, and not of the right side valves, the murmur produced at the diseased valve is often unaccompanied by any normal sound which ought to have been produced at the corresponding healthy valve. Thus in cases with incompetence of an abnormal mitral valve, and an apparently normal tricuspid valve, a systolic murmur of

mitral regurgitation may be heard, with no tricuspid first sound. Or, again, in aortic incompetence, and apparently a normal pulmonary valve, a diastolic aortic murmur is not infrequently heard with no pulmonary second sound.

These facts may mean either that the tricuspid or the pulmonary valve is unable to produce a first sound of its own, which is contrary to common belief, or that the first and second sounds produced at the normal right side of the heart have not sufficient intensity to be heard through a murmur.

Incompetence through dilatation of a valve.—It is interesting to consider why, if the curtains of an *a.-v.* valve are normal, but through dilatation of the orifice a murmur is produced, there is not always a *sound of tension also.* There often is, but not uncommonly it happens that a soft systolic murmur of muscle-failure incompetence is heard with no first sound. This may be explained in one of several ways:

1. In a normal heart the *a.-v.* curtains are "*floated*" up into apposition by the filling of the ventricle; then—

2. At the very onset of *v.s.* they are competently locked, and this provides the resistance for the "explosive" contraction of the ventricle, which throws the curtains into sound-producing tension, and also causes a note of muscle contraction.

3. If the valve curtains are unable to meet across a dilated orifice they cannot be competently locked at all by *v.s.*, but allow blood to slip away and so prevent the necessary resistance for the normal explosive contraction of the ventricle and its sound-producing (curtain and muscle tension) results. The more gradual tension of the curtain and muscle fails to produce a first sound, a murmur taking its place. In support of this contention

is the fact that a systolic murmur sometimes grows in intensity *pari passu* with the abnormally slow development of the maximum of *v.s.*

Influence of mobility of curtains on sounds.—(*a*) *Mitral and tricuspid valves.*—It does not follow, then, that when a systolic murmur of regurgitation is heard the absence of a first sound denotes loss of mobility of the mitral or tricuspid (*a.-v.*) valve curtains.

(*b*) *Aortic and pulmonary valves.*—Absence of a second sound when an aortic or pulmonary murmur of regurgitation is heard does mean almost certainly loss of mobility (atheroma) of the valve curtains. This is probably explained by the fact that the normal semilunar valve curtains are thrown more suddenly into tension by the pressure in the large arteries, which is at its maximum at its onset, than are the *a.-v.* valves by the slower-acting muscle of the ventricles. A leak in the semilunar valve will, then, not have the same effect on the development of sound-producing tension in them as will a leak in the *a.-v.* valves.

The presence or absence of a second sound with an aortic (or pulmonary) incompetent murmur, therefore, gives more information as to the mobility of the valve curtains than is the case with a similar condition at the *a.-v.* valve.

V

AREAS OF AUDIBILITY OF HEART MURMURS

Situation of the valves.—Broadly speaking, the murmurs produced at the various valves of the heart are

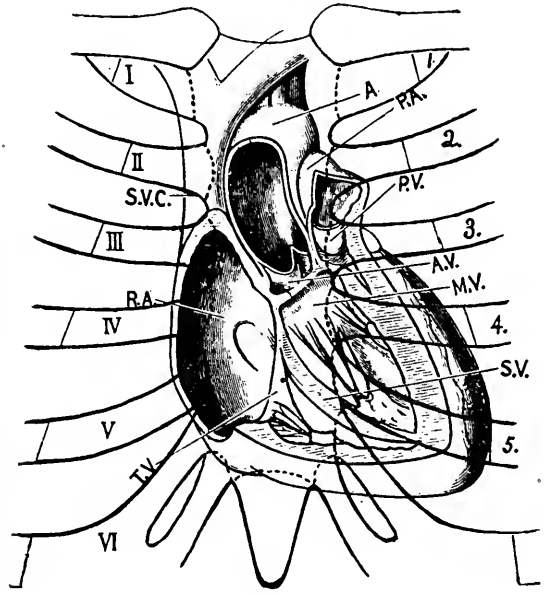

FIG. 15.

A., Aorta; *A.V.*, aortic valve; *M.*, mitral valve; *P.A.*, pulmonary artery; *P.V.*, pulmonary valve; *R.A.*, right auricle; *S.V.*, septum ventriculorum; *S.V.C.*, superior vena cava; *T.V.*, tricuspid valve. (Cunningham.)

heard best at those parts of the anterior chest-wall which are named after the individual valves. These

areas are not all directly over the valves, the situation of which may be stated accurately enough for all practical purposes to be as follows: Pulmonary valve at the level of the third left costal cartilage; aortic valve, third interspace; mitral valve, fourth costal cartilage; and tricuspid valve, fourth interspace, and in a line running obliquely downwards from the third left cartilage, where it joins the sternum, to the midsternal line at the level of the sixth rib.

Mitral area.—Murmurs produced at the mitral valve are best heard at the apex of the left ventricle—*i.e.*, the apex-beat in the fifth interspace $\frac{3}{4}$ inch internal to the mid-clavicular line, or $3\frac{1}{2}$ inches from the midsternal line.

If the heart is enlarged or displaced, the apex must be located as accurately as possible in its new situation by the methods described previously.

The danger of using the nipple as a landmark must be remembered. Thus it may be out of place in relation to the midsternal line through abnormalities in the shape of the chest, and in women, naturally, it is generally no guide at all.

Tricuspid area.—Tricuspid murmurs are heard best at, and to the left of, the lowest part of the body of the sternum.

Aortic area.—The area at which experience shows aortic *systolic* murmurs to be generally best heard is that of the first intercostal space against the sternum, behind the second right costal cartilage at its junction with the sternum, or on the sternum at this level. This is where the arch of the aorta comes nearest to the anterior chest-wall, and sounds produced at the aortic orifice during systole of the ventricle are conducted along the aorta.

Diastolic aortic murmurs, however, may be inaudible here, and are generally heard best down the left margin of the sternum or over the tricuspid area. They may be audible at any small area of the præcordia only; so an aortic diastolic murmur, when suspected, must be sought for carefully, square inch by square inch, all over the chest anterior to both ventricles.

Pulmonary area.—The pulmonary area is situated at the junction of the third left costal cartilage and sternum, or in the third left intercostal space against the sternum.

Whilst the above-mentioned principles relating to the area of audibility of murmurs produced at the different valves hold good in most cases, it must be remembered that *a murmur produced at any one of the orifices, if intense enough, may be heard all over the præcordium* (see Section VI.).

Effect of Change from the Upright to the Lying Down Position on Murmurs.

It is important to note that some organic murmurs, especially mitral systolic murmurs, *are most marked when the patient is lying down.* They may, indeed, only be heard when he is in this position, so in doubtful cases examination in the erect posture only is not conclusive.

VI

CONDUCTION AND TRANSMISSION OF MURMURS

Conduction means the carrying of heart sounds and murmurs from their seat of origin to other parts of the heart (or præcordia) and into the large vessels by means of the heart muscle or the blood.

Transmission means the carrying of the sounds and murmurs beyond these limits by means not thoroughly understood.

Conduction.—***A murmur is conducted best in the direction of the flow of blood which causes it.*** We can get fairly close with our stethoscopes to the direction of the flow of blood through the tricuspid, pulmonary and aortic valves.

The tricuspid and pulmonary valves, being on the right side of the heart, which forms the anterior and most superficial part of the organ as it lies in the chest, are nearer to the anterior portion of the chest than are the mitral and aortic valves.

1. **Tricuspid murmurs.**—A *systolic* murmur ought to be conducted towards the right of the sternum, the right auricle, especially when dilated, extending well beyond

the right border of the sternum. It is heard over the præcordia anterior to the right ventricle.

A *diastolic* murmur is conducted towards the apex of the right ventricle.

Both murmurs are difficult to distinguish from mitral murmurs.

2. **Pulmonary murmurs.**—The pulmonary artery at the base of the right ventricle is nearer the anterior wall of the chest than the aorta, but its divisions are deeply seated within the chest.

Systolic pulmonary murmurs are audible to the left of the sternum in the second intercostal space.

A *diastolic* pulmonary murmur is rare, but, when heard, may be conducted down the sternum or over the right ventricle.

Aortic murmurs.—*Systolic* murmurs are conducted upwards into the large arteries, subclavian and carotid, and therefore these murmurs are conducted to more accessible regions than any other heart murmur.

When being sought for over the vessels in the neck, care must be taken to avoid pressure on the artery, otherwise a murmur will be produced from the pressure itself.

They may be easily heard over the apex-beat, though less distinctly there, as a rule, than at the aortic area. When traced from the aortic cartilage to the apex-beat over the præcordia they gradually decrease in loudness.

Diastolic murmurs will be conducted back into the left ventricle, and, as said before, may be audible anywhere over that part of the chest-wall lying in front of both the ventricles. Their usual line of conduction is down the left edge of the sternum below the level of

the aortic area, and *they may be heard at the tricuspid region only.*

They may also be heard over the sternum towards the pulmonary area.

Mitral murmurs.—*Systolic* murmurs should be conducted towards the sternum and left auricle, but the left side of the heart is too deeply situated for their conduction to be traced. They may be transmitted round through the axilla to the back, and be audible at the lowest part of the left scapula.

Diastolic murmurs are best heard at the apex of the left ventricle, which is in the direction of the flow of blood causing them. They are scarcely ever transmitted into the axillary region.

Crescendo murmurs are audible, as a rule, only over the apex-beat, but they are occasionally conducted more or less widely over the præcordia. As a rule, however, the terminal portion or accentuated first sound only is heard away from the apex or at the back.

Transmission.—The conditions which favour the transmission of cardiac murmurs are not fully understood; but I think that there are certain factors predisposing to a wider area of audibility.

1. *Condition of chest-wall.*—A thin chest-wall, with no fat, in close apposition to a heart without intervening lung, will tend to favour transmission. Thus a largely dilated heart in a child will have its murmurs transmitted more widely than if the same heart were in an adult with thicker or more rigid chest-wall and more intervening lung. The *ribs* may have something to do with transmission of mitral murmurs, especially if one be immediately in contact with the apex of the heart.

2. The *nature of the murmur*—that is, if produced by *vibration of the valve*—will have some effect. In my opinion, such a murmur will be conducted and transmitted more widely than one produced by the small column of blood passing into a large chamber (fluid vein).

Thus a systolic mitral murmur is likely to be transmitted further through the axillary region if it is produced by regurgitation through valve curtains distorted, thickened, and made nodular by chronic endocarditis, and capable of being thrown into vibration by the blood-stream, than if the regurgitation is through a dilated orifice with normal valve curtains, such as we meet with in cardiac muscle failure, or through a rigid thick cartilaginous funnel, such as is met with sometimes in mitral stenosis.

But *loud, vibratory murmurs are not always conducted or transmitted the farthest.* Thus, I have several times noticed a soft systolic murmur produced at the mitral or aortic orifice which was audible all over the chest, and even down over the lumbar region below the ribs. Several of these cases were malignant endocarditis, with involvement, especially, of the aortic cusp of the mitral valve.

Whilst murmurs produced at any of the valves may, if intense enough, be transmitted in the opposite direction to their path of conduction, those produced during systole at the aortic and mitral valves are the murmurs most commonly transmitted. An aortic systolic murmur is frequently transmitted to the apex of the heart, whilst a mitral systolic murmur is often transmitted to the axillary region.

VII

SPECIAL CHARACTERISTICS OF THE DIFFERENT VALVULAR MURMURS

Mitral Murmurs.

Lesions of the mitral valve are the commonest of all valvular lesions.

Mitral systolic murmurs vary very much in character according to the condition of the curtains of the valve at which regurgitation takes place. If they can be thrown into vibration by the regurgitant stream of blood the murmurs are more intense, harder, rougher, and more penetrating than when the leak is between normal curtains which are incompetent through dilatation of the valve orifice, when they are softer, more "blowing," and have less carrying power. A projecting tag of fibrous tissue may give rise to a loud "musical" murmur.

The murmur is of *uniform pitch*—that is, it arises at a leak which is constant (in relation to each *v.s.*) in its area whilst the murmur is being produced. In intensity it fades with the cessation of *v.s.*—that is, it is *decrescendo*. The mitral crescendo murmur has characteristics peculiar to itself.

In time it is synchronous with the throb in the carotid artery. The murmur may or may not be accompanied by a first sound. In either case, if the murmur be not transmitted round the back, the first sound will often be heard where the murmur is inaudible. (Figs. 16 and 17.)

Best heard at the apex-beat, a mitral systolic murmur may be *conducted* with diminishing intensity more or less widely over the præcordia.

The murmur may be *transmitted* to varying extent, according to its nature and causation, through the axillary regions to the angle of the left scapula.

FIG. 16.—FIRST SOUND, SYSTOLIC MURMUR; DOUBLE SECOND, DIASTOLIC MURMUR, WITH A SOUND-FREE INTERVAL BETWEEN DOUBLE SECOND SOUND AND THE MURMUR.

Significance.—Many students think a mitral systolic murmur indicates mitral stenosis, but it *only means regurgitation through a defective valve.* It means this definitely; but the actual rhythm of the murmur gives no information as to the condition of the valve which allows of leakage. How this is determined must now be considered.

Condition of the mitral valve at which regurgitation takes place.—In investigating the condition of an incompetent mitral valve the medical history of the patient is of the greatest importance. Causes of cardiac muscle failure such as anæmia, alcohol, increased arterial tension, diphtheria, etc., likely to produce mitral dilatation, must be sought for. In the absence of such a cause of

incompetence, clinical experience and pathological findings have shown that *a mitral systolic murmur in an adult who has had endocarditis in childhood means regurgitation through a stenosed and incompetent valve* in the great majority of cases.

Rheumatism in childhood.—The indication of endocarditis in childhood is a history of acute rheumatism, chorea, "growing pains," or scarlet fever, and careful inquiry will show that, where mitral stenosis exists in adults, nearly every one of the patients will give such a history.

Cases do, however, occur in which there can be

FIG. 17.—SYSTOLIC MURMUR WITH NO FIRST SOUND, ACCENTUATED SECOND (REDUPLICATED IN SECOND CYCLE).

obtained no history of any aches or pains which generally accompany rheumatism with endocarditis, but the question about "growing pains" must always be put. It is astonishing how often an adult of fifty, with mitral disease, will deny having had rheumatism in any form, but will remember distinctly severe pains as a child, which were called "growing pains." They may have been bad enough to interfere with school or work, but they were not called rheumatic either by the doctor or by the parents.

It must be remembered that *in infancy the tendency in acute rheumatism is for the joints to be less involved, for there to be less pain and more endocardial*

trouble, than in older ages, in which the opposite conditions tend to prevail. There may even be no trivial aches or pains, and the sole indication of an illness in which the slowly progressing valvulitis is set up is entirely confined to the heart, with the disease so insidious and slow in development that even breathlessness and debility are not prominent. It is probable, however, that in nearly all these painless endocarditis cases there is at least some debility, if it only be recognized.

A severe general affection of the heart, endocarditis, myocarditis and pericarditis may also be set up and lead to changes which can not be made good, and which cause death at the time or in a few years.

Rheumatic Nodules are sometimes, but not commonly, seen in children suffering from acute rheumatism. They are almost always associated with serious heart muscle complications, and generally prognose a fatal termination before long.

The absence of a "rheumatic" history need not therefore exclude the diagnosis of mitral stenosis in an adult in whom the regurgitation is found.

The reason why the diagnosis of mitral stenosis in an adult who has had rheumatism in childhood can be made is that the simple valvulitis set up by the endocarditis then is a very slowly progressing disease and only causes cardiac symptoms after some years, whilst the endocarditis with myocarditis and pericarditis which not uncommonly complicates rheumatism in childhood and causes dilatation of the valve orifice through severe muscle failure and adherent pericardium, ends fatally at the time or before adult life is reached.

Further indications of the condition of the incompetent mitral valve.—Apart from the above evidence as to the condition of the valve at which regurgitation is taking place, clinical experience and pathological findings show that mitral regurgitant murmurs which result from *disease of the valve curtains* are audible *more widely* than those which result from *cardiac muscle failure dilatation of the valve ring, the curtains remaining normal.*

This is probably because valve vibration murmurs are transmitted farther than those of fluid-vein origin, and are heard in the axilla whilst the latter are not.

If an incompetent stenosed valve be so stiff and cartilaginous that its curtains cannot be thrown into vibration by a regurgitant stream of blood, the smooth murmur so

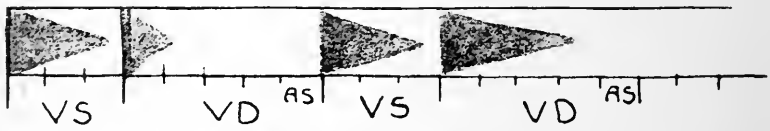

FIG. 18.—SYSTOLIC MURMUR, SECOND SOUND CONTINUED BY AORTIC DIASTOLIC; SYSTOLIC AND DIASTOLIC MURMURS WITH NO SOUNDS.

produced will not be propagated towards the axilla farther than one of ordinary fluid-vein production.

Cardiac muscle failure regurgitant murmurs, as has been stated before, are not transmitted into the axilla like those of curtain disease.

The prognosis of mitral systolic murmurs depends entirely on the condition of the cardiac muscle; the less this is affected, the better the prognosis. With a regular, slow pulse the prognosis may be quite favourable; it is bad with a quick, irregular pulse.

As it has been said on a previous page, a loud murmur does not necessarily indicate serious disease of any of the cardiac valves.

Mitral diastolic murmurs.—These are best heard at the apex-beat, and are not transmitted towards the axilla. They may be heard towards the tricuspid region.

Time.—They almost always distinctly follow a second sound, which is frequently reduplicated, *a sound-free interval occurring between the sound and the murmur.* This is an important point in distinguishing them from aortic diastolic murmurs with which often no second sound is heard, or which follow immediately and uninterruptedly on a second sound, if one be present (see Figs. 16 and 18).

The reason why mitral diastolic murmurs generally follow *at a later phase* of *v.d.* than aortic diastolic murmurs is that the conditions which cause them are later in developing than with the aortic murmur. Thus the main forces which move the blood from auricle to ventricle, namely, active ventricular relaxation or diastole and auricular systole, are at their minimum at the beginning of ventricular diastole, whereas those driving blood back through an incompetent aortic valve into the ventricle (elasticity of the aorta) are at their maximum then.

The diastolic murmur varies from soft, blowing, to hard, thrill-producing characters, and is decrescendo in force.

Cause.—Mitral diastolic murmurs are almost invariably due to obstruction at the stenosed valve to the flow of blood from auricle to ventricle during diastole of the ventricle, and often can be palpated as a *thrill.*

The force moving the blood through the valve is that of ventricular aspiration with auricular systole helping at the end of ventricular diastole when the auricle is strong enough to contract.

A diastolic murmur does not occur in every case of stenosis, probably because the contraction of the auricular wall, which aids in its production, is often very feeble or entirely absent in mitral stenosis.

A few cases have been described in which a diastolic murmur was produced at a dilated mitral valve. An explanation of them is difficult to find.

The Prognosis of mitral diastolic murmurs is not good, as their presence indicates some definite obstruction to the flow of blood through the heart which may lead to auricular muscle failure and pulmonary and, finally systemic venous congestion.

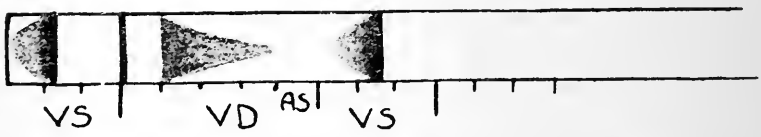

FIG. 19.—CRESCENDO MURMUR (EARLY *v.s.*) ACCENTUATED FIRST SOUND (DELAYED), ACCENTUATED SECOND, PAUSE, DIASTOLIC MURMUR.

The Mitral crescendo murmur is characterized by rising pitch, increasing force, and abrupt termination with accentuated first sound at its highest pitch and maximum force.

It has been argued on a previous page that, judging from its characteristic of *rising pitch*, this murmur must arise at an orifice which closes whilst it is being produced. The sound *oo-ip*, produced at the lips only, demonstrates this point clearly.

The mitral valve when stenosed is often so diseased that it cannot close at all during ventricular systole, when an ordinary systolic murmur of uniform pitch and decrescendo force is heard; or it can only be closed at a later phase of ventricular systole when a sufficient

amount of power has been developed to force together the stiffened rim of the valve. Whilst this force is gathering, blood regurgitates through the open valve and produces a systolic murmur, to which a rising pitch character is imparted by the closing of the valve.

With the gathering force which closes the valve the murmur *increases in vigour* or becomes crescendo in force.

The *accentuated first sound* is caused by forcible co-aptation of the thickened margins of the valve, and has been shown to arise at an abnormally late phase of *v.s.*

Time.—The *crescendo murmur, therefore, is early v.s. or presphygmic in rhythm,* and occurs *immediately before the carotid pulse.*

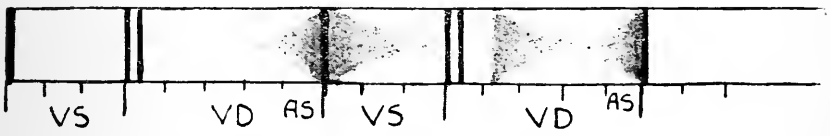

FIG. 20.—ACCENTUATED FIRST AND DOUBLE SECOND SOUNDS: WITH CRESCENDO MURMUR OF SO-CALLED PRE-SYSTOLIC RHYTHM, THAT IS, AURICULAR SYSTOLIC; SYSTOLIC MURMUR; DOUBLE SECOND, MITRAL DIASTOLIC APPARENTLY CONTINUOUS WITH FOLLOWING PRE-SYSTOLIC MURMUR.

The crescendo murmur varies in type from a soft "oo-ip" sound to one of a rougher valve vibration character.

It is usually taught that this murmur is *late diastolic, auricular systolic,* or *pre-systolic* in rhythm (Fig. 20), but the manner of its causation, in my opinion, negatives this.

Location.—Crescendo murmurs are usually *only heard at the apex of the heart,* and *this must be carefully located* and the stethoscope applied *exactly over it.* They are

rarely transmitted into the axilla, possibly because the murmur is often really of a fluid-vein type and has not wide carrying powers. The accentuated first sound is generally heard in the axilla and at the back. Occasionally, however, a crescendo murmur is both conducted towards the sternum and transmitted into the axilla. In these cases the valve probably is made to vibrate by the regurgitant current of blood which causes the murmur.

N.B.—Whilst a crescendo murmur always means stenosis, the absence of one does not necessarily mean no stenosis.

The Association of a crescendo murmur with other murmurs in mitral stenosis is important.

1. It may be the *only murmur* heard in mitral stenosis.

2. *It may follow a diastolic murmur* (see Figs. 19, 20 and 21). As a rule there is a sound-free interval between the decrescendo diastolic murmur and the crescendo murmur. Sometimes, however, there is, to the ear, no interval between the murmurs, and a continuous murmur apparently runs from after the second sound to the accentuated first sound. This is one reason advanced in support of the auricular systolic or presystolic rhythm theory of the murmur, for it is said that such an apparently continuous sound must be produced by blood flowing in the same direction through the valve. This argument can be used to prove that it is early systolic in rhythm (see paragraph 3 below).

When the diastolic murmur is apparently continuous with the crescendo murmur, it probably means that the *auricle is able to contract* in its proper rhythm—namely, just before *v.s.*, and so keep up the true diastolic murmur.

Very often in mitral stenosis *the auricle is so dilated and weakened as to be unable to contract with sufficient*

force to help in causing a diastolic murmur. I believe that this explains the frequent absence of this murmur in mitral stenosis.

3. *A crescendo murmur may be uninterruptedly followed by a systolic murmur.* This occurs when the curtains are so distorted by disease that, although their edges are brought into apposition abnormally late in ventricular systole (crescendo murmur), they still are unable to completely close the orifice, and a leak is left through which blood continues to regurgitate throughout ventricular systole and continues the crescendo murmur as a systolic murmur (see Fig. 14, E and F). These two murmurs are produced at the mitral valve by the continuous action

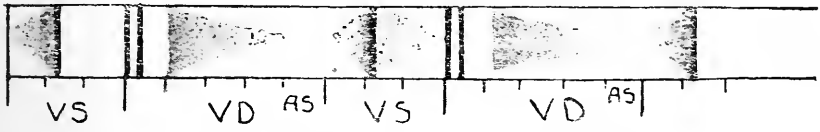

FIG. 21.—CRESCENDO MURMUR (EARLY *v.s.* RHYTHM), DOUBLE SECOND MITRAL DIASTOLIC, SOUND-FREE INTERVAL, THEN CRESCENDO MURMUR AGAIN, FOLLOWED BY SYSTOLIC MURMUR, ETC.

The first is the commoner condition, the continuous diastolic and crescendo murmur, depicted in the second cycle, being not so common.

of the ventricle in its successive stages of systole, and are continuous, though so very dissimilar in character. The impact of those portions of the thickened valve curtains which can come into apposition causes the accentuated first sound which delimits these murmurs.

4. *A systolic murmur may change into a crescendo murmur* within a few heart-beats and *vice versa.* When the ventricle is not contracting with sufficient force to close the valve, a systolic murmur of regurgitation is heard. If the force of contraction be increased, as by making the patient sit up in bed, the valve is then closed and the crescendo murmur developed. When the

patient lies down and the extra force of ventricular systole is reduced, the systolic murmur again appears. To put it in another way, a systolic murmur may be heard when the patient is up and about and overworking the heart, which is replaced by a crescendo murmur after rest in bed and treatment by digitalis.

5. *Crescendo, systolic,* and *diastolic murmurs may all be heard* during one cardiac cycle, with accentuated first and double second sounds (see Fig. 21).

Prognosis of crescendo murmurs.—The presence of a crescendo murmur means that the ventricle is contracting well and that regurgitation through the valve is reduced to a minimum, and less than when a systolic murmur is heard. I look upon it as a favourable sign, for it often is only heard after overworked heart muscle recovers its tone through rest in bed and treatment.

Tricuspid Murmurs.

Lesions of the tricuspid valve are much less common than those of its fellow valve, and are often difficult to diagnose. They are, however, often accompanied by evidence of back-pressure in the pulmonary arterial or systemic venous circulations, and this evidence is useful.

Tricuspid systolic murmurs are best heard over the heart between the apex and the tricuspid region. They are similar to mitral murmurs in origin and characteristics, but are not transmitted through the axillary region. They are often hard to differentiate from mitral murmurs, and often heard with them.

When a systolic murmur is heard to the right of the sternum, and there is evidence of enlargement of the

right auricle and pulmonary and general venous congestion, it is almost certainly due to tricuspid incompetence.

Tricuspid diastolic murmurs are similar in characteristics to mitral murmurs, but may be conducted slightly to the right of the sternum, especially with dilatation of the right auricle. They are not common.

The tricuspid crescendo murmur is of similar origin and characteristics to the mitral murmur of the same name.

With both diastolic and crescendo murmurs there would be evidence of systemic venous congestion, enlargement of the liver and general œdema, and also of pulmonary circulation congestion.

The prognosis of tricuspid murmurs is not good, as disease of this valve impedes the return of venous blood to the lungs and induces general œdema and secondary cardiac muscle failure.

Aortic Murmurs.

Lesions of the aortic valve are not so common as those of the mitral valve.

Aortic systolic murmurs vary in nature according to their causes. In relative stenosis, due to dilatation of the aorta, they ought to be of the softer fluid-vein type; but, as a matter of fact, when relative stenosis occurs at this valve, there is generally some atheroma of the aorta as well which affects the curtains of the valve also, roughening and stiffening them more or less, and giving rise to the rougher, more intense vibration murmur of actual stenosis. Sometimes they are of a musical char-

acter, in which case a tag of fibrin may be vibrated by the blood-stream. When actual stenosis exists, or when the curtains are much changed, a *thrill* can often be felt over or above the aortic area.

They are best heard in the aortic area, whence they are conducted into the great arteries, and are especially well heard over the carotids without any pressure from the stethoscope.

They may be heard down the cardiac area to the apex-beat, gradually diminishing in intensity all the way. It is therefore important to listen to an apex murmur over different portions of the præcordia between the apex and the aortic area. A murmur which *grows in intensity* when followed in this way indicates an aortic (or pulmonary) murmur, and one which diminishes a mitral (or tricuspid) murmur.

Aortic systolic murmurs are of a decrescendo nature, fading away with cessation of *v.s.*

If an aortic systolic murmur be due to actual stenosis, the radial pulse is regularly delayed.

Time.—Aortic systolic murmurs are synchronous with the carotid pulse, and should always be timed by it, because a loud diastolic murmur may alone be present and simulate one.

Aortic diastolic murmurs vary in intensity from a loud, rough, easily audible sound to the faintest whiff, which is the most difficult of all cardiac murmurs to recognize. The difficulty of hearing the short faint aortic diastolic murmurs is increased by the fact that they may be audible only at the tricuspid region of the heart, and they are very commonly overlooked. Their presence may be suspected by the change in the second sound when they are associated with one. The sound is no

longer distinct and clear; it is impure, and apparently prolonged into a whiff or faint blow, as if the "bottom were knocked out" of the sound (see Fig. 18).

An aortic diastolic murmur is particularly difficult to hear if there be a co-existent mitral diastolic murmur.

This short diastolic murmur may be caused by a similar mechanism to that which produces an *a.-v.* crescendo murmur, and have some of the rising pitch characteristic of this murmur (p. 59). The short duration of it can hardly be due to inability of the contracting aorta to keep up the murmur, for aortic diastolic murmurs are generally more prolonged.

The important difference in rhythm between aortic and mitral diastolic murmurs—namely, that the latter begin at a later phase of ventricular diastole than the former—has already been pointed out, and reasons why such should be so given (see Figs. 16 and 18).

Condition of the aortic valve at which regurgitation occurs.—The diastolic murmur may continue a distinct aortic second sound, in which case the curtains are mobile and more or less normal, perhaps with *dilatation* of the valve ring; or there may be no aortic second sound, in which case the leakage results from a *stenosed* valve with thickened, rigid, immovable curtains. The latter form of murmur is rougher than the former.

Sometimes the aortic diastolic murmur is the only murmur present, and, unless it be timed by the carotid artery, will very likely be mistaken for a systolic murmur, especially by the student, who is inclined to look upon all murmurs as systolic unless they prove themselves to be diastolic.

Like all other murmurs, except the crescendo murmur, they are of diminishing intensity, or decrescendo.

Crescendo aortic murmurs.—The possibility of an aortic diastolic murmur having some rising pitch character has already been referred to above and in the paragraph on crescendo murmurs (p. 85).

See-saw or "To-and-fro" murmur.—Not uncommonly the aortic diastolic murmur follows immediately an aortic systolic murmur, giving a to-and-fro characteristic known as the "see-saw murmur." If the lesion be one of absolute stenosis with incompetence, an aortic second

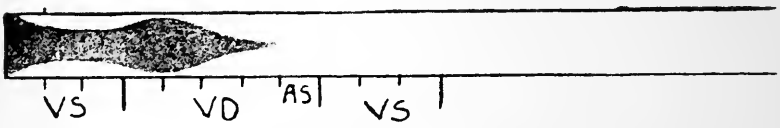

FIG. 22.—SEE-SAW AORTIC MURMUR, CONTINUOUS, NO SOUNDS, BUT THE DIASTOLIC PORTION ARISES IMMEDIATELY AT THE END OF *v.s.*, AND NOT AFTER A SOUND-FREE INTERVAL, AS IN MITRAL DIASTOLIC MURMURS.

sound is rarely heard. If it be one of relative stenosis with aortic dilatation or aneurysm, a sound may intervene.

Prognosis of aortic murmurs.—*Aortic systolic murmurs* generally indicate actual obstruction to the flow of blood from, and increase in the work done by, the left ventricle. Slight obstruction may be easily compensated by ventricular hypertrophy, and if this is adequate the condition may not affect the duration of life. When greater, the ventricle may be unable to respond adequately; it fails or dilates, and the outlook is bad.

When aortic systolic murmurs indicate *relative obstruction* of the orifice with a dilated aorta or an aneurysm beyond, the prognosis is serious.

Aortic diastolic murmurs, indicating incompetence of the valve, are more serious. Slight degrees of regurgitation

may be adequately counteracted by efficient ventricular hypertrophy, and may be present for years without adversely affecting the health. When the leakage is greater the strain on the left ventricle is very great, and adequate compensation becomes impossible. The outlook is then most serious, sudden overstrain of the heart with fatal syncope being a common ending.

The *condition of the muscle of the left ventricle* is the determining factor in prognosis of both forms of aortic disease, as it is in all other forms of valvular disease.

Posture murmur.—A very well-marked systolic murmur, simulating exactly an aortic murmur, is sometimes heard over the manubrium sterni and base of the heart in quite healthy persons who are holding the shoulders well back, and probably thereby causing some pressure of the collar-bone on the subclavian artery. Such a murmur may be heard when the subject being examined is holding his vest well up for auscultation at the base of the heart. It disappears at once if the shoulders are brought forward to remove the pressure. The murmur is so deceptive that it may create the impression of some aortic valve disease. (See also p. 67.)

Pulmonary Murmurs.

Pulmonary murmurs are very much like those produced at the aortic valve in nature. Structural changes in the valves do not often arise in adult life, and when met with then are almost always congenital in origin.

Pulmonary systolic murmur.—This is not uncommonly heard in marked anæmia, when it is caused by relative stenosis of the valve orifice, the pulmonary artery beyond the valve frequently dilating, for some reason or other not understood, in anæmic conditions of the blood.

It may be met with in apparently healthy persons, and may mean nothing; and it is also commonly heard in mitral stenosis. It is frequently heard in Graves' disease.

A systolic murmur due to structural changes of the curtains is by no means common in adult life, but is more often heard in childhood.

Actual stenosis of the valve occurs as a congenital deformity, but generally causes death before adult life. It may be present in young adults, when cyanosis will be a marked feature of the circulation.

Conduction.—Pulmonary systolic murmurs are not conducted into the carotid or subclavian arteries, but are conducted or transmitted over the left second intercostal space well away from the sternum.

Pulmonary diastolic murmurs are very rare, but when heard have much the same area of conduction, down the left margin of the sternum, as the analogous aortic murmur, with, however, the pulmonary cartilage as the theoretical seat of maximum intensity.

Prognosis.

Prognosis in pulmonary murmurs.—If due to anæmia the prognosis is that of the primary cause. If due to structural changes it is bad, and subjects of pulmonary valve disease rarely reach adult life.

VIII

HÆMIC MURMURS

Cardiac hæmic murmurs, which are of a soft, blowing character, are met with in anæmic conditions of the blood, and without there being any disease of the valve curtains. They are systolic in rhythm, and not conducted widely. Their causation has been much debated. It is very doubtful, as has already been stated, whether the diminished viscosity of the blood itself has anything to do with the production of the murmur. Moreover, it is important to remember that in chlorosis, in which these murmurs commonly occur, there is sometimes *two or three times the normal volume of blood* present, and this may lead to abnormal distension of cavities and so to the easier production of fluid-veins, especially at the semilunar valve orifices.

The commonest situation for them to be heard is at the base of the heart over the pulmonary cartilage and in the second left intercostal space. The murmur thus heard is probably due to there being relative stenosis of the pulmonary valve, experience having shown that this artery immediately beyond its valve is often dilated in marked anæmia.

A hæmic murmur may also be heard over the apex in severe anæmia. In these cases there is generally some dilatation of the ventricle, and the murmur is one of valvular incompetence arising from muscle failure.

They are often heard best with the quicker heart's action of the erect position, and disappear when the patient lies down, or the heart's action slows.

Vascular hæmic murmurs are heard over the large vessels with or without slight pressure from the stethoscope. If pressure is used the constriction of the vessel produces a fluid vein.

Bruit de diable.—This is best heard over the veins about the attachment of the sterno-mastoid to the sternum, and without any pressure from the stethoscope. It is of a continuous humming or buzzing character and is probably only heard in chlorosis where we have an increased volume of blood, sometimes more than double the normal amount, in addition to diminished viscosity. It is possible that the continuity of the hum is due to the abnormal distension of the large veins in this disease forming large masses of blood into which comparatively small streams pass from the tributary veins with an abnormal head of pressure produced in the overfull capillaries. Diminished viscosity may also contribute in some unknown way to its production.

The hum can also be heard by placing the stethoscope lightly over the ball of the eye, the mastoid region, or on the occiput over the site of the torcular Herophili. In these situations the stethoscope is close to the cavernous sinuses of the skull.

The *bruit de diable* is rarely met with in severe anæmia without increased volume of blood; in these cases, instead of a continuous hum, the murmur over the veins is of a broken, swishing, rhythmic nature.

Like true chlorosis, it is uncommon in Manchester.

IX

EXOCARDIAL SOUNDS

These sounds are produced outside the cavities of the heart.

Pericardial sounds.—Normally the smooth surfaces of the visceral and parietal layers of the pericardium move over each other without any audible sound, but when this membrane is inflamed, as it is in pericarditis, and becomes rough, then the movement of the two roughened surfaces against each other causes a sound known as a pericardial rub or friction sound.

This pericardial rub varies greatly in character from a very slight, almost inaudible sound to a marked, rough, leathery creaking, the latter being occasionally so well developed as to be palpable to the hand.

The pericardial rub frequently simulates an endocardial sound, but usually is easily differentiated from it.

It is generally a double or to-and-fro sound, corresponding roughly with systole and diastole of the heart, and is well imitated by rubbing the balls of the thumbs over each other and in a to-and-fro movement with varying degrees of pressure.

In some instances one or other parts of the sound may be double, imparting to it a shuffling character.

Pericardial friction is generally best heard over the *base of the heart,* and *often can be intensified by gentle*

pressure with the stethoscope, which has for its object the bringing of the two layers of the pericardium more closely together. It is unchanged by holding the breath, and may only be heard for a day or so.

When fluid develops and, by distending the pericardial sac, separates the two sufaces the rub will disappear, possibly to reappear with the absorption of the fluid.

The sound is not conducted away from its seat of origin unless it be very loud, in which case it may be transmitted slightly beyond the limits of the præcordia.

Cardio-respiratory sounds.—Occasionally sounds are produced by the movements of the heart acting on the lungs or pleura which may simulate endocardial or pericardial sounds; but they are, as a rule, easily differentiated from the endocardial sounds because they vary with the respiratory movements, and because the sounds of the heart are heard as usual.

Cardio-pulmonary sounds.—Respiratory sounds synchronous with systole of the ventricle may be heard when an emphysematous lung comes forward over the heart and is compressed by the movements of the heart.

Such a sound may simulate an endocardial murmur, but it is loudest with expiration and disappears when the breath is held on full inspiration. It may also resemble the creaking of a saddle.

Cardio-pleural sounds.—With inflammation of the pleura over the front of a lung a sound of friction may be caused by the movements of the heart pressing the two pleural surfaces together. Such a sound may closely simulate a pericardial rub and will not vary with inspiration.

X

SEPTIC ENDOCARDITIS, PERICARDITIS, ANGINA PECTORIS

Septic, malignant, or ulcerative Endocarditis is not uncommonly met with, especially in the practice of a general hospital. It is also, unfortunately, not uncommonly unrecognized during life, as an elevated temperature, often in the form of rigors, and evidence of great debility are the only physical signs present. The heart sounds may be quite free from murmur.

In typical cases with elevated temperature, murmurs, enlarged spleen, hæmaturia and infarcts the diagnosis is straightforward.

Its presence must always be suspected when there is prolonged elevation of temperature in cases of chronic valvular disease.

Treatment is very unsatisfactory. In some of the cases complicating chronic endocarditis the acuteness subsides and the chronic condition remains much as before, but the acute cases arising *de novo* almost invariably end fatally. Vaccine treatment by autogenous vaccines where the organism can be isolated, which is, however, very difficult to do, is reported to have relieved a few cases, but all treatment is as a rule unavailing.

Pericarditis generally occurs as a complication of rheumatic infection, but may be found in gout, cachectic conditions and (N.B.) *acute pneumonia.*

Simple acute pericarditis results in the exudation of lymph which in the most favourable cases is soon reabsorbed without any permanent damage. Its presence is recognized by the associated endocarditis, by some respiratory distress, and by pericardial friction (*q.v.*), which may be heard with the stethoscope. Pain is not a prominent symptom, and for this reason pericarditis is often overlooked. In less favourable cases the inflammation may become dry and persist for a longer time, clearing up eventually or leading to the formation of adhesions and adherent pericardium. Dry pericarditis may be felt by the hand, causing a rubbing sensation behind the præcordia, and it can be heard by the stethoscope.

The danger to the patient in most cases is not the actual pericardial inflammation itself, but the fact that it is evidence of an acute inflammation of the whole of the cardiac tissue—endo-, myo-, and pericardium. A patient with acute pericarditis is always in danger, the termination being not infrequently remarkably sudden.

Pericardial effusion.—The inflammation of the pericardium may lead to effusion of serous fluid in greater or less amount. Fluid may be suspected if a rub passes off in a day or so and reappears in a few days. If it is poured out in large quantity it causes increased cardiac dulness, especially in an upward direction, by distending the sac into a pear-shaped body which can be made out by percussion, the broad part being about the line of the fourth intercostal space. The intercostal spaces of the præcordia may be distended. The impulse of the apex-beat diminishes in intensity and is internal to the limits

of cardiac dulness. The heart sounds are muffled or weakened, and the apex may be pushed up and out. There is a good deal of respiratory distress, due to the interference with the heart's action, which makes the sitting-up (orthopnœa) position necessary. The pulse is also quick, 120 or more, and there is much pallor. Serous effusions tend to be absorbed in time, and operative interference—*paracentesis pericardii*—is not often advisable.

Purulent pericarditis is often only recognized post mortem, and its diagnosis in life is quite uncertain.

Adherent pericardium is most likely to be met with before adolescence, but it is more often found in the post-mortem room than diagnosed in the wards. Patients in whom it is present rarely reach adult life. It may be suspected when severe rheumatic inflammation of the endocardium, myocardium, and pericardium has led to considerable dilatation of the heart which persists permanently after subsidence of the acute symptoms, and in spite of treatment by prolonged rest in bed and especially by digitalis. Uncomplicated dilatation of the ventricles often recovers very well after such treatment, but if the inflamed pericardium covering the enlarged ventricles adheres to the inflamed outer layer of the pericardial sac subsequent shrinking of the muscle becomes impossible.

In adherent pericardium there may be systolic retraction of the intercostal spaces between the left lateral sternal line and the region of the apex of the enlarged heart, with synchronous protrusion of the apex of the heart. Such a condition, however, may be met with when there is considerable cardiac enlargement with no pericardial adhesion, the systole of the base of the

enlarged right ventricle causing negative pressure in the chest which leads to retraction of the intercostal spaces in front of it. When the heart is much enlarged and the two layers of the pericardium are coherent contraction of the heart may pull on the diaphragm, and consequently on its attachments to the ribs in the axillary and posterior regions of the chest, causing systolic indrawing of the regions of the diaphragmatic attachment.

On auscultation a systolic murmur, indicating incompetence through dilatation of the mitral valve, will generally be heard.

There are also present signs of interference with the action of the heart muscle leading to back pressure in the pulmonary and systemic circulations.

Pericardial adhesion may be a part of a process leading to a general mediastinal inflammation which has a special group of symptoms, or of a process leading to inflammation of all the serous membranes of the trunk, —the pericardium, pleuræ and peritoneum.

Angina pectoris is the term given to the extremely distressing and painful condition which results from interference with the circulation of blood through the heart muscle itself by some obstruction of the coronary arteries or by overworking a heart with enfeebled muscle. The pain is of two kinds—a heavy, suffocating feeling behind the sternum or of something gripping the heart and giving rise to a sensation of impending death; and shooting neuralgic pains down the arm, especially the left arm, to the elbow or even to the little and ring fingers, up the neck behind the ear, and into the left shoulder-joint.

Diagnosis.—Angina pectoris may occur without there being the slightest auscultatory evidence of any cardiac

disease; when there is evidence of aortic disease, atheroma, or aneurysm; of mitral stenosis or of severe cardiac muscle failure or debility. It is not uncommonly fatal in its first appearance, but more often it recurs at intervals over longer or shorter periods of time. It is generally made worse by exertion, but it may be brought on through the interference with the heart's action which a stomach distended with flatulence often gives rise to.

XI

CARDIAC IRREGULARITY RESULTING FROM DISTURBANCES OF STIMULATION

Electrocardiogram.—The different phases of the cardiac cycle can be timed with great accuracy by means of the property which muscle has of giving rise to currents of electricity by its contraction. These currents are very slight, and they need the most delicate instruments for receiving and recording them. The instrument now used, the string-galvanometer, is an elaboration and improvement of that originally used as a receiver in the first days of transatlantic telegraphy. The principle of it is that the minute current of electricity generated by the muscle contraction is carried on a silvered filament of rock crystal or on some suitable wire between the live poles of a strong electro-magnet and according to whether the current passing is + or − so will the string be deflected to the − or + pole. The movements of the string are recorded by the shadow which is cast by it, on a moving sensitized roll of photographic paper between which and a light it also lies. The tracing is very small, and has to be magnified subsequently to get the record which is to be used for observation.

With a normal heart contracting, a regular type of

electrocardiogram is obtained showing movements which are caused by the currents induced by contraction and relaxation of the auricles and ventricles. Departures from the normal rate, rhythm and method of contraction of the auricles or ventricles show corresponding departures from the normal cardiographic tracing.

An electrocardiogram is generally taken synchronously with one or more other tracings taken from the carotid or radial artery or the jugular vein; a phonoscope recording the sounds produced in the heart may be used at the same time as well. The most elaborate tracings give all of these synchronously with a time record also.

The electrocardiograph is a very valuable instrument for the investigation of cardiac disease, but its cost (£200) and the laboratory accommodation necessary for its use practically restricts its employment to hospitals.

Tachycardia means quick heart's action, over 130 a minute, but its use as a term is best restricted to a more rapid action which is the result of some pathological change in the heart's nervous mechanism or musculature.

The apprehensive heart.—A very rapid beat is often met with in a heart unaffected by disease when the patient is of a nervous, apprehensive type. The so-called insurance heart and the legal neurasthenic heart are of this nature. I have counted a pulse of above 160 in a girl who was being treated with zinc ionization for œsophageal ulcer. The heart was structurally normal, and slowed down to its proper rate within a couple of hours after the exciting experience. A rate of 130 is not uncommon in insurance work. In this type of rapid heart action the quickness varies even within a few seconds. The heart may slow down almost to normal after a time, or when the subject lies down, only to race

off again within a few seconds. The relative spacing of the heart sounds is normal. The increased stimuli do not originate in the heart.

In Graves' disease the pulse is often very rapid—from 120 upwards. I have counted it at 150 or more when the patient has been excited, as by a visit to a physician. The rhythm is regular, and the rate persists uniformly, without slowing down to near the normal as with the apprehensive heart. The rapid action is not caused by abnormal conditions in the heart.

Paroxysmal tachycardia is the term given to a condition in which the whole heart, auricles and ventricles, jumps from about a normal rate to one of 150 or more, and continues at this rate for a longer or shorter period, and in spite of all attempts to slow it down by treatment. Thus, I was once listening to a heart to make sure if a murmur was present when it instantaneously jumped from a rate of 80 to 170 a minute. It continued in hospital for three weeks at this pace, in spite of complete rest in bed and all drug treatment, and then stopped of its own accord, and, as far as I could gather, equally suddenly. I subsequently saw this case several times, and during the last illness, and I thought it was one of mitral stenosis; but it proved to be one of a congenital septum in the left auricle, with only a small aperture, about half an inch in diameter, for the blood to pass through. There was great enlargement of the liver during the longer paroxysms.

The paroxysms of rapid action may last from a minute or so to days or months. The rate is not materially altered when the patient changes from the erect to the lying position or *vice versa*. The stimuli to contraction arise from some other part of the heart than the sino-

auricular node, but there is one beat of the ventricles to every one of the auricles. There is generally some lesion in the muscle or nerve tissue of the heart.

Abnormally slow pulse.—Many healthy people of a phlegmatic, stolid type have a pulse-rate below 60, and a similar slow beat is often found in athletes used to prolonged exertion. I have seen it in several cross-country runners, and tennis or racquet players, and also after acute illnesses with a high temperature. The whole heart, auricles as well as ventricles, partakes of the abnormal slowness.

Bradycardia is the term applied to a very slow pulse (40 or less), and its use is best confined to cases in which there is some pathological cause, such as heart-block (*q.v.*), acting to produce the slow pulse. The beat of the auricles is more frequent than that of the ventricles.

Heart-block means the interruption of the passage of stimuli from the *a.-v.* node through the *a.-v.* bundle to the ventricles, with the result that the ventricle contracts only a fraction of the number of times stimuli from the auricles try to pass to it. If the block be complete so that no stimuli pass from auricles to ventricles, the latter continue to beat a rhythm of their own, which is usually about 30 per minute. In its severe forms it is due to some disease, of sclerotic, calcareous, or gummatous nature, interrupting the integrity of the *a.-v.* bundle, and the number of beats of the ventricles may be reduced to 9 or 10, as in a case under my care. It may be associated with epileptiform convulsions in the type of disease known as Stokes-Adams disease.

Irregular or intermittent pulse—Premature or extrasystole.—In feeling a pulse it is a very common

occurrence to find that a beat is missed, and simultaneously the patient has an unusual sensation in the chest as if the heart stopped for a second and then gave an extra forcible beat. To this condition the term "dropped beat" is often given, but it does not correctly describe what has happened. If the stethoscope be used whilst the pulse is being felt, a very feeble beat of the heart will be heard to follow prematurely a normal beat, and then a long pause comes before another normal beat. This early beat is best called a *premature* systole, as it is not an *extra systole*. It may be too feeble to open the aortic valve, when a feeble first sound alone is heard; or it may be strong enough to open the valve and cause a slight second sound but no real pulse-wave. Any accompanying murmur will be modified also in a similar way. The premature systole is brought about by a stimulus arising in some part of the heart other than the sino-auricular node and before its proper time.

The premature systole form of cardiac irregularity of itself, that is, provided there is no evidence of cardiac muscle disease, namely, abnormal frequency of heart-beat, want of diastolic rest (tic-tac rhythm), valvular disease, muscle failure, need not alarm, as it is met with very frequently in apparently normal hearts. A very common cause is a reflex action of some functional disorder of the stomach, especially flatulence. Tobacco is probably another cause, acting directly on the heart or reflexly through disturbance of the digestion. A single premature systole, occurring occasionally, is the simplest form of the condition, and the next is a more frequent repetition of the premature beat, varying to a state in which beats are dropped regularly every few or more (4 to 20) heart-beats, and which persists for years, or in which three or four premature systoles will occur con-

secutively with a fluttering sensation. Such a condition often runs in families, tending to be more persistent as its subjects, often liable to suppressed gout, grow older, when there are degenerative changes in the cardiac muscle. By suppressed gout I mean a diathesis in which the metabolism of food is not carried to its proper end-formation of urea, but stops in a variation of the sodium biurate formation of true articular gout. Suppressed gout may lead to a variety of ailments, affecting every system of the body, which develop after a period of consumption of food which, though not immoderate, is in excess of the demand for the body in later adult years. Premature heart-beats is one of the commonest of these suppressed gout manifestations.

Such irregularity generally passes off on exertion, and may pass off during an acute illness, or in older people may be made worse. I have seen an old patient of seventy-three, whose heart had been more or less irregular for many years, pass safely through an attack of acute pneumonia in which there were very few successive regular cardiac cycles for three or more days. The heart seemed to roll over and over in a state of delirium cordis. With convalescence the usual degree of irregularity returned.

Whilst cardiac irregularity means nothing serious with healthy heart muscle, it is serious when other signs of muscle failure, such as breathlessness on exertion, absence of diastolic rest, etc., are present.

Sinus arrhythmia.—Another form of irregularity is that arising from the sinus; it occurs chiefly in young people, and may be produced by such a physiological action as swallowing. Here diastole only is prolonged, systole being of the normal length.

It has no evil prognostic significance.

Auricular fibrillation is the term given to a condition in which the contraction of the auricles is so feeble and indefinite that the muscle is thrown into a state of fibrillation or twitching of a very rapid nature. The auricle now ceases to contract as a whole and the *a.* wave disappears from a jugular tracing. In many if not all of the cases there is some stretching of the muscle fibres and dilatation of the cavities of the auricles, with also some chronic inflammatory process of a fibrotic nature. The auricular tissue becomes very excitable and responds to stimuli which arise with very great rapidity (200 to 300 per minute) from somewhere else than the sino-auricular node, possibly in the auricular muscle itself. The conducting tissue between the auricles and ventricles in the majority of cases is not damaged and conducts a greatly increased number of stimuli to the ventricles. These stimuli are of unequal strength and catch the ventricles at phases of their cycle when they cannot respond properly to any stimulus. The result is usually increased frequency (90 to 150) of ventricular action with extreme irregularity of rhythm and of completeness of contraction. The pulse becomes quick and very irregular, with scarcely two beats of a similar nature following successively. The irregularity of force of ventricular contraction may only be fully appreciated by using the stethoscope at the same time that the pulse is being felt. A very irregular pulse of over 120 in rate is almost certain to be of auricular fibrillation origin. Increased strain on the heart, such as is caused by exercise, increases the number of beats and also the irregularity of the heart's action.

Auricular fibrillation occurs most commonly in hearts affected by rheumatism, but it may occur when there is no evidence of this disease. In the rheumatic cases it is

very common in mitral stenosis and between the ages of twenty and sixty, whilst in the non-rheumatic cases its incidence is later—between fifty and seventy. The quick, irregular pulse of mitral stenosis is a typical example of the condition. Although the pulse is generally quick, it may be slower and more regular; but there will always be found some irregularity in the duration of the cardiac cycle if careful measurement of the length of the arterial pulse-waves be made. In a few cases with impaired conduction through the *a.-v.* bundle the pulse-rate may be below normal.

With auricular fibrillation there are generally present some of the usual signs of cardiac failure and back pressure on the circulation.

It may persist for several years in cases of mitral stenosis which react to digitalis and are carefully treated and dieted; the subjects of it may be quite able to keep up and about and follow some clerical occupation.

Auricular flutter is the term given to a much less common condition which occurs chiefly in the later years of life, over fifty, when there is some senile arteriosclerosis and cardiac muscle degeneration with or without valvular disease. The auricle contracts so very rapidly (260 to 320 per minute) to stimuli which arise—probably from some single focus in its own tissue—that it is in a state of flutter. It is almost always associated with some degree of heart-block, so many stimuli being unable to pass through the *a.-v.* bundle, and the ventricle only contracts one-half, one-third, or a less fractional number of times of the auricular rate. It is difficult to distinguish from paroxysmal tachycardia. The ventricular beating is often regular in rhythm and completeness of contraction. Even when irregular, the irregularity is

more or less regular in its appearance. The attacks may last for a few hours or for months or years; in the former case they may occur from time to time for several years. In one of Dr. Lewis's cases the ventricular rate was 160 for four years. If the ventricles take on the full auricular rate the attack is very serious, causing faintness, and if it should persist for more than a few hours death is likely to ensue. Firm pressure on the carotid artery passing on to the vagus produces a slowing of the pulse. Digitalis often controls the rapidity of the heart-beat very effectively.

Further information on the cardiac affections referred to in this section should be sought for in Dr. Thomas Lewis's valuable small work, "Clinical Disorders of the Heart-Beat," or in Sir James Mackenzie's much larger book on "Diseases of the Heart." I am indebted to Dr. Lewis's work for some of the information in this section, especially in the paragraphs on Auricular Fibrillation and Auricular Flutter—subjects which he has studied very closely.

XII

TREATMENT OF CARDIAC DISEASE

The treatment of affections of the heart ***centres chiefly round the cardiac muscle,*** and has for its objects: (1) The prevention of weakening of the muscle—that is, of cardiac muscle failure—or (2), if the cardiac muscle is already weakened or has failed, to bring about compensation or prevent any further weakening. In other words, treatment in heart affections may be called for (*a*) before muscle failure, or (*b*) after muscle failure.

Treatment of Cardiac Affections before Muscle Failure develops.

Many slight cases of valvular disease occur in which there is no evidence of cardiac muscle failure. In the most favourable cases the heart-rate is normal, there is no shortness of breath, and the patient leads a life like the average person. Slight murmurs not uncommonly occur without the patient knowing of their existence. No treatment is called for in these cases.

In the next class of case the lesion is ***a little more severe,*** and, although the heart may stand well a quiet life, there is no margin for meeting the extra demands of the muscular exertion required in periods of excitement, in going up hills or stairs quickly, or in playing

games. In other words, there is danger of cardiac muscle failure developing.

The treatment in these borderland cases must be directed to helping the heart muscle to keep up with its work. It must be saved from the influence of adverse forces acting directly on it, chiefly muscular over-exertion, and indirectly or reflexly from the nervous and alimentary systems.

Mental excitement, which tends to quicken the action of shaky hearts, must be avoided, and a proper amount of sleep should be secured.

Diet.—In the alimentary system the diet should be so ordered as to avoid the development of flatulence in the stomach which tends to excite an abnormal heart. This is most surely brought about by the avoidance of too much starch and sugar food, and especially by not taking these foods with the heavy meals of the day in which flesh foods are consumed. It is surprising what a difference the dyspeptic feels when he takes a meal consisting only of fish or meat, green vegetables, toast, and even potatoes, as compared with when he takes in addition even small amounts of "sweets" in the form of milk pudding, or any other pudding made with starch and sugar. The discomfort of the second type of meal arises partly from the greater amount consumed at it, and partly from the flatulence which such a proteid and starch meal produces. The carbohydrates and fats should be taken at breakfast in the form of porridge, toast, brown bread, butter, fat bacon, milk, or cream. Tea, coffee, or cocoa with mixed meals comprising partly flesh foods are very liable to cause flatulence.

The bowels should be kept regular, using, if necessary, some simple non-irritant aperient, two of the best of

which are cascara, or the pill of colocynth and hyoscyamus in two and a half grain or larger doses. Purgation must be avoided, and saline aperients often cause considerable gastro-intestinal disturbance.

Exercise should be encouraged, but this must be well within the capacity of the heart. The pulse-rate should not be raised too high by it, and should subside to the normal rate within reasonable time after resting. Shortness of breath should especially be avoided. Too much is being done if the patient cannot take his food and sleep properly. Golf is a useful form of exercise if taken leisurely and in moderate amount, but not if played too vigorously or too long.

Medicinal treatment in these cases is directed towards keeping the heart from getting overstrained, and should be of a general nature calculated to keep the whole body in as healthy a condition as possible. The digestion should be helped if necessary by stomachic mixtures, tincture of nux vomica, or strychnine, being specially useful; and the blood should not be allowed to become anæmic, arsenic and iron preparations being used for this purpose.

Treatment of Cardiac Muscle Failure.

Rest in bed.—As the muscle failure is the result of overwork, the first indication for its treatment is rest, and the only way to secure this properly is to keep the patient in bed altogether, even using the bed-pan for the bowels. It is no use letting the patient get up and lie on the couch. The exertion of dressing is considerable, and any exertion increases the heart-rate considerably in muscle failure. The saving of the work of the heart which complete rest in bed, as compared with the

"up and about" condition, insures, can be seen by a simple arithmetical sum. There is frequently a difference of 20 and 30 beats per minute between the two conditions in cardiac debility—that is, 1,200 to 1,800 an hour, or 14,000 to 20,000 per twelve hours. To save the heart this work means good progress on the upward journey. It is astonishing to those seeing it for the first time how complete rest in bed, without any drugs, in cases of general œdema from muscle failure, especially if due to alcoholic poisoning, will lead to elimination of the fluid by *spontaneous diuresis*. It is not uncommon to see patients, who on admission pass only 20 ounces of urine a day, begin, after three or five or so days in bed, to pass 50, 100, 150, or 180 ounces per day with almost visible shrinking to normal bulk.

Diet.—The diet in cases of cardiac muscle failure must be on the same lines as that ordered in the earlier stages of heart affections. It suits the patients best to be kept more rigidly to flesh foods. The lighter forms of flesh foods are the best. No duck, goose, salmon, pork, veal, or high game, should be allowed. Made-up dishes with rich gravy, and sauces or soups thickened with flour and burnt fats are all very productive of flatulence.

It is also important to keep the diet *as free from fluid as possible*, so as to try and promote the absorption of the excess of fluid which tends to remain in the tissues.

The bowels must be kept acting daily, without purging, but with fluid evacuations if there is œdema present. When the liver is enlarged, some blue pill is useful. It can be given in the form of Pil. diuretica (Guy's pill) three times daily, or else in larger single doses, 3 grains at night twice or three times a week. It should be followed in the morning by a saline aperient.

Sleep.—One of the most distressing results of severe cardiac muscle failure, especially with much enlargement of the heart, is inability to sleep restfully, or even to sleep at all. This is due in great part to the tumultuous contraction of the big heart, and, with aortic incompetence, to throbbing of the carotid arteries as well. These movements are so marked that they constantly disturb the patient, and even if sleep comes from exhaustion it is not restful and sooner rather than later the patient wakes up in a fright with a sense of suffocation. There is no chance of sleeping in any but the sitting position, and not uncommonly only when the trunk and head incline forward. In the worst forms bed is impossible, and an armchair the only resort.

In milder cases a glass of hot whisky and water may help, and the addition of a drachm of ethyl nitrite when there is any high tension will give some relief. Chloral and bromide are useful in the more advanced stage, and the former drug can be given without any fear, as it is not really depressant to the heart. Even if chloral were depressant, no harm would be done to a vigorously overacting heart. Chloralamide may also be tried; paraldehyde is useful, but it is objectionable to take. Unfortunately, very often none of these drugs help matters, and then morphia must be used. When given hypodermically in quarter-grain doses with $\frac{1}{100}$ grain of atropine some restful sleep may be obtained. Alcohol may help towards securing sleep.

Drugs.—The medicinal like the general treatment of heart failure is primarily directed towards strengthening the muscle and removing the œdema, and the groups of drugs used for these purposes, with their most useful members, are—

1. ***Drugs acting on the cardiac muscle***—digitalis, strophanthus, and caffein citrate.

2. ***Drugs acting as vessel dilators***—the nitrite group, especially sodium and ethyl nitrites.

3. ***Diuretics***—digitalis, caffein citrate, theobromine and sodium salicylate, theocin sodium acetate.

Drugs acting on the Cardiac Muscle.

Digitalis is the most useful drug in the treatment of heart disease. It slows the heart by prolonging diastole, and it also tends to raise the blood-pressure. Its indication is cardiac muscle failure, and not any special valvular disease. It may also act well in muscle failure from degeneration, fatty or fibroid, but its use in these conditions must be carefully watched.

If digitalis will do good it can almost always be tolerated, but sometimes it disagrees from personal intolerance of the drug before any question of overdosage arises, and causes nausea, vomiting, and looseness of the bowels. True overdosage may cause cardiac irregularity or coupling of the heart-beats, which will return to normal after discontinuance of the drug and treatment in bed alone.

When digitalis has been given for some time the urine often becomes diminished in amount before nausea appears—a warning to stop its use.

A watery extract, especially the freshly prepared infusion (one to two drachms three times daily) is the best; but it is very convenient to prescribe it in granule form ($\frac{1}{240}$ milligramme of digitalin), people with mitral disease and secondary muscle failure, especially, sometimes taking one every morning with great advantage for years. If it is desired to push the drug a granule may be given

twice or three times daily. The tincture in ten to fifteen minim doses three times daily is also useful.

Digitalis acts best in cardiac muscle failure and dilatation of the ventricles from overwork in which there are signs of back pressure or œdema so often associated with mitral disease or alcoholism. Whilst it often acts well in cases with a quick irregular pulse associated with auricular fibrillation and with some œdema, it is impossible to tell definitely whether it will act or not. No instruments can supply us with this knowledge. It is also useful in aortic incompetence with ventricular dilatation, provided the pulse is above 80 when the patient is in bed.

Cases of valvular disease in which the heart is beating slowly (70 to 80) and regularly, and there is no shortness of breath, do not need digitalis. A simple general tonic like nux vomica is enough, with a healthy mode of life and a diet which will avert flatulence.

Digitalis must be used with caution in hearts which give evidence of senile decay, fatty degeneration, or cardiosclerosis, but when so used is often invaluable. If it is desired to use it with high tension, which it increases, some vessel dilator should be given at the same time. An excellent prescription for high tension is a grain and a half each of sodium and potassium iodides, a quarter to half a grain of sodium nitrite and water to half an ounce three times a day.

If digitalis does not act on the cardiac muscle other drugs, in my experience, are unlikely to do so, but strophanthus or caffein citrate may be tried.

Strophanthus acts on the heart like digitalis, but it does not raise the blood-pressure. It may be useful, therefore, if there is a tendency to high tension, which digitalis will emphasize, and also in cases which cannot

tolerate digitalis. The new tincture is much stronger than the old one and the dose is 2 to 5 minims.

Caffein citrate stimulates the heart by increasing the frequency of its beat and decreasing diastole. Its diuretic effect helps. But there are only a few cases which will react to it and not to digitalis and strophanthus. Two to three grains in water thrice daily is a sufficient dose.

Strychnine or nux vomica even in large doses, in my opinion, is not of any real value as a heart muscle stimulant and restorative. These drugs act as tonics—that is, they tend to stimulate the stomach and to enable it to start the processes of digestion of food better. With improved digestion there is improved absorption of food and consequent improvement of the whole system, including the heart.

Vessel Dilators.

The nitrites are most useful drugs whenever high tension is present, as indicated by the condition of the radial pulse or by dyspnœa of the asthmatic or paroxysmal type. For prolonged treatment of high tension a mixture containing sodium nitrite (one quarter to half a grain), with small doses of iodides of potash and soda (of each one and a half grains), in half an ounce of water, three times a day is most valuable. For more rapid relief the solution of ethyl nitrite is very effective. Many heart cases are liable to attacks of dyspnœa at night and these are relieved by a teaspoonful of ethyl nitrite solution in a little water, followed immediately by a tablespoonful of whisky in hot water. The ethyl nitrite is very volatile, must be kept in well-stoppered bottles, and must not be given in hot water.

Diuretics.

If digitalis fails to remove dropsy, and there is no nephritis, theobromine and sodium salicylate (gr. x. t.d.s. for an adult) often will. Theocin sodium acetate (gr. v. t.d.s.) is also often useful.

Complete rest in bed on light diet is one of the best diuretics, especially in alcoholic cardiac muscle failure with general œdema. After four or five days the urinary output begins to go up rapidly, with gradual decrease in the œdema. Guy's pill, or Pil. diuretica, consisting of a grain each of powdered digitalis leaves, powdered squill, blue pill, and extract of hyoscyamus, given three times a day, is a most useful method of treating these cases. The diuresis that follows its administration is often very marked.

Treatment of Special Forms of Heart Disease.

Mitral disease.—Mitral stenosis with more or less incompetence is the most common form of heart disease in adult life. Its presence may not be suspected and may only be recognized when an examination is made for some purpose not connected with a suspicion of any heart trouble, as, for instance, a life insurance proposal. Such people lead an active life like their fellows with perfectly sound hearts. In these cases the leakage is small and has no bad effects.

Many other cases in which there is evidence of cardiac weakness, quickened heart-rate, breathlessness, especially on exertion, but without œdema, can get about quite comfortably for years with the help of digitalis; when the drug acts it acts well, and a granule of $\frac{1}{240}$ grain of Nativelle's digitalin, given daily at breakfast-time, or

one of $\frac{1}{600}$ grain given three times daily, has kept people on their legs for years. Such patients must, however, rest as much as possible in the day-time.

The drug can be used as long as it does not cause nausea, or does not slow the pulse below 70. With œdema present digitalis still acts well, but rest in bed is necessary in addition.

The quick, irregular heart of auricular fibrillation met with in these cases is often considerably steadied by this treatment, especially if œdema be present, but if there are no back-pressure symptoms it is often very disappointing to find that no treatment whatever improves the condition. The only thing to be done is to give cardiac and general sedatives, especially the bromides. These are as difficult cases to deal with as any in cardiac disease.

All patients taking digitalis must be watched carefully, as the drug is cumulative in its action, and if given for too long a time may slow the pulse too much, and produce coupling of the beats and gastro-intestinal symptoms.

Aortic disease.—This disease does not contra-indicate digitalis if there is any muscle failure. It can be safely given, and should be given, even in extreme regurgitation, provided the pulse-rate does not slow down below 80.

Alcoholic heart failure, with œdema, and with or without valvular incompetence, does splendidly on the rest in bed alone, a diuresis coming on which clears off the œdema. Digitalis, especially the infusion, acts at its best in these cases, and soon strengthens the heart muscle and hastens the diuresis. Rest in bed is, however, also essential for this action of the drug.

Angina pectoris.—During an attack one of the vessel dilators, the nitrites, must be used. In the milder cases they will relieve the pain. Many patients carry amyl nitrite capsules in their pockets and inhale the drug during the attack. In less severe cases a trinitrin tablet is carried, and taken if there are threatenings of pain. In the most severe cases it is necessary to give a hypodermic injection of morphia.

Between the attacks treatment is directed to removing calcareous deposits from the arteries of the heart and to preventing any further deposition of them. Phosphoric acid is very useful for this; twenty minims of the dilute acid with infusion of calumba and spirits of chloroform, given three times a day after food for some weeks, has been followed by great freedom from attacks.

If the disease occurs in people below forty-five or fifty, syphilis must be suspected. The Wassermann reaction should be done, and, if positive, treatment with mercury inunction and iodide of potash instituted; salvarsan is dangerous in these cases.

When there is evidence of cardiac muscle failure with dilatation of the heart this must be treated carefully by approved measures. The question of exercise in people liable to angina pectoris is a very important matter. Only that which cannot throw a strain on the heart should be allowed, and golf is not such an exercise.

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