Communicable Diseases: A Manual for Rural Health Workers

by: Jan Eshuis and Peter Manschot

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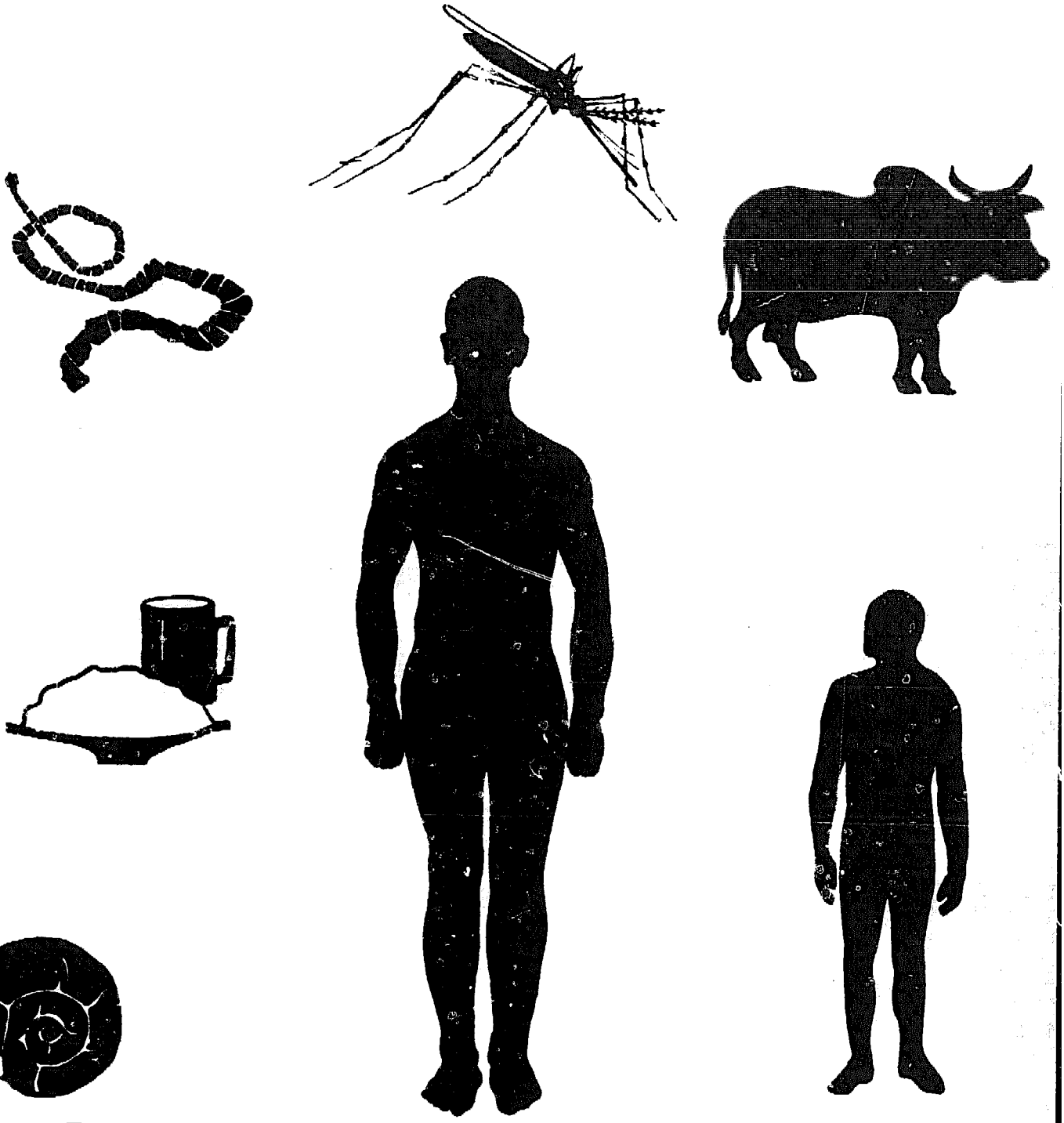

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RURAL HEALTH SERIES 7

AMERICAN MEDICAL ANSO RESEARCH FOUNDATION

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A MANUAL FOR RURAL HEALTH WORKERS

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Illustrated by
SISTER JEAN LORENZ, S.C.M.M.

AFRICAN MEDICAL AND RESEARCH FOUNDATION

ACKNOWLEDGEMENTS

The expansion of training programmes for rural health workers has made obvious to all involved, the need for suitable books adapted to the local environment. The work of drafting this manual has been shared by many people. Copies of the drafts were circulated to all medical assistant training centres, and discussed at workshops held at Mwanza in 1975, Moshi in 1976, and Dar es Salaam in 1977, and we are grateful to all the following who have spent time and energy dicussing the drafts and making suggestions for improvement: Drs Bhachu, Brooke, Dahoma, Georgiev, Hart, Huenges, Kagimba, Kisanga, Massawe, Mavura, Mbilu, Mkumbwa, Ngoda, Safe, Seda, Shao, Swai, Vaughan, and the members of the National Leprosy Advisory and Co-ordinating Committee.

In drafting the manual we drew on a number of other publications—in particular those written by ALERT, AMREF, King, Manson-Bahr, and Morley. Our special thanks go to Dr Rosemary McMahon for her continuous support and constructive criticism, and for scrutinizing the draft word by word. We have tried to incorporate as many as possible of the suggestions made to us.

We are grateful to Mrs Polly Morris who did many of the original illustrations for the draft edition; to Mrs Elizabeth Wood who corrected the English; to Miss Nicolette Dames who did the layout; and to Drs de Glanville and Wood and their staff at the African Medical and Research Foundation who undertook extensive editing throughout all stages of the production of the book. We are also grateful to Misereor whose grant to AMREF subsidized the production and made possible the first free distribution to those who need the book most—rural health workers.

Finally, our thanks go to our students, who by their continuous requests for the drafts gave us the stimulus to go on with book in addition to our normal duties.

Jan Eshuis
Peter Manschot

TO THE READER

This book has been written for you, the rural health workers of Tanzania, who are faced with the task of giving primary medical care to farmers and other workers in health centres and dispensaries. The majority of Tanzania's population expects help and advice from you. Many of you have to bear this heavy responsibility without having access to medical books and periodicals, and without opportunity to ask advice from other professionals. We hope this book will help you.

The sections of the book on management and treatment are written with the facilities available in health centres in mind, but for the benefit of those whose health centres are far away from a hospital, or who are unable to refer patients because of transport problems, the basic treatment provided in a hospital has also been included.

As well as treating disease, rural health workers must concern themselves with preventing it. This is achieved mainly through health education, vaccination programmes, and environmental sanitation. To organize and run such programmes successfully, the interest and co-operation of community leaders outside the medical field is essential. Suggestions on how this interest can be stimulated are included in the Action section for control of each disease.

Another responsibility of the rural health worker is the early detection and reporting of outbreaks of epidemic diseases. By detecting these in time and taking the appropriate immediate action many lives can be saved.

We have tried to base the contents of this book on currently prevailing concepts. Medicine is a dynamic science, however, and what is advanced today may be old-fashioned tomorrow. So journals and periodicals must be read to keep in touch with new developments.

If this book stimulates you to keep in mind the medical needs of the whole community rather than just satisfying the demands of a few, then it will have served the purpose for which it was intended.

Jan Eshuis
Peter Manschot

September 1978

FOREWORD

I am happy to commend this latest edition to the series of manuals for Medical Assistants and Rural Medical Aides. For the first time all the essential information on communicable diseases, from both clinical and public health aspects, has been collected in one volume, adequate for the training of paramedical staff. The presentation is beautifully clear, the essential facts being put plainly and concisely in a way that should simplify both learning and easy reference when in doubt.

With the emphasis that is now rightly being placed both on rural health facilities (now fashionably called 'primary health care') and on the training of paramedical workers such a book is essential and it is my hope that it will materially assist in the production of soundly trained and well motivated workers in this field.

I do congratulate the authors on a task so well fulfilled, and hope the book will be of real value to many generations of students. Lastly I wish to record my appreciation of the untiring efforts of the African Medical and Research Foundation whose continuing devotion to the cause of medical education has made possible the publication of this excellent and practical manual.

L. D. STIRLING, M.P.
Minister for Health, Tanzania

August, 1978

INTRODUCTION

Diseases can conveniently be divided into a few large groups according to their main causes—see table below.

Most of the common diseases in Africa are environmental diseases due to infection by living organisms—viruses, bacteria, protozoa, or metazoa. These are called communicable diseases because they spread from person to person, or sometimes animals to people. Together with malnutrition they are today the major cause of illness in Africa. They occur at all ages but are most serious in childhood and they are to a great extent preventable. In countries where they have been prevented, other conditions such as accidents and the degenerative and malignant diseases, which occur mainly at an older age, have taken their place and become the commonest.

Main groups of disease, by cause

Disease group	Cause	Example
Genetic	inherited	sickle cell anaemia
Deficiency	shortage of intake of essential ingredients	iron deficiency anaemia
Environmental	most commonly due to biological organisms	tb, tetanus
	sometimes due to physical factors, e.g. heat	heat cramps
	sometimes due to chemical factors, e.g. insecticide	insecticide poisoning
	sometimes due to social factors, e.g. stress	anxiety, peptic ulcer
Degenerative	due to wear and tear of the body	heart failure of old age senile arthritis
Malignant	change in cell growth due to mainly unknown causes	cancer of the liver

Communicable diseases are very important in Africa because:

1. many of them are very common;
2. some of them are serious and cause death and disability;
3. some of them cause widespread outbreaks of disease—epidemics;
4. most of them are preventable by fairly simple means.

Organisms and agents of disease

The living organisms that cause communicable diseases are of different sizes and sorts. The largest like tapeworms, or the filariae, are visible to the naked eye. They are themselves made up of many cells and are called metazoa. Complicated but single-cell organisms, like malaria parasites and amoebae are called protozoa. They are smaller and can only be seen when magnified by a microscope.

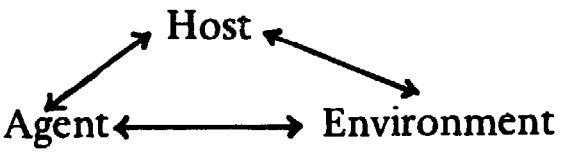
Smaller still are bacteria which are simple, single cells best seen under a microscope after they have been stained with dyes. Smallest of all are viruses which cannot even be seen with an ordinary microscope.

Patterns of communicable diseases

Different diseases are common in different places and at different times. To understand why this happens we need to consider the living organisms of disease, the *agents*, the people they infect, the *hosts*, and the surroundings in which they live, the *environment*.

The agents need a suitable environment in which to grow and multiply and thus be able to spread and infect another host.

If they are not successful in doing this they die out. There is therefore a balance between the agent, the host and the environment which can be shown diagramatically.



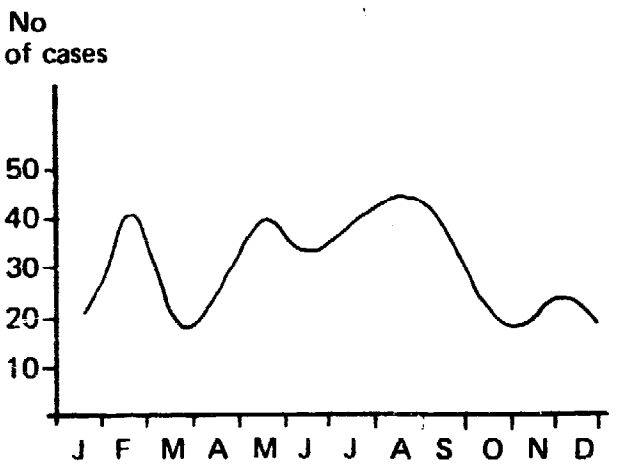
Hosts (people) are affected by their environment; they may live in a hot and wet climate in which there are many mosquitoes, but they can also change it; they may cut grass or drain swamps. Similarly the environment can affect the agent—disinfectants, sterilization; or the agent may affect the environment.

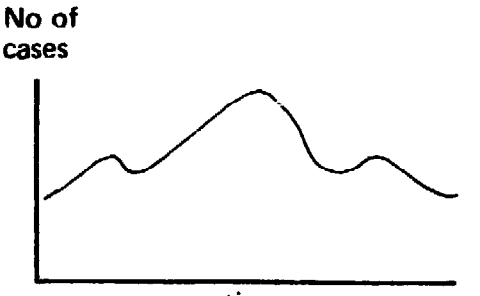
When the balance between these three is constant there will be a fairly steady number of people getting sick all the time. When this happens a disease is said to be *endemic*.

Where the balance is shifted in favour of the organism, for example when a lot of non-immune children have been born in an area since the last measles epidemic, a large number of cases of measles will occur in a short time. When all the non-immune have had the disease it will die down again, this being called an epidemic.

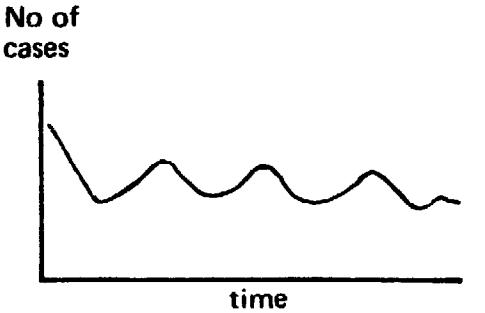
If the balance can be shifted against the agent the disease will be controlled and the number of cases will go down.

Example of graphs showing the number of recorded patients in a health centre





Pattern of epidemic disease
e.g. measles



Pattern of disease control
e.g. trypanosomiasis after
village establishment programme

The management of communicable diseases

The proper management of communicable diseases involves both trying to stop people getting diseases (prevention and control), and looking after those who have them (treatment and cure). The two are frequently closely related and doing one without the other is only doing half the job.

To be able to prevent communicable disease it is necessary to understand the factors which affect the balance between the host, the agent, and the environment, and to know what can be done to tip the balance in favour of control.

The host and infection

Most of the communicable diseases in Africa have people as their main host. There are, however, a few important animal infections which may sometimes spread to humans such as rabies and plague. These diseases are called *zoonoses*.

When an organism infects a person there are several stages to

consider. The time between infection and the appearance of symptoms and signs of illness is called the *incubation period*. The shorter the incubation period the more rapidly the disease can spread or die out in the community. Infection with some organisms nearly always leads to detectable symptoms and signs—a *clinical infection*. Others are able to infect people without always producing obvious symptoms or signs—these are called *sub-clinical infections*. This is important because people with symptoms and signs are ill and therefore they come for help from the medical services or they can be found, but people with subclinical infections do not know they are infected and that they are a danger to other people. It is also very difficult to detect them in the general population without special tests. These people are called *carriers* because they have a subclinical infection and are excreting organisms which are dangerous for other people. This can occur during the incubation period (e.g. measles), or following recovery (e.g. typhoid).

Infection with a micro-organism may result in	clinical infection
	subclinical infection but spread of the organism by the infected person
	the organism being killed by person infected. No danger.

People are susceptible to many diseases. To others they may have or develop *resistance*. The resistance of the human body is provided by its various defence mechanisms: the defensive properties of the skin, antibacterial action of secretions, white blood cells, and immmunity. Immunity is due to special body cells and to antibodies circulating in the blood (see immunization chapters in *Community Health* and *Child Health* manuals). Immunization procedures are an artificial way to raise the people's resistance to certain infections by giving them vaccines.

The *reservoir* of the infection is the animal or place in which a

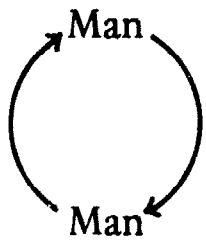
particular organism usually lives and multiplies. For most of the important communicable diseases man himself is the main reservoir. For brucellosis it is cattle, for rabies wild carnivores, and for a few, e.g. tetanus, it is the soil. The *source* of the infection is the animal or place from which the particular organism spreads to its new host.

The way in which an organism leaves the infected host or source, and travels to a new susceptible is called the *route of transmission*. Each disease organism has particular routes and these therefore play a large part in how these organisms spread in the community. For example, some spread in water and food and other by vectors like mosquitoes and snails.

Transmission cycle

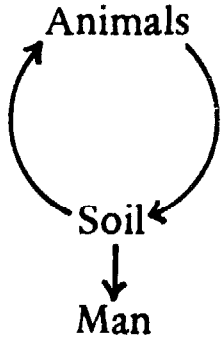
The *transmission cycle* describes how an organism grows, multiplies, and spreads. Man may be the only host and infections spread directly from man to man, e.g. measles.

Measles

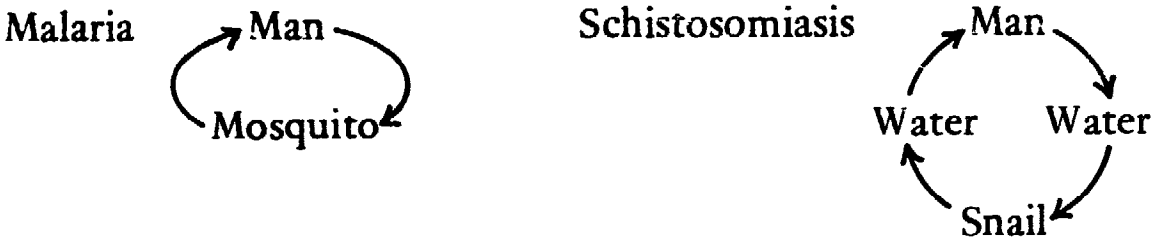


Or man may be the final host from whom the organism has no chance to pass further, e.g. tetanus.

Tetanus



Man is more usually an integral part of the transmission cycle and he then is also the main reservoir, e.g. malaria or schistosomiasis.



The three main parts of the transmission cycle for an agent or organism can be illustrated thus:

SOURCE ⟶ TRANSMISSION ⟶ SUSCEPTIBLE HOST

Source

This can be an infected person or animal, or soil. People and animals may have clinical diseases, subclinical infection, or be carriers. If there is a reservoir, it should be considered with the source.

Transmisssion

The main routes are by:-

- direct contact
- vectors
- faecal contamination of soil, food, water
- contact with animals and their products
- air-borne

Susceptible host

This is a host with low resistance to the particular infection which may be due to:-

- not having met the organism before and therefore not having any immunity to it, e.g. when a child has lost the protection of the antibodies inherited from his mother, after 6-12 months,

and comes in contact with a child with measles for the first time, as he has no immunity against it he will develop the disease.
- having another serious illness like diabetes at the same time; such people have a high risk of developing tuberculosis.
- malnutrition, which can make infections worse.

Principles of communicable disease control

The aim of control is to tip the balance against the agent. This may be done by:-

- attacking the source
- interrupting the route of tranmission
- protecting the host.

The main methods of control are:-

Attacking the source	*Interrupting transmission*	*Protecting host*
Treatment of cases and carriers	Environmental sanitation	Immunization
Isolation	Personal hygiene	Chemo-prophylaxis
Reservoir control	Vector control	Personal protection
Notification	Disinfection and sterilization	Better nutrition

Attacking the source

1. *Treatment of cases*

If clinical cases can be treated with drugs that are effective against the organism, then fewer organisms are available to spread to new hosts. The effectiveness of treatment as a control measure depends on how many of the cases in the country can be reached. It is an important method in the control of tuberculosis and leprosy.

Treatment of subclinical cases and carriers

For treatment to be effective, subclinical *cases and carriers*

must also be treated. But special efforts have to be made to find them first, as they do not usually present with any apparent illness, e.g. subclinical infections of cholera or ankylostomiasis.

Mass treatment

Where a high percentage of the population are known to have a disease, it is sometimes advisable to treat everybody, without checking whether individuals have the disease or not. This is callled *mass treatment* and has been used, for example, in the treatment of schistosomiasis in school children.

2. *Isolation of cases*

Isolation means that the case is not allowed to come into close contact with other people, except those who are looking after the patient. The organisms therefore cannot spread. Isolation was one of the methods successfully used in the control of smallpox.

Isolation is very difficult to enforce, however, and has a number of disadvantages; in particular people are frightened of being isolated and this stops them coming for treatment, and so increases the spread of the disease.

3. *Reservoir control*

In those diseases that have their main reservoir in animals, mass treatment or chemoprophylaxis or immunization of the infected animals can be carried out, e.g. in brucellosis. Other ways include separating Man from animals or killing the animals and so destroying the reservoir, e.g. plague and rabies.

4. *Notifications and reports*

Although these do not directly affect the source, notifications are an essential means of keeping a watch—*surveillance*—on the number of new cases and thereby monitoring the effectiveness of the control programme (see Appendix B).

Interrupting Transmission

1. *Environmental sanitation*

   Many organisms are able to spread through contaminated food and water, particularly those that are dependant on the faecal-oral route. Other diseases are spread through refuse and dirty living conditions. The airborne diseases are more likely to spread when housing is inadequate and people live in crowded rooms. The main methods of improving environmental hygiene are described in the manual on *Community Health.*

2. *Personal hygiene*

   Many careless or dirty personal habits help to spread some diseases, particularly the contact and venereal diseases and those that may be spread by faecal contamination of hands, food, and water.

3. *Vector control*

   Any organism that requires a vector, like a mosquito or snail, for its transmission cycle may be affected if the vectors are killed off or reduced. Methods of vector control include altering the environment so that it is unfavourable to the vector (e.g. draining swamps), using toxic substances (e.g. larvicides), and using other living organisms that attack the vector (biological methods), e.g. introducing snail-eating fish into irrigation schemes to control schistosomiasis.

4. *Disinfection and sterilization*

   These aim at destryoing the organism when it is in the environment, e.g. the use of chlorine in wells, and sterilization of surgical instruments.

Protecting the host

1. *Immunization*

   Increasing host resistance by immunization is one of the most effective methods of control for some communicable diseases. It has been responsible for the worldwide control of smallpox

and for the control of poliomyelitis, diphtheria, and other diseases in many countries. There are many diseases in Africa—polio, whooping cough, tuberculosis, tetanus—in the control of which immunization plays a critical role. An Expanded Immunization Programme is being developed throughout the world by WHO.

To be effective in control of a disease in a community immunizations have to be given to a high proportion of the people—80% or more ('80% coverage'). The protective effect obtained when a high proportion of the population has been immunized is called *herd immunity*.

Herd immunity may be produced by:

(a) many people having a disease (active natural immunization)

(b) giving vaccinations to most of a population (active artificial immunization).

Unfortunately many communicable diseases cannot be prevented by immunization and some can only be partially prevented.

For further information on immunization, refer to the *Child Health* and *Community Health* manuals.

2. *Chemoprophylaxis*

Drugs that protect the host may be used for suppressing malaria, and for preventing infection with such diseases as plague and cerebrospinal meningitis.

3. *Personal protection*

The spread of some diseases may be limited by the use of some barrier agent against being infected, e.g. shoes to prevent getting hookworms from the soil, nets and insect repellants to prevent getting bitten by mosquitoes.

4. *Better nutrition*

Malnourished children get infections more easily and suffer more severe complications (e.g. measles), therefore the promotion of better nutrition will help to control the spread of

communicable disease. When famine is present epidemics are more likely to occur.

Effective control of a disease is most likely when a combination of methods—attacking the source, interrupting transmission, and protecting the host—are used at the same time.

Starting a disease control programme

In almost all areas there are diseases which could be reduced by relatively simple measures. The difficulty is to know how and where to begin.

One of the most important starting points is the understanding that something can be done. Too often people are unaware that high morbidity and mortality rates can be reduced. Much can be achieved both by preventive measures and by prompt simple treatment, for instance, of malaria, diarrhoea, pneumonia, and other diseases. However, without the understanding and active co-operation of individuals and communities few disease control programmes will succeed.

Before a communicable disease programme starts it is best if the following can be done:-

1. *Study the problem*
   Start with a survey to find out the extent and distribution of the disease and what people think and do about it—this is called community diagnosis.

2. *Prepare yourself for a programme*
   Study the information colllected. Work out yourself what needs to be done and discuss this with others. Make sure that the necessary equipment, drugs, transport, are available.

3. *Make a plan with your community*
   Make sure that the necessary understanding and motivation are there so that there will be co-operation. Ensure that you will have the support of other health workers.

4. *Follow-up*

Make a plan to measure what progress is made so you can compare the situation with what it was before and see what has been achieved—evaluation.

The actual application of the control methods can be undertaken by different groups of people and the responsibility for them is best thought of at three levels: individuals and villages, Health Centres, and District and other higher levels.

Individuals and villages responsible for:

Personal and household hygiene
Protective barriers—shoes, mosquito nets
Chemoprophylaxis—malaria
Avoiding infected venereal contacts
Protection of water supplies
Building and use of latrines
Rubbish collection and disposal
Vector control—clearing the surroundings, drainage
Avoiding bilharzial water

Health Centres responsible for:

Health Centre staff should support and encourage all of the above and with help from the district medical officer or health officer also set about:

Immunization
Reservoir control
Larviciding, molluscicide
Water protection and purification
Inspection of food supplies, markets and shops
Sanitary control of public toilets

District, Regional, and Central Ministry Services responsible for:

Mass immunization campaigns
Mass chemotherapy
Vector control schemes

Health legislation
Research into control methods
Famine relief
Epidemic control

ALL THESE ACTIVITIES NEED SUPPORTING HEALTH EDUCATION

The best communicable disease programmes start in a small way. They follow the wishes of communities stimulated by their own health worker. Then they grow and, with extra support, may be extended.

THE MOST IMPORTANT THING TO DO IS TO START

TABLE OF CONTENTS

Chapter one

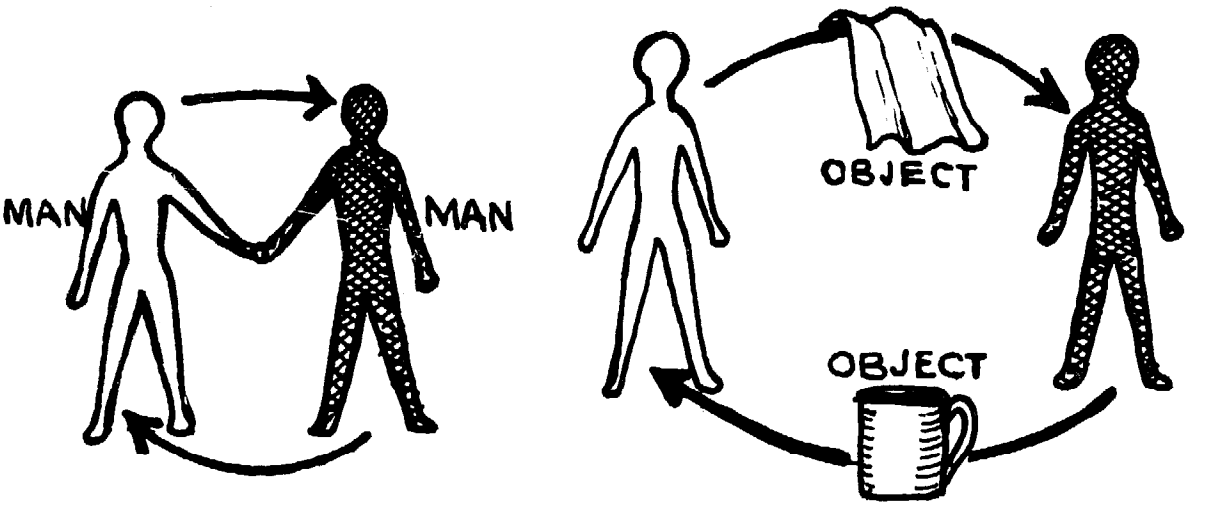
CONTACT DISEASES

Introduction

The diseases belonging to this group are transmitted by direct or indirect contact.

Direct contact is by skin-to-skin contact, for example by touching an infected person.

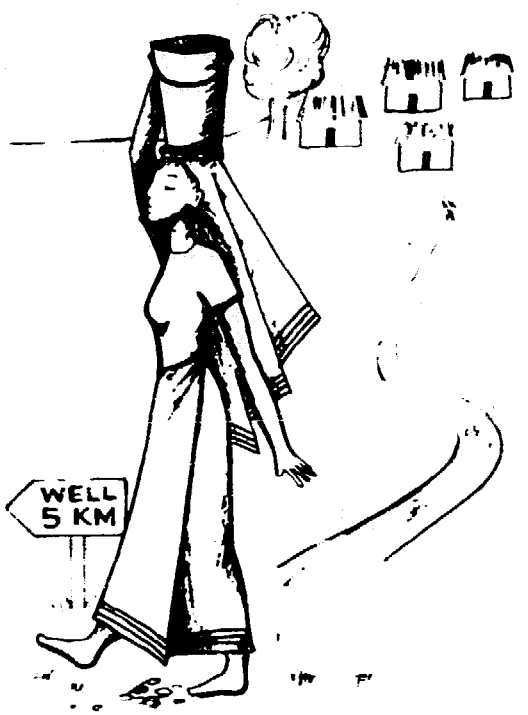
Indirect contact is through the handling of contaminated objects, such as clothing, bedding, dressings, eating utensils, but always by close contact.



Transmission of contagious diseases.

Contagious diseases tend to occur in *clusters* within households and within groups of people in close contact with each other, e.g. children's play groups, schools, and factories. Such groups of infected people are called 'clusters'.

Transmission of contagious diseases is encouraged by:



1. high population density (urban areas)
2. overcrowding
3. poor personal hygiene; an important factor in this is poor water supply or having to go far to fetch it. The poor personal hygiene so frequently seen in the dry rural areas is often due to the small amount of water available for home use
4. a special form of transmission through direct contact is that which occurs during sexual intercourse. Most of the diseases transmitted by skin-to-skin contact can be transmitted at the time of sexual intercourse.

A few diseases, of course, are only acquired through sexual contact (see Chapter 2 on Sexually Transmitted Diseases).

Control of contagious diseases can be directed at:

1. the source, e.g.
   - elimination of the reservoir by case-finding
   - treatment of individual cases
   - mass treatment
   - isolation of cases
2. the route of transmission, e.g.
   - environmental improvements
     - —proper refuse disposal
     - —water supply
   - improved living conditions
     - —better houses
     - —correction of overcrowding
   - personal hygiene
3. the susceptible person, e.g. personal protection.

Diseases transmitted by contact are:

Organism	Diseases	Remarks
Arthropods	Scabies Lice Other infestations, e.g. fleas, bedbugs	See *Note* below (some of these are not contagious but are considered here for convenience)
Fungi	Ringworm—tinea capitis —tinea corporis —tinea pedis —tinea unguium Candidiasis	(when in mouth = thrush)
Bacteria	Impetigo	See diseases caused by streptococci and staphylococci in Chapter 7 on Airborne Diseases
Chlamydiae	Trachoma	
Other	All diseases which can be transmitted through sexual intercourse	See Chapter 2 on STD

Note Infestation is the lodging, development, and reproduction of arthropods on the surface of the body or in the clothing.
Insects which may do this on the human body are:

Human lice
1. the body louse (Pediculus corporis)
2. the head louse (Pediculus capitis)
3. the pubic louse (Phthirus pubis)

Bedbugs blood-sucking parasites living in cracks of walls, woodwork, and furniture

Fleas
1. the human flea (Pulex irritans)
2. the jigger flea (Tunga penetrans)
3. rat fleas (Xenopsylla)

Flies the larvae of the tumbu fly and under special conditions larvae of other flies develop in the tissues of warm-blood living beings including Man.

Mites scabies (Sarcoptes scabiei).

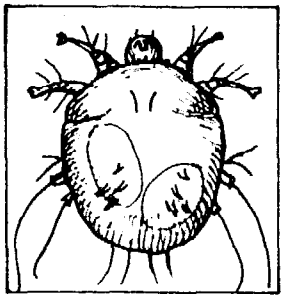
SCABIES

1. Scabies is a parasitic infestation of the skin characterized by severe itching of typical distribution.
   Synonym: *Upele*.

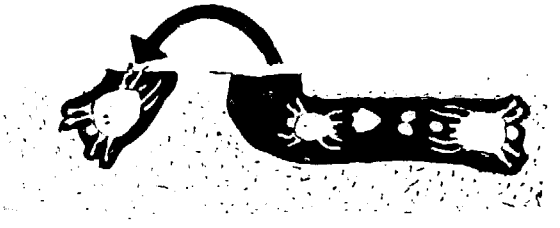
2. *Occurrence and importance*
   Scabies is very common in rural Tanzania. The prevalence in some villages is very high, especially if there is shortage of water. Infection and symptoms are more severe in children than in adults.

3. *Epidemiology*
   Scabies is caused by a small arthropod, the itch mite—Sarcoptes scabiei. The female mite enters the skin and makes a small tunnel or burrow.

   The burrow is always superficial, never below the horny layer of the skin. The skin selected for burrows is always thin and wrinkled giving the scabies rash its typical distribution. In the burrows eggs and faeces are produced. The eggs hatch in 4–5 days. The larvae leave the parent tunnel and bury themselves in the skin in other places. They do not make tunnels.



The itch mite (Sarcoptes scabiei) actual size just visible with the naked eye



Female mites in burrows in skin.

The infection is spread by direct close body contact as in bed, or in the contact between children and their parents. Transmission is also possible indirectly through bed clothes and clothing. Low socioeconomic conditions favour the spread of this disease.

SCABIES IS A DISEASE OF FAMILIES

Scabies is not a very contagious disease, and is uncommon in people who bathe regularly.

4. *Clinical picture*

Very often patients with scabies do not seek medical attention. The skin lesions may be so common that they are not considered to be a disease.

The skin lesions itch severely especially at night; this itching leads to scratching. So secondary infection with bacteria is almost inevitable. The diagnosis is made by finding an itchy rash with the typical distribution (place on the body).

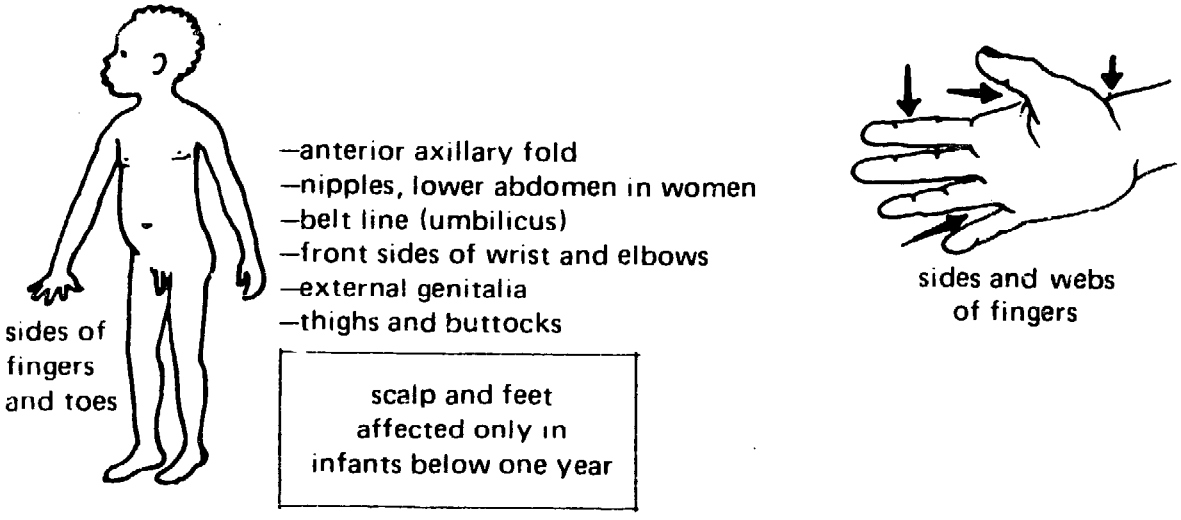
SEVERE ITCHING

+

TYPICAL DISTRIBUTION

↓

SCABIES



Commonest places for infestation.

Important notes:

i) Scabies is a contraindication to smallpox vaccination.

ii) Patients who are suffering from leprosy or other diseases

which interfere with normal sensation may not feel the itching caused by scabies. In these cases scabies can be very extensive. Thick crusts can form and even obstruct normal movement.

5. *Management of the individual patient*

The drug most often available is 10% benzyl benzoate emulsion (BBE). After the patient has taken a warm bath, a handful of BBE should be rubbed over the whole body by the mother or a nurse, preferably using her bare hands. After 24 hours the patient should bathe again and put on clean clothes. BBE has little effect on the eggs; therefore treatment should be repeated after 4–7 days to kill those larvae which have hatched since the first treatment.

BATHE AND RUB BBE OVER WHOLE BODY
BATHE AGAIN AFTER 24 HOURS
CLEAN CLOTHES
REPEAT AFTER ONE WEEK

The itching will not disappear immediately. Treat the itching symptomatically with powder or calamine lotion. Do not repeat the treatment too soon.

Tetmosol solution or soap is a more convenient alternative treatment but not always available.

Note 1. in infants the scalp should be treated as well, but protect the eyes carefully

2. in some patients secondary infection is so severe that a course of PPF is advisable. The penicillin has no action, however, against the scabies mites themselves.

6. *Control*

Regular bathing, washing of clothes, and frequent use of soap will control the disease. Health education, stressing the use of soap and regular bathing of children is essential in combating

this disease. Piped water supplies will greatly help to reduce the incidence.

SOAP CONTROLS SCABIES

Treatment of a single child infected with scabies is useless. As soon as he returns home he will be reinfected by the other members of the family.

TREAT THE WHOLE FAMILY

Scabies lesions in an adult may not be visible clinically. Therefore treat the whole household, even the apparently healthy. If Tetmosol soap is available, the simplest way to treat a family is to encourage every member to use it for bathing.

7. *Action*

If you treat scabies,

—treat the whole body
—treat the whole family
—give health education at the same time.

If scabies is a problem in a village,

—treat as many families as possible by making house-to-house visits
—do a survey of the children of the primary school.

Stress the importance of improvement of water supply in the ward development committee. When water supply improves you can also expect less of other diseases caused by water shortage (e.g. trachoma, louse infestations, gastroenteritis, dysentery).

8. *Summary*

Scabies is characterized by severe itching of typical distribution.
It is treated with BBE or Tetmosol.
Treat the whole body.
Treat the whole family.
Regular use of water and soap will control scabies.

PEDICULOSIS

1. Pediculosis is the infestation of the scalp, hairy parts of the body, or clothing with adult lice, blood-sucking parasites, and their larvae or nits (eggs).
   Synonym: Lice, *chawa.*

2. *Occurrence and importance in Tanzania*
   The head louse is common in schoolchildren and people with long hair which is seldom washed.

   Lice are especially common when people are crowded in bad hygienic conditions such as occur after natural disasters, in wars, and in refugee camps.

   Pediculosis is more common in cold areas where people seldom wash or change clothes (e.g. Rwanda). The body louse is the vector of some important diseases like epidemic (louse-borne) typhus and relapsing fever. Both diseases are very rare or do not occur at all in Tanzania.

   Pubic (crab) lice do not transmit disease.

3. *Epidemiology*
   Three different kinds of lice can cause pediculosis. The head louse and body louse do not differ very much and distinction is of no practical value. Both are transmitted by direct contact with an infested person or his personal belongings, especially clothing.

   Pubic lice are usually acquired during sexual intercourse.

   Life cycle. The female parasite produces several hundred eggs daily, each of which is attached to a hair with a special glue which makes it very difficult to remove the eggs (called nits).

   A larva hatches in 6–9 days and develops into a mature louse in 1–2 weeks.

4. *Clinical picture*
   Lice cause irritating bites, itching, and scratching with chronic secondary infection.

   Pubic lice live on pubic and sometimes other body hairs; head lice on scalp hairs; and body lice on clothing, from which they bite the covered areas of the body. Impetigo of the head and

neck may be a manifestation of head lice.

5. *Management of individual patients*

Head lice: apply 10% DDT dusting powder; cover hair with towel or cap for several hours; wash well afterwards, repeat after one week.

Body lice: take a hot bath and put on clean clothes; wash and iron clothing and bedding, or dust all clothing for the next 10 days with DDT powder 10%.

Crab lice: dust hairy parts of the body with 10% DDT powder and bathe after 24 hours; repeat after one week.

Against DDT-resistant lice, lindane can be used as a treatment.

Shaving of hair is a common and good practice, but not strictly necessary.

6. *Control*

Health education on the importance of water and soap to maintain cleanliness, and the laundering of clothes to destroy nits and lice.

Case finding by direct inspection of bodies and clothing of schoolchildren and adults living in camps.

Examination of household contacts.

7. *Action*

—Organize a school survey in co-operation with the headmaster.
—Examine household contacts of children with lice.
—Check all newcomers in refugee camps for lice as a routine.

8. *Summary*

Pediculosis is infestation with lice. The main theoretical medical importance of lice is their ability to transmit epidemic typhus and relapsing fever; the practical importance is the irritation they cause and the lack of cleanliness they indicate. Young adults with pubic lice should be suspected of also having a sexually transmitted disease.

OTHER INFESTATIONS WITH ARTHROPODS

Bedbugs are blood-sucking insects that are notoriously common in

seaports; they are not known to cause any disease. After the bite an itching papule may develop, which disappears within several hours. Treatment is not necessary. Powdering beds and floors with DDT is very effective. Bedbugs hide in the daytime in cracks in walls, beds, furniture.

Control
Systematic dusting of the infected and neighbouring houses with insecticides.

FLEAS
Synonym: *Viroboto.*

Pulex irritans is the human flea; it breeds in dwellings and visits human beings to take a blood meal; it will also attack animals if no human is available. The bite causes an erythematous spot in the centre of which a petechia can be seen. Treatment is not necessary.

Control
Eradication of fleas of man and of domestic animals with DDT. Periodic bathing of dogs and cats in antiseptic solution (dips).

Flea dermatitis (not contagious)
Allergic reactions to fleas and bugs can cause severe dermatitis, especially in children. It is characterized by small firm papules with an erythematous and sometimes swollen area around them. The condition may at first sight resemble chickenpox but the papules are firm and do not develop into vesicles and pustules.

Very often there is secondary infection and the differentiation from scabies may be difficult (but remember the distribution of scabies).

Treatment: calamine to relieve the itching, and powdering with DDT.

*Tunga penetrans** Synonym: Jiggers, *funza.*
Both male and female jigger fleas are blood-suckers of all warm-

*Jiggers and myiasis are not contagious diseases. Transmission is not possible direct from man to man as in other infestations, but is always through the external environment.

Three types of larvae can be distinguished, producing

obligatory myiasis: the larvae can only develop in living tissues

facultative myiasis: the larvae usually develop in decaying tissues but may also invade necrosing wounds

accidental or *intestinal myiasis:* the eggs or larvae are ingested; these are not killed in the intestine, where they may even develop further.

The tumbu fly is an obligatory parasite causing myiasis.

Tumbu fly or mango fly

Adult larva of tumbu fly.

The eggs of the tumbu fly are laid on dry soil, sand, or clothing which has been contaminated by the sweat or urine of man or animals, particularly rats, the normal hosts. The larvae hatch in two days. If a host comes into contact with the larvae, they are activated by the body heat of the host and penetrate the skin, producing a furuncle-like swelling. In this subdermal cavity the larvae will mature in 12 days. Contact with the host is often achieved through the eggs being laid on clothes that have been left to dry in the sun and not properly ironed afterwards.

Treatment: put Vaseline on the furuncle. The larva will try to come out to get air and in so doing, will lubricate its hole. Now it can be gently squeezed out or pulled out with a pin.

Control

Ironing of clothes.

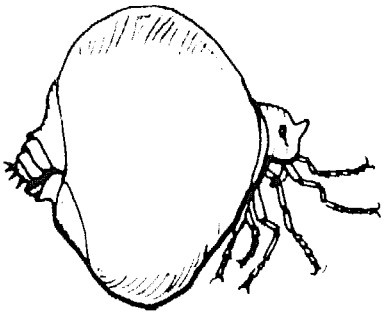
FUNGAL SKIN INFECTION (Dermatomycosis)

1. Dermatomycosis is a term applied to fungal infections of the skin and mucous membranes. Several types are identified according to causative organisms, site, and clinical appearance.

2. *Occurrence and importance*

   Fungal skin infections are usually problems of appearance

blooded animals. The male leaves its host after obtaining its meal but the female, when pregnant, burrows herself into soft skin, commonly between the toes. There they are in an environment rich in food, so they lay many eggs.

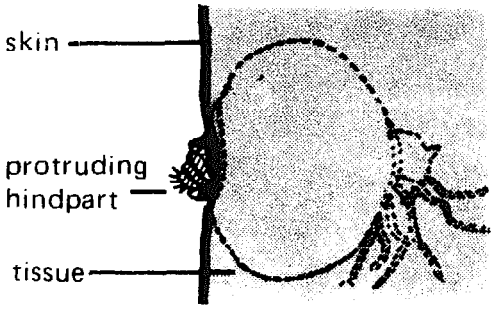


Female jigger flea with swollen abdomen full of eggs.

The hindpart of the flea protrudes through the skin so the eggs can be released.

The result is a small, round, itching, painful tumour with a point (the hindpart of the flea). Secondary infection is very common, even at times with tetanus.

Treatment: mechanical removal of the flea with a sterile pin, followed by an antiseptic dressing. Application of kerosene will kill the flea but results in ulceration of skin until the dead flea is expelled.



Position of flea in tissue.

Jiggers are common in Kigoma, Kahama, Ngara, Songea and Tanga.

Prevention: This flea cannot jump very well. Therefore only the feet are affected, and shoes provide protection. In children other parts of the body may be infected (buttocks). Keeping a house clean will reduce contact between man and fleas.

Xenopsylla are known as tropical rat fleas; they may also feed at times on human beings and are the vectors of plague.

FLIES: Tumbu fly or mango fly

Flies may produce a condition known as myiasis, which can be defined as the disease caused by fly larvae invading living tissues.

rather than illness, but it is important to distinguish them from leprosy and syphilis.

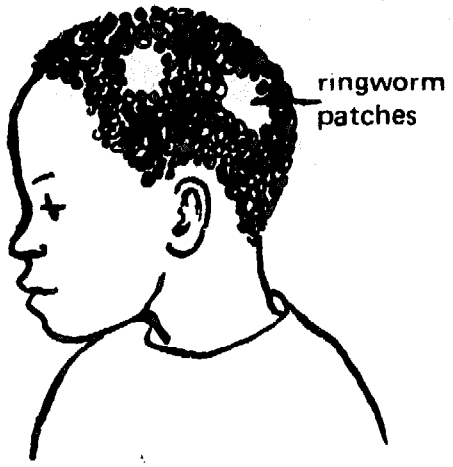
3. *Epidemiology*
   All fungi may be spread by direct or indirect contact. Genital infection (balanitis and vulvo-vaginitis) may be spread during sexual intercourse.

4. *Clinical picture* (see table p 15).

TINEA INFECTIONS

Synonym: Ringworm.

Tinea capitis (ringworm of the scalp) begins as a small papule which spreads to involve a larger area; hairs in the affected skin become brittle and break off easily. Occurs mainly in children under 10. It disappears after puberty.



Tinea capitis.

Tinea corporis (ringworm of the body) is characterized by flat, ring-shaped, spreading lesions. The ring lesions are reddish, vesicular or pustular, and may be dry and scaly, or moist and crusted. The central area often clears, leaving apparently normal skin.

Tinea pedis (ringworm of the foot or athlete's foot) is characterized by scaling and cracking of the skin between the toes, particularly the 4th and 5th toes.

Tinea unguium (ringworm of the nails) is characterized by thickening, discolouration, and brittleness of one or more of the nails. There is an accumulation of caseous material beneath the nail, which becomes chalky and disintegrates.

Tinea versicolor or *pityriasis* is a very superficial infection. The skin of the lateral sides of the face, neck, and chest shows many irregular, round, light-coloured areas.

CANDIDIASIS

Synonyms: Moniliasis, thrush, yeast infection.

There are four main manifestations:

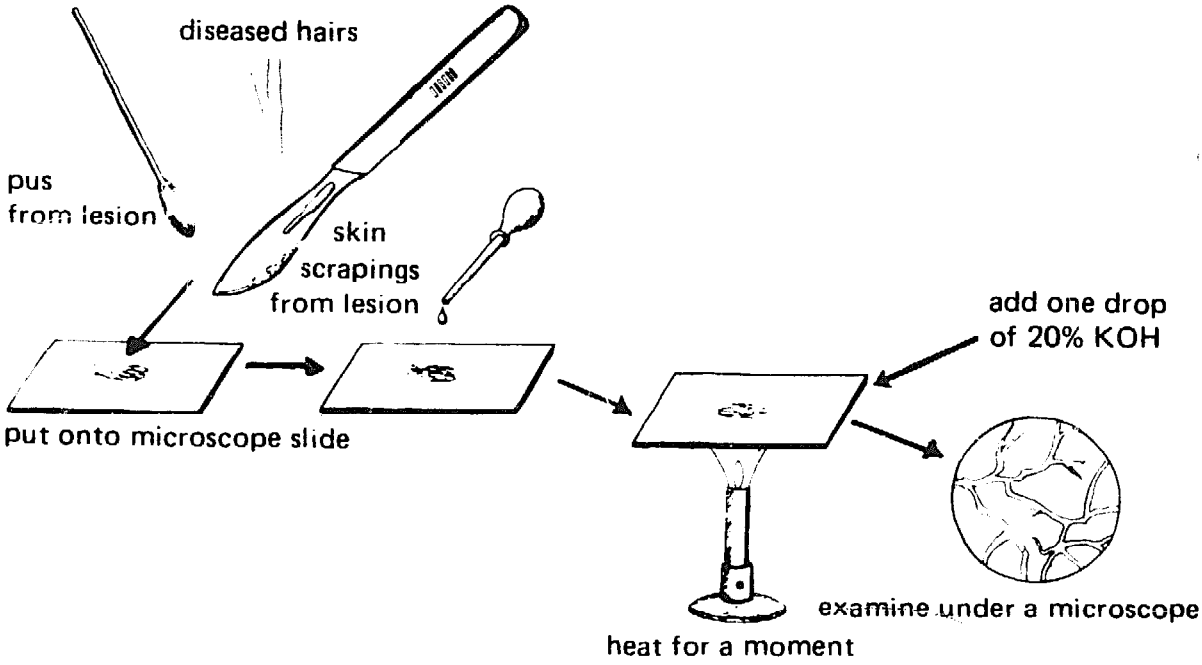
i) *Oral thrush:* White pseudomembrane on the mucous membranes of the mouth: the underlying epithelium is bright red; ulcers are rare; common in newborns, malnourished children and after antibiotic therapy.

ii) *Vulvo-vaginitis:* Pruritus and vaginal discharge, thick and white.

iii) *Balanitis:* Itching and redness of glans penis, swelling of prepuce, sometimes phimosis, eventually secondary infection with ulcers.

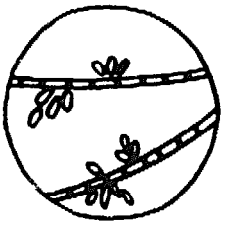
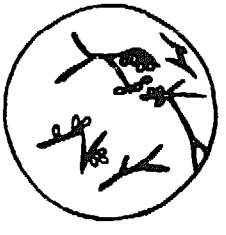
iv) *Intertrigo:* In deep body folds, e.g. axillae, under the breasts, groins, anus. The skin is erythematous, moist, and eroded. The boundaries of affected areas are sharp; watch for predisposing factors (urine for sugar).

Diagnosis: The diagnosis of fungal infection can be confirmed by laboratory investigations. The technique is easy and materials needed are simple:



Diagnosis of fungal skin disease.

Table summarizing characteristics of fungal skin diseases

Fungus	Risk factors	Main symptom	Diagnosis	Treatment
Ringworm				
Tinea capitis	Children under 10	Brittle hair areas of broken hair on scalp	 *branching filaments crossing borders of cells*	Whitfield's (Ung. acid. benz. co.) b.d. for 3 weeks
Tinea corporis	Excessive perspiration, hot humid areas	Ring-shaped lesions with central healing		
Tinea pedis (athlete's foot)		Scaling and cracking of skin between toes		Keep dry and clean, apply Whitfield's ointment daily
Tinea unguium	Nail injuries; corticosteroids	Thickening, discolouration or brittleness of nails		Surgical removal of nails plus Whitfield's ointment daily for 16 weeks
Tinea versicolor	Excessive perspiration, hot humid areas	Superficial small round light-coloured areas	 *short filaments, round thick-walled cells, budding*	Personal hygiene
Candidiasis				
Oral thrush	Measles Newborn: vulvo-vaginitis of mother; after antibiotic treatment	Pseudomembranes on mucous membranes		G.V. paint
Vulvo-vaginitis	Pregnancy; Diabetes	Vaginal discharge white and thick, itching		Nystatin vag. tab. G.V. paint
Balanitis	Lack of personal hygiene. (Acquired by sexual intercourse.)	Itching and redness	 *long filaments, oval cells, budding*	Hygiene G.V. paint
Intertrigo	Fatness; Diabetes; humid areas (armpits, breasts folds, between toes)	Intertriginous areas of redness (like eczema)		G.V. paint (Diet in diabetes.) Keep areas dry by exposure and talcum powder

5. *Management of individual patient*

All patients with fungal skin diseases should be given health education on personal hygiene.

Scrubbing with water and soap, followed by the application of fungicidal drugs is the basis of treatment. Fungicides need to be applied for a long time, at least 3 weeks.

For specific treatment see table.

6. *Control*

By general improvement of personal hygiene, and treatment of individuals. When many children with tinea capitis are seen, mass school treatment may be rewarding. Oral thrush of the newborn can be prevented by treating maternal vulvo-vaginitis in the third trimester of pregnancy.

7. *Action*

—Check your laboratory for diagnostic facilities.

—Check your knowledge of leprosy; do a KOH slide with appropriate skin diseases but also test for anaesthesia of skin in doubtful cases.

—Refer all skin diseases of uncertain diagnosis for investigation, but treat fungal infection yourself.

8. *Summary*

Dermatomycosis is caused by a wide variety of fungi. Infections should be differentiated from skin manifestations of leprosy and syphilis. Diagnosis and treatment can be done at HC and dispensary level.

TRACHOMA

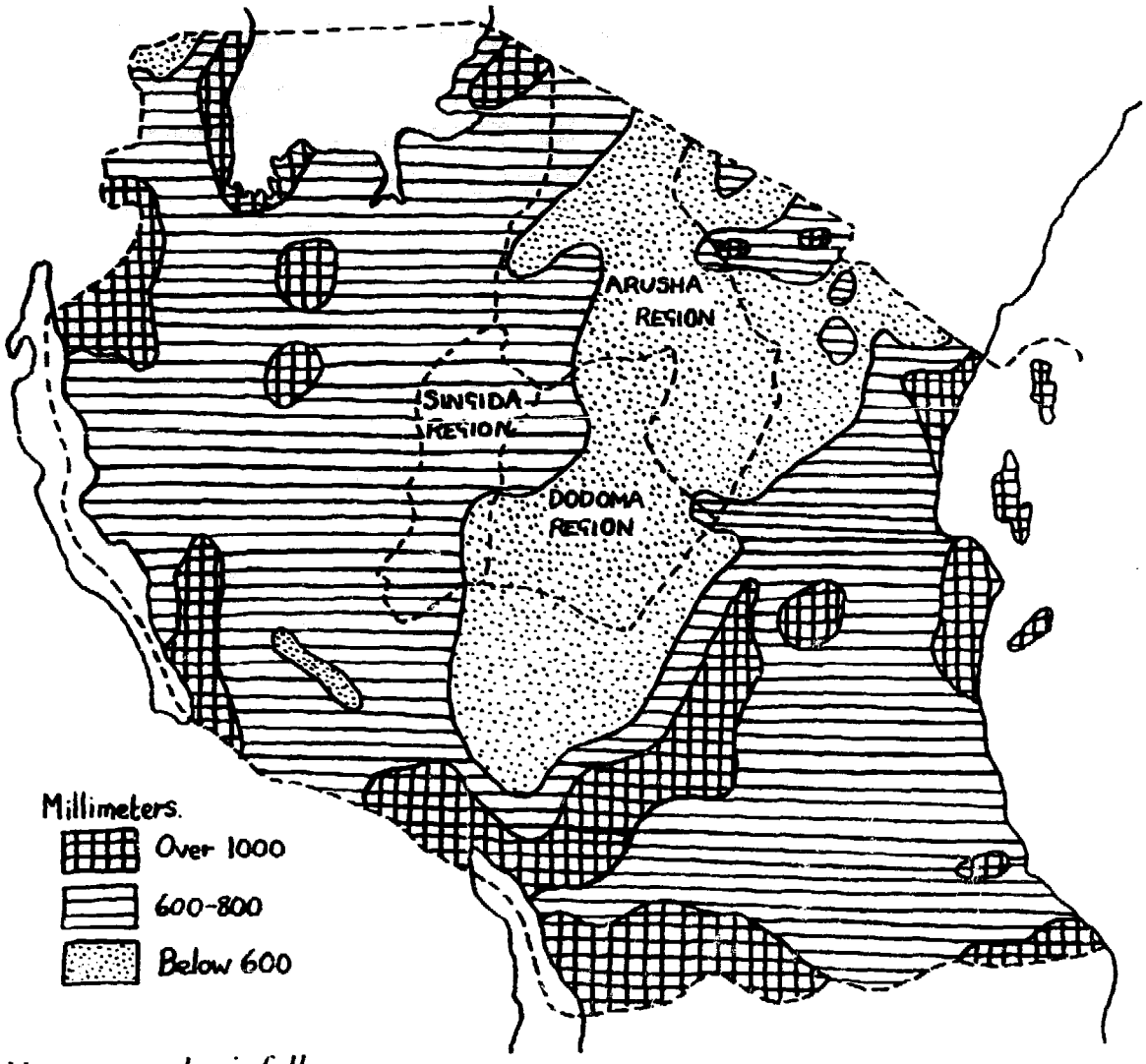
1. Trachoma is a chronic inflammation of the conjunctiva and cornea. It is characterized by follicles in the conjunctiva, followed by vascular invasion of the cornea (pannus), and in its later stages by scarring of the lids, leading to blindness.

2. *Occurrence and importance*

Trachoma is especially common in dry, dusty areas with poor hygiene, poverty, and crowded living conditions. It is mainly

seen in areas where water shortage is a daily problem.

Trachoma is common in Dodoma, Singida and Arusha Regions. The incidence of trachoma correlates well with mean annual rainfall.



Mean annual rainfall.

Trachoma is a very important cause of blindness, causing severe, permanent disability and a lot of human suffering.

3. *Epidemiology*

Trachoma is caused by the TRIC agent (from Trachoma and Inclusion Conjunctivitis), Chlamydia trachomatis, a very small gram-negative bacterium belonging to the group of Chlamydiae.

Other organisms belonging to this group cause psittacosis, NGU*, and lymphogranuloma venereum. It is now agreed that this agent is not a virus although it can only exist inside cells and can pass a bacterial filter. It differs from viruses in being sensitive to ordinary antibiotics, and in several other ways.

Transmission occurs by direct contact with ocular discharges of infected persons.

C. trachomatis is present in large numbers during the early stages of the disease and disappears gradually.

Secondary bacterial infection increases the risk of corneal ulceration. If corneal ulceration occurs, scarring is more severe and more rapid, possibly resulting in perforation of the cornea and so leading to blindness in an early stage of the disease.

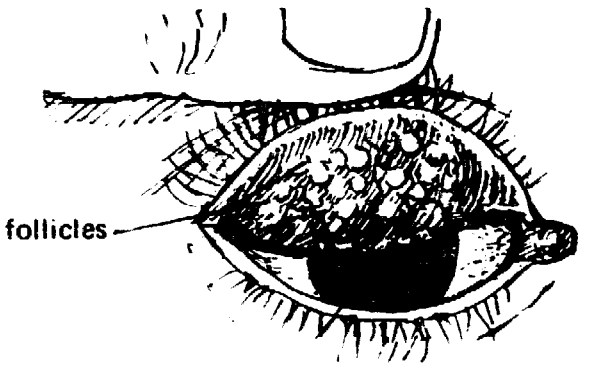
Flies are important in transmitting the secondary infection, but their role in the transmission of the trachoma is uncertain.

In endemic areas children get the infection at pre-school age. Although the disease normally progresses very slowly, blindness by the age of 10 is not uncommon. Girls tend to be more affected than boys, probably because the boys more often leave the house where water is short, and get more opportunity for bathing.

Because the early stages are the most infective, transmission is high among children.

4. *Clinical picture*

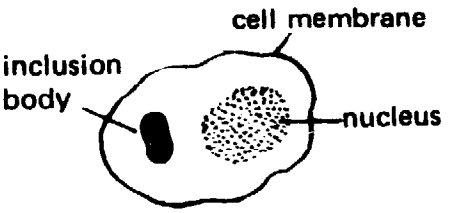
Trachoma develops in four stages.



Stage I Early trachoma

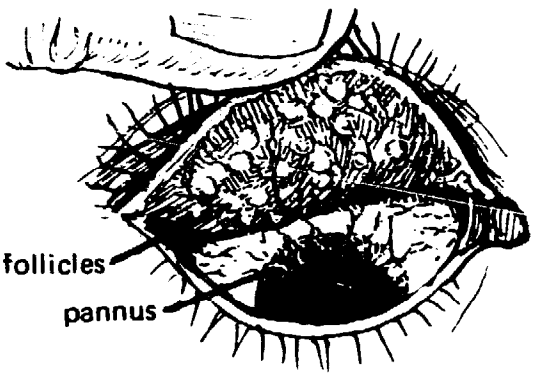
- Trachoma begins with red, watery eyes like ordinary conjunctivitis.
- After a month or more, small pinkish, gray lumps called follicles, form inside the upper lids. To see these, turn back

*NGU is non-gonococcal urethritis or non-specific urethritis (NSU) (see p 36)



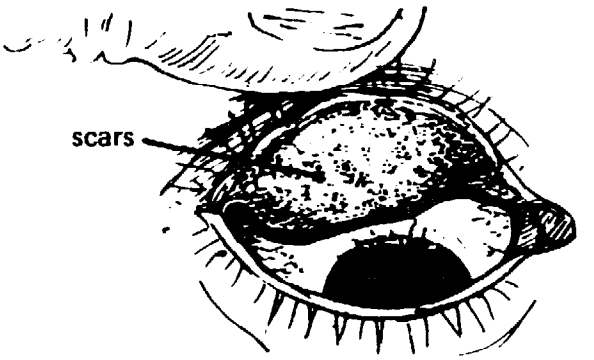
the lid as shown in the drawing.

- The white of the eye is mildly inflamed.
- At this stage there is little pus unless bacterial superinfection has come in. The pre-auricular glands are also not enlarged.
- Confirmation can be made by demonstrating the TRIC inclusion bodies in scrapings from the conjunctiva stained with Giemsa and/or Lugol's.



Stage II

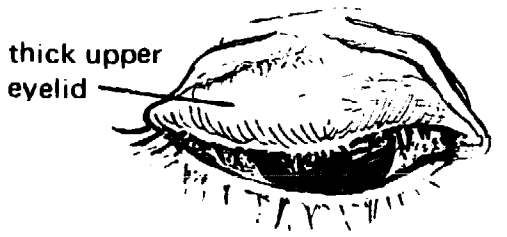
- At this stage of the disease if you look very carefully or with a magnifying glass, you may see that the top edge of the cornea looks greyish because it has many tiny red blood vessels in it (pannus).
- The combination of both follicles and pannus is almost certainly trachoma.



Stage III

- Trachoma is a self-limiting disease. After several years the follicles begin to disappear leaving whitish scars on the conjunctiva.
- In the cornea the small vessels retrogress. A diffuse haze remains on the cornea, but some vision might remain unless gross damage like rupture of

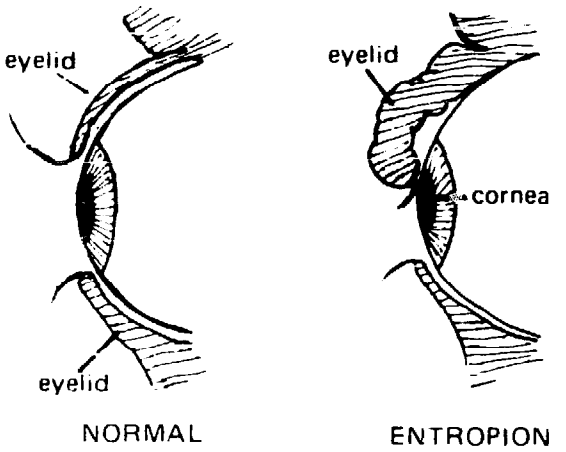
the cornea from corneal scars had occurred.



Stage IV

It is the scar formed after the healing process years after the onset of the disease, that does most damage. The scars are on the inner side of the conjunctiva.

- Scar tissue always retracts so the eyelid becomes thick, and turned inwards, this is known as entropion.



Side view of eyeball and eyelid.

These scars make the eyelids thick and short and may keep them from opening all the way, or they may pull the eyelashes down into the eye, scratching the cornea whenever the patient blinks. This is called trichiasis. The combination of entropion and trichiasis will completely destroy the cornea resulting in blindness.

5. *Management of the individual patient*

C. trachomatis is sensitive to tetracycline and sulfonamides. The treatment of choice is 3% tetracycline topical eye ointment* and oral sulfonamides systemically for two weeks. Sulfonamides are potentially dangerous drugs and can only be given under guidance and close supervision.

Effects of the treatment are slow and the success of treatment can only be judged after 3 months.

Patients in the third stage of the disease with entropion must be treated surgically. It is essential to do this as soon as possible

*Note. This is stronger than the usual 1% tetracycline eye ointment used for general eye infections, and it stings, but it is important to use it whenever it is available because it is much more effective.

because every blink increases the damage.

If no surgery is possible in your health centre, you can remove the inturned eyelashes by pulling them out with forceps.

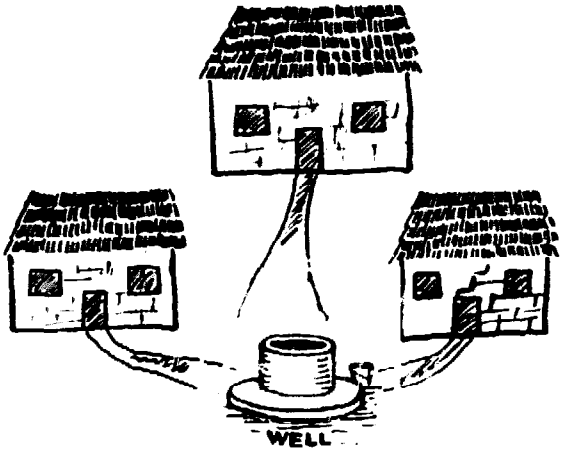
This should be done before you refer the patient. Entropion operations are simple and can be carried out at HC level, but pannus and opacity of the cornea have to be dealt with by an eye specialist.

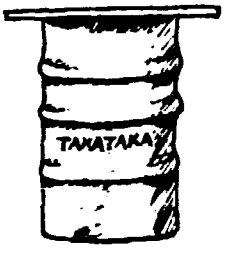
6. *Control*

Trachoma is a clear example of a disease caused by lack of water. The quantity of water is more important than the quality. The most effective way of dealing with the trachoma problem is to have enough water near people's homes. (If water was supplied to every house it would eradicate trachoma completely.)

IMPROVE WATER SUPPLY

Until such time as water supplies are improved, other preventive measures must be taken. Health education can teach people





CONTROL OF TRACHOMA

—supply of water near every house

—washing faces of children with soap and water

—regular bathing

—proper disposal of refuse

a few basic principles that will go a long way towards reducing the incidence of the disease.

Health education

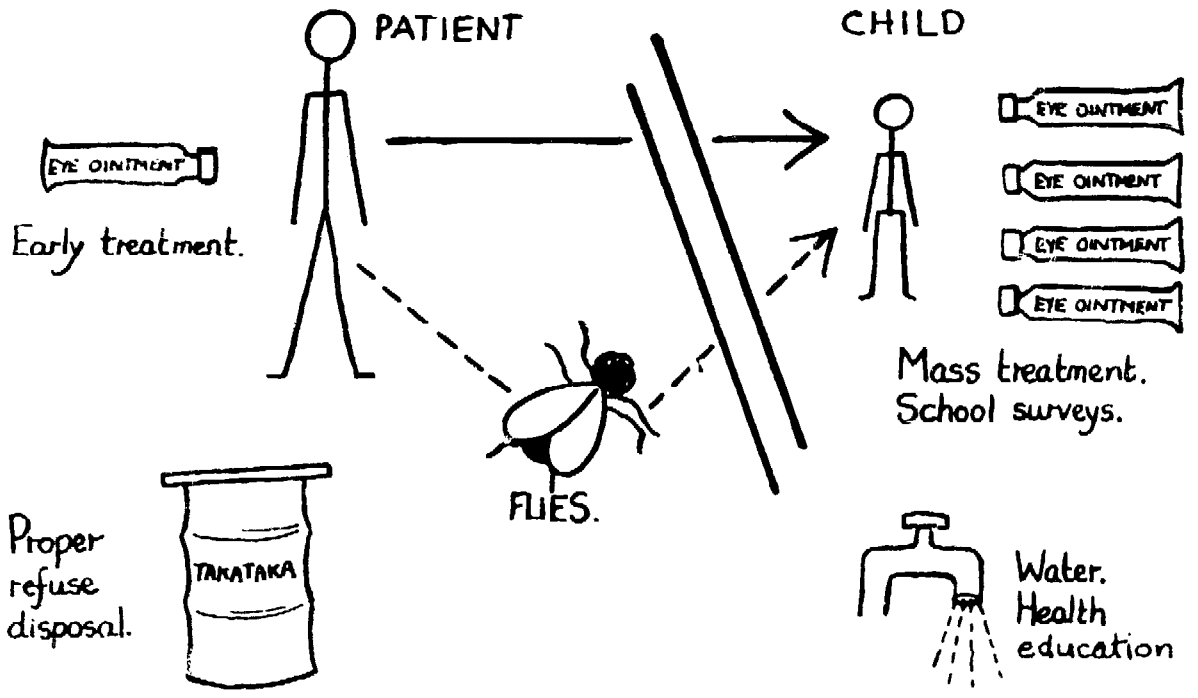
The importance of regular bathing and especially of washing the faces of children with soap and water should be stressed.

Improper refuse disposal will encourage fly breeding. Reduction of the fly population will reduce bacterial superinfections, and the severity of the disease as well.

Early treatment will reduce transmission because trachoma is most infectious during the early stages.

To detect as many cases as possible school surveys should be done yearly in endemic areas. A school teacher can give treatment to all children regardless of whether they are already infected or not.

For such mass treatment WHO recommends intermittent application of 3% tetracycline eye ointment for 3–5 days per month for 6 months.

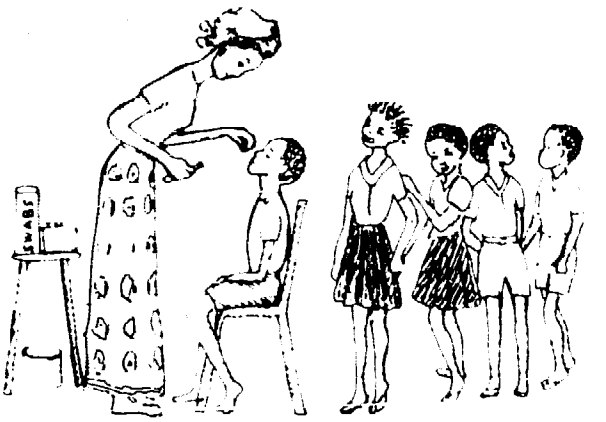


Breaking the trachoma transmission cycle.

7. *Action*

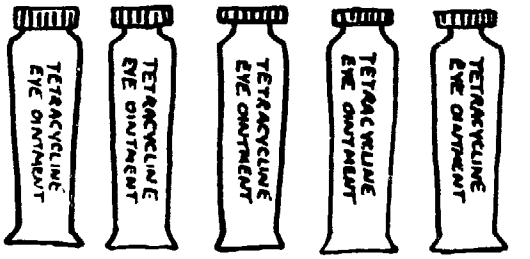
When trachoma is a problem in the catchment area of your HC do the following:

i) Arrange with head teachers for a day to do a school survey. If you find many trachoma cases ask the help of the teacher in treating them. Explain to him how to clean the eyes and how to apply eye ointment.



Explain to the head teacher how to clean eyes and how to apply eye ointment.

ii) Supply the head teacher with enough tetracycline eye ointment to treat all children for 3–5 days each month for 6 months.



Supply the head teacher with enough tetracycline eye ointment to treat all children.

iii) Stress in the ward development committee the importance and the urgent need of improvement of the water supply to provide *plenty* of water *nearby.*

iv) Give health education talks at MCH clinics on the transmission of trachoma and how it can be prevented and treated. Stress the danger of the complications.

8. *Summary*

Trachoma is an important cause of blindness in Tanzania.

Treatment with 3% tetracycline eye ointment should start early.

Main control measures: improvement of water supply, mass treatment.

ACUTE BACTERIAL CONJUNCTIVITIS

1. Acute bacterial conjunctivitis is a clinical condition starting with watery eyes, redness of the conjunctiva, followed by oedema of the eyelids and mucopurulent discharge.

   Synonyms: Pink eye, sore eye.

2. *Occurrence and importance*

   Conjunctivitis is common and widespread throughout Tanzania, particularly in hot and dry areas. It is usually self-limiting and does not lead to corneal or lid scarring.

3. *Epidemiology*

   Transmission is by contact with ocular discharges or secretions from the upper respiratory tract of infected persons through contaminated fingers, clothing, and other articles.

   Flies and eye-gnats may transmit the disease. Transmission is favoured when living conditions are poor, where rainfall is low, and where dust and flies are plentiful.

4. *Clinical picture*

   The patient presents with red eyes because of vascular injection of the conjunctiva. Usually there is oedema of the eyelids. The eyes are watery and the patient complains of a gritty feeling. There is no actual pain or loss of vision. Normally both eyes are affected. If this is not so, other conditions causing a red eye must be carefully excluded, particularly a foreign body in the eye.

5. *Management of the individual patients*

   Cleaning the eye regularly with water is often sufficient and should be the first priority in treatment. Penicillin eye ointment should not be used since it causes penicillin allergy. Chloramphenicol (or 1% tetracycline) eye ointment is best. Drops are more convenient for the patient but their action is short-lived. A relative of the patient should be instructed on how to apply the ointment or drops.

6. *Control*

   Personal hygiene; care and treatment of affected eyes. Proper refuse disposal to prevent fly breeding; health education.

7. *Action*

When conjunctivitis is a problem, give health education in schools and at the health centre, stressing the importance of personal hygiene and the dangers of improper refuse disposal.

8. *Summary*

Pink eye is caused by a number of different organisms, especially in hot areas where hygienic conditions are poor. Treat by cleaning the eye regularly with boiled water and eventually with chloramphenicol or tetracycline eye ointment.

Note: Most cases of conjunctivitis are caused by viruses (conjunctivitis is a common feature in systemic viral infections, e.g. measles). They do not need application of antibiotics. Only frequent cleaning of the eyes is important. Bacteria are only likely to be the cause when there is a mucopurulent discharge so only give antibiotic eye ointments to *purulent* conjunctivitis. In these cases the patient must use the antibiotic ointment for at least 5 days. Application of eye ointment only once is useless.

ANTIBIOTIC EYE OINTMENT
ONLY FOR PURULENT CONJUNCTIVITIS

Chapter two

SEXUALLY TRANSMITTED DISEASES

Introduction

The diseases belonging to this group are usually transmitted during sexual intercourse; hence the name sexually transmitted diseases or STD.

Some of these diseases have already been discussed in the previous chapter because they are contagious. During sexual intercourse there is close body contact, which is an ideal situation for transmission.

The causative organisms of the STD are very easily killed by drying or by cooling to below body temperature. Therefore transmission of these agents from one person to another can only occur under very special circumstances, mostly during sexual intercourse.

STD are very common in adults, but they are often hidden for fear of the opinion of others. Treatment is sometimes sought from private practitioners, pharmacists, nurses, or traditional healers*. This results in under-reporting of STD.

Single young men are a high-risk group for STD, as they satisfy their sexual needs with women who have many sexual partners (promiscuity). They may be professional prostitutes, barmaids, or persons who in other ways gain from casual sexual relationships. This group is called the promiscuous women pool (PWP). They are the reservoir of STD.

Risk factors are:

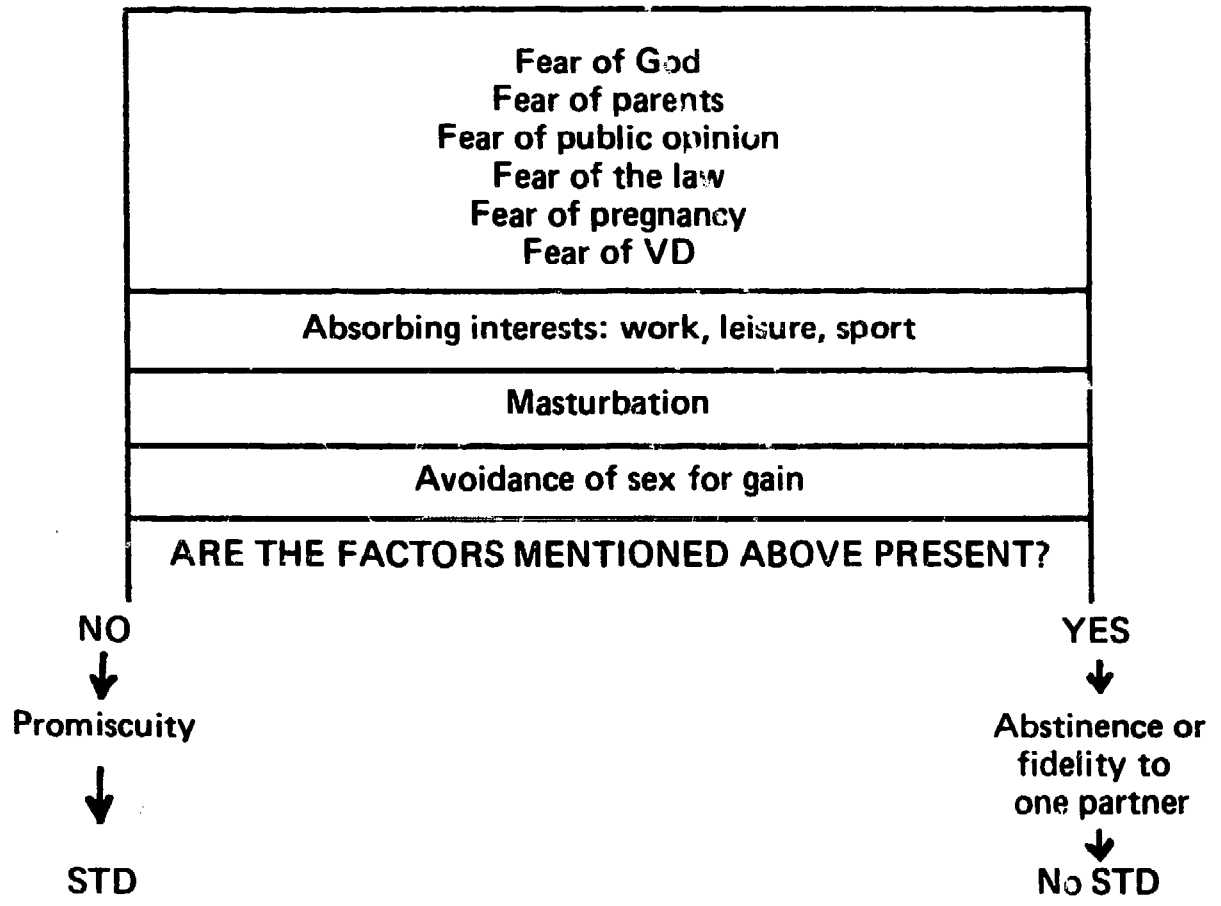
1. *Age:* 15 years and older.
2. *Marital status:* Unmarried people change their sexual partners more often and are more frequently exposed. Most of the

Note STD is the most important cause of illegal purchase and use of antibiotics.

women in the PWP are unmarried or divorced.

3. *Occupation:* Soldiers, policemen, students, seasonal labourers, and other people who are temporarily away from home tend to expose themselves more easily.
4. *Residence:* Due to industrialization and consequent urbanization there is usually a large group of single young men in towns. Women in towns may have more difficulty in earning their daily living than women in rural areas and may take up prostitution for money.
5. *Promiscuity:* Whether the sexual need of an individual is satisfied in promiscuous sexual behaviour or not depends on certain factors. The more of these factors present; the more the individual tends to abstain from promiscuity and the smaller the risk of acquiring STD.

   The factors are illustrated in the diagram.



STD are very difficult to control because the co-operation of those infected is needed and this is difficult to get.

Control of STD

1. ***Importance of early diagnosis and treatment***

This is the most important measure and not difficult to achieve. Facilities for diagnosis and treatment of STD must be freely accessible to everyone; confidential service is essential. One-by-one treatment and examination must be available in every HC, dispensary, and hospital OPD.

Patients should be encouraged to bring their contacts (including their husbands or wives) for treatment. Failure to do this will result in a high number of reinfections.

2. ***Elimination of the reservoir***

The reservoir is exclusively human; it includes untreated patients and especially unsuspected infections in the PWP. Regular examination and treatment of known prostitutes and other promiscuous women (barmaids) will reduce the reservoir, but will not completely eliminate the risk of infection.

CONTACTS MUST BE TREATED

3. ***Sex education***

This is a form of health education and should be directed at the groups at risk (sailors, students, soldiers, labourers etc). It is important to stress:

(a) dangers of sexual promiscuity
(b) the early signs and symptoms of STD
(c) the possibilities for individual prophylaxis
(d) normal sexual behaviour
(e) the dangers of antibiotic chemoprophylaxis.

(c) The possibilities for individual prophylaxis include: the use of rubber condoms (protecting both against STD and against undesired pregnancy); careful toilet of the genitals with soap

and antiseptic creams after exposure is advisable but will not give complete protection—also care should be taken not to overdo this or urethral irritation (chemical urethritis) may result, which may be mistaken for STD.

(d) Normal sexual behaviour: masturbation is a normal activity; probably 100% of adolescents masturbate. It does not cause disease, but if it is accompanied by feelings of guilt, there may be psychosomatic complaints.

(e) Dangers of antibiotic chemoprophylaxis: chemoprophylaxis may suppress the acute clinical manifestations but the disease may remain latent and progress silently. The widespread use of antibiotics in sub-curative doses is the major cause of the occurrence of drug-resistant strains.

4. *Other measures*
Provide quarters for married couples in work camps; provide recreational facilities for soldiers, students etc.

What action can you take from your HC to reduce the incidence of STD in the community?

—Arrange the line of flow in your OPD in such a way that confidential service is guaranteed for every individual patient.

—Arrange with the head teachers of the primary schools for lectures to be given on sexual behaviour, birth control, and STD to the standard six pupils before they leave school. Do this every year.

—Give health education on STD to groups at risk (students, soldiers, labourers).

—Suggest periodical check-ups for STD for barmaids and other women at risk (this would be ideal, but is very difficult in practice).

Clinical picture of STD

STD show in two different ways:

1. STD showing as genital discharge
   - Gonorrhoea
   - Non-gonococcal (non-specific) urethritis
   - Trichomoniasis
   - Candidiasis
2. STD showing as genital sores or lumps
   - Syphilis
   - Chancroid
   - Condylomata acuminata (venereal warts)
   - Herpes simplex
   - Molluscum contagiosum
   - Scabies
   - Pediculosis pubis (crab lice infestation).

It has been a frequent practice to diagnose all genital sores on clinical grounds as syphilis and all cases of genital discharge as gonorrhoea. This leads to waste of penicillin.

A better approach is to make gram stains of all cases of genital discharge.

STD important in Tanzania are listed in the table opposite:

When the only generally recognized STD were syphilis, gonorrhoea, and chancroid, these used to be called the *venereal diseases* (love diseases), because they, with NGU (NSU), are only transmitted by sexual intercourse. The name STD is preferred these days, however, to include also the other conditions mentioned which are *mainly* transmitted sexually but *can* also be transmitted in other ways.

GONORRHOEA

1. Gonorrhoea is an acute or chronic purulent infection of the urogenital tract.

   Synonym: 'Clap', *kisonono*. Abbreviated: gc

Causative agent		Disease
Bacteria:	spirochaete	syphilis
	gonococcus	gonorrhoea
	H. ducreyi	chancroid
	chlamydiae (TRIC agent)	non-gonococcal (non-specific) urethritis (NGU, NSU)
Viruses:	herpes virus	herpes simplex of genitals
	other	condylomata acuminata
		molluscum contagiosum
Protozoa:	Trichomonas vaginalis	trichomoniasis
Fungi:	Candida albicans	candidiasis (moniliasis, thrush)
Parasites:	Sarcoptes scabiei	scabies
	Phthirus pubis	pubic lice

2. *Occurrence and importance*

Gonorrhoea is by far the commonest STD especially in rural areas. It can cause sterility in both males and females and cause a serious decline in birth rate in some communities. 'One-child sterility' is frequently seen in Tanzania and is most often caused by gc. During sexual contact a woman may become pregnant and acquire gonorrhoea as well. During and after delivery pelvic invasion is easy and the result is a bilateral salpingitis with tubal obstruction. One-child sterility is one of the major problems of gynaecology clinics.

Gonococcal ophthalmia of the newborn may cause blindness. Gonorrhoea in females may cause death due to salpingitis and peritonitis; in males it may cause urethral stricture resulting in urine retention, hydronephrosis and uraemia, also causing death. The frequency of these fatal complications is not known.

3. *Epidemiology*

Gonorrhoea is caused by the gonococcus, a gram-negative kidney-shaped diplococcus, which can only grow intracellularly (Neisseria gonorrhoeae).

The gonococcus is not able to penetrate the skin or squamous epithelium. Contact of mucous membranes with gonococcal pus is usually only possible during sexual intercourse.

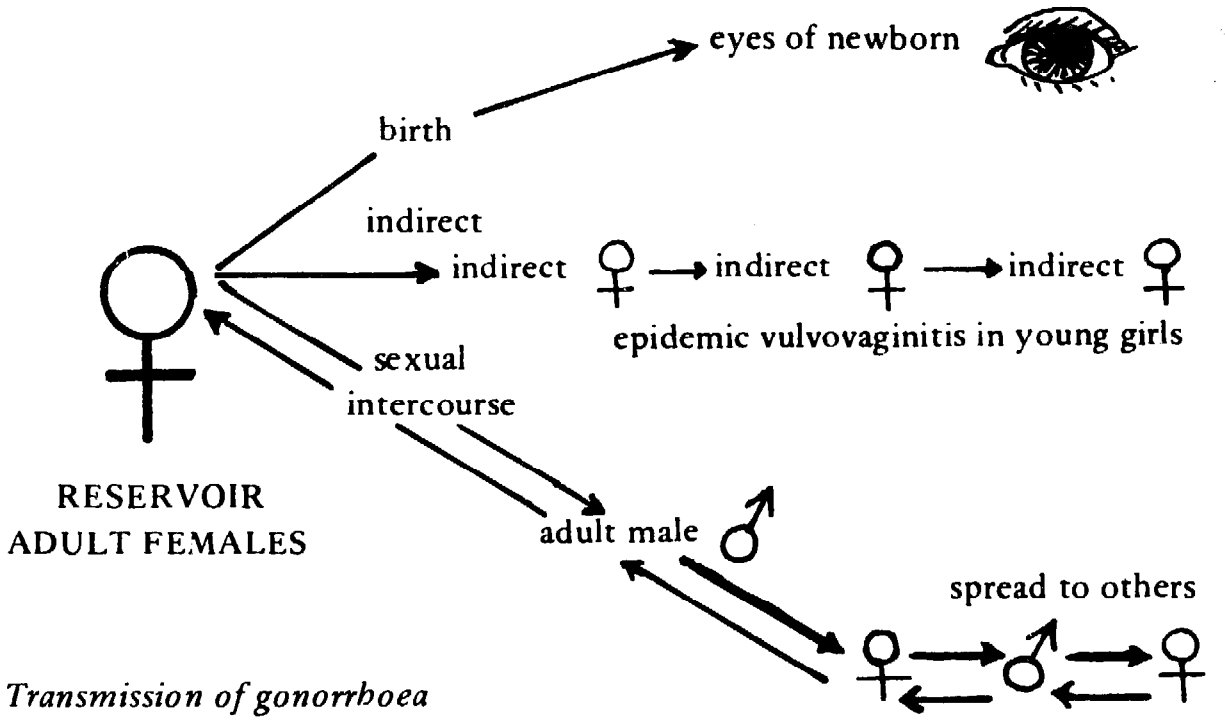
The following conditions are exceptions to this rule:

(a) *Gonococcal ophthalmia neonatorum* This is an acute inflammation of the conjunctiva of the newborn. The infection is contracted during passage through the birth canal.

(b) *Gonococcal vulvo-vaginitis* The vagina of adult women is lined with squamous cells which contain glycogen. Through bacterial lysis this glycogen is metabolized into lactic acid, thus producing a low pH. This low pH protects the vaginal wall from invading gonococci.

The glycogen content of the cells is determined by oestrogen levels and is therefore low before puberty. So before puberty the vagina is less resistant to gonococcal infections.

Through use of shared towels or clothing the infection can spread from one girl to another.

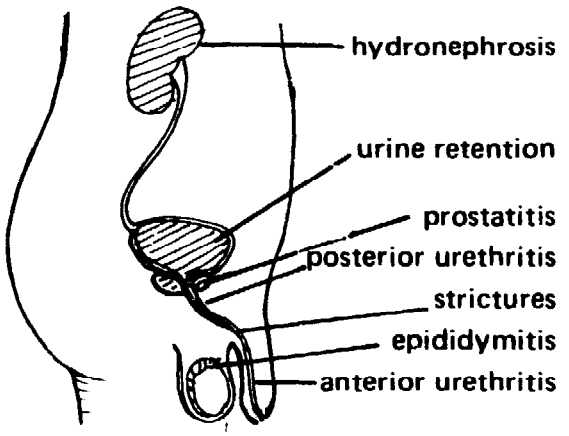


Transmission of gonorrhoea

4. *Clinical picture*

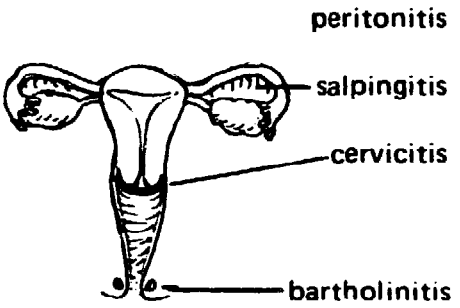
Gonorrhoea in males starts 2–10 days after the coitus in which the infection was acquired. The initial symptoms are painful micturition followed by usually thick yellow purulent discharge from the urethra.

In females the urethra is short and urethritis is often not noticed. Cervicitis may cause vaginal discharge but very often some discharge was present anyway and an increase in discharge is not regarded as abnormal.



Males

Anterior urethritis with thick yellow purulent discharge; complicated by posterior urethral sinuses, epididymitis, prostatitis, and infertility. Urethritis may cause urethral stricture resulting in urine retention and eventually in hydronephrosis and kidney failure.



Females

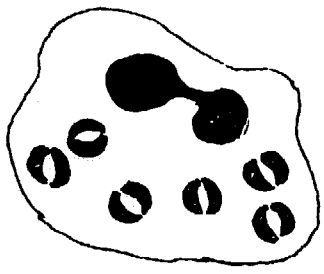
Initial urethritis or cervicitis often unnoticed; later Bartholinitis and pelvic invasion especially after menstruation or childbirth, resulting in salpingitis or pelvic peritonitis, both of which can give tubal obstruction with the danger of ectopic pregnancy or infertility afterwards.

IN PID AND STERILITY LOOK FOR GC

In both *males* and *females* gc can be complicated by septicaemic spread, resulting in arthritis, endocarditis or meningitis, but these are rare.

The diagnosis can be confirmed with a urethral smear (man),

or a cervical smear (woman). Gram stain will show: gram-negative diplococci lying inside pus cells (intracellular) (Some may escape and lie outside cells but the diagnosis should not be made unless intracellular diplococci are definitely seen.)



Gonococcal ophthalmia is characterized by a short incubation time (3 days or less) and mucopurulent discharge from one or both eyes.

5. *Management of the individual patient*

Uncomplicated cases should be treated with a single or double dose of antibiotics. If you prescribe a 5-day course it will not be completed by most of the patients and will lead to insensitivity, with all the dangers of antibiotic-resistant gonorrhoea in the community.

First choice: 4.8 mU PPF (2.4 mU in each buttock); repeat same dose next day.*

Second choice or in suspected resistance: males and females 8 x 500 mg tetracycline, swallowed in the presence of the prescriber or dispenser.

All complicated cases should be admitted and given a 7–10-day course of high doses of penicillin.

There are four important causes of treatment failure in gonorrhoea:

i) wrong diagnosis: non-gonococcal urethritis, chemical urethritis, venereophobia
ii) too low dose of antibiotic
iii) no treatment given to sexual partner, resulting in reinfection
iv) post-gonococcal urethritis, see p 36.

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*If probenecid is available, give 1.0g orally half-an-hour before the penicillin. Probenecid blocks the excretion of penicillin in the kidneys. This will result in a longer-lasting high level of penicillin in the blood. It is the high blood level of penicillin which is the essential part of the treatment of gc.

DOUBLE OR SINGLE DOSE TREATMENT IN UNCOMPLICATED CASES

Note: Treatment of gc with penicillin can mask concurrent syphilis (see p 41)

Ophthalmia neonatorum

Specific treatment: parenteral crystalline penicillin plus 1% tetracycline eye ointment.

Some ophthalmologists prefer the continuous application of crystalline penicillin eye drops. Use common crystalline penicillin powder. Dilute to 10,000 IU per ml and put one drop in each eye at least every 10 minutes for 24 hours. Teach the mother to apply the drops.

6. *Control*

Gonorrhoea in adults—see introduction.

Ophthalmia neonatorum can be completely prevented; ideally this should be done by the use of one drop of 1% silver nitrate in each eye of every newborn. The problem with silver nitrate solution is that it has to be freshly prepared every week. If fresh silver nitrate is not available the eyes of the newborn can be washed out with normal water; this will remove the gonococci because they will remain on the surface of the mucous membranes for some hours before they invade the tissues.

EYE WASH FOR EVERY NEWBORN

7. *Action*

—Do not treat urethritis routinely as gc; confirm diagnosis with smear; non-gonococcal urethritis is as or more frequent than frank gonorrhoea.

—Check all gc cases for concurrent syphilis after 8 weeks.

—Treat all cases of salpingitis and PID as complicated gc (admission or referral).

8. *Summary*

Gc is an infection of the urogenital tract transmitted by sexual intercourse; ophthalmia neonatorum is an eye infection of newborns, easily prevented by routine eye washing at birth.

Uncomplicated gc is treated with a single or double dose of penicillin. Prevention is concentrated on early diagnosis and treatment of patients and contacts.

NON-GONOCOCCAL (NON-SPECIFIC) URETHRITIS

1. Non-gonococcal urethritis is an inflammation of the urethra not caused by the gonococcus. It is still commonly called 'non-specific' urethritis, but as there are several specific causes (e.g. chlamydiae, trichomonas) it is better to call it non-gonococcal.

2. *Occurrence and importance*

Although NGU is at least as common as gonorrhoea, urethral discharge is generally automatically diagnosed as being gonorrhoeal and treated with penicillin, which has little or no effect on NGU. This leads to unjustified doubts as to the value of penicillin in gonorrhoea.

3. *Epidemiology*

NGU may be caused by mechanical or chemical irritation of the urethra or by micro-organisms such as chlamydiae (see Trachoma p 16) or trichomonads transmitted by sexual intercourse. Patients successfully treated for gonorrhoea may also have a slight continuing urethral discharge for a time. If no gonococci are to be found in this discharge, it is probably because the urethral epithelium has not fully recovered (post-gonococcal urethritis).

Fear of having caught an STD (venereophobia) may also lead to excessive use of antiseptic or squeezing of the penis, and so to slight discharge, apparently justifying the patient's fear.

4. *Clinical picture*

The discharge of NGU is often thinner and whiter than typical gonorrhoeal discharge, but it may look just like typical thick

yellow gc pus, and on the other hand gonorrhoea may sometimes present with a thin watery discharge. There is no way of being certain which is which without microscopy. A careful history may help the diagnosis (history of catheterization, foreign bodies introduced into the urethra, excessive masturbation, use of antiseptics to prevent STD, recently treated gonorrhoea). If there is no apparent cause of chemical or mechanical urethritis, and no gonococci are seen microscopically, the urethritis is most likely to be chlamydial.

5. *Management of the individual patient*
   Chlamydiae are sensitive to tetracycline; 250 mg qid for one week is sufficient in the male. The other condition will improve when further trauma of the epithelium is avoided. Reassurance and rest (that is, abstinence from sexual intercourse) are all that are needed. A sedative might help.

6. *Control*
   As for other STD.

7. *Action*
   Always order a wet smear (for trichomonads) and a gram stain in all cases of urethritis; treat according to findings.

8. *Summary*
   NGU is inflammation of the urethra due usually to chlamydiae but sometimes to non-specific irritants.

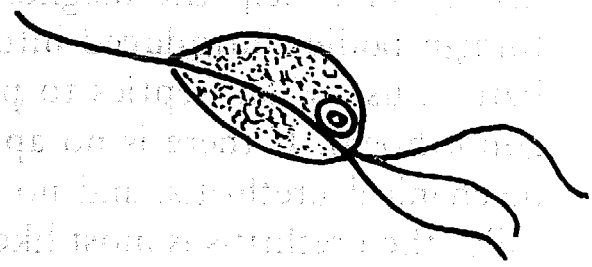
TRICHOMONIASIS

1. Trichomoniasis is a protozoal infection of the genital tract of males and females.

2. *Occurrence and importance*
   Trichomoniasis is a very common infection of the female genital tract, 10% of all women are infected. In males it may cause a urethritis, often misdiagnosed as gc and consequently wrongly treated with antibiotics.

3. *Epidemiology*

Trichomoniasis is caused by Trichomonas vaginalis. Transmission is by sexual intercourse or by indirect contact through contaminated clothes and other articles. The parasite likes an acid environment (pH 4) as found in the vagina of adult women during the reproductive period.



Trichomonas vaginalis.

4. *Clinical picture*

Trichomonas infection in women is usually symptomless, but it may cause itchy vaginitis and increased vaginal discharge. The discharge is white and foamy. In males the infection causes urethritis, often diagnosed as gc and treated with penicillin. As the causative organism is a protozoon, this treatment has no effect and only results in a disappointed patient running from one practitioner to another in order to get rid of his complaint. The diagnosis is easily confirmed once one thinks of it. A fresh drop of vaginal or urethral discharge is examined directly for moving protozoa. It is essential to do the examination with a fresh specimen; therefore it is better to examine the specimen in the OPD than to send the specimen to the laboratory.

If a patient complains of urethral discharge it is best to make a gram stain to exclude gc and at the same time examine a fresh specimen for trichomonas.

5. *Management of the individual patient*

The parasite will disappear when the urine is alkaline; therefore Mist. potassium citrate is the treatment of choice for males. Vaginitis can be treated topically with di-iodohydroxyquinoline (Floraquin) pessaries (to be inserted in the vagina). Disadvantage: expensive. Alternatively males and females can be treated with a single dose of metronidazole (Flagyl) 2 g (that is 10 tablets), swallowed under supervision. Disadvantages: incompatible with alcohol, difficult to obtain at HC level.

If neither Flagyl nor Floraquin is available and the vaginitis is troublesome, the vagina can be painted daily with G.V. This treatment is messy and laborious but effective. Treatment will only be successful when both sexual partners are treated and when they abstain from sexual intercourse for two weeks.

6. *Control*
   —As for other STD—see introduction.
   —Improvement of general hygiene.

7. *Action*
   Always order a wet smear together with a gram stain in all cases of urethritis; treat according to findings.

8. *Summary*
   Trichomoniasis is a common infection of the genital tract in both males and females. It may cause urethritis in males. Treatment when indicated should be cheap and simple. Antibiotics are of no use.

SYPHILIS

1. Syphilis is a disease characterized by a primary lesion, a later secondary eruption on the skin and mucous membranes, then a long period of latency, and finally late lesions of skin, bones, viscera, CNS and cardiovascular systems.

   Synonyms: *Kaswende*, lues.

2. *Occurrence and importance*
   Yaws and syphilis are closely related. In a community with yaws there will be no syphilis, but as yaws has been practically eradicated in East Africa, the incidence of syphilis is increasing. Syphilis is said to occur more often in urban societies, although a high incidence is also found in some rural communities.

   Syphilis is a very slowly progressing disease, but it is disabling and may be fatal after 10–20 years.

   The same groups are at risk as for other STD—see introduction—but medical and laboratory workers have to be included as

they can, rarely, acquire the infection accidentally.

3. *Epidemiology*

Syphilis is caused by a spirochaete (Treponema pallidum). The spirochaete enters the body through the mucous membranes and the intact skin. Outside the body the spirochaetes die rapidly.

Conditions for transmission are optimal during sexual intercourse but skin-to-skin contact can be enough for transmission.

Spirochaetes invade the blood-stream after infection and so blood may be infective.

Summary: usual transmission—*sexual intercourse*
unusual transmission—*accidental,* by touching infective tissues (dentists, midwives, and other medical personnel)
—*via blood transfusion*
—*congenital* infection may occur before birth, in the case of an infected mother.

4. *Clinical picture*

The incubation time varies from 10 days to 10 weeks. Most frequently 3 weeks. Patients usually present with a genital sore, the hard chancre (hence *Kaswende ngumu*). This is part of Stage I of the disease.

Primary syphilis

This consists of the hard chancre, the primary lesion of syphilis, together with a regional lymphadenitis.

The hard chancre is a single, painless, ulcer on the genitalia or elsewhere (lips, tongue, breasts); even when not treated it will heal spontaneously in a few weeks. The lymph glands are bilaterally enlarged and not painful. There will be no suppuration (see also comparison between soft and hard chancre, p 45).

Secondary syphilis

Four to six weeks after the primary infection a generalized secondary eruption appears, often accompanied by mild constitutional symptoms. These early rashes tend to be symmetrical,

quickly passing, and do not itch. These early skin lesions are very infective and many spirochaetes can be demonstrated in them.

The secondary manifestations will also disappear spontaneously within a few months. A period of latent infection then precedes the third stage of the disease. In this latent period later lesions of skin and mucous membranes may occur. These lesions are not symmetrical and are more lasting than the early lesions.

CNS signs and eye lesions may develop.

Tertiary syphilis

This stage is characterized by destructive, non-infectious lesions of the skin, bones, viscera, and mucosal surfaces.

Other disabling manifestations occur in the cardiovascular system (aortic incompetence, aneurysms), or central nervous system (dementia paralytica, tabes dorsalis).

Syphilis in pregnant females

According to the severity congenital syphilis can result in congenital abnormalities (early or late manifestations), stillbirth, or repeated abortions.

Notes:

—Primary syphilis in females is often missed because the chancre is symptomless and not easily seen.

—No genital lesion will be present when infection is acquired through infected blood (congenital, blood transfusion).

—Gonorrhoea and syphilis are sometimes acquired at the same time. When the gc is treated promptly with PPF, the first stage of syphilis may be suppressed. The patient will feel cured and safe but his syphilis will progress into the second and third stages.

Therefore it is wise to screen cases of gc routinely for concurrent syphilis. A Kahn test or VDRL should be done 8 weeks after infection.

AFTER GC WATCH FOR SYPHILIS

Diagnosis

(a) Serological tests are based on antibodies formed. These will be positive only 6 to 8 weeks after infection. Most hospital laboratories are able to perform these tests (Kahn or VDRL).

(b) The spirochaete causing syphilis is difficult to stain. It can be demonstrated in smears by using a special illumination technique: dark ground illumination. Smears from the primary lesion (hard chancre) or from skin lesions in the early secondary stage will show the spirochaetes. As the technique requires special skills only a few laboratories will perform this examination.

(c) Herxheimer's reaction: after penicillin injection, due to a large number of killed spirochaetes a patient with late syphilis may react with fever. This may be considered as confirming the clinical diagnosis.

5. *Management of the individual patient*

During treatment the patient should of course abstain from sexual intercourse.

Drug of choice PAM (oily penicillin); dose 3.0 mU + 1.5 mU on the 1st, 4th, and 7th day. Alternative PPF (watery suspension) 0.6 mU for 10 days.

Syphilis is cured by low blood levels of penicillin, but only if the penicillin is in the blood for a period of 10 days.

SYPHILIS: LOW LEVEL, LONG TIME

GONORRHOEA: HIGH LEVEL, SHORT TIME

6. *Control*

In general the same methods apply as in the control of gonorrhoea. Condoms give a better protection against gonorrhoea

than syphilis because the spirochaetes can penetrate the intact skin.

Medical workers and midwives should be careful in examining suspect lesions and should preferably wear gloves when doing pv examinations.

Blood transfusion donors can be screened routinely but it is cheaper to give the receiving patient a single dose of PPF 0.6 mU. Blood which has been stored in the fridge for three days or more is safe for use.

If there are many cases of syphilis in your area it may be wise to screen all pregnant women routinely in order to prevent congenital syphilis. Arrange for this with the nearest laboratory which is able to perform the VDRL or Kahn test.

7. *Action*
   - —Check all gc cases for concurrent syphilis.
   - —Think of syphilis in all poorly understood skin diseases, heart disease, psychiatric and neurological disorders.

8. *Summary*
   Syphilis is a long-lasting disease. It is transmitted by sexual contact. Early diagnosis and treatment are essential for control.

CHANCROID

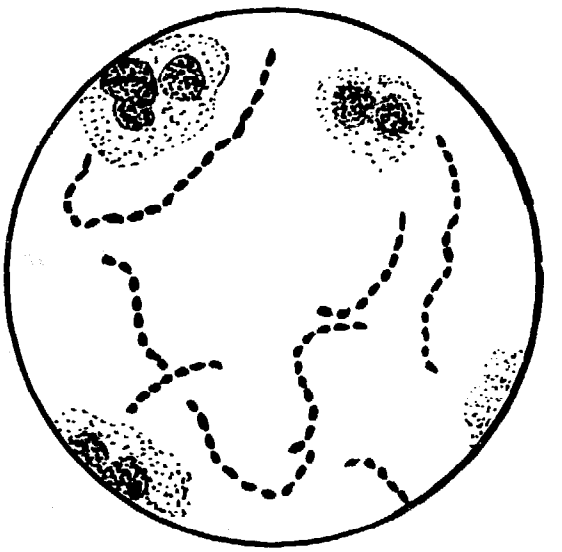
1. Chancroid is an acute venereal infection which typically presents as a ragged, painful, necrotizing ulcer on the genitalia, frequently accompanied by inflammatory swelling and suppuration of the regional lymph nodes.

   Synonym: Soft sore

2. *Occurrence and importance*
   Most common in tropical countries, especially seaports. Importance in Tanzania unknown, estimated incidence about one case for every 10 cases of gc.

   For high-risk factors, see Introduction.

3. *Epidemiology*



Chancroid is caused by a gram-negative bacillus with the form of a small rod, lying in chains (Haemophilus ducreyi). These bacteria can penetrate the intact skin very easily. Women may harbour the organisms in the vagina without symptoms. They form the reservoir of infections. Transmission is by sexual intercourse. Infected males usually do not pass the infection further because of the painful ulcer. Accidental infection by non-sexual contact can occur (children, medical workers).

4. *Clinical picture*

Three to five days after a sexual contact an ulcer develops on the skin of the penis. The ulcer can enlarge in every direction. Often from the first ulcer other parts of the skin are infected and new ulcers develop. The ulcers are painful, and soft on palpation. There may be a suppurative painful regional lymphadenitis. In most cases only one side is affected.

For differences between the soft chancre (chancroid) and the hard chancre that is the early sign of syphilis, see table opposite.

Diagnosis is confirmed by a smear from the ulcer. Gram stain will show the typical rods in chains.

5. *Management of the individual patient*

Sulfa is the drug of choice. Give sulfadimidine two tabs (1 g) qid for one week. Do not incise lymph glands, even when there is fluctuation; they will heal completely with sulfa only.

6. *Control*

Condoms will not prevent chancroid. When condoms are used an ulcer may develop at the base of the penis which is not covered by the condom: 'condom chancre'. Thorough washing of geni-

	Chancroid	Syphilis
Synonyms	Soft sore	Hard chancre, lues
Ulcer	Single or multiple painful, soft, irregular edge, pus and oedema	Usually single, painless, hard, well-defined edge, clear discharge
Lymph glands	One-sided or bilateral, painful enlargement, often suppurative	Bilateral, painless enlargement, never suppuration
Lab	Haemophilus ducreyi in smear	Kahn positive 6–8 weeks after infection
Incubation time	3–5 days	3 weeks or longer

talia with soap and water promptly after intercourse is very effective.

7. *Action*
   See Introduction.

8. *Summary*
   Chancroid is an acute venereal infection characterized by painful, soft, destructive, growing, multiple ulcers.
   Treatment of choice: sulfa.
   Prevention is as for other STD.

OTHER GENITAL SORES OR NEW GROWTHS

Many skin diseases can be localized in the skin or mucous membranes of the genital organs. Some infectious skin diseases are transmitted during sexual intercourse. The table on the next page summarizes the most common ones.

Pathogenic organism	Disease	Main sign/symptom
Viral	Herpes simplex	Blisters
	Venereal warts	Tumours
	Molluscum contagiosum	Papules
Fungal	Candidiasis	Redness and itching
Bacterial	Balanitis	
Parasitic	Scabies	Papules and tunnels, itching
	Pediculosis pubis	Itching, pyoderma

Herpes simplex

Herpes can be sexually transmitted. It shows as recurrent crops of painful vesicular lesions on external genitalia or cervix (in which case symptomless). The first attack is the worst.

Recurrence cannot be prevented. Herpes is thought to be partly responsible for cancer of the cervix.

Treatment is symptomatic with G.V. paint.

Venereal warts or condylomata acuminata

Venereal warts are caused by a virus and may be transmitted during sexual intercourse. The warts show as irregular-shaped growths often in the coronary sulcus of the penis.

Treatment depends on distribution and extensiveness.

Drug of choice is 25% podophyllin in spirit applied to the warts (cover the surrounding tissue with ointment!), and washed off after 4 hours; cautery is possible at hospital level. Circumcision is a useful additional measure.

This condition must be differentiated from condylomata lata which are a secondary manifestation of syphilis.

Molluscum contagiosum

Shows as a round, smooth, red papule. The papule has a central

depression (umbilicated).

Treatment is local application of pure phenol or trichloroacetic acid (carefully!), or cautery (hospital).

Balanitis

Balanitis is an inflammation of the glans penis. It may be caused by candida or bacteria. Lack of personal hygiene is the main promoting factor. Phimosis may be the reason that the glans penis cannot be deaned. In candida infection diabetes mellitus must be excluded. Candida infection may be acquired from a woman with a vulvo-vaginitis caused by candida (see also Chapter 1 on Dermatomycosis).

Therapy: Improved personal hygiene, health education, eventually circumcision.

Antibiotics are not indicated.

Chapter three

VECTOR-BORNE DISEASES

Introduction

Definition of vector: Generally speaking a vector is any carrier of disease, but in the case of the 'vector-borne diseases' we restrict the word to those invertebrate hosts (insects or snails) which are an essential part of the life cycle of the disease organism. A house fly just carrying bacteria or amoebic cysts on its feet to food is not regarded as a vector, this would be simple mechanical spread.

Insect vectors usually acquire the disease organism by sucking blood from infected persons, and pass it on, later, by the same route. There are other routes, however; infection may enter skin cracks or abrasions either from infected faeces deposited when feeding, or from body fluid when an insect is crushed.

By definition the disease organism undergoes a period of development inside the vector, and the time taken for this is called the *extrinsic incubation time.*

Distribution

Vector-borne diseases are limited to areas where suitable conditions exist for the vectors. Most vectors have quite specific breeding, feeding, and attacking behaviour.

Classification of sample vector-borne diseases according to causative organisms.

Viruses	Bacteria	Protozoa	Worms/flukes
Yellow fever	Plague Relapsing fever	Malaria Trypanosomiasis	Filarial diseases: filariasis bancrofti; onchocerciasis Schistosomiasis
Diseases and their vectors			
Mosquitoes	Anopheles – funestus – gambiae		Malaria
	Culex and Anopheles		Filariasis bancrofti
	Aedes sp		Yellow fever
Buffalo gnats	Simulium damnosum		Onchocerciasis
Biting flies	Glossina palpalis G. morsitans		Trypanosomiasis
Lice	Pediculus humanus		Relapsing fever (louse-borne variety)
Fleas	Xenopsylla cheopis		Plague
Ticks	Ornithodorus moubata		Relapsing fever (tick-borne variety)

Classification of vector-borne diseases according to distribution pattern in Tanzania.

Epidemic:	Plague	*Endemic:*	Malaria
	Yellow fever		Schistosomiasis
	Relapsing fever (louse-borne)		Filariasis
			Trypanosomiasis
			Relapsing fever(tick-borne).

Control of vector-borne diseases

All organisms causing vector-borne disease have a stage in their transmission cycle outside the human being.

This offers an opportunity of controlling the disease without interfering directly with human beings and their behaviour, but this task is not as easy as it may seem. To be successful a thorough study of the behaviour of the vector is essential; e.g. in the case of a flying insect breeding places, resting places, feeding patterns, and flying distance should all be known.

Diagram of control of vector-borne diseases

Host	*Vector*	*Susceptible individual*
Reduce human reservoir by mass treatment	**vector control**	Protect host by – screening houses – protective clothing – repellents
Reduce animal reservoir		– vaccination – chemoprophylaxis

Vector control is divided into three main methods:

(a) killing of adults—insecticides
(b) killing of larvae—larvicides
(c) prevention of breeding by environmental sanitation.

(a) *Insecticides*

Insecticides fall into two groups: residual and non-residual. Of the non-residuals the best known is pyrethrum which is the base of ordinary 'Flit'. Pyrethrum is a natural insecticidal mixture, derived from pyrethrum flowers. The dried powdered flower petals can be used as dusting powder. An extract of the powder is often used in solution in kerosene as a spray. The insecticidal components of pyrethrum are unstable in light and air and therefore have no residual effect. Pyrethrum is still the quickest-

acting insecticide known and is of considerable use in the immediate handling of a biting nuisance.

When pyrethrum is sprayed in a room which is then kept closed for 10 minutes it will kill most insects present at the time of spraying, but when the room is opened again new insects entering will be unaffected and further spraying will be necessary. In most tropical houses it is impossible to keep a room closed. Efficient screening and daily use of pyrethrum are recommended.

Larger insects like cockroaches tend to recover from the almost immediate knockdown by pyrethrum. Therefore a residual insecticide is added to most products for domestic use. Pyrethrum is also incorporated in slow-burning insect coils. These coils produce an insecticidal smoke which is both toxic and repellent to insects. Such coils burn for about nine hours and are widely used.

Residual insecticides are stable organic chemicals which, when applied to a surface, remain toxic for some time, usually several months. Particles of these insecticides are picked up by the feet of the insects when they rest or walk on the surface. The particles dissolve in the waxy outer layer of the insect and then penetrate into it. These insecticides affect the nervous system of the insect causing paralysis and death. Their action is slow, often contact of several minutes is necessary and death may not occur for several hours. Thus a room treated with residual insecticides may not show any dead insects because the affected insects may leave the room and die elsewhere.

Two main types of residual insecticides are used:

Chlorinated hydrocarbons:	DDT (dicophane) Dieldrin Gammexane (gamma-benzene hexachloride, gamma-BHC)
Organophosphorus compounds:	Diazinon Malathion Parathion

Chlorinated hydrocarbons, particularly DDT, are irritating to insects so they may leave the treated surface before they have picked up a lethal dose. Most of the organic phosphates are highly toxic for man and therefore cannot be used in house spraying. The newer ones, such as malathion and diazinon, are less toxic, but also less effective especially when used on mud surfaces.

(b) *Larvicides*

The residual insecticides have little residual effect when used as larvicides in water. This may be due to dilution, absorption by mud and vegetation, or the insecticide being washed away. Therefore the application of these insecticides has to be repreated regularly. Oil works by spreading over water surface as a continuous film. Therefore it can be used only in open water; when there is vegetation, emulsions or pellets must be used. Stomach poisons such as paris green are effective only against larvae which feed on the water surface—this includes all mosquito larvae.

(c) *Environmental sanitation*

Prevention of breeding of mosquitoes is the most effective and cheapest form of mosquito control. It also has the advantage that most measures can be carried out at village level by self-help.

These methods are best applied in densely populated areas where the number of breeding places is limited.

The measures have to be carried out as far as the flight range of the mosquito (1–2 km for anopheles).

Control measures

—Draining water holes, ditches, and any accumulation of water around the village or filling in holes and ditches so that water will not accumulate (effective against A. gambiae and Culex).

—Clearing bush and grass along water banks and in the village (A. funestus).

—All containers likely to hold water to be collected and disposed of (Aedes).

—No water container should have water in it longer than one week. This can be done by having a dry day every week (Aedes).

—Clearing bush, replacing the bush by shamba, will prevent breeding of Glossina.

—Snails can be controlled by disturbing their habitat: changes in water level, filling or draining habitat, clearing habitat.

Personal protection

Individuals should be advised to screen their houses with mosquito gauze and to make ceilings in their houses. Mosquito nets should be used at night. Screens and nets should be of proper construction. Bed nets should be rectangular in shape because when they are cone-shaped the sleeper is more likely to come into contact with the net and so risks being bitten.

When screening of the whole house is too expensive, a part may be screened, e.g. the bedroom. Screening of only part is less effective because it does not keep out insects which are in search of the kitchen (notably flies).

Repellents give a valuable form of protection for those who have to be out in tsetse-infested areas or at night in mosquito-infested areas. Repellents act for about 3–5 hours. In such circumstances it is advisable to wear long trousers and long-sleeved shirts.

MALARIA

1. Malaria is an acute infection of the blood caused by protozoa of the genus Plasmodium.

   The infection is accompanied by attacks of fever. The periods of fever depend on the species of the causative malaria parasite.

   Synonym: *Homa ya mbu*

2. *Occurrence and importance*

   Malaria is the commonest disease in Tanzania. In 1973 it ac-

counted for 12% of all sick people (total morbidity). It is the most common disease seen at OPDs and hospitals. It accounted for 4.5% of all deaths (total mortality) recorded in hospitals (the third commonest cause of hospital deaths).

The geographical distribution depends on the climatic conditions necessary for the survival of the vector (the anopheles mosquito). The vector needs a humid climate, a warm average temperature, and suitable breeding places.

3. *Epidemiology*

Malaria in Tanzania

(a) In some areas transmission goes on throughout the year, e.g. in the coastal belt and the area around Lake Victoria.

(b) Other areas are free of malaria, e.g. those above 1500 meters altitude or with very dry climate, e.g. Arusha region.

In the rest of Tanzania the situation is between (a) and (b), and malaria transmission occurs during part of the year (the rainy season).

Spleen rate and endemicity: To classify malaria endemicity in an area we survey the spleen rate. The spleen rate is the percentage of persons with an enlarged spleen in a certain age group. On the balance of the results of the spleen rate the areas are classified as hypoendemic (low transmission), hyperendemic (high transmission), or holoendemic (transmission all year). This correlates more or less with the annual transmission period and with the clinical picture of malaria.

The differences in clinical manifestations seen at different ages are attributed to the development of malarial antibodies.

When a person is exposed to frequent malaria infections, he develops antibodies. The result is that after some time the clinical signs become less impressive, but he still may be infected. This is called *semi-immunity*.

When we take the life history of a child in a holoendemic area we see the following pattern:

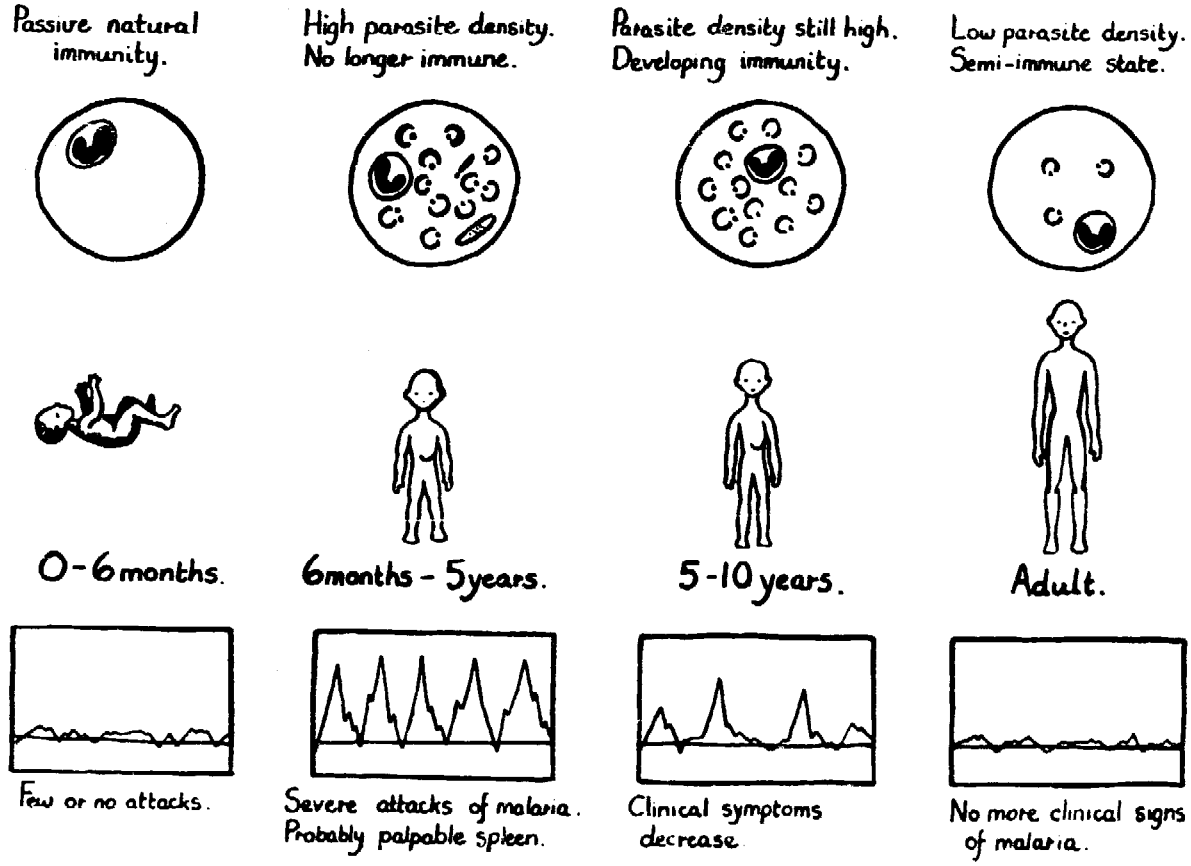
Birth to 6 months: Few or no malaria attacks, due to the malaria antibodies inherited from the mother: passive natural

immunity.

6 months to 5 years: No more antibodies present: severe attacks of malaria, and the spleen usually palpable. Blood slides show a high parasite density (number of infected erythrocytes high).

5 to 10 years: Clinical symptoms decrease: parasite density still high (the immunity is developing).

Adult: No more clinical signs of malaria, spleen no longer palpable, a blood slide may reveal few parasites (low parasite density) without the person suffering from malaria: semi-immune state.



Developing immunity in a holoendemic area.

During pregnancy the general resistance of the expectant mother is lowered. Her immune system is also altered. This makes a pregnant woman more susceptible to attacks. The malaria parasites thrive in the placenta. This may result in abortion, stillbirth or lower birth weight of child. Immunity against

Malaria endemicity, annual period of transmission, and clinical picture

Malaria epidemicity	Spleen rate in children 2–10	Spleen rate in adults	Months of transmission annually	Clinical features of malaria patients	Immunological description
Hypo-endemic	⟨ 10%	Low	⟨ 3 months	Clinical attacks severe in all age groups	Non-immunes
Hyper-endemic	⟩ 50%	High	3–6 months	Clinically severe malaria only in children (occasional attack of disease in adults)	Adults semi-immune
Holo-endemic	⟩ 75%	Low	⟩ 6 months	Severe malaria in children; no clinical malaria in adults but always few parasites in blood	Adults semi-immune

malaria is always against the local type of parasite. Newcomers and travellers should therefore be regarded as non-immune.

The people who are especially at risk of getting malaria are:

—under-fives

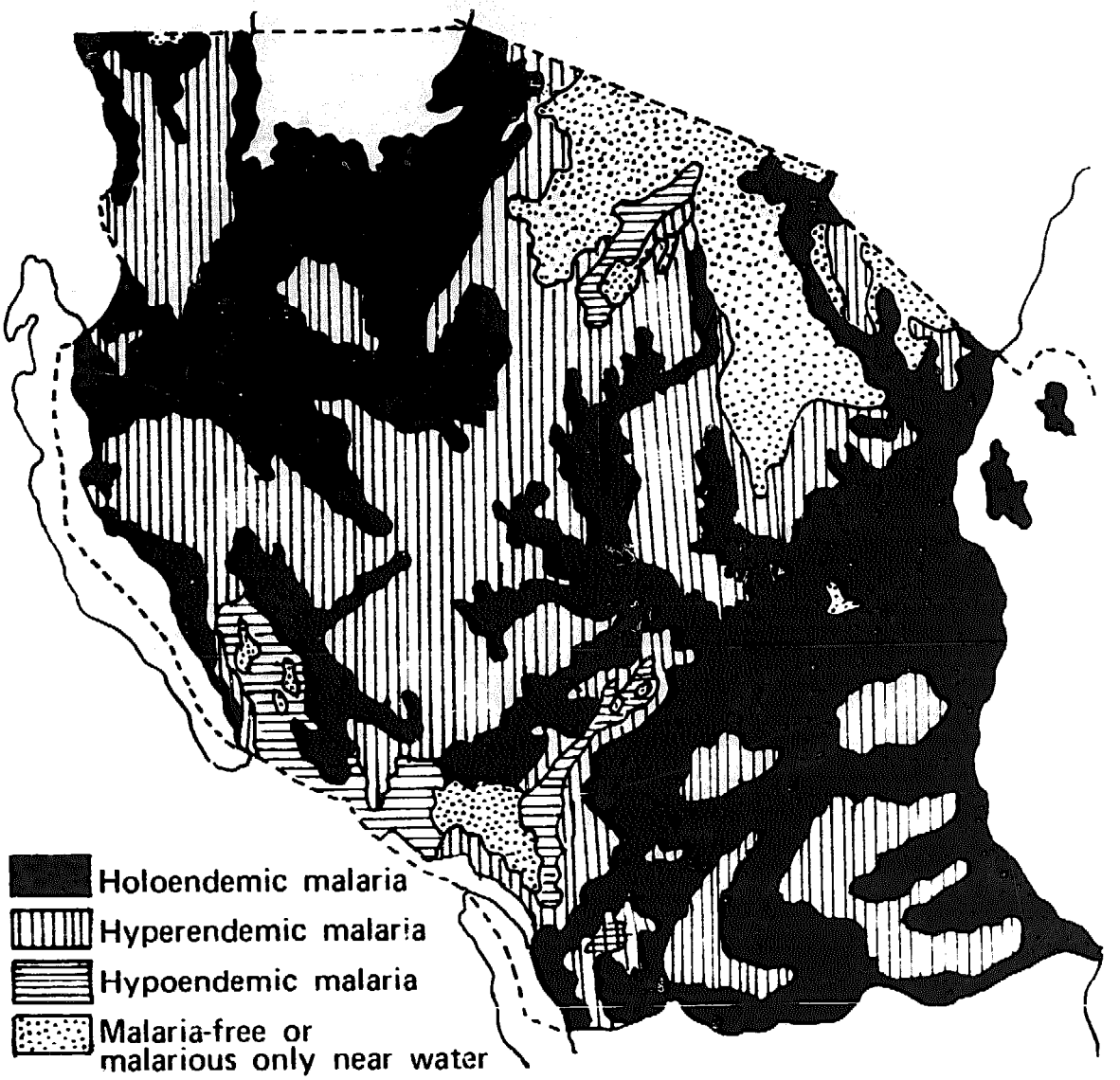
—pregnant women

—travellers and newcomers (people from non-endemic areas).

Malaria is caused by protozoal parasites of the genus Plasmodium. Four different species (types) occur in man.

The division into different species is not of practical importance in the management of malaria in Tanzania. Treatment is the same for all species.

Transmission: Malaria is transmitted by the female mosquito.



Distribution of malaria

The male does not suck blood.

Two types of mosquitoes are of importance in Tanzania:

(a) Anopheles gambiae: breeds in temporary water bodies, exposed to sunlight (pools and puddles) which are mostly found during the rainy season. This mosquito is responsible for seasonal malaria transmission.

(b) Anopheles funestus: breeds in permanent vegetation such as swamps and rice fields.

Other ways of transmission are by blood transfusion and congenital malaria.

The different malaria species affecting Man

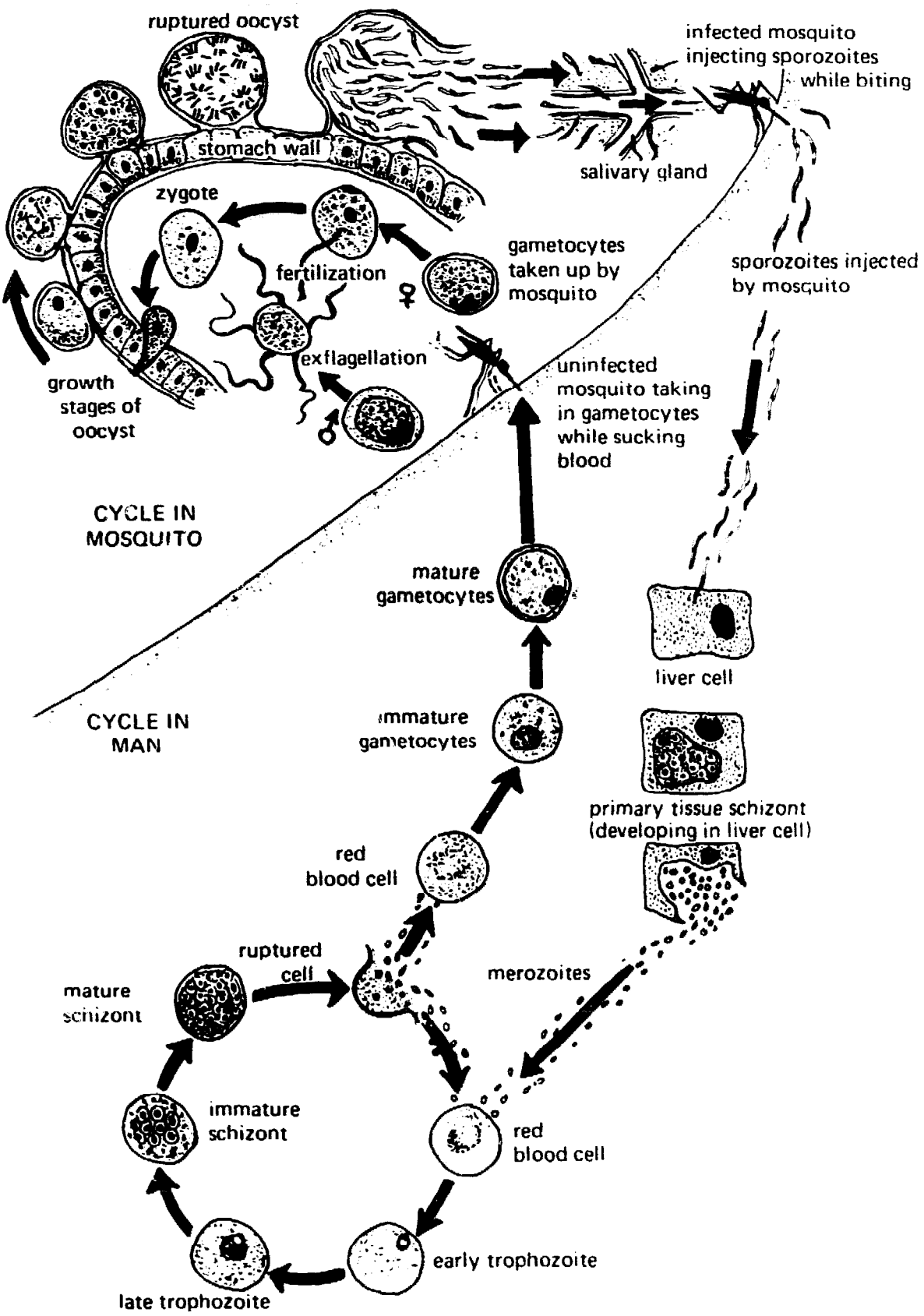
	Frequency as cause of malaria in Tanzania	**Description**
Plasmodium falciparum	90%	Causes malignant tertian malaria; high mortality in non-immunes
Plasmodium malariae	15%	Causes quartan malaria; periodical fever every fourth day
Plasmodium vivax Plasmodium ovale	5%	Benign tertian malaria Periodical fever every third day
Total	110%	This means 10% of all malaria cases are double infections

4. *Clinical picture*

Pathology: The merozoites from the exo-erythrocytic schizonts invade the erythrocytes and continue dividing in the erythrocytes: erythrocytic schizonts. Then they burst out of their erythrocytes and invade new ones and start dividing again. When the erythrocytes burst pyrogens are released causing fever.

The merozoites of Plasmodium falciparum are able to enter erythrocytes of all ages (young and old). The merozoites of the other plasmodia prefer either only young erythrocytes or only old erythrocytes.

Therefore with Plasmodium falciparum more erythrocytes are invaded than in other malaria infections—there is a high parasite density. In Plasmodium falciparum infection the infected erythrocytes may clot together in the capillaries and in this



Transmission cycles of malaria

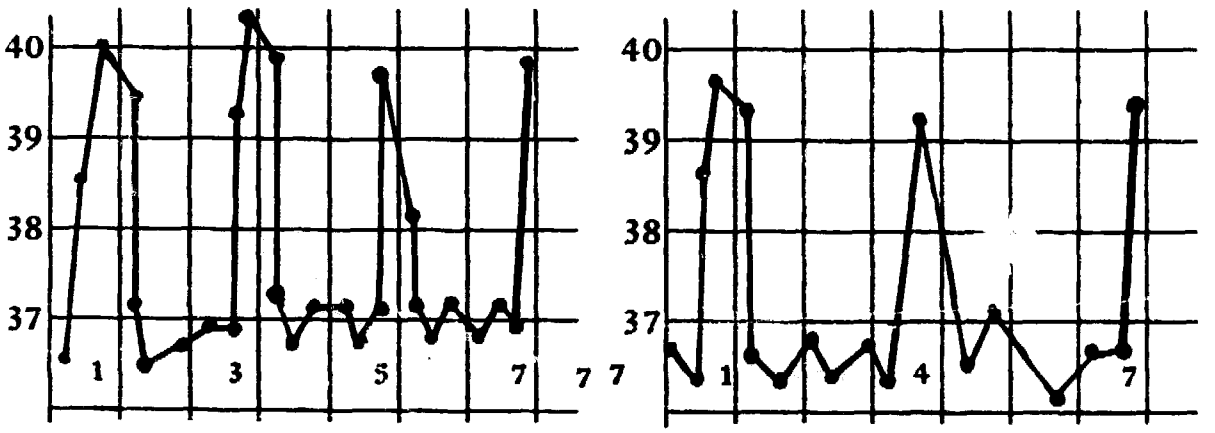
way the blood supply to the organs is disturbed.

The combination of high parasite density and the severe signs from the organs make falciparum infections very dangerous; hence the name malignant tertian malaria.

Symptoms	*and*	*Signs*
Fever		High temperature, rigors
General malaise		periods of sweating
Joint pains		anaemia (haemolytic)
Nausea, vomiting		jaundice (haemolytic)
Chills		spleen enlargement
Headache		± complications

Fever: Periodic fever is a well known aspect of malaria, but fever periods are most evident in the rare and less dangerous forms of malaria caused by P. ovale, P. malariae and P. vivax.

P. vivax and P. ovale cause tertian malaria; that is, there is fever on day 1, day 3, day 5 etc.



Tertian malaria

Quartan malaria

P. malariae causes quartan malaria: there is fever on days 1, 4, 7 etc. In the most common form of malaria, caused by P. falciparum all types of fever are possible, especially at first, and if one waits for a typical fever pattern to appear the diagnosis of this severe form of malaria may be missed.

A typical fever attack is divided in three stages: (a) cold; (b) hot; (c) sweating.

(a) Cold: the temperature is rising and the patient shivers; lasts 1–2 hours. In this stage the red cells are bursting and many ring forms are seen in the blood smear.

(b) Hot: the temperature is high (about 40°C), the skin is dry and hot, there is severe headache, often nausea and vomiting; lasts 3–4 hours.

(c) Sweating: the temperature falls rapidly; the patient sweats profusely—his bed clothes are soaked; lasts 2–4 hours. The patient is relieved but exhausted. In between attacks the patient has few complaints (in uncomplicated malaria).

Spleen: The spleen enlarges with each malaria attack. In early infections it may not be palpable but in later attacks it is enlarged and may be painful during the attack.

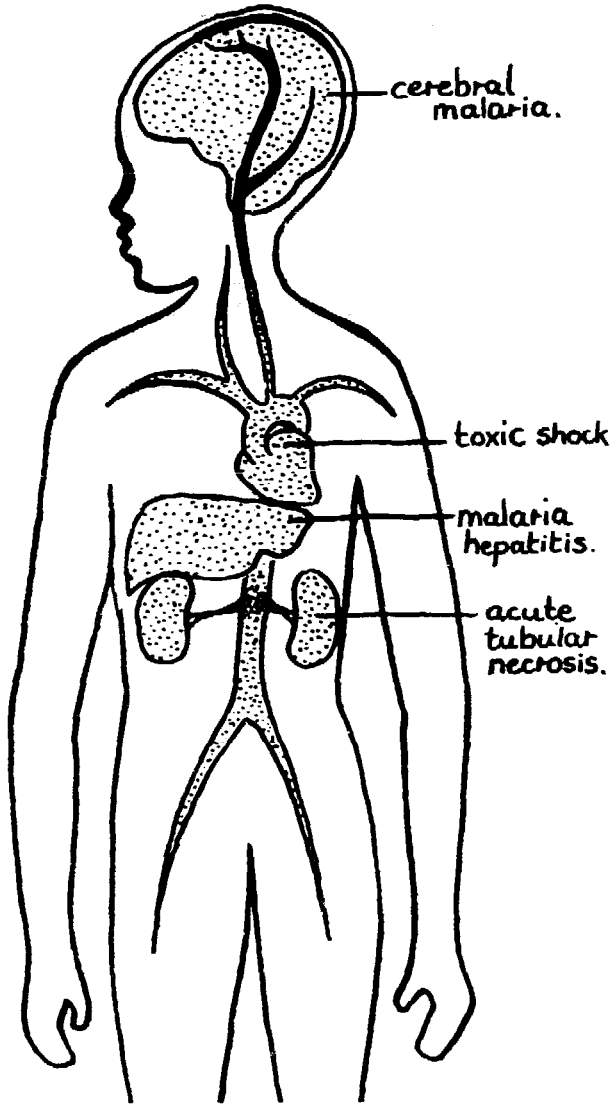
Anaemia: Anaemia is the result of destruction of infected erythrocytes. Therefore the anaemia is of the haemolytic type —that is, normocytic. Later, as a result of repeated attacks using up the folic acid stores for production of new erythrocytes, the anaemia may develop macrocytic features.

Anaemia develops more rapidly and is more severe in P. falciparum infections due to high parasite density.

Jaundice: Jaundice is the result of haemolysis. Usually the jaundice is slight.

Complications: Due to the obstruction of capillaries within organs the blood supply may be hindered, resulting in severe complications (see next page).

When a child in a holoendemic area gets older he develops semi-immunity. The spleen becomes smaller and probably no longer palpable. In a few cases, however, the spleen continues to increase in size. This is thought to be an abnormal immunological reaction to malaria. The condition is called tropical splenomegaly syndrome (TSS). The syndrome occurs in all age groups but especially in young females. The features of TSS



Brain:
Mental disturbance appearing as acute psychosis; meningitis-like symptoms; coma.
Kidney:
Acute tubular necrosis due to anoxia. Result: anuria with consequent uraemia.
Liver:
Malarial hepatitis with hepatomegaly and jaundice.
Shock:
Development of a shock syndrome caused probably by the amount of toxins produced (toxic shock).
Spleen:
The normal function of the spleen is to take away old and abnormal erythrocytes. Due to the infection of many erythrocytes the spleen has to absorb a huge amount of cells and increases in size. Therefore splenic enlargement is a common finding in malaria.

Severe complications of malaria.

are:

—hypersplenism resulting in thrombocytopenia, anaemia, decreased immunity, increased bacterial infections

—attacks of haemolysis resulting in anaemia and haemolytic jaundice. The attacks are more common in pregnancy and may be fatal. The diagnosis can be confirmed by liver biopsy (REFER).

Diagnosis: Malaria may be diagnosed in several ways:

Clinical diagnosis—presenting symptoms suggest malaria;

Therapeutic diagnosis—patients' complaints respond to malaria treatment;

Laboratory diagnosis—malaria parasites in blood smear.

Each method has its limitations as is shown in the stories below.

Story one: In a holoendemic area an adult presented himself with fever at the health centre. The nurse sent him to the laboratory for blood examination. The report—'malaria parasites present'—was back before the patient was examined clinically.

The medical assistant was busy and so prescribed malaria treatment straight away. The next day the patient still complained of fever and slight abdominal pain. He was ordered to finish his full chloroquine course before returning to the health centre again. On the fourth day he was brought in on a stretcher by his relatives. He now had manifest signs of generalized peritonitis. He was referred to the district hospital where laparotomy was done. Diagnosis: peritonitis due to a perforated appendix.

Moral: **IN A HOLOENDEMIC AREA THE PRESENCE OF A FEW PARASITES IN ADULTS IS ONLY AN EXPLANATION FOR FEVER IF YOU HAVE RULED OUT OTHER CAUSES BY EXAMINING THE PATIENT.**

Story two: In a holoendemic area a healthy-looking man donated one pint of blood for his sister. She was undergoing a caesarean section for obstructed labour.

During the operation she was given one bottle of blood, one bottle of Dextran, the rest saline. Two days after the operation she developed some fever. Suspected diagnosis: postoperative sepsis. Treat-

ment was started with penicillin. After two days there was no improvement and the therapy was changed to broad-spectrum antibiotics. During the night the patient got high fever. The nurse on duty took a blood slide as a routine. Result: malaria parasites seen. After chloroquine treatment the patient did well.

Moral: **EVERY PATIENT HAVING A BLOOD TRANSFUSION IN A HOLOENDEMIC AREA SHOULD GET MALARIA TREATMENT AS WELL. THE DONORS MAY BE FIT BUT MAY STILL HAVE SOME LATENT PARASITES IN THE BLOOD. THE RECIPIENTS MAY BE LESS RESISTANT DUE TO PREGNANCY OR TO SURGICAL STRESS.**

Story three: A man from Kilimanjaro reported to the health centre complaining of having felt unwell for some weeks. The man looked ill and had a temperature of 38.5° C. Nothing significant was found and a thick blood film was negative.

The patient was sent home on a standard chloroquine course and when seen 3 days later he was better. Malaria seemed to be proven. Three months later the same patient was admitted, severely wasted, febrile, dyspnoeic and with obvious pain in the lower right chest. He had a massive right pleural effusion, the liver was enlarged to the umbilicus, smooth, and very tender. Diagnosis: amoebic liver abscess.

Moral: **MOST FEVERS RESOLVE SPONTANEOUSLY, CHLOROQUINE HAS A POTENT ANTI-INFLAMMATORY ACTION AND HAS A NON-SPECIFIC EFFECT IN MANY ILLNESSES, AND A SPECIFIC EFFECT IN SOME OTHER PARASITIC DISEASES (e.g. HEPATIC AMOEBIASIS).**

The following facts should be kept in mind:

(a) The diagnosis 'clinical malaria' is nothing other than a diagnosis 'pyrexia of unknown origin' (see below, differential diagnosis).

(b) Everyone in a holoendemic malaria area may have a few parasites in his blood.

(c) Results of treatment do not always confirm a diagnosis.

If malaria is suspected the diagnosis should be confirmed by the laboratory, especially in areas where relapsing fever or trypanosomiasis occur.

Differential diagnosis: Fever in adults and children is common and can have many causes. Viral infections are a common cause of fever, but the diagnosis is difficult to prove. A careful history and proper examination may result in the right diagnosis.

The most common diagnosable causes of fever, *apart from short virus infections*, are listed below.

(In females conditions related to childbirth must be excluded:
septic abortion
puerperal sepsis
pelvic inflammatory disease.)

CHILDREN

	Disease	**Diagnostic criteria**
Probabilities	Malaria	Blood slide
	Early measles	Koplik's spots
	Otitis media	Ear drum examination
	Upper respiratory tract infection	Physical examination
Possibilities	Meningitis	Neck stiffness, bulging fontanelle, CSF
	Urinary tract infection	Urine sediment examination
	Tonsillitis	Pus on tonsils
Rarities	Relapsing fever	Blood slide
	Trypanosomiasis	Blood slide

ADULTS

	Disease	Diagnostic criteria
Probabilities	Malaria	Blood slide
	Relapsing fever	Blood slide
	Meningitis	Signs of meningeal irritation, CSF
	Pneumonia	Dyspnoea, fine crepitations
Possibilities	Typhoid fever	Continual fever, blood culture, Widal test
	Acute bronchitis	Cough, sputum examination
	Rheumatic fever	Arthralgia
	Urinary tract infections	Urine sediment examination
	Hepatitis	Jaundice
	Subacute bacterial endocarditis	Heart murmurs, enlarged heart, blood culture
	Filariasis	Eosinophilia, night blood slide
	Septicaemia	White count, blood culture, abscesses
	Necrotizing enteritis and/or peritonitis	Acute abdomen
Rarities	Amoebiasis	Liver enlargement, white count, ESR
	Trypanosomiasis	Blood slide, lymphadenopathy
	Leptospirosis	Jaundice, bleeding, meningism
	Hodgkin's disease	Lymphadenopathy
	Leukaemia, reticulosis	White count, bone marrow examination
	Appendicitis	Pain and rebound tenderness Rt lower abdomen

5. *Management of the individual patient*

Treatment of the malaria attack:

Chloroquine is available as two different salts. In chloroquine phosphate (trade names Resochin, Aralen, Malarex) the total weight of the tablet is 250 mg. The active component ('base') is 150 mg.

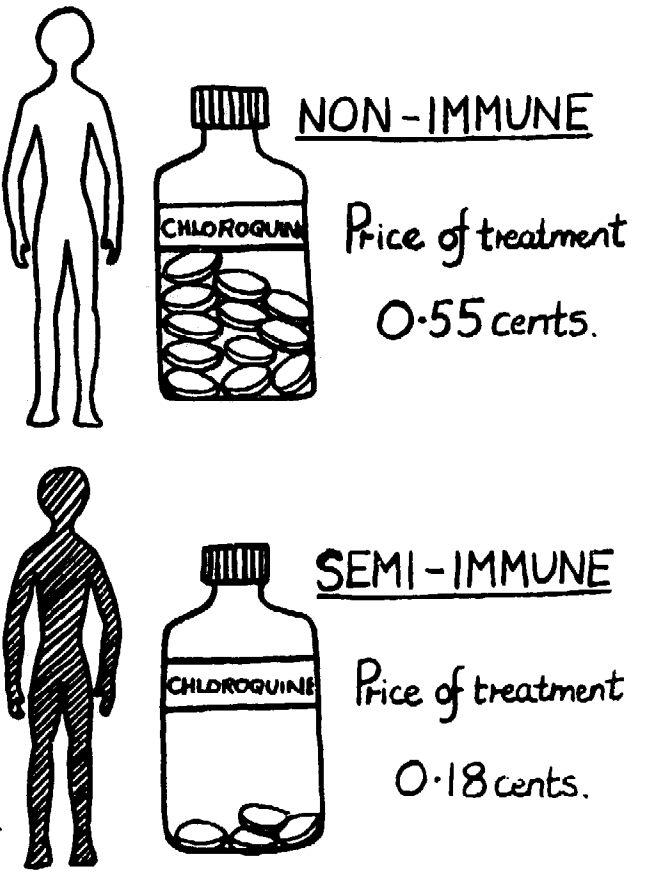
The other salt is chloroquine sulphate (trade name Nivaquine) of which *200* mg are equivalent to 150 mg base. Chloroquine is therefore always prescribed in terms of base, the same in the two types of tablet.

To save tablets and to save money it is important to differentiate between non-immunes and semi-immunes. A semi-immune never has a severe attack of malaria.

Non-immunes: all children under five; all pregnant females; all who come from malaria-free areas or hypoendemic areas (malaria transmission less than 3 months/year).

Semi-immunes: all others (see also epidemiology, p 54).

Chloroquine	
Semi-immunes	**Non-immunes**
600 mg (4 tabs) start or 300 mg daily (2 tabs) for two days	600 mg (4 tabs) start; after 6 hours 300 mg (2 tabs), then for three days 300 mg (2 tabs) daily
Total	
600 mg (4 tabs)	1800 mg (12 tabs)
Price	
0.28 cents	0.84 cents



In case chloroquine is not available or the person is allergic to chloroquine, amodiaquine (Camoquin) may be used in the same doses of base of chloroquine as above.

If you make this differentiation between non-immunes and semi-immunes, your supply of chloroquine, an essential drug, may last three times as long.

Chloroquine injection: There is a widespread malpractice of giving chloroquine injections routinely to start the treatment,

probably based on the mistaken belief that injections work quicker.

For other drugs this might be true, but it is not so in the case of chloroquine.

It has been proved that the blood levels after injection rise *more slowly* than after oral consumption of chloroquine tablets. Chloroquine is very well absorbed within 30 minutes.

There are disadvantages to chloroquine by injection:

(a) It is dangerous: there have been many reports of acute cardiac arrest after chloroquine injections, especially in children.

(b) It causes abscesses.

(c) It involves quite a lot of staff doing unnecessary work, sterilizing syringes and giving the injections.

(d) Chloroquine injections are far more expensive than tablets. When money is scarce, this should always be kept in mind. 200 mg chloroquine as an injection costs 25 cents; 200 mg chloroquine as a tablet costs 8 cents.

The only indications for chloroquine injection are as follows:

(a) The patient who is unconscious and unable to swallow, i.e. the patient with cerebral malaria.

(b) In the very few patients who are vomiting so much that they are not able to absorb the tablets.

Dose of chloroquine injection: 5 mg base/kg, maximum 200 mg; the injection may be repeated after 6–8 hours. Give intramuscularly or subcutaneously, never intravenously.

CHLOROQUINE INJECTIONS ARE DANGEROUS

Treatment of cerebral malaria: Chloroquine im (dose see above) together with hydrocortisone iv (5 mg/kg) and iv drip, saline or glucose.

Treatment of TSS (tropical splenomegaly syndrome): Attack of haemolysis with anaemia—REFER. (Corticosteroids are given; blood transfusion is of no use and may aggravate the haemolysis.)

Long-term treatment: malaria prophylaxis for at least one year or sometimes for life. The best prophylaxis in these cases is causal prophylaxis with proguanil (see control). Splenectomy is not indicated. The immune disorder is not cured and hepatomegaly, severe malaria attacks, and sepsis may follow as a result of removal of the spleen.

Resistance to chloroquine: Unfortunately, but not unexpectedly, some strains of malaria parasites have become resistant to chloroquine. So far, this has not been proved anywhere in Africa, but it may develop in the coming years.

If you suspect a person of having a chloroquine-resistant malaria it is advisable to refer him for examination and treatment.

Usually the resistance is partial and high doses of chloroquine may cure the patient. About one-third of all malaria parasites in Tanzania are resistant to pyrimethamine.

6. *Control*

Possibilities of control:

(a) Vector control; see Introduction.
(b) Individual protection; see Introduction.
(c) Chemoprophylaxis; why? who? how? what?

Why

In the past some malaria specialists have been opposed to chemoprophylaxis in holoendemic regions. They were afraid that it merely postponed the period of immunity development. It seemed to be use of money without long-term benefit. It has

now been proven that chloroquine prophylaxis reduces the mortality of malaria and at the same time does not interfere with the building of immunity. So there is no reason for withholding chemoprophylaxis.

Who

There is a need to give malaria prophylaxis only to people who have not enough immunity. These are:

(a) Children under five: the passive immunity from the mother will wear off in about six months. From this time onwards the child is exposed to severe malaria attacks, up to the age of 5 years. If the child survives these 5 years he will have developed immunity.

(b) Pregnant females: in pregnancy the general resistance and immunity is lowered; therefore a pregnant mother has to be regarded as non-immune. Malaria attacks are severe and may result in abortion, premature labour, and small-for-dates babies.

(c) Travellers and newcomers: people coming from non-malaria areas are non-immunes and are advised to take malaria prophylaxis on an individual basis. Immunity against malaria parasites is only against local strains—travellers therefore must also be regarded as non-immunes.

How

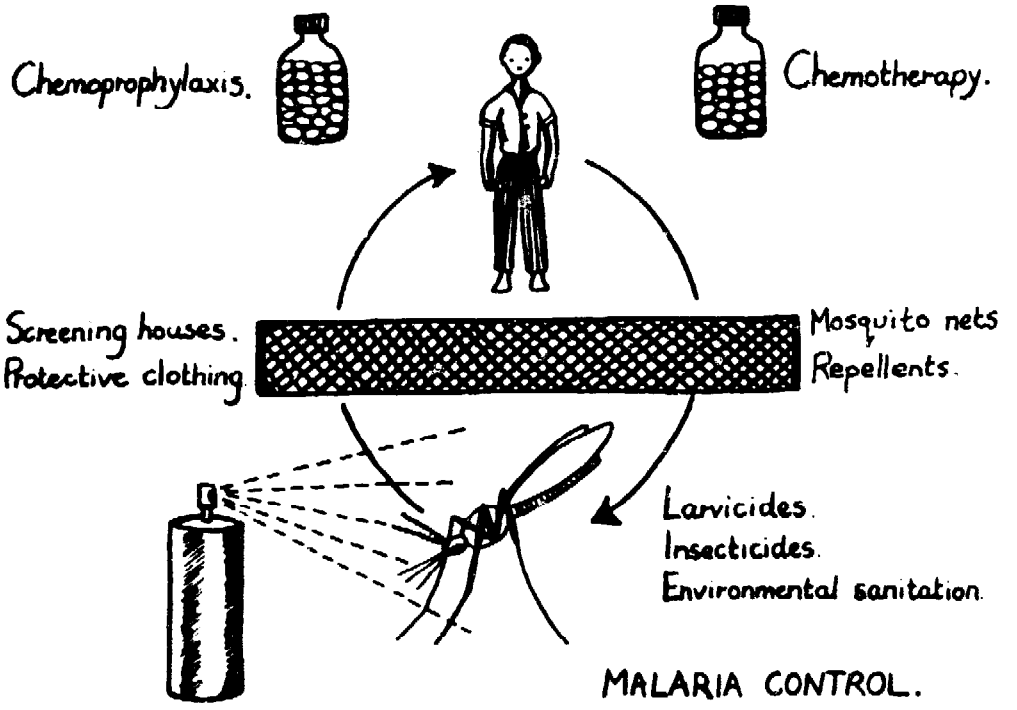
—All pregnant mothers and all children under five should be given malaria prophylaxis when attending the MCH clinic.

—Another method of giving malaria prophylaxis is by mixing chloroquine with domestic cooking salt. This method was used as a pilot study in Mto wa Mbu (Arusha Region). It worked but was difficult to maintain in practice.

—Taking malaria drugs on an individual basis. Most shops sell antimalaria tablets. It is a good habit to take some chloroquine in case of fever.

Drugs and doses for chemoprophylaxis of malaria

Drugs	Price per week	Dose in adults	Place of action	Description
Chloroquine tabs 150 mg base tabs 100 mg base	0.14	300 mg/weekly	Schizonticidal in erythrocytes	Suppressive
Proguanil (Paludrine) tabs 100 mg	0.20	1^0 mg/daily	Primary tissue schizonticidal	Causal prophylaxis (resistance common)
Pyrimethamine (Daraprim) tabs 25 mg	0.20	25 mg/weekly	Primary tissue schizonticidal	Causal prophylaxis (resistance common)



Malaria policy in Tanzania

In theory it would be possible to eradicate malaria in Tanzania. In practice it is not so easy. This is due to the large amount of transport, equipment, staff, and money needed to do it.

Antilarval measures are justified in towns, in industrial estates, and other places with high population densities. In villages the population should be stimulated to start with environmental sanitation as part of their self-help activities.

Individual chemoprophylaxis is *indicated in all MCH clinics.* Further, the availability of chloroquine tablets in shops all over the country may also contribute to a gradual decline in the incidence of malaria in Tanzania.

The policy for MCH clinics is that chloroquine should be given *weekly.* If all children under five and all pregnant mothers were given chloroquine, this would consume 10% of all money available for drugs and supplies. Although this would be a very wise use of the available money the amount of chloroquine allocated to your health centre may not be enough to give *weekly* prophylaxis.

The best you can do then is to give all MCH patients chloroquine *once monthly*, to be swallowed in your presence. Chloroquine once monthly does not completely prevent all malaria attacks, but it prevents people from dying of malaria.

7. *Action*

–Ensure malaria prophylaxis is given to all under-fives and all pregnant women at your health centre MCH clinic.

–Advise the local shopkeepers to stock chloroquine.

–Discuss with the health auxiliary and the ward development committee the feasibility of an anti-mosquito campaign. Such a campaign is of use only if, after the initial phase, a maintenance phase can be established.

Start the campaign in the most densely populated area of the ward, e.g. in the village around your health centre. If this is successful the area may be extended.

Check the dormitories of schools and other institutions—are the windows screened? Do the pupils have mosquito nets and are these maintained? Discuss possibilities for improvement with the head teacher or other authorities.

—Check the use of chloroquine injections in your health centre. Discuss the strict indications of the use of this drug in staff meetings, and also the right dose of oral chloroquine. Stress the difference between treatment of semi-immunes and non-immunes.

—Give health education on individual protection by chemoprophylaxis and use of mosquito nets.

—Advise ceilings in houses made with local materials.

CHLOROQUINE FOR ALL UNDER-FIVES AND PREGNANT WOMEN

8. *Summary*

Malaria is an acute infection of the blood with plasmodia, characterized by fever, joint pains, headache, and resulting in anaemia because of haemolysis. Malaria is the commonest disease in Tanzania. Malaria may be hypo-, hyper-, or holoendemic.

Transmission is mostly by A. gambiae and A. funestus.

Semi-immunes should be treated with a single dose of chloroquine.

Eradication of malaria has proved to be very difficult. Malaria prophylaxis should be given routinely to those most susceptible —pregnant women and under-fives.

RELAPSING FEVER

1. Relapsing fever is an acute, infectious, bacterial disease, characterized by alternating febrile periods.

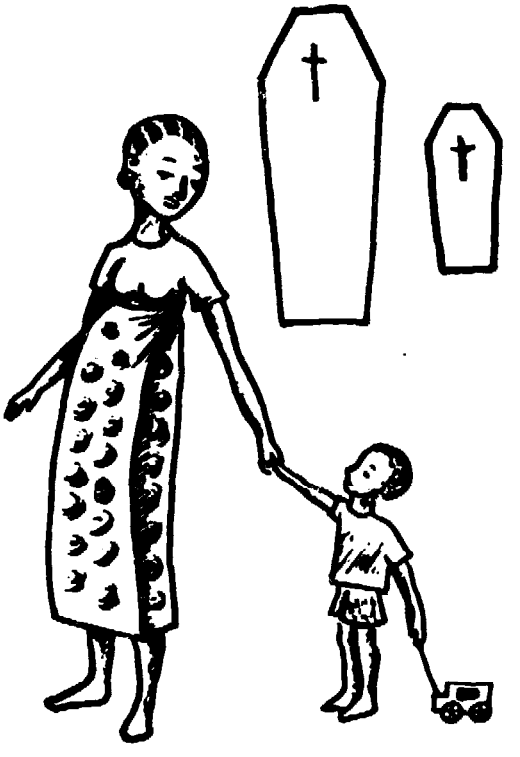
Synonym: Recurrent fever, tick fever, famine fever, *Homa ya papasi;*

There are two types of relapsing fever:
(a) Tick-borne relapsing fever transmitted by ticks.
(b) Louse-borne relapsing fever transmitted by lice.

2. *Occurrence and importance*

Louse-borne: Only 3% of all cases of relapsing fever reported in Tanzania in 1973 were louse-borne type, a total of 55 cases. No deaths reported. Louse-borne relapsing fever is rare. The disease is transmitted by lice. The lice involved are the human head louse, Pediculus capitis and the common body louse, Pediculus corporis.

The latter lives in clothes which are worn continuously. So the disease is only present in conditions where many people are together in cold weather without changing or washing clothes, e.g. in war, famine, natural disasters. The louse is not able to survive without being in contact with the human body. This gives the disease an epidemic character.



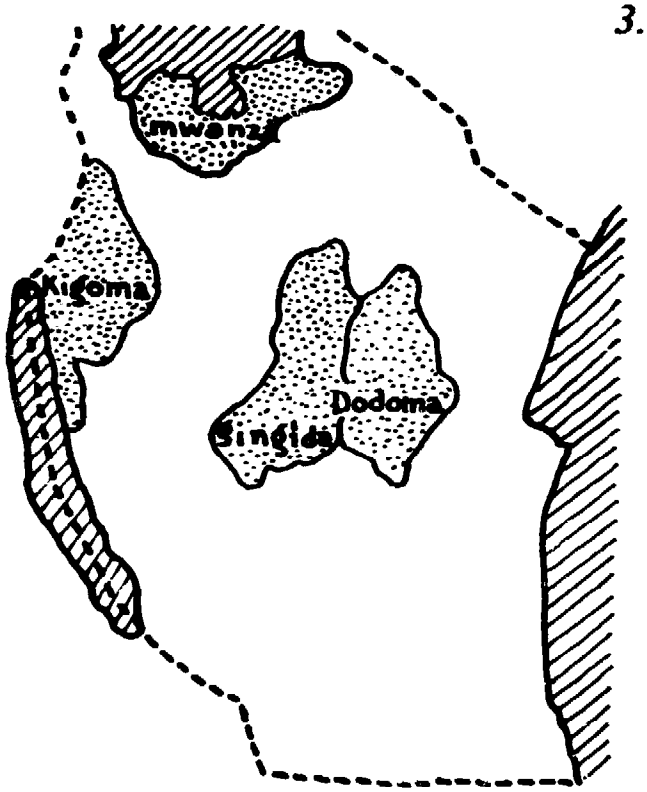
Most deaths from relapsing fever occur among pregnant women and children.

Tick-borne: The tick-borne form is much more common in Tanzania.

As in malaria, adults in endemic areas are semi-immune. In pregnancy resistance is lowered, therefore most deaths from relapsing fever occur among pregnant women, children, and newcomers.

The disease is transmitted by ticks. The ticks live in cracks and crevices of walls and floors. This means that the disease is endemic in people with bad housing conditions.

Distribution in Tanzania: Most frequently diagnosed in Singida, Dodoma, Kigoma, and Mwanza regions.



Distribution of relapsing fever.

3. Epidemiology

Both types of relapsing fever are caused by spirochaetes of the genus Borrelia (louse-borne: Borrelia recurrens; tick-borne: Borrelia duttoni).

Mode of transmission

Louse-borne: When lice suck blood from an infected person the spirochaetes multiply within their bodies, but are not present in saliva or coxal fluid.

When lice are crushed on the skin some parts containing borreliae are rubbed into the bite wound, infecting the person.

The organisms are not transmitted to the offspring of the lice (in contrast to the cycle in ticks).

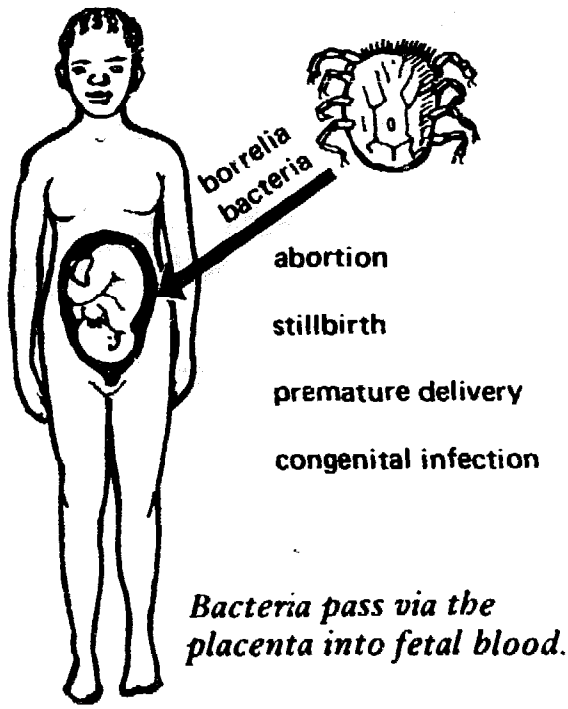
Tick-borne: When the tick sucks blood from an infected person the spirochaetes enter the body of the tick. There they multiply and are present after one week in the salivary glands and in the coxal fluid.

During feeding usually some saliva and coxal fluid is deposited on the skin of the person bitten. The bacteria enter the wound of the bite. The bacteria are also able to enter the body via the mucous membranes (e.g. in laboratory infections; Dutton, the discoverer of the disease, died from it).

The tick, Ornithodorus moubata, involved in the transmission of the disease in East Africa, needs a blood meal only occasionally.

The bacteria pass into the ovary of the tick and offspring of an infected tick are automatically infected without having sucked infectious blood themselves.

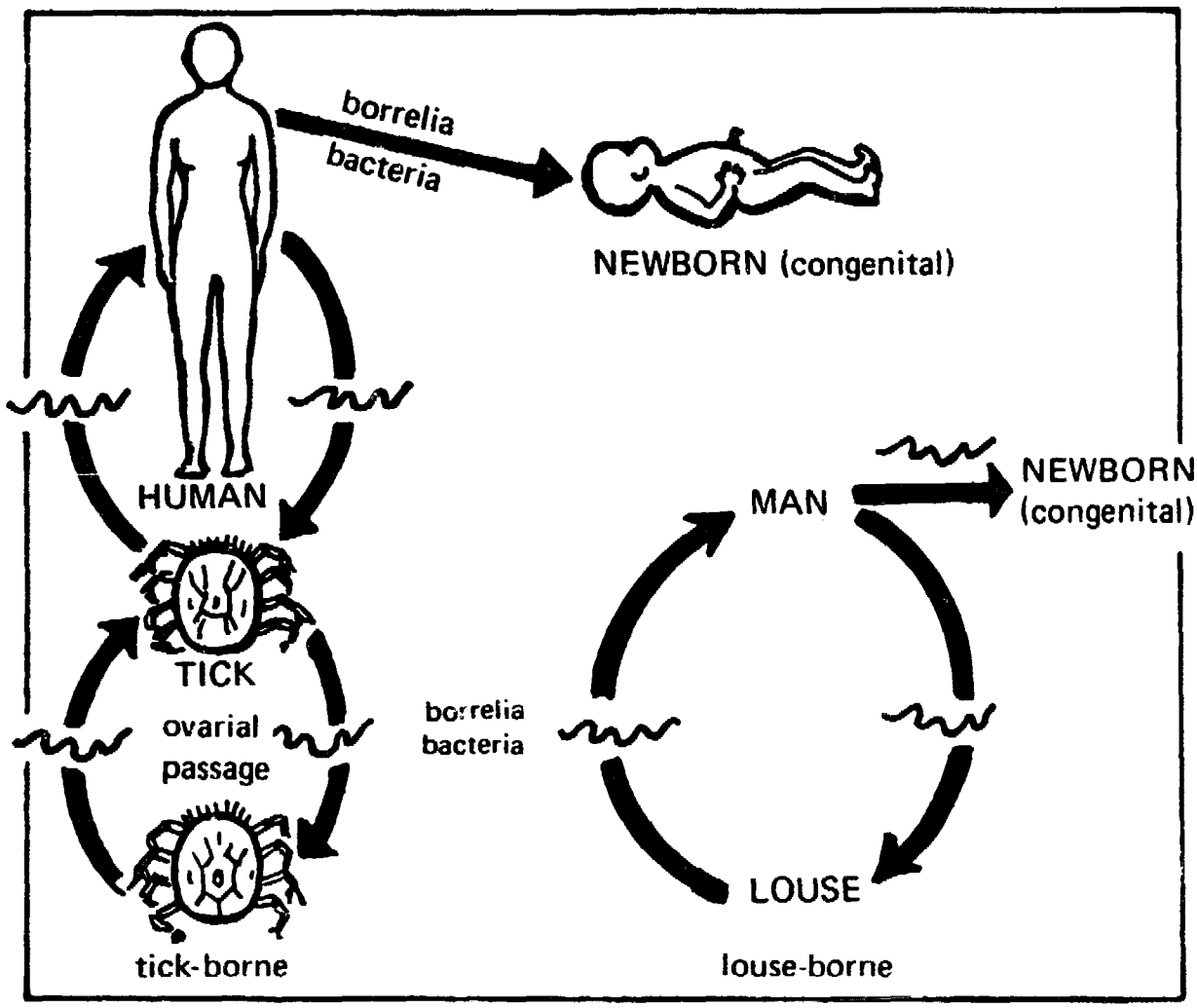
In this way a house once inhabited by infectious ticks



Bacteria pass via the placenta into fetal blood.

can remain dangerous for up to 10 years. There have been reports of people getting relapsing fever in a rest-house which was only visited for a few days throughout the year.

In humans the spirochaetes can pass via the placenta into the fetal blood. This may cause abortions, stillbirths, premature deliveries and relapsing fever infections in the newborn (compare syphilis).



Transmission routes of the relapsing fevers.

4. *Clinical picture*

The borreliae multiply in the body fluids. They produce endotoxins when they die. The toxins affect the cells of the liver, spleen, and endothelial cells of the capillaries.

The patient presents with:

–high fever starting with chills, headache, and other body pains (joints).

There may be:

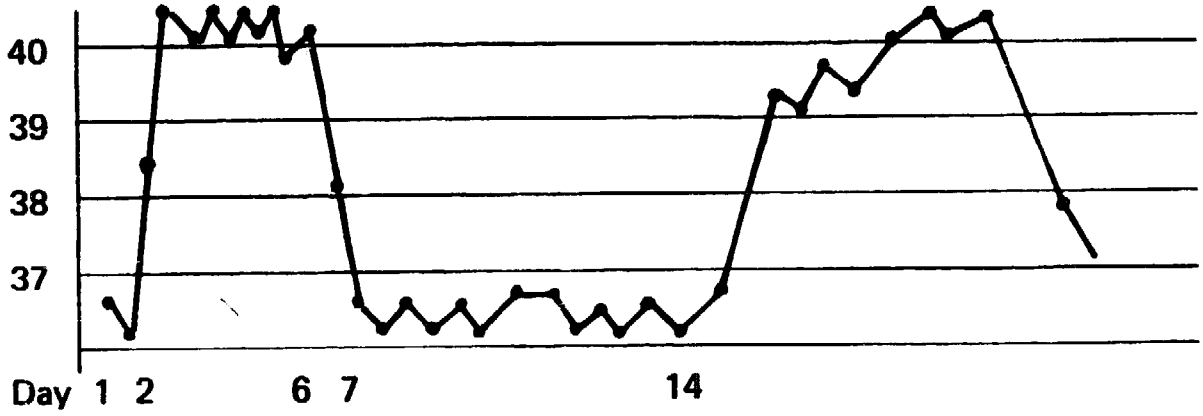
–nausea, vomiting

–jaundice

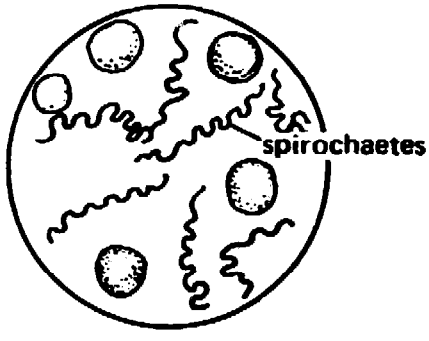
–liver swelling.

After 4–5 days the temperature comes down, the patient stays free of fever for 8–12 days and then a relapse follows with the same signs only less intense.

In untreated cases there may be up to ten relapses. The clinical picture is so similar to malaria that it can only be differentiated by a blood smear.



Complications:



(a) Coma in patients with little immunological resistance, pregnant women, children. The first attack may be very severe leading to coma and death.

(b) Meningitis: a lymphocytic meningitis.

(c) Eyes: iritis and atrophy of optic nerve.

Diagnosis:
Thick-drop blood slide examination as is done for malaria. In fact most diagnoses are made when the patient is suspected of suffering from malaria and is sent for a blood slide examination. Differentiation between the two types of relapsing fever is not possible by microscopy.

5. *Management of the individual patient*
Treatment:
First choice: Tabs tetracycline 250 mg q i d for one week.
Price: 1.26.
Second choice: Injection PPF 0.4 mU for 5 days. Price: 1.75.

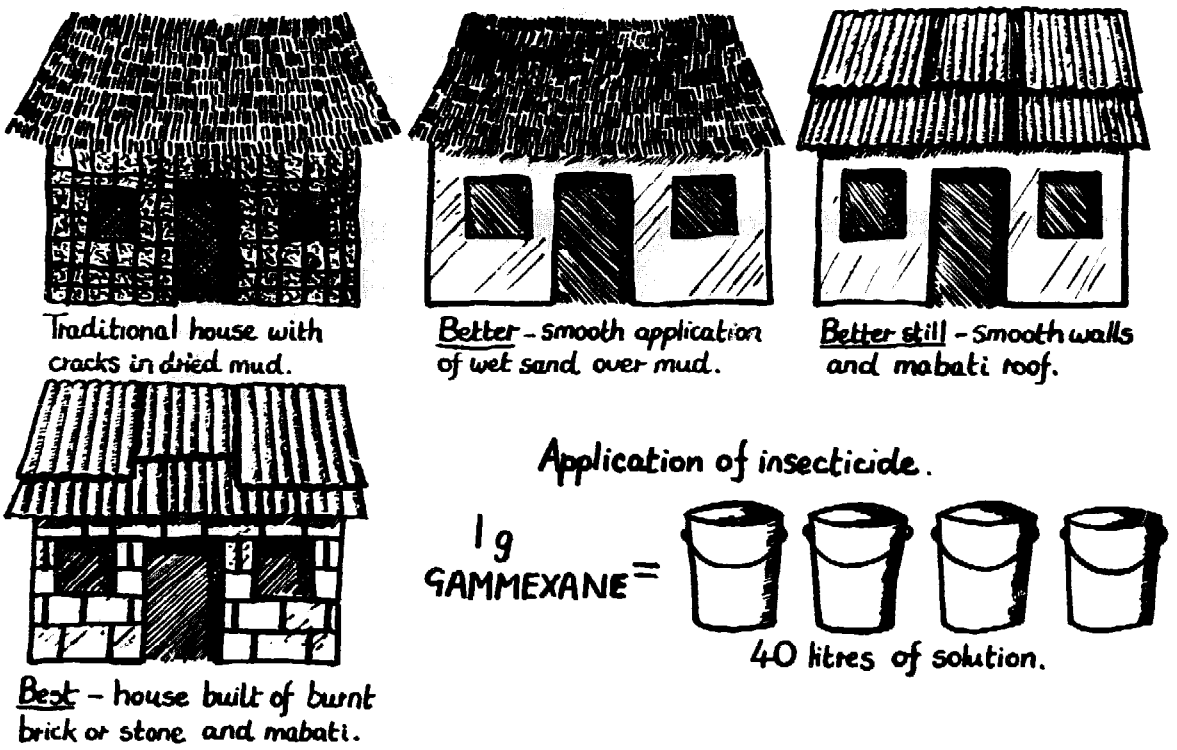
Attention:
Quite a lot of deaths have been described after the start of the treatment. This is explained as a Herxheimer reaction. The antibiotic suddenly kills a large amount of bacteria, with highly toxic effect. To avoid this it is best to give a very low dose.

LOW DOSE TO AVOID HERXHEIMER'S REACTION

Tetracycline is contraindicated in pregnant mothers and in children.

6. *Control*
Tick-borne relapsing fever:
(a) The best way to control the disease is to improve housing conditions.

The present way of building houses in Tanzania is to make walls of a wooden framework which are then filled with stones and mud. When the mud dries cracks appear. Cracks are hiding places for ticks. The application of wet sand on



Control of relapsing fever by better housing.

the mud walls produces a nice smooth surface without cracks. This method is within everyone's reach.

(b) Another hiding place for ticks is in roofs made of grass. Corrugated iron roofs are an important improvement of a house. In the long run the building of houses from burnt bricks and stone will reduce disease. With a rise in socio-economic levels the disease should disappear. Application of insecticides which kill ticks is also advisable. Recommended is gammexane (Gammatox plus). 1 kg is enough to make 40 litres of solution. Price: 16/-.

(c) Control of tick fever is also achieved by reducing risk of infection. People sleeping on the floor have the greatest incidence of the disease.

(d) Mass treatment of patients is not useful, as the important reservoir is the tick; once infected a tick is able to produce offspring which are also infectious. One infected person can make a tick family infectious for over 10 years.

Louse-borne relapsing fever:

Reducing *vector* by use of insecticides; DDT powder to disinfect clothes; boiling of clothes to kill eggs.

Reducing risk of being infected: frequent changing and washing of clothes; water and soap for body hygiene.

7. *Action*

—Order a routine thick blood smear in all cases of clinical malaria if you are in a relapsing fever area. This will increase your number of diagnoses of relapsing fever.

—Tell every patient to apply insecticides to the walls of his house in order to avoid reinfection.

—Give every patient diagnosed as having relapsing fever health education on the improvement of his house (walls, roof, bed).

—Pregnant women and under-fives: Attacks of fever in these groups are very dangerous when caused by relapsing fever. Be on your guard; take blood smears.

8. *Summary*

Relapsing fever is an acute infectious disease caused by spirochaetes of the borrelia group. It is characterized by attacks of fever.

Transmission is by ticks or, rarely, by lice. Treat with low doses of antibiotics to avoid Herxheimer reactions. Prevention is mainly by vector control.

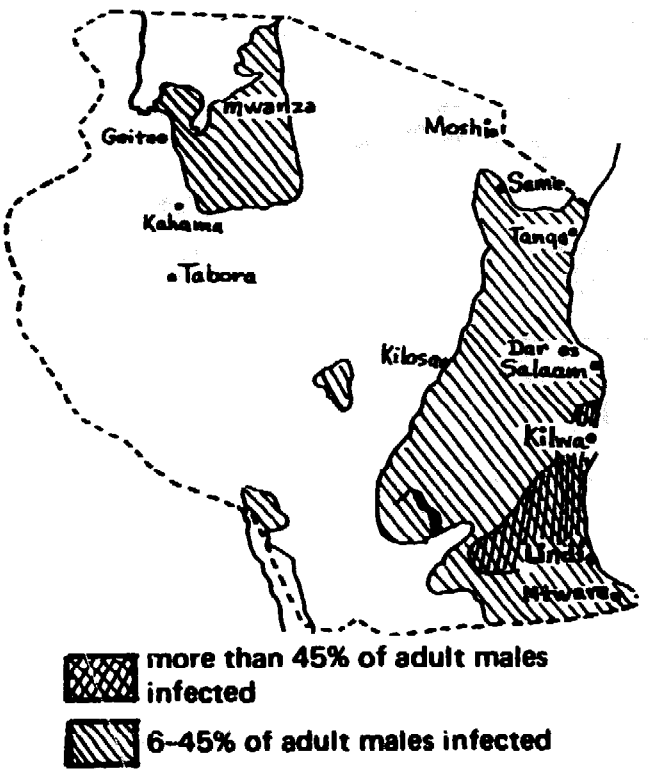
BANCROFTIAN FILARIASIS

1. Bancroftian filariasis is a disease caused by the reaction of the body to the presence of worms in the lymphatic system.

Synonym: Wucheriasis bancrofti.

2. *Occurrence and importance*

Since the disease is transmitted by mosquitoes it is rare in high altitudes and dry regions. In Tanzania filariasis is most frequent in the coastal belt and the lake regions. The disease is more



Distribution of Bancroftian filariasis in Tanzania

disfiguring than dangerous. In some coastal places over 30% of adult males have hydroceles, a late manifestation of the infection. The treatment for hydrocele is surgical and this may be a severe drain on the limited hospital resources.

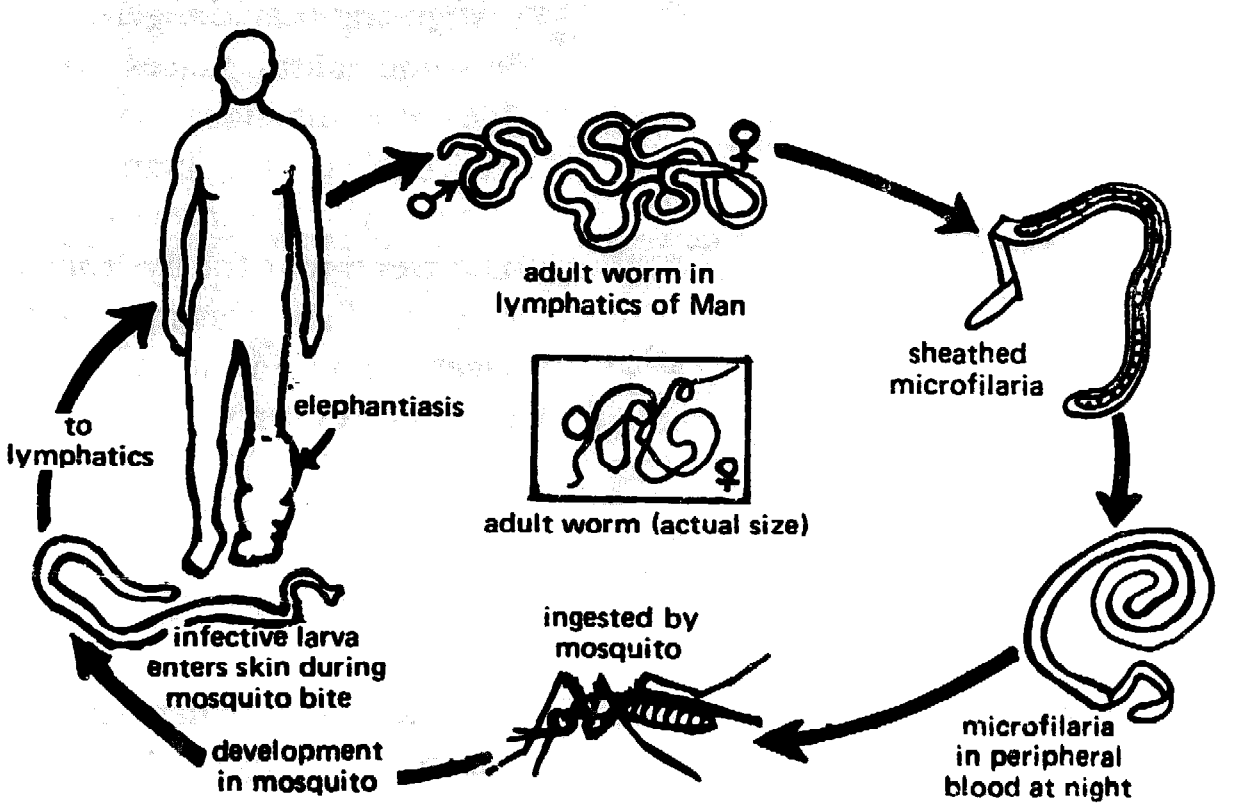
3. *Epidemiology*

Filariasis bancrofti is caused by Wuchereria bancrofti, a worm belonging to the nematodes.

The size of the adult worm is 4–8 cm long and 0.2 mm thick, long and thin, like a hair. Mosquitoes are the vectors of the disease: mainly Culex species in urban areas and Anopheles species in rural areas. The infective mosquito injects some infectious microfilariae into the subcutaneous tissue of the patient. These larvae find their way to the lymph vessels where they mature in about a year.

After this time the female worms give birth to live microfilariae (viviparous).

During the day the microfilariae hide themselves in the central large vessels. During the night when the vector mosquitoes suck blood they migrate to the peripheral blood. When a mosquito sucks blood from an infected person it will be infected with these microfilariae. The microfilariae undergo several changes in the body of the mosquito. After 10 days the mosquito is able to transmit the infection to other human beings.



Life cycle of Wuchereria bancrofti.

4. *Clinical picture*

The presence of the worms in the lymph vessels gives rise to a foreign-body reaction. After the death of the worm, more proteins are released; the reaction then is even more severe. Three stages may be distinguished:

Acute phase:

Starts within a few months after infection
–lymphadenopathy
–fever
–eosinophilia.

In this stage microfilariae are not yet demonstrable in the peripheral blood because the worms are not yet mature. The acute phase is mainly due to a hypersensitivity reaction.

Subacute phase:

(After about one year.) In this phase worms have matured and

microfilariae are present in the peripheral blood. Reactions to the adult worms cause attacks of fever with lymphangitis, funiculitis, and/or epididymitis. Recurrent attacks will sooner or later lead to hydrocele.

Lesions caused by the microfilariae are less common and are associated with hypereosinophilia and lung symptoms (tropical pulmonary eosinophilia syndrome).

Chronic phase:

After many years of repeated attacks, lymph glands and lymph vessels become obstructed. As a result lymphoedema develops. Lymphoedema is most often seen in the legs or scrotum (elephantiasis) but may also be present in vulva, breasts, or arms.

Since the adult worms have usually died, microfilariae are no longer seen in the blood.

Summary:

Bancroftian filariasis	**Main signs and symptoms**		**Blood smear**
Acute phase (first months)	Fever Eosinophilia Lymphadenopathy Lymphangitis		Negative
Subacute phase	Adults cause: Fever Lymphadenitis Funiculitis Epididymitis Hydrocele	Microfilariae cause: Hypereosinophilia Asthma-like attacks Fever	Positive
Chronic phase	Lymphoedema Elephantiasis Chlyuria Hydrocele		Negative

Diagnosis:

During the daytime microfilariae are not present in the peripheral blood. To demonstrate the microfilariae blood slides

should be taken:

either (a) between 22.00 h and 02.00 h

or (b) 45 minutes after a provocative dose of diethylcarbamazine 100 mg.

Puncture fluid from swollen glands or a hydrocele may also be examined.

Differential diagnosis:

Always suspect filariasis in a feverish patient in endemic areas. Suspicion is strengthened by eosinophilia and/or presence of one of the clinical symptoms. Elephantiasis is often not caused by filariasis but may be due to chronic damage of the feet by absorption of mineral crystals leading to obstruction of lymph flow. This condition (called lymphoconiosis) is prevented by wearing shoes.

5. *Management of the individual patient*

The drug of choice is diethylcarbamazine (Hetrazan or Banocide). This kills the microfilariae and has also some effect on the adult worm. It should not be given during the acute attacks—only after acute phase has subsided.

The dose is 6 mg/kg body weight in three divided doses, i.e. 150 mg (3 tabs) tds for an average adult. Treatment should be given for 2–3 weeks.

To avoid reactions antihistamine drugs and/or steroids can be given during the first days of treatment.

In the chronic phase of the disease chemotherapy is no longer useful since there are no active worms. Surgical treatment is indicated for hydrocele and is sometimes possible for elephantiasis of legs and scrotum (REFER).

6. *Control*

(a) Reducing the vector population: Anopheles, see Introduction (p 52). Culex often breeds in septic tanks and latrines. Therefore septic tanks and latrines have to be treated regularly with larvicides.

(b) Reducing the human reservoir: this is possible by way of

organized mass campaigns. Campaigns should be continued for at least 10 years. Before starting, a good cost-benefit analysis should be made.

(c) Reducing risk to host of infection: see Introduction p 53.

7. *Action*
   - —If your health centre is located in an endemic area ensure that it is possible to take thick blood smears in the night between 10 p.m. and 2 a.m.
   - —Suspect filariasis in all cases of unexplained eosinophilia (with fever.
   - —Discuss the possibilities of a mosquito campaign with your health auxiliary and the ward development committee.
   - —Give health education on personal protection (screening houses, mosquito nets).

8. *Summary*

   Bancroftian filariasis is a chronic disease caused by the body's reaction to the presence of Wuchereria bancrofti, a nematode worm. Transmission is through mosquitoes. Diagnosis by night blood slide. Control is difficult. Individuals may protect themselves by screening their houses or using mosquito nets.

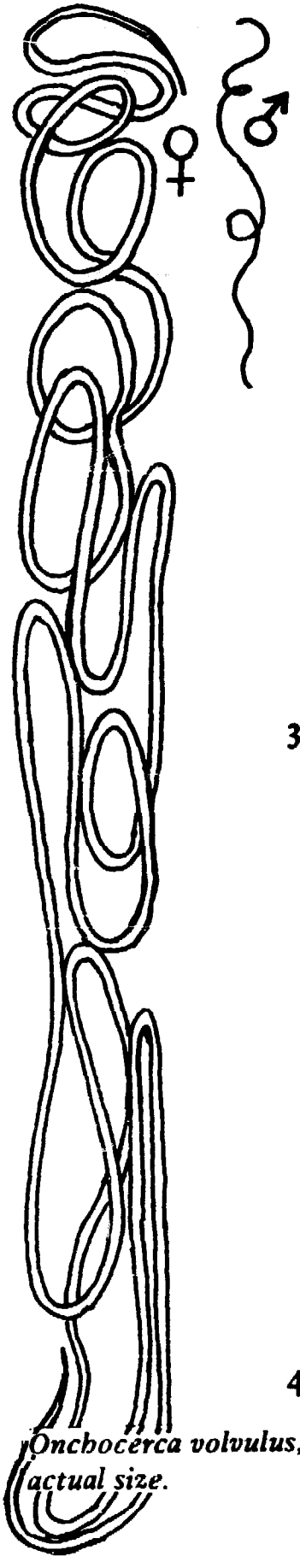
ONCHOCERCIASIS

1. Onchocerciasis is a chronic disease caused by a filarial nematode worm. The disease shows itself by nodules in the skin at places where bones are near the surface, and by eye lesions.

   Synonym: River blindness.

2. *Occurrence and importance*

   The disease is a major cause of blindness in areas of tropical Africa where people are exposed to heavy and repeated infection. Where infection is light the eyes may not be affected. Importance in Tanzania has almost certainly been underestimated and the disease under-reported, apart from the cases in which it is undiagnosed. The disease is transmitted by a small



Onchocerca volvulus, actual size.

black fly named Simulium. The fly lays eggs in fast-running rivers. When the river does not run quickly enough there is not enough oxygen present in the water to make the eggs develop into larvae. The average flying distance of the fly is about 40 km (maximum 150 km) so the disease is only present or likely to be present in the neighbourhood of such fast-running mountain rivers.

Distribution in Tanzania:
Morogoro region: Mahenge-Morogoro
Iringa region: Njombe-Ubena
Ruvuma region: Songea
Tanga region: Usambara Mountains
Mbeya region: Tukuyu.

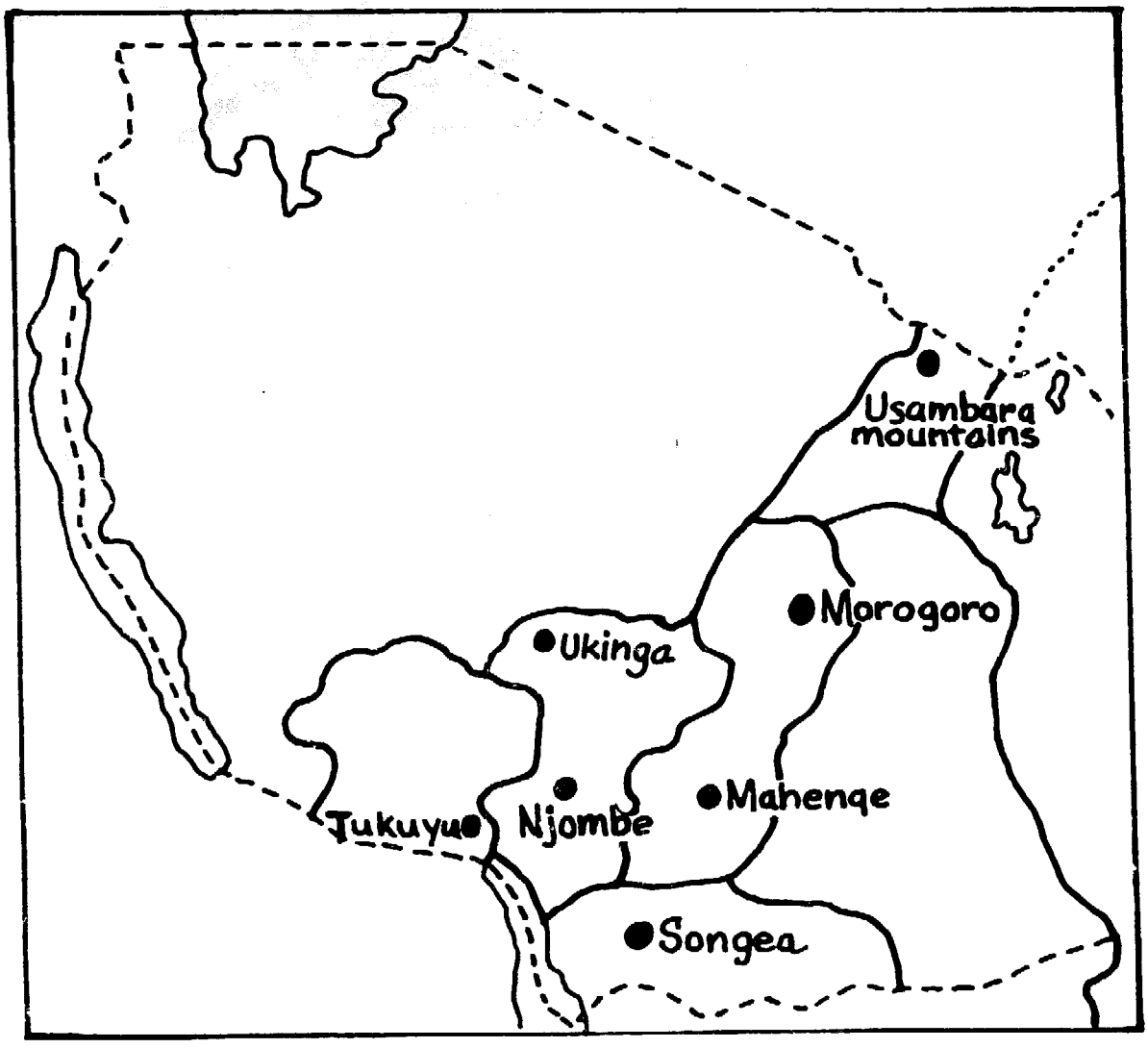
3. *Epidemiology*

River blindness is caused by Onchocerca volvulus, a filarial worm belonging to the nematodes. The size of an adult female worm is about 40 cm long and 0.4 mm thick. The male worms are about 4 cm long and 0.2 mm thick.

The vector of onchocerciasis is the female Simulium fly (buffalo gnat). When she takes blood from an infected person the microfilariae develop into infectious larvae. The Simulium fly attacks outdoors during the day but not in bright sunlight. Most bites occur around sunrise or sunset, or on cloudy days or in the shade.

4. *Clinical picture*

Pathology: the infectious larvae injected into the subcutaneous tissues develop into adult worms. The mature males and females then

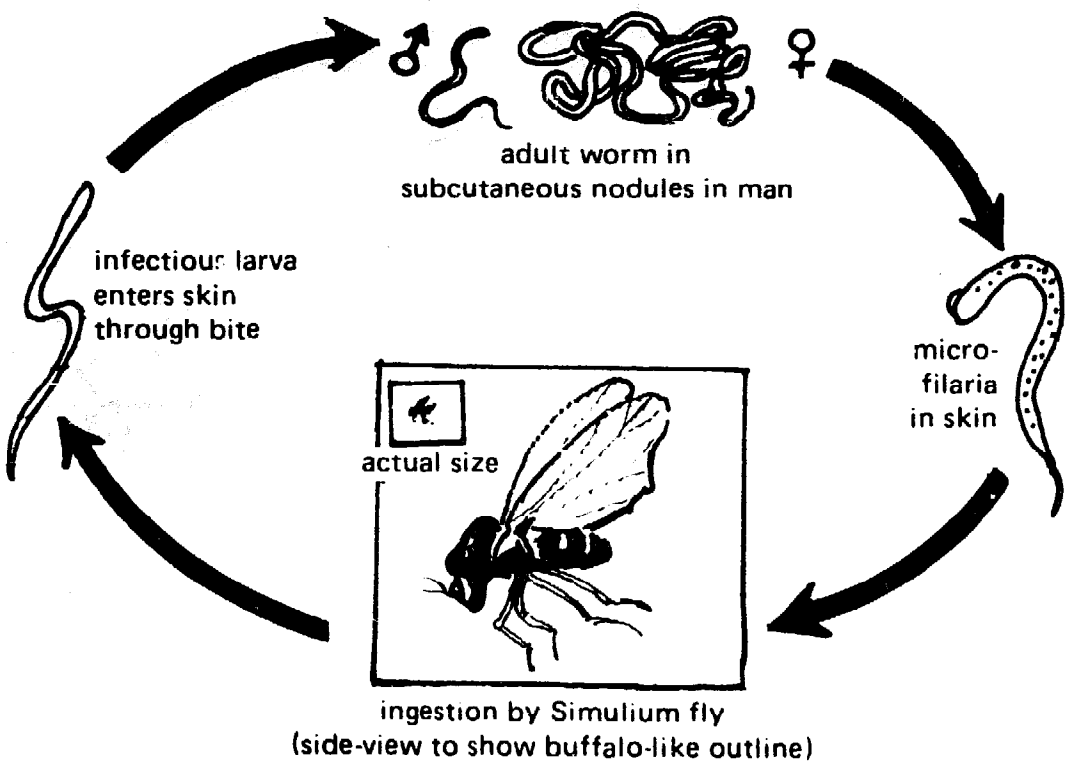


Incidence of onchocerciasis.

collect in balls, bound together by fibrous tissue which forms the typical nodule. The nodules are best seen in places where the bone is near to the skin, e.g. elbow, shoulder, scapula, skull ribs, iliac crests.

After one year the adult worms start giving birth to microfilariae. The microfilariae migrate in the epidermis, subcutaneous tissues and in the eye. Onchocerciasis presents itself in three different clinical pictures:

(a) Nodules caused by the adult worms: non-tender, rubbery, firm nodules, size from 3 mm to 3 cm.

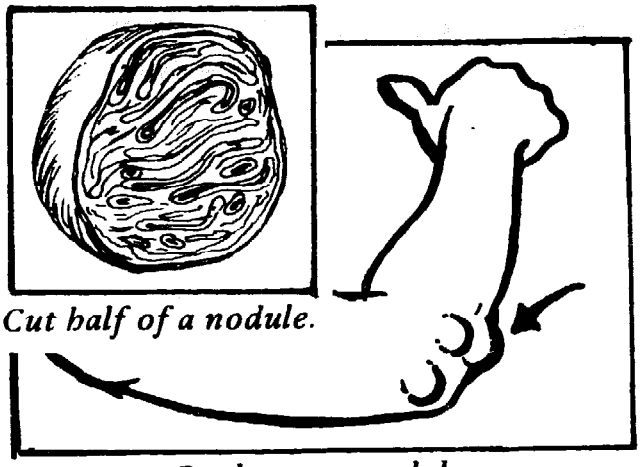


Onchocerca cycle

(b) Dermatitis: caused by reaction to the presence of microfilariae in the epidermis
—papules
—macules
—itching
afterwards the skin becomes loose,atrophic, and depigmented

(c) River blindness: the microfilariae invade the cornea and the anterior chamber of the eye causing first:
—oedema of conjunctiva
—corneal spots and pannus starting at the lower limbus (unlike trachoma, which affects the upper limbus)
afterwards:
—glaucoma leading to blindness
—cataract.

Diagnosis:
The microfilariae of Onchocerca volvulus are not found in the blood. The diagnosis is made by examination of a skin snip,



Cut half of a nodule.

Onchocerca nodules.

which is teased out in a drop of saline on a slide. Moving microfilariae are then looked for under the microscope.

Whenever you remove a node surgically in an onchocerca area, cut the node into two and inspect the cut half. Many nodules are removed and sent to pathology departments under provisional diagnoses of sarcoma, carcinoma, etc., when the diagnosis could have been made by just cutting, looking, and seeing the entwined threads of the adult worms inside.

5. *Management of the individual patient*

Onchocerciasis is not a killing disease, while the treatment of the adult worm may cause severe side effects. If the patient has no serious complaints and is likely to be reinfected there is no need for treatment.

Groups in need of treatment:

(a) patients with eye lesions
(b) patients with severe skin lesions
(c) patients with heavy infections.

Two different kinds of treatment are needed:

(i) To kill the microfilariae: diethylcarbamazine (Hetrazan)—use the same schedule as for bancroftian filariasis. This treatment, however, does not affect adult worms.
(ii) Removal of adult worms:
  (a) by surgical removal of nodules
  (b) by use of suramin. This may cause severe side effects; for schedule of treatment see textbooks.

6. *Control*

In the past, trials to control the disease by massive treatment of

patients have not proved successful. The most promising method is by adding insecticides continuously to the water of the rivers known to be breeding places of the Simulium fly. An onchocerciasis focus was eradicated on Mt Kenya using this method.

7. *Action*
If river blindness is occurring in the catchment area of your health centre discuss with the DMO and his health officer what you can do.

8. *Summary*
Onchocerciasis is a chronic disease characterized by fibrous nodules in the skin and, though rarely in Tanzania, by eye lesions. It occurs only near fast-running rivers in the highlands: hence the name river blindness. The vector is the Simulium fly.

Treatment is by combined action against the microfilariae and the adult worms by Hetrazan and suramin. Control of vectors is the only method of prevention.

YELLOW FEVER

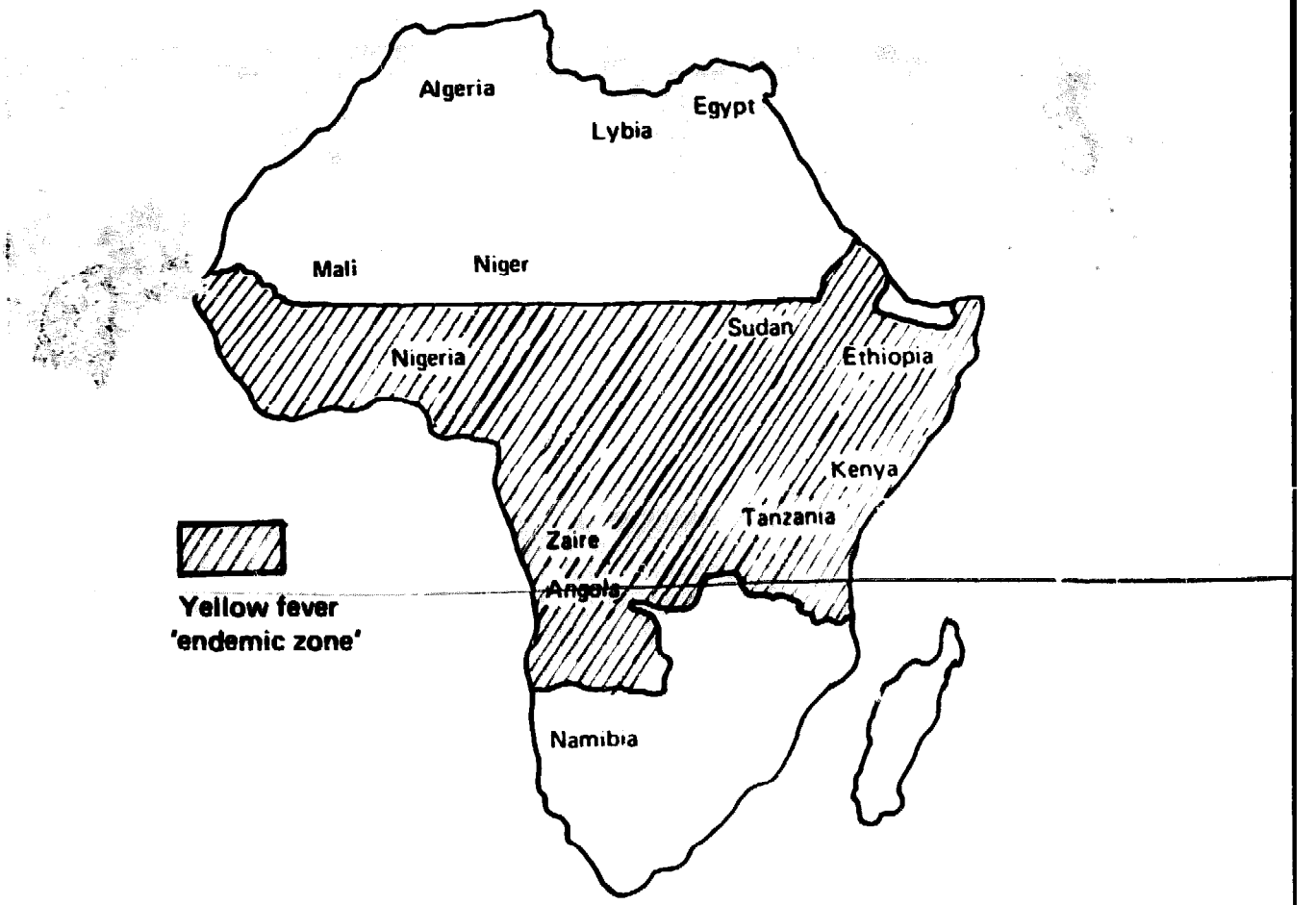
1. Yellow fever is an acute infectious disease characterized by sudden onset of fever, hepatic and renal failure, and jaundice.

Synonym: Jungle fever.

2. *Occurrence and importance*
Tanzania is within what is internationally regarded as the yellow fever 'endemic zone', though in fact there is no yellow fever in Tanzania. The vector, however, is present, and if infection were introduced the results might be disastrous. Spread can be very rapid with up to 85% case fatalities in a non-immune population.

3. *Epidemiology*
Yellow fever is a zoonosis of forest monkeys. The disease is transmitted among them by mosquitoes living in the tree tops. Man may be infected by these mosquitoes when going into the

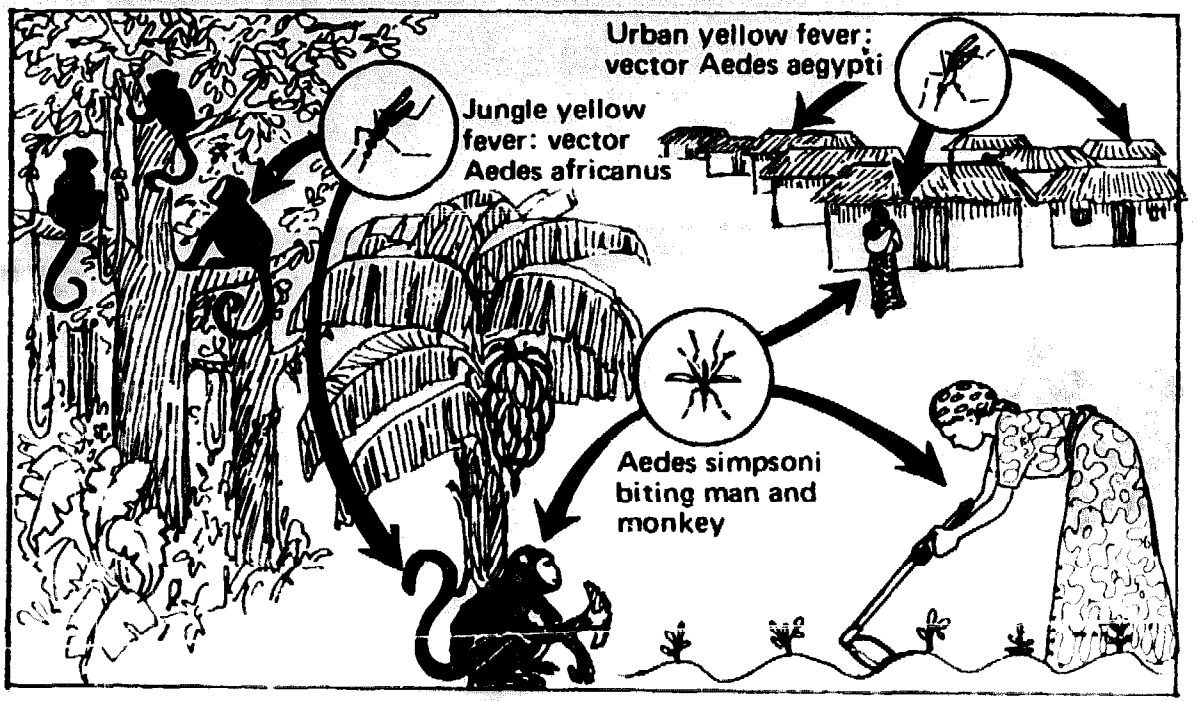
forest, or monkeys may infect human mosquitoes when leaving the forest to feed on banana plantations.



4. *Clinical picture*

Many infections are inapparent, leading to immunity without definite illness. In a classical case of yellow fever the onset is sudden with fever, headache and backache, nausea and vomiting. Bleeding tendency is common resulting in epistaxis, bleeding of gums, haematemesis, melaena. Jaundice occurs due to liver cell necrosis and this may result in liver failure and death. Albuminuria occurs due to nephrosis and this may result in kidney failure and anuria. Patients surviving the seventh day of the disease usually recover.

Diagnosis: Yellow fever should be suspected when patients die from a feverish disease with jaundice especially when there was albuminuria.



Differential diagnosis: Viral hepatitis, malaria, leptospirosis, relapsing fever, Marburg disease (see viral hepatitis, p 165).

5. *Management of the individual patient*

There is no specific treatment. Infection of further mosquitoes should be avoided by treating patients in screened rooms or under mosquito nets. When a case of yellow fever is suspected the DMO should be informed immediately. Do not refer the patient but request the DMO to see the patient to confirm diagnosis.

6. *Control*

To avoid importation of the yellow fever virus, travellers from an infected area to a potential receptor area need valid international vaccination certificates. Vaccinations against yellow fever are carried out at regional hospitals usually once a month. The vaccine contains a live attenuated virus and gives good protection for at least 10 years. Side effects are rare.

To avoid importation of infected mosquitoes, aircraft coming from infected areas should be sprayed with aerosol insecticides.

Provided these measures are carried out correctly an outbreak of yellow fever in Tanzania is unlikely to occur and therefore routine vaccination of the entire population is not necessary.

7. *Action*

If a case of yellow fever is seen:

—isolate contacts in screened quarters

—inform DMO

—start a mass vaccination campaign starting with persons most exposed and those living in areas infested with aedes mosquitoes

—spray the houses of the patient and his neighbours with insecticides

—extend the insecticide spraying to all other houses and apply larvicides to breeding places of aedes mosquitoes.

8. *Summary*

Yellow fever is an internationally notifiable disease. It does not occur in Tanzania but the vector is present. Control is through vaccination of travellers, and spraying of aircraft to avoid importation of infected mosquitoes carrying the virus.

TRYPANOSOMIASIS

1. A disease caused by protozoa, characterized by fever, followed by general weakness, cerebral involvement and frequently leading to death. There are two forms of differing acuteness and severity (see below).

   Synonym: Sleeping sickness, *malale*.

2. *Occurrence and importance*

Trypanosomiasis is not so common in Tanzania. In 1973 about 500 cases were reported. 50 people died.

The disease is found where human beings live in tsetse-infested areas. The main importance of the Trypanosoma parasites lies in the fact that one species, the Trypanosoma brucei,

causes serious damage to cattle. Because the economic and agricultural potential of these tsetse areas is greatly reduced, about one-half of the land area of Tanzania is scarcely or not at all populated.

3. *Epidemiology*

The most important species of trypanosomes are:

(a) Trypanosoma gambiense: causing the West Africa picture of 'sleeping sickness'. No animal reservoir known.

(b) Trypanosoma rhodesiense: causing the more severe trypanosomiasis *without* 'sleeping sickness'. Reservoir in game animals (bucks). It causes disease in humans and in cattle.

Glossina, distribution in Africa.

(c) Trypanosoma brucei: causes disease in animals only. Not pathogenic for man.

There is no visible difference between these trypanosomes when under the microscope. They are transmitted by tsetse flies (Glossina species [*mbung'o*]). The adult flies, male and female, feed exclusively on blood with a preference for ungulates (hoofed animals).

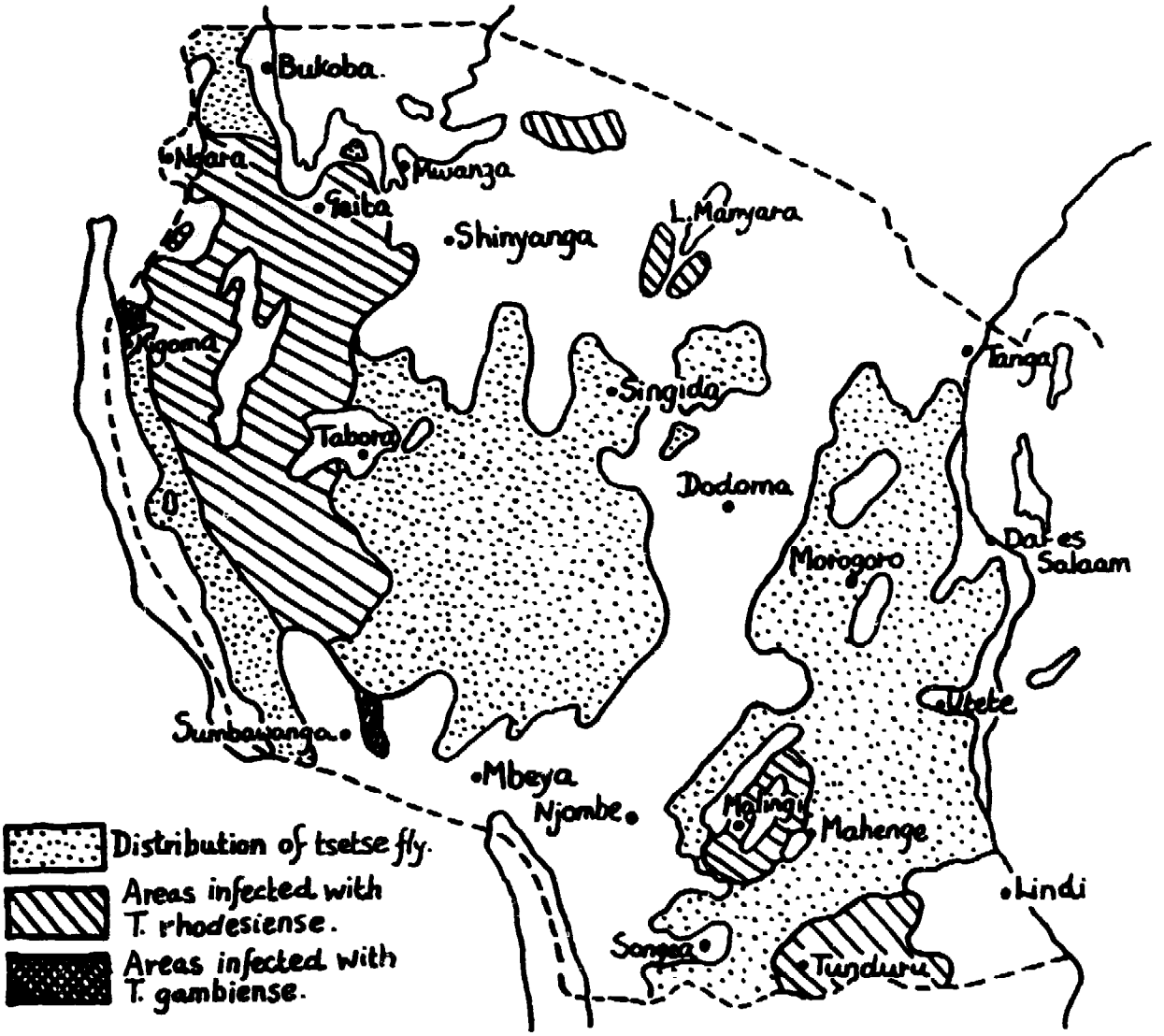
Tsetse flies need wooded vegetation and are not found on flat plains, closely cultivated areas, or areas densely inhabitated by human population.

Two types can be seen:

–riverine, breeding along rivers and lakes

–woodland, living in open lightly wooded parkland away from water.

The river species are the main vectors of T. gambiense. In East Africa only one species is important: G. palpalis. Trypanosomiasis gambiense is a disease of man only; transmission occurs especially at river crossings and water holes where contact between man and fly can be very intense.



Distribution of vector and parasite in Tanzania.

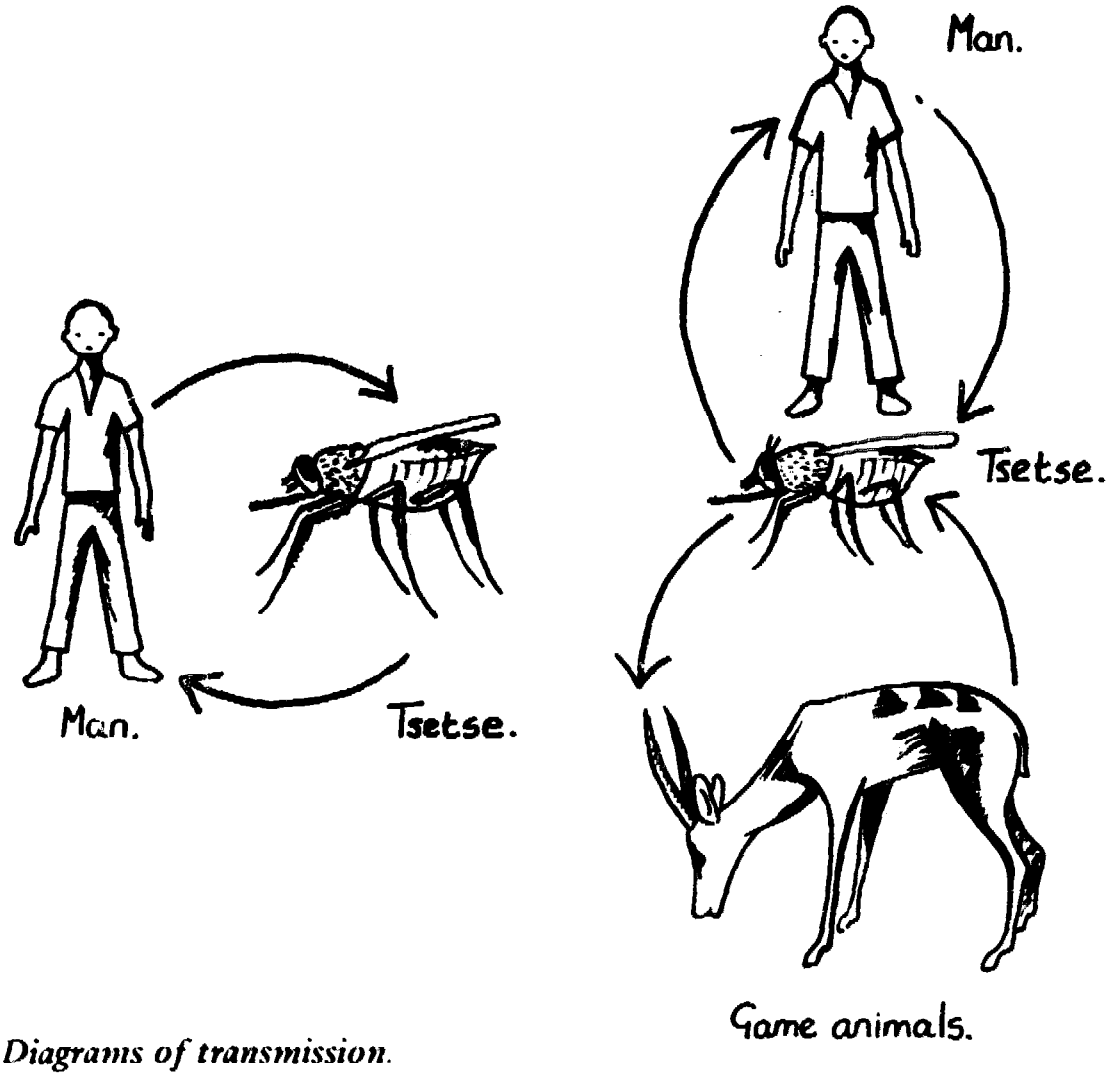
The main vector of T. rhodesiense is G. morsitans; it prefers to bite cattle and game but will also bite man.

In the dry season the fly population is at a minimum and concentrated in certain limited areas which are known as permanent breeding places, primary foci or 'homes'.

Tsetse flies hunt by sight and are attracted by moving dark objects. Hence they follow buffaloes and vehicles.

Tsetse flies become infected with trypanosomes when they take a blood meal from infected men or animals. In the tsetse fly the trypanosomes undergo transformation during three weeks. After this period the flies may transmit infection when

taking another blood meal. In heavily infested areas about one fly in every 3000 is infected with trypanosomes. Contact between Man and fly is not frequent because of the behaviour of the Glossina species and therefore trypanosomiasis only occurs sporadically in people who live close to infested areas or people who, due to their profession, enter tsetse fly infested areas, e.g. wood cutters, honey collectors, game rangers, and hunters.



Diagrams of transmission.

4. *Clinical picture*

Since rhodesiense trypanosomiasis is more serious, and more frequent in Tanzania, most emphasis will be laid on it. For comparison with gambiense trypanosomiasis see appendix to this section (p 101). Since the disease may mimic many other organic diseases, it is advisable always to suspect trypanosomiasis when

you are working in an endemic area.

Trypanosomiasis develops in stages, according to the places where the parasites are found (see table).

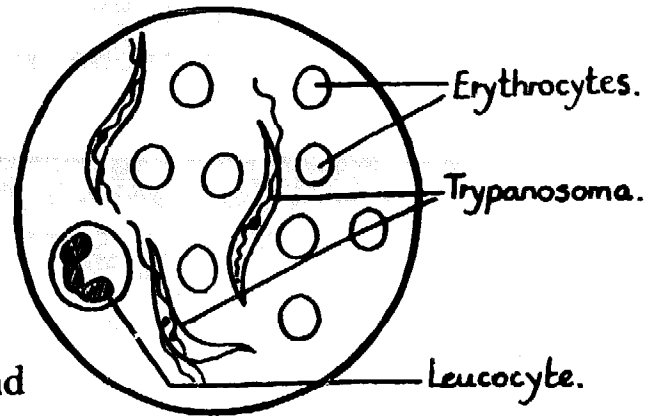
Time after bite	Parasites found	Pathology	Clinical features	Diagnosis from
1–3 weeks local stage	Only at place of bite	Trypano-some chancre	Local swelling painless; spontaneous recovery	Examination of aspirated fluid from boil
1–2 months fever stage	In blood and lymph	Parasit-aemia	Irregular fever; Headache; General weak-ness	Thick blood film (freshly taken and repeated several times) Aspiration of lymph gland fluid (LP is contra-indicated in this stage)
From 4 months	In organs: Heart	Pancarditis	Quick irregular pulse; Oedema; CCF	Repeated examination of fresh blood smear
	Bone marrow	Anaemia	Headache; Anaemia signs; CCF	Bone marrow biopsy
	Lymphoid tissues		Lymphaden-opathy; Splenomegaly	Lymph gland fluid
	Kidney	Glomerulo-nephritis	Oedema; Proteinuria	
	Brain	Meningitis; Encepha-litis	Severe head-ache; Apathy; Dizziness; Difficulties in walking, speaking; Tremors of fingers, tongue	CSF: raised protein, raised cell count *Note:* In case of serious suspicion, the patient should be referred to the trypanosomiasis ward in Tabora Hospital. There the diagnosis can be made by inoculation of labora-tory animals

Diagnosis:

The diagnosis is confirmed in the early stages by repeated examination of fresh blood slides (especially useful in T. rhodiense); lymph gland puncture (more useful in T. gambiense); and in the later stages by bone marrow examination. Inoculation of sus-

ceptible animals can be done in trypanosomiasis laboratories (Tabora).

Differential diagnosis:
Fever stage: malaria
relapsing fever
typhoid
tuberculosis
Visceral stage: meningitis
leukaemia and reticulosis
psychosis
hookworm anaemia
cachexia from other cause (Tb, malignancies).



Trypanosomes in blood smear.

5. *Management of individual patient*
Early cases: Berenil im (suramin formerly used)
Advanced cases: mel B

For doses and ways of administration see circular of Ministry of Health, *Sleeping sickness notes* (available on request from Sleeping Sickness Service, PO Box 482, Tabora), or textbooks, e.g. *Manson's Tropical Diseases.*

As the treatment may have severe side effects, if you have not had experience with it yourself, refer the patient to special treatment centres.

6. *Control*
The control of trypanosomiasis focuses on:

(a) early detection and treatment
(b) vector control.

Bush clearing is the most effective method of vector control. Use of insecticides is expensive and only of temporary benefit. The same is true for fly-catching and larvae destruction.

The establishment of villages will in the long run reduce the number of breeding places within flying distance of the villages.

By turning bush land into agricultural land the habitats of the tsetse will be destroyed. There are no effective drugs available to prevent the development of the disease.

Suramin, useful in the prevention of gambiense trypanosomiasis, is not effective in the prevention of rhodesiense trypanosomiasis. It is even dangerous, since it suppresses the early symptoms.

7. *Action*

At health centre level:

—Enquire at the veterinary department if tsetse flies are present in the catchment area of your health centre. If so, be on the look-out for trypanosomiasis.

—When villages are built in tsetse-infested areas advise the ward development committee
  (a) to surround the village with cultivated ground;
  (b) to have the cattle pens at the outskirts of the village; if tsetse flies come for a blood meal they will be satisfied by sucking blood from animals and not from humans.

—Give health education about protective clothing and early symptoms to people who are in danger of infection due to their profession, e.g. game wardens.

At national level:

Co-operation between Ministries of Agriculture, Natural Resources (Veterinary Department), Health, and Land and Housing is necessary in order to co-ordinate eradication schemes of tsetse flies.

A trypanosomiasis service is established in Tabora (PO Box 482). In Tanga there is a tsetse research station.

Note: Trypanosomiasis is a notifiable disease.

8. *Summary*

Trypanosomiasis in Tanzania is mostly caused by Trypanosoma rhodesiense. The disease does not present as sleeping sickness,

but may mimic other febrile diseases.

The disease is transmitted by tsetse flies. The flies are of economic importance due to the transmission of Trypanosoma brucei which is pathogenic for cattle.

Control consists mainly of destruction of the habitat of the vector by bush clearing, and establishing agricultural land.

PLAGUE

1. Plague is a highly infectious bacterial disease which can kill many people within a short time. The first case has the clinical picture of bubonic plague. Afterwards it may spread as pneumonic plague.

   Synonym: *Ugonjwa wa tauni.*

2. *Occurrence and importance*

   Plague was one of the major diseases recorded in the Middle Ages with extensive epidemics. These epidemics do not occur nowadays. But East Africa continues to be one of the few remaining foci of plague in the world. Small outbreaks occurred in Arusha in 1969 and in Moshi in 1972.

   The disease is rare, but its importance lies in the fact that it may spread at an enormous speed, unless the very first case is recognized and appropriate action taken. This first identification will usually be the duty of an RMA or MA, who normally has to provide primary care in outbreaks.

   The fatality rate can be as high as 60% in some outbreaks.

3. *Epidemiology*

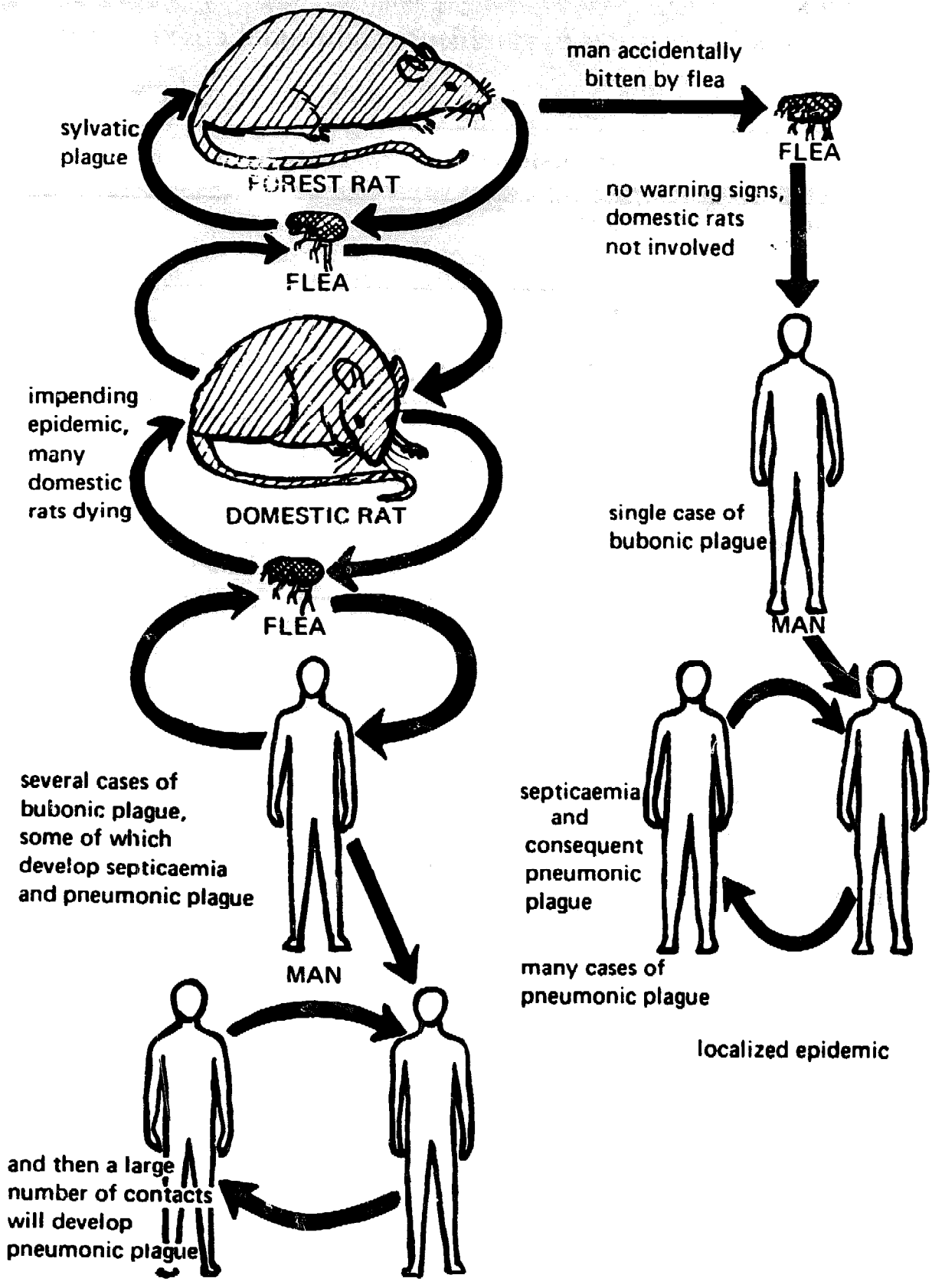
   Plague is caused by a gram-negative bacilli named Yersinia pestis (formerly Pasteurella pestis). The disease is endemic in wild rodents living in forests in the highlands. It is spread by fleas from the rats.

   The former great epidemics of plague were caused by the fact that when forest rats died from the disease, the fleas looked for other rat hosts and took domestic rats as substitutes. When the

Appendix

Table comparing features of gambiense and rhodesiense trypanosomiasis.

	Rhodesiense	Gambiense
Importance in Tanzania	Great	Limited
Reservoir	Man Wildlife Cattle (zoonosis)	Man (Disease of Man only)
Vector	Glossina morsitans palidipes swynnertoni	Glossina palpalis
Environment	Savanna bush land	Water holes River banks
Fatality rate	High	Low
Clinical course	Acute 4–8 months Severe	Chronic two years Mild until late stages
Cerebral symptoms 'sleeping sickness'	Not frequent	Frequent
Involvement of lymph glands	Minimal	Predominant
Diagnosis most probable from	Blood	Lymph gland puncture
Chemoprophylaxis	None	Pentamide (suramin)
Main control method	Prevention of breeding of vector	Treatment of human reservoir Reduction of Man-fly contact
Treatment: Stage I Stage II	 Suramin Melarsoprol = mel B	 Melarsen Melarsen or tryparsamide



Plague cycle.

domestic rats also died the fleas started to bite human beings. In the first human being infected the disease causes bubonic plague, but in the terminal stages of this there is septicaemia and plague pneumonia, which can spread directly from man to man. For example, a person working in the forest is bitten by an infected flea. He may develop symptoms of bubonic plague and later develop septicaemia and pneumonic plague. By coughing infective plague bacilli he infects his relatives and suddenly many people die from an unknown disease.

4. *Clinical picture*

Bubonic plague:

bacilli in lymph gland only

Characterized by bubo swelling of the lymph glands (bubo). Mostly the glands of the groins, sometiines armpit or other places. Swelling may be size of an egg, tender or non-tender.

bacilli in blood: septicaemia

Sudden high fever
Shock
Prostration
Coma
Death on 3–5th day.

In the septicaemic stage the bacilli are everywhere in the organs, including the lungs. The patient may cough and spread the bacilli to his attendants. They develop:

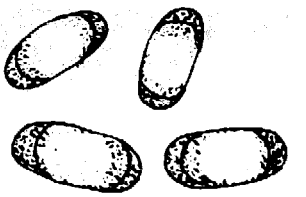
Pneumonic plague:
- acute onset
- severe prostration
- watery sputum quickly followed by blood-stained sputum
- pleural effusion
- death within 1–2 days.

Difference from normal pneumonia: extreme illness of patient without definite physical signs.

Minor plague: A few people who contract bubonic plague may

not appear to be very sick. The fever comes down when the bubo bursts and starts discharging pus. These patients may walk around and infect others. The lesion can be differentiated from lymphogranuloma venereum by making a smear of the pus.

Diagnosis: The presence of vast numbers of gram-negative bacilli in sputum or pus from lymph nodes.



The bacilli are short and thick with rounded ends; the extremities stain a deeper colour than the inter-polar part, giving a bipolar appearance.

5. *Management of the individual patient*

The bacilli are very sensitive to all antibiotics or chemotherapeutics. Early treatment with streptomycin, sulfa, or tetracycline is indicated.

6. *Control*

Early diagnosis is most important. In cases of suspected plague, the patients should *not* be referred to the hospital but IMMEDIATE assistance of the DMO must be asked for. Plague is an internationally notifiable disease (see Appendix B, p 344).

—Chemoprophylaxis of all contacts must be started forthwith (sulfadimidine).

—The area where the disease occurs must be quarantined (isolated from outer world); the police or army must do this.

—Insecticides may be distributed to kill fleas.

—A vaccination programme may be started by the DMO.

—Encourage people to kill rats.

It is hoped that you will never see a case of plague, but remember that most probably the initial recognition of the dis-

ease must be by an MA or RMA, so your quick action may save the lives of many people

EARLY RECOGNITION OF PLAGUE IS A MATTER OF LIFE OR DEATH

7. *Action*

If you recognize a case of plague do the following:

(i) Notify your DMO; ask him to come immediately; plan to inform the public.

(ii) Treat the patient with antibiotics.

(iii) Give sulfa prophylaxis to all contacts (including yourself).

8. *Summary*

Plague is a disease of rats. It is transmitted by the rat flea. When Man becomes infected, the first case(s) present as bubonic plague. Septicaemia may result in pneumonic plague. Pneumonic plague spreads very rapidly. Early recognition and notification to the authorities is vital.

SCHISTOSOMIASIS

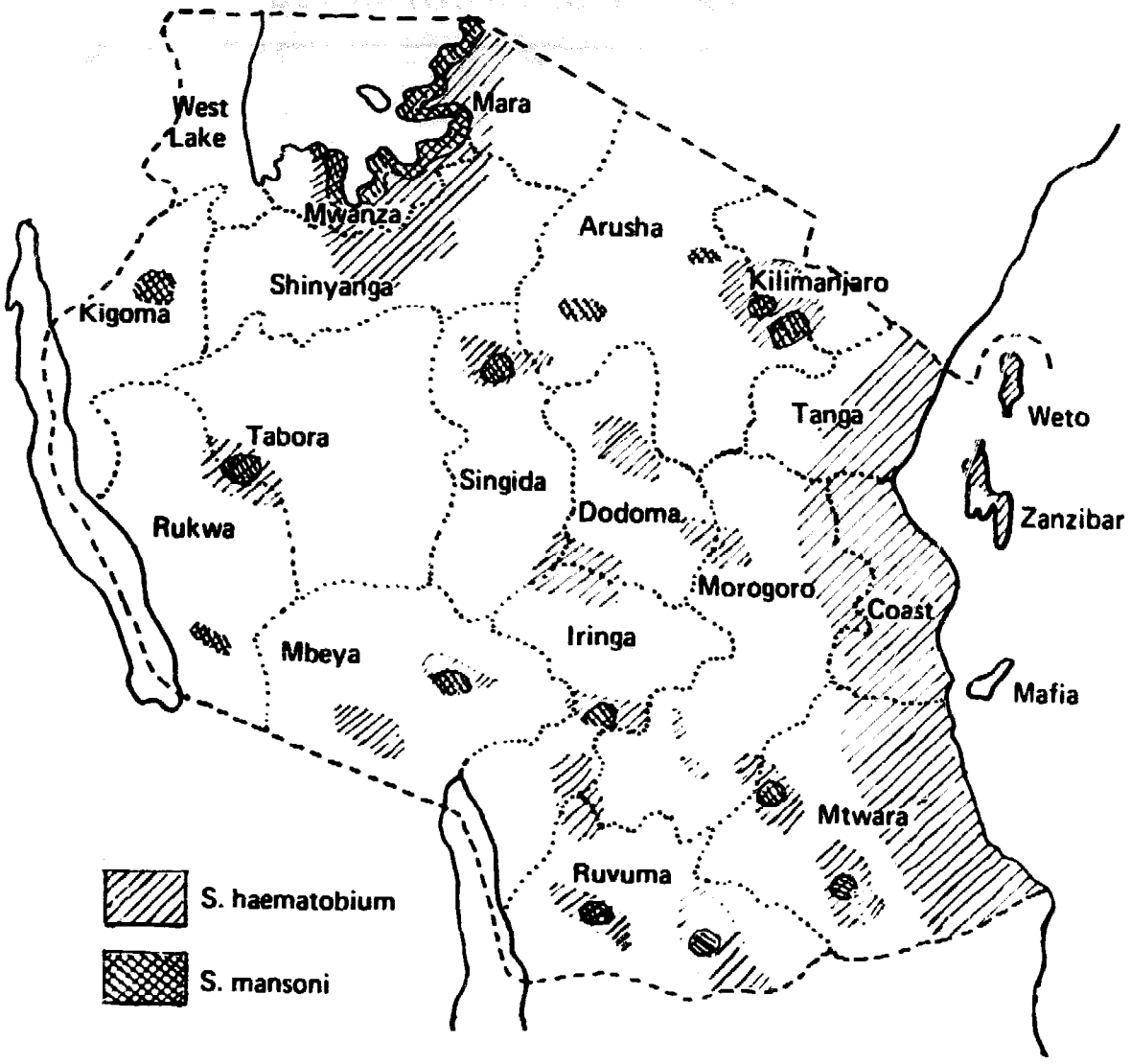
1. A chronic disease caused by the reaction of the body to the eggs of a worm. The clinical picture depends on the location of the eggs in the body.

   Synonym: Bilharzia, *kichocho.*

2. *Occurrence and importance*

Figures from surveys indicate that schistosomiasis is the second most frequent disease in Tanzania (after malaria). Infection, however, does not necessarily lead to clinical disease. This depends on the worm load, duration of infection, and immune state of the patient. Although it is known that schistosomiasis can result in killing conditions such as portal hypertension, uraemia and heart failure, it is not known how often these

occur. There is still no general agreement about the effect of schistosomiasisis on the general health of the people. Opinions vary from life-threatening to harmless. Mild infections may indeed be quite symptomless and cause no ill effects at all. For distribution—see map.



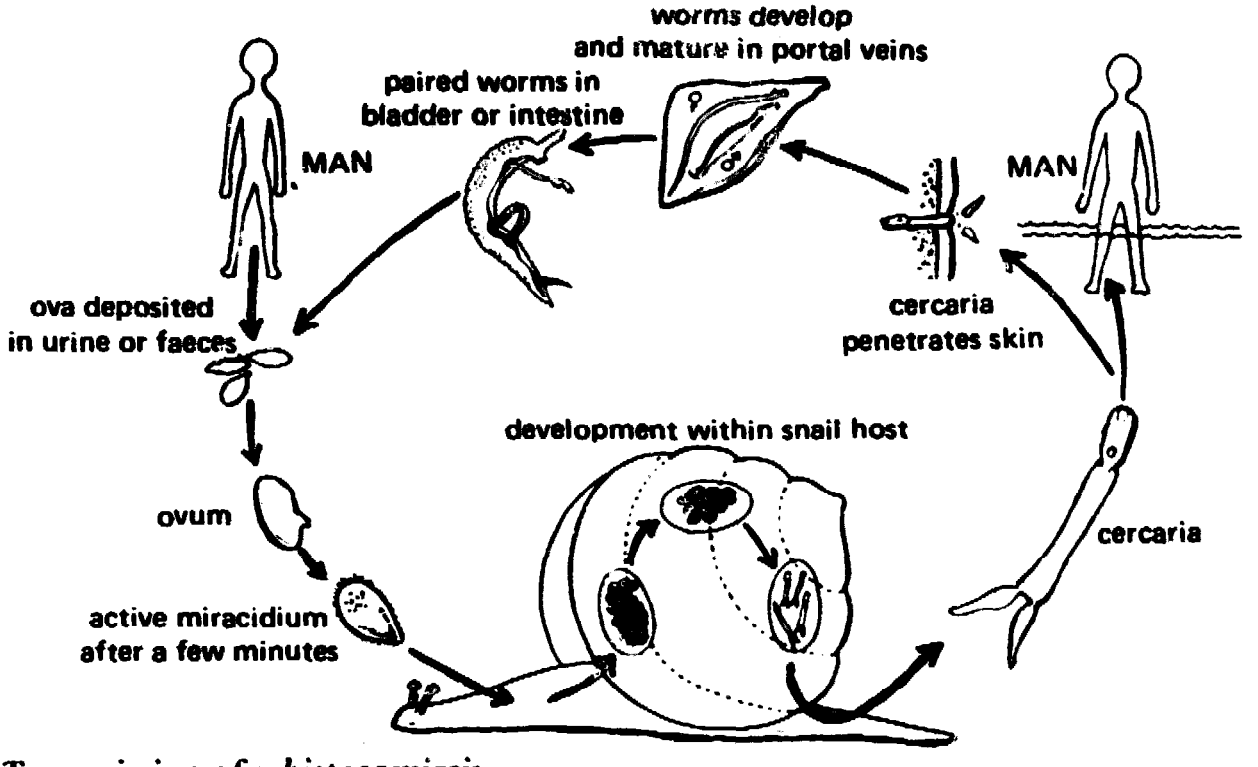
Schistosomiasis in Tanzania.

The incidence of schistosomiasis is related to water use. Development of water projects for irrigation or electricity provides the habitat for the snail vector. Such projects may cause epidemics of schistosomiasis. The rise of socio-economic levels with improved agricultural techniques has, all over the world, been accompanied by an increased incidence of schistosomiasis. It is quite likely that this will also happen in Tanzania.

3. *Epidemiology*

Schistosomiasis is caused by the tissue reaction against eggs of the schistosome worm which is a type of fluke (flat worm). Depending on the species, the eggs are extrected in urine or faeces.

When the eggs reach water a miracidium hatches. This is a free-swimming larva which has to reach a snail host within 24 hours. In the snail, development and multiplication take place to form many cercariae. The cercariae are shed from the snails. They live up to 48 hours. Man is infected by the cercariae present in water. Infection takes place during cultivating, bathing, fishing, and other water-related activities.



Transmission of schistosomiasis.

Two types of schistosomiasis can be seen, each with a different clinical picture, depending on where the adult worms live in the body, and a difference of distribution, dependent on snail host behaviour.

Schistosoma haematobium lives in the venous blood plexus of the bladder; eggs are excreted in urine. The vector snails

belong to the Bulinus species which lives in temporary bodies of water like ponds, dams, and paddy fields. It will adapt to adverse conditions during the dry season by inactivity (aestivation).

Schistosoma mansoni lives in the mesenteric plexus of the lower intestine; eggs are excreted with faeces. The vector snails belong to the Biomphalaria species which prefers permanent water like streams, irrigation schemes, and lakes.

The differences between S. haematobium and S. mansoni are shown in the diagram on p 115.

4. *Clinical picture*

The status of schistosomiasis are: (a) invasion; (b) maturation; (c) established infection; and (d) late stage.

(a) *Invasion* In this stage the cercariae penetrate the skin. This causes cercarial dermatitis with itching papules and local oedema. It is more severe when caused by bird and animal schistosomes (swimmers' itch). Very often, however, it is not noticed, or not reported.

The cercariae remain in the skin for 5 days before they enter the lymphatic system, and reach the liver via the right side of the heart and the lungs.

(b) *Maturation* The schistosomes mature in the liver. This stage is associated with fever, eosinophilia, abdominal pain, and transient generalized urticaria. It is known as the Katayama syndrome. After maturation the worms descend the portal vein. S. mansoni migrates to the mesenteric veins in the intestinal wall, and S. haematobium finds its way to the bladder plexus. In rural inhabitants, again, this stage may pass largely unnoticed--perhaps diagnosed as 'clinical malaria'.

(c) *Established infection* This is the stage of egg production. To continue the life cycle the eggs have to reach the lumen of bladder and bowel. Eggs are produced by the female worms

after moving as far down the small veins of bowel and bladder wall as they can go. Some eggs do not penetrate the tissues and are carried with the blood-stream to the liver and lungs. Other eggs penetrate into the tissues with the help of their spines but fail to reach the lumen of the bowel or bladder. The eggs which do not reach the lumen provoke an inflammatory reaction. It is this inflammatory reaction and the resulting fibrosis that cause the signs and symptoms of schistosomiasis: signs of colitis with bloody diarrhoea and cramps in S. mansoni infection; terminal haematuria and dysuria in S. haematobium infection.

(d) *Late stage* This is the stage of fibrosis, which occurs where there are eggs in the tissues. Around the bladder this may result in

—stricture of the urethra leading to urine retention or fistula

—dilatation of the ureters (hydroureter) and kidney (hydronephrosis) possibly leading to kidney failure

—calcification of the bladder.

In the liver the fibrosis is periportal resulting in portal hypertension. Portal hypertension leads to hypersplenism and anaemia, eosophageal varices and bleeding.

In the lungs the fibrosis results in pulmonary hypertension and so leads to congestive cardiac failure (CCF).

Carcinoma of the bladder is more common in persons with S. haematobium infection than in the general population.

Diagnosis: The diagnosis of schistosomiasis is confirmed by finding eggs in stools or urine; if these are repeatedly negative a biopsy can be done (rectal snip, liver biopsy, bladder biopsy).

5. *Management of the individual patient*

Drugs in common use are tabled on the next page.

Drug	Dose	Remarks
Stibophen (Fouadin) im	Adults: 1st day 1.5 ml 3rd day 3.5 ml 5th day 5.0 ml 7th day 5.0 ml Continue 5 ml on alternate days until a total of 60 ml has been given (that is on day 25).	Price of full course TShs 3/-. This schedule is long, and therefore difficult, but cheap. Do not use if liver is damaged.
Stibocaptate (Astiban) im	10 mg/kg; full course consists of 5 doses on alternate days; toxic effects are less if doses are given twice weekly.	Price of full course TShs 12/-. *Note:* Expensive, not to be used in hepatitis, myocarditis, nephritis, pregnancy. Avoid heavy exercise.
Niridazole (Ambilhar) oral	25 mg/kg/day for one week, generally 2½ tabs daily; side effects gastrointestinal disturbances, psychosis. Combine with phenobarbitone. Not always effective in S. mansoni infection.	Price of full course TShs 11/-. Contraindications: poor general condition, epilepsy, liver disease, psychosis. Do not combine with INH (e.g. with thiazina).
Metrifonate (Bilarcil)	7.5 mg/kg bodyweight as single dose. If symptoms persist, repeat after one month.	Cheap (3/-); effective; non toxic; for S. haematobium only.
Hycanthone (Etrenol) im	Single injection.	Expensive (40/-); serious side effects; for hospital use only; for S. mansoni only.

Notes:

1. The finding of S. mansoni eggs in a routine examination is not always an indication for treatment. The drugs used in the treatment of S. mansoni have serious side effects and are dangerous. These drugs may do more

harm to the patient than the worms. Reinfection is likely to occur.

2. Stibophen, stibocaptate and niridazole are effective against both S. mansoni and S. haematobium.

3. The choice of drug to use in S. mansoni is difficult and there is no agreed policy. Stibophen is the cheapest drug. However, it requires a long course and it is difficult to persuade patients to complete it. Stibocaptate is four times as expensive, and only suitable for individual patients. (Oxamniquine is a new drug, effective only against S mansoni. Side effects are few. Dose 15 mg/kg body weight b d for 2 days [total 60 mg/kg] . It is not yet available in Tanzania.)

Summary:
S. mansoni, drug of first choice: stibophen; alternative: stibocaptate.
S. haematobium, drug of first choice: metrifonate; alternative: stibophen.

6. *Control*

Infection of snails may be prevented by using pit latrines. However, only a small amount of contamination of the water is needed to keep the infection going. So pit latrines are more important in preventing other diseases than in control of schistosomiasis.

(a) *Control of snails*

The control of snails is one method for reducing transmission of schistosomiasis, but snail control has proved to be very difficult. In fact schistosomiasis is increasing due to new agricultural methods, e.g. irrigation schemes form an ideal breeding place for snails and very often the introduction of irrigation methods is followed by an epidemic of schistosomiasis.

Chemicals (molluscicides)
These are not likely to give complete eradication of snails. Disadvantages of molluscicides: expensive and may kill fish. Application of molluscicides should be timed in such a way that it causes maximum damage to the snail population es-

pecially when, due to climatic conditions, pronounced fluctuations in density of snail population occur (Bulinus). The most important molluscicides are:

Copper sulphate ($CuSO_4$). Safe to handle, kills both snails and their eggs, not as toxic to fish as other compounds, is most effective at low pH, ineffective in high pH, corrosive to equipment, absorbed by soil and organic material.

Niclosamide (Bayluscide). Safe to handle and use, kills both snails and eggs, cheapest chemical per volume of water treated, tends to clog in equipment used. Effectiveness is slowly reduced by sunlight, absorption by mud, or high concentration of minerals (hard water).

(b) *Environmental sanitation*

Prevention of snail breeding may be more important than killing of snails. This requires radical alteration of snail habitats by:

—draining or filling of water bodies

—clearing of vegetation in water bodies to deprive snails of food and resting places

—irrigation schemes: intermittent irrigation will result in sudden changes of water level and wave action

—flooding (harmful for all snails)

—changing speed of water flow in channels and rivers

—protecting ponds

—straightening and deepening margins of water bodies

—when irrigation schemes are planned, these should be organized in such a way that at least no increase in snail population will occur. Sanitation engineers or public health specialists should be involved in planning

—the supply of piped water to the people will not directly affect snail breeding but is most effective in reducing contact between man and snail and so will greatly reduce tranmission of schistosomiasis.

(c) *Biological*

Introduction of snail enemies can result in disappearance of vector snails.

—A certain large bug is an obligatory snail-eater and a single bug will kill about 125 snails in its life.

—Some enemy snails will eat eggs and the young of the vector snail and will compete for other food.

Disadvantage: Some enemy snails can act as intermediate hosts of the human lung fluke, a parasite not known in Tanzania. Introduction of such an intermediate host would therefore be a serious potential health hazard.

Snail control through environmental sanitation and or molluscicides needs maintenance over a long period (at least 10 years). It is impossible to use either of these methods generally in Tanzania because of prevailing economic problems.

(d) *Prevention of infection of human beings*

The most effective way to protect human beings from schistosomiasis is to give them an ample supply of safe water. Building of public water kiosks has the highest priority in the prevention of the disease. Water from other sources can be made safe by keeping it in a container for 48 hours—the three-pot system. Within this period the cercariae will have died.

Mass treatment

Due to the lack of cheap, effective, and safe drugs, mass treatment has not been possible until recently. Some experts now advocate the mass use of metrifonate (WHO Report to Zanzibar).

7. *Action*

—When there is a high prevalence of schistosomiasis do not use all the available money for treating people with only a positive

stool examination haphazardly.

—Give frequent health education on how the disease is spread.

—Stress the importance of a safe water supply.

—Emphasize the need for building pit latrines.

8. *Summary*

Schistosomiasis is a common disease in Tanzania. Its public health importance is uncertain.

A cheap, safe, and effective drug is now available for the treatment of S. haematobium; but treatment of S. mansoni is still a problem. Control is difficult and expensive. No single measure has been effective.

	S. haematobium	S. mansoni
Egg		
	Terminal spine found in urine	Lateral spine found in faeces
Adult worm	Plexus of bladder	Mesenteric veins of bowel
Vector	Bulinus (actual size)	Biomphalaria (actual size)
		
	Temporary water bodies, e.g. paddy fields, ponds, dams	Permanent water bodies, e.g. lakes, rivers, irrigation schemes
Early disease	Haematuria	Bloody diarrhoea 'mansoni dysentery'
Late disease	Bladder calcification; Carcinoma of bladder; Hydronephrosis; Kidney failure; Pulmonary hypertension leading to CCF	Bowel fibrosis; Portal hypertension with hypersplenism; Oesophageal varices; CCF
Treatment	Metrifonate	Stibophen or stibocaptate

Chapter four

DISEASES CAUSED BY FAECAL CONTAMINATION

Introduction

The diseases in this group have in common that the causative organisms are excreted in the stools of infected persons (or, rarely, animals).

The portal of entry for these diseases is the mouth.

Therefore the causative organisms have to pass through the environment from the faeces of an infected person to the gastro-intestinal tract of a susceptible person. This is known as the faeco-oral transmission route.

Faeco-oral transmission occurs mostly through inapparent faecal contamination of food, water, and hands. Very small

amounts of faeces can carry enough organisms to establish infection. So sparkling clear water may be dangerously polluted; contaminated food may taste, smell, and look normal but harbour disease; and apparently clean hands may carry and transmit enough micro-organisms to spread disease. As shown in the diagram on the next page food takes a central position; it can be directly or indirectly contaiminated via polluted water, dirty hands, contaminated soil, or flies.

Water can be polluted directly by faeces, or faecal material may be washed in from polluted soil from the river banks.

Hands are contaminated after defaecation or by touching contaminated objects.

The house fly is very likely to carry faecal material because of its habit of starting a meal on faeces and finishing off on human food. The fly can transfer the germs from faeces to food by carrying them on its body, by vomiting on solid food in order to liquify the food, and by defaecating on food. Flies defaecate every few minutes and the faeces and vomitus may contain surviving organisms from human faeces.

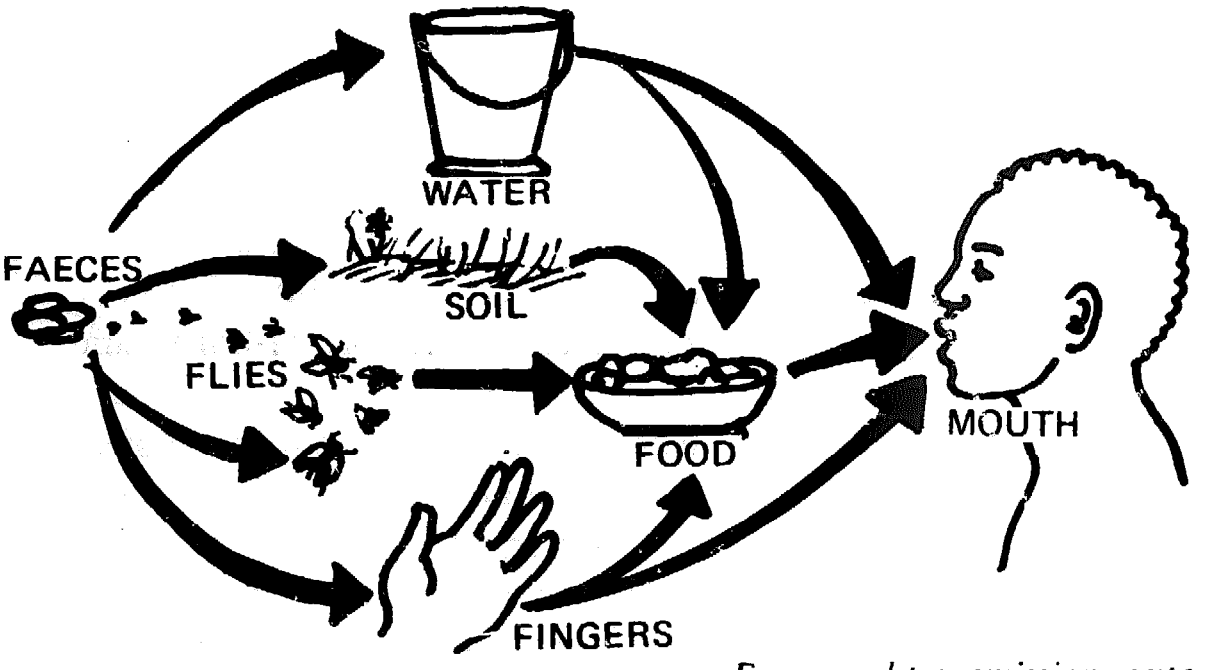
In polio and viral hepatitis infections the virus is detectable in faeces by using special techniques. But faeco-oral transmission is of minor importance in these two diseases, as recent investigations have shown the polio virus is present in nasopharyngeal discharges. So droplet spread is likely to occur and is nowadays regarded as an important way of transmission. The way in which viral hepatitis is spread is the subject of many discussions. Viral hepatitis is regarded as contagious, airborne, faecal-borne, vector-borne and even sexually transmitted.

The eggs of the intestinal worms (and of Schistosoma mansoni) also of course leave the body with the faeces but the portal of entry is not always the mouth, and because of their related life cycles the intestinal worms are dealt with in a separate chapter. Schistosomiasis has already been considered as a vector-borne disease. Food poisoning is not always due to faecal contamination but certainly belongs to the food-borne diseases and is therefore included in this chapter.

We list the diseases for which the gastrointestinal tract is the portal of entry by type of causative organism as follows:

Viral	Bacterial	Protozoal	Toxins	Worms
Polio Hepatitis	Typhoid and paratyphoid	Amoebiasis	Staphylo-coccal food poisoning	Ascariasis Enterobiasis Trichuriasis Taeniasis Hydatidosis
Viral diarrhoeas (entero-viruses)	Cholera Bacillary dysentery* Other salmonelloses Anthrax			See Chapter 5
Unspecified diarrhoeal diseases				

*Dysentery means diarrhoea with blood. Dysentery is a clinical syndrome with different causes. Most well known are: bacillary dysentery—shigella; amoebic dysentery—E. histolytica; mansoni dysentery—schistosomiasis.



Faeco-oral transmission route.

As stated above, the mode of transmission of the viral gastro-intestinal infections is not firmly established. For other infections

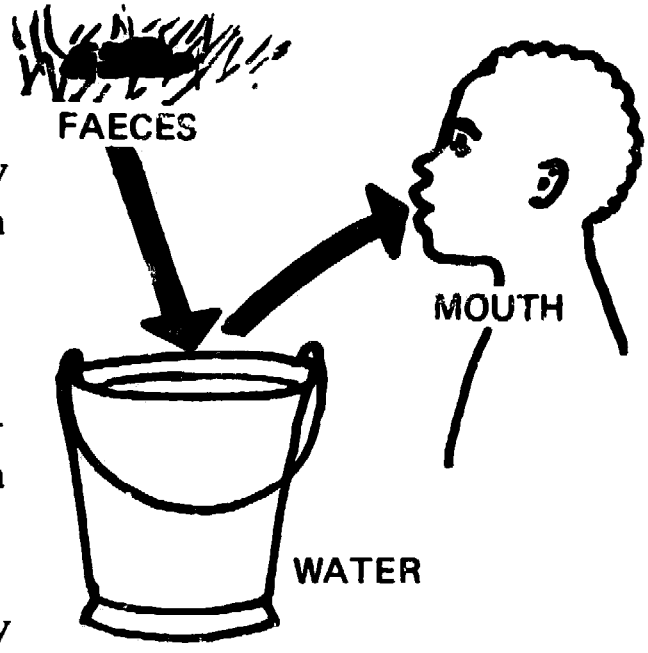
the number of organisms which have to be ingested in order to cause disease have been calculated.

Number of germs necessary to cause disease			Main route of transmission
Vibrio cholerae	Cholera	10^9, = 1,000,000,000	Water
Salmonella typhi	Typhoid	10^5, = 100,000	Water
Salmonella paratyphi	Paratyphoid	10^3, = 1,000	Food/Water
Shigella sp	Dysentery	10^2, = 100	Food/Water

The explanation for the marked differences in the threshold for disease is the sensitivity of the organisms to gastric acid.

This natural defence mechanism is most effective against cholera vibrios and typhoid bacilli (see table). As a result very large doses of vibrios or salmonella typhi have to be ingested in order to cause disease. This makes accidental infection with typhoid or cholera by contact or contamination of food by flies very unlikely and both diseases are therefore almost exclusively water-borne.

When only a very small number of organisms is required to cause infection, as in bacillary or amoebic dysentery, this is very easily done accidentally by the fingers or by contamination of food and eating utensils by flies. Indeed many dysentery cases are usually found during the season when flies are plentiful. Of course infection through contaminated water occurs as well. Contamination of fingers and eating utensils is most likely to occur when water for hand-washing and cleaning is in short supply.



Main way of transmission of typhoid and cholera.

Therefore diarrhoeal diseases may be expected to occur more often in the dry season. These diseases are associated more with *lack* of water than with *contaminated* water and will disappear when the amount of available water is increased. It is the quantity rather than the quality which is important.

Natural defences

(a) One of the natural defence mechanisms against infections of the gastrointestinal tract is already mentioned above (gastric acid).

(b) Bacteria normally living in the intestinal tract (intestinal flora) form an important barrier. As long as they live and multiply in the bowel other organisms have less chance of thriving. This is called bacterial antagonism. One way to predispose animals to bowel infection is to treat them with broad-spectrum antibiotics, which kill their intestinal flora.

(c) A third natural defence mechanism is normal bowel motility, which acts as a cleansing mechanism. As with coughing and vomiting, diarrhoea can also be regarded as a normal body response to get rid of harmful substances.

Thus it may not be surprising that wilful suppression of these mechanisms may be harmful. Studies have shown that treatment of shigellosis patients with opiates (or Lomotil), even when antibiotics are given, will delay recovery. Stool-solidifiers do not decrease the amount of water lost in diarrhoea, they only make the stools look more solid. These are not as harmful as the bowel paralytic drugs, but there also exists the danger that these drugs will be given in place of proper treatment.

Death from diarrhoeal disease is largely due to dehydration and disturbances of electrolyte balance. The bar diagram opposite from hospital data (1973) shows 1,400 deaths caused by gastroenteritis and a total of 200 other deaths from diarrhoeal diseases (dysentery and food poisoning). Typhoid fever and hepatitis look unim-

portant with 160 deaths.

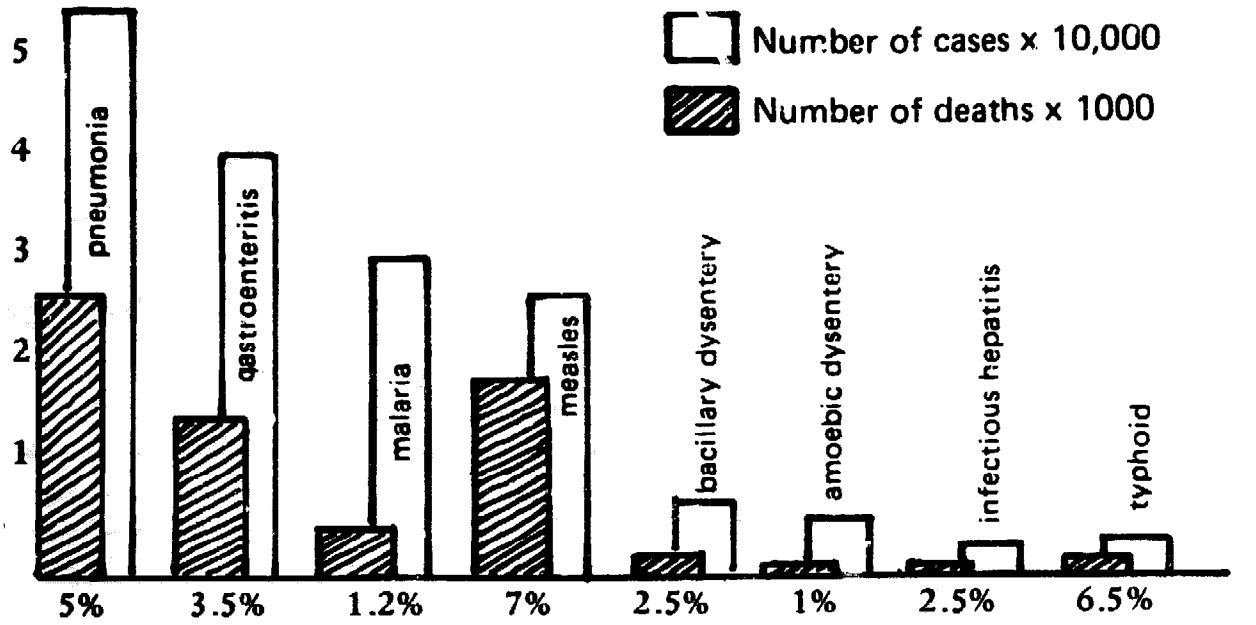
Other facts

From the bar diagram it can be seen that gastroenteritis is the second commonest cause for admission to hospital. Compare with pneumonia, measles and malaria.

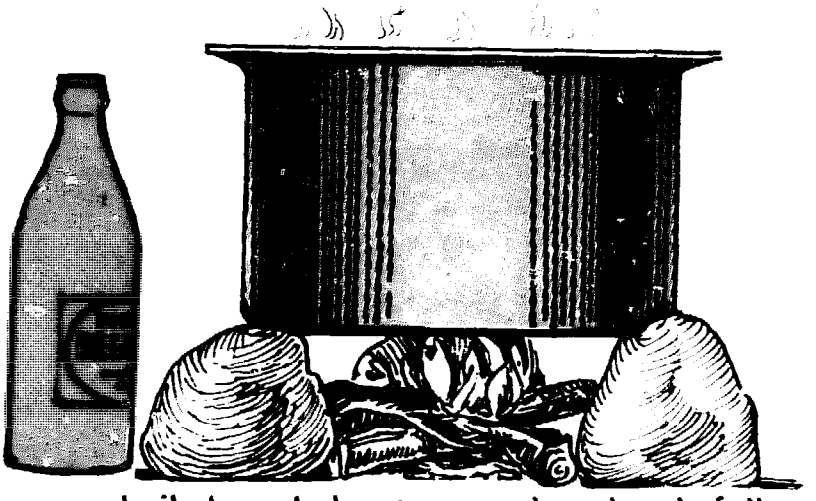
Gastroenteritis is the most frequent form of diarrhoea. It accounted for more than 40,000 hospital admissions, compared with less than 5000 caused by bacillary dysentery. It should be realized that these figures show hospital data only. The real extent of the problem is unknown; there are no figures available from the villages where most probably a large number of untreated and fatal cases occur.

Therefore, prevention and early treatment of dehydration should be the first priority in the treatment of all diarrhoeal diseases.

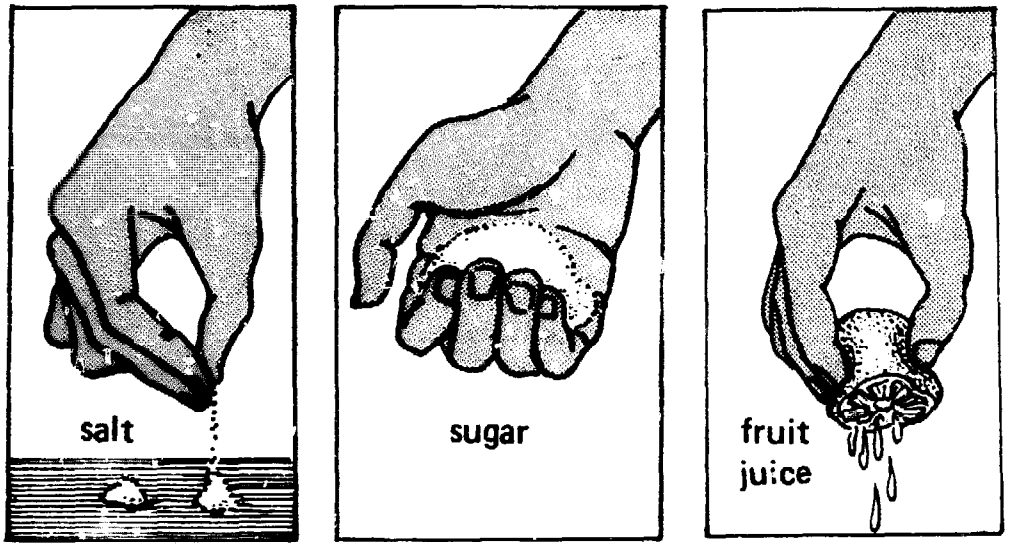
REHYDRATION FIRST PRIORITY



Treatment In mild cases oral rehydration with home-made fluids is best. (Tea.is also a good oral rehydration fluid.)



boiled, cooled water, one beer-bottle full



THIS IS HOW SIMPLE IT IS TO SAVE A LIFE

*Preparation of home-made oral rehydration solutions**

One pint of boiled water (1 beer-bottle full), two thumb-and-two-finger pinches of salt, one four-finger handful of sugar and some fruit juice.

Give every child as much as possible of this rehydration mixture. Continue even when children are vomiting.

In severe dehydration parenteral rehydration is necessary. If

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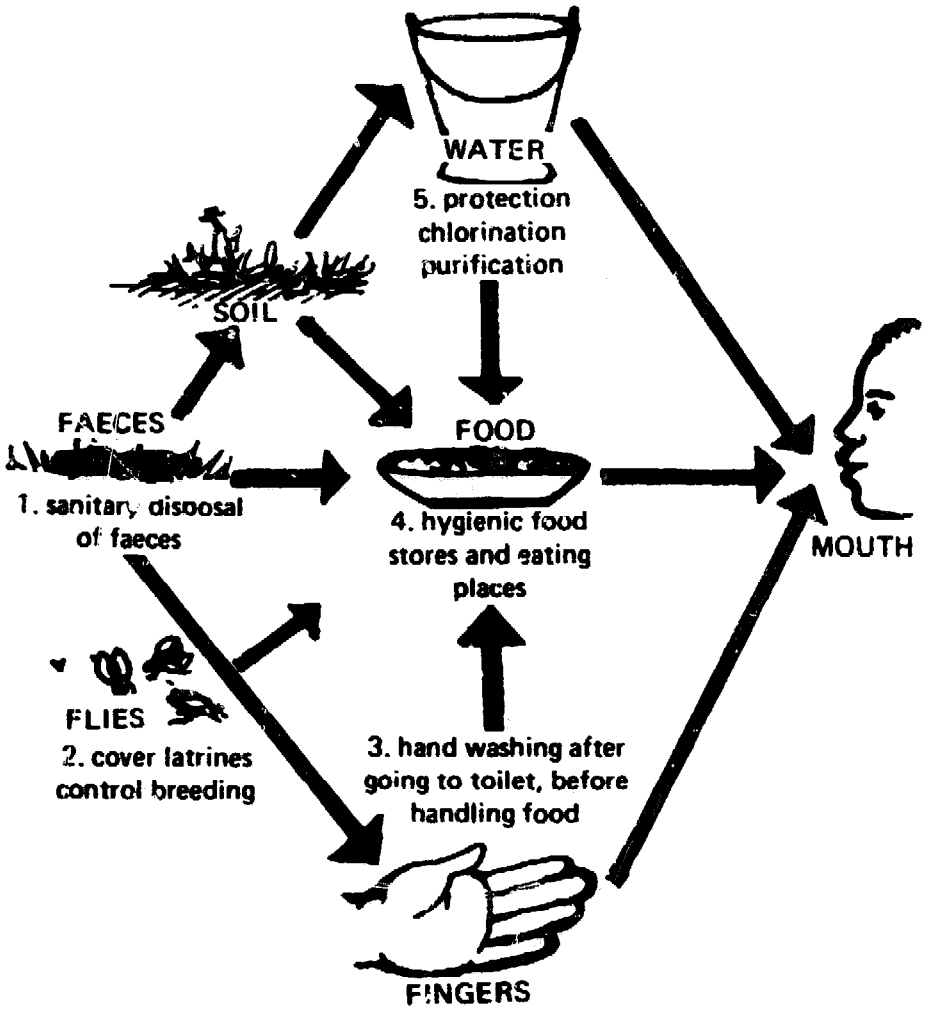
***UNICEF is preparing a mixture of salts and glucose to be dissolved in drinking water. It will be named Oral Rehydration Salts or ORS. It may become available in Tanzania. Because this mixture contains glucose in place of sugar, it is better than the home-made solutions. See also p 149.**

dehydration is severe blood circulation is poor and fluids given subcutaneously or intraperitoneally are not rapidly absorbed. Therefore *intravenous* rehydration is best.

It is a widespread practice to suppress vomiting by the use of phenothiazine drugs (Largactil, Plasil). These drugs frequently cause neurological side effects. They act by making the child drowsy. This interferes with oral fluid intake.

TREAT VOMITING BY SUPPLYING EXTRA AMOUNTS OF REHYDRATION FLUID

Prevention Prevention depends on breaking through the faeco-oral transmission cycle.



1. *Faeces*
   Control of diarrhoeal diseases including dysentery is only possible when the problem of stool disposal is solved (deep pit latrines in rural areas). Hand-washing facilities should be provided with toilets.

2. *Fingers*
   Providing hand-washing facilities at toilets. Wash hands after going to toilet, wash hands before cooking or eating.

3. *Flies*
   Fly control by proper refuse disposal and proper disposal of faeces.
   –Screen toilets, cover latrines.
   –Screen kitchens and food stores.
   –Store left-over food where flies cannot reach it.
   –Spray with residual insecticides, use baits and traps.

4. *Food*
   Food should always be properly cooked. Raw vegetables and fresh fruits without intact skins are unsafe*. Milk should be boiled or pasteurized. Eating utensils should be cleaned and dried after use. These include feeding bottles (see also *Child Health* manual, Chapter 12.1 and *Nutrition for Developing Countries*, King, Chapter 8). Sanitary supervision of processing, preparation and serving of foods in public eating places is important.

5. *Water*
   Protection, purification, and chlorination of public water supply. Well water can be made safe with lime (see p 151). Deep well water is usually uncontaminated if the well-head is protected and if there are no pit latrines nearby. Drinking water from

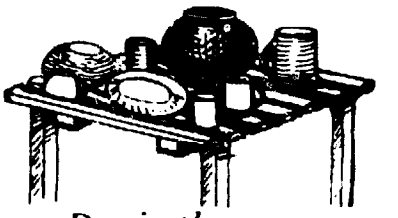
*Green salad leaves can be made safe by immersion for 15 minutes in undiluted vinegar or sodium hypochlorite solution 1% (Milton). Fresh intact fruits should be peeled at the time of consumption.

other sources should be boiled when there is an outbreak of any water-borne disease. Small groups of people may treat water with iodine or chlorine (Halazone) when travelling. Piped water near each house would greatly contribute to general and personal cleanliness, and diminish the risk of infection from contaminated water.

6. *Health education*
   (i) Educate people about dangers of bottle-feeding; encourage cup/spoon feeding methods; encourage prolonged breast-feeding.
   (ii) Explain sources of infectious diarrhoea, encourage the use of latrines (also for children); explain importance of hand-washing, and of cleaning utensils.
   (iii) Demonstrate prevention of dehydration by home-made sugar/salt solutions.

Feeding with cup and spoon.

Washing of hands and utensils.



Dry in the sun.

All these control measures have to be continued indefinitely.

Checkpoints for sources of water-borne diseases

A. Primary school
   (a) Drinking water sources; how and where is the water stored?
   (b) Latrines (number of children): cleanliness of latrines, etc.
   (c) Facilities for washing hands.
   (d) If the school provides food, check on the cleanliness of the cook, kitchen, and equipment; also check the place where children eat food brought from home.

B. Pombe shop
   (a) Source of water: possible contamination, and whether water is stored in clean vessels.

(b) Brewing process: cleanliness of equipment, hygiene of the process and disposal of waste materials.
(c) Staff: personal hygiene, septic sores.
(d) Consumption: care of the drinking cups, etc.

C. Hotel

(a) Food; water source; storage of water. How is the food stored and for how long? Rats and mice around?
(b) Food preparation: kitchen and cleanliness of utensils. Where is waste food disposed of?
(c) Staff personal hygiene, septic sores.
(d) Customers' facilities: latrines, washing facilities, including the cleanliness of towels.

D. Private homes

(a) Water supply: from what distance must the water be collected? Is the supply enough throughout the year? Water sources for domestic animals. Is there a slab for washing clothes?
(b) Water collection: private or common bucket? Who takes care of the cleanliness of the bucket?
(c) Water storage in the house: tins or pots; covered and cleaned regularly; emptied before refilling; protected against animals; advise the family on the three-pot system, boiling of drinking water.
(d) Latrines: each private home should have a latrine; look for the care of each latrine and whether it is used by each member of the family.
(e) Are there washing and bathing facilities?

E. Irrigation furrows

(a) Site: chance of contamination; possibility of protection along its course.
(b) Use: speed and volume of water; fluctuations of amount of water during various periods of the year; use for Man and/or animals; maintenance.
(c) Quality of water: take sample for microscopy and its appearance.

F. Shamba

(a) Where do people working on the shamba get water for drinking?

(b) Where do people working on the shamba go to latrine?

(c) Where is waste and refuse from the shamba put?

DIARRHOEAL DISEASES UNSPECIFIED

1. Acute diarrhoeal disease is a clinical syndrome of diarrhoea (more than five liquid stools in 24 hours), nausea and/or vomiting, and often fever.

   Synonym: *Kuharisha.*

2. *Occurrence and importance*

   Diarrhoeal diseases affect all the population but severity varies in different age groups. Dehydration occurs rapidly in children and is a common cause of death (see *Child Health* manual, Chapter 12). Especially at risk are:

   Infants: Low-birth-weight children and premature children easily get E. coli infections. In the prematures the case fatality rate may be as high as 40%.

   Weanlings: In the weaning period new types of food are introduced to children. They are then exposed to a variety of micro-organisms, pathogenic and non-pathogenic. In this period of their life malnourished children have a low resistance to infection. Incidence of diarrhoea in this period has been reported as 275 attacks in 100 children in one year, almost 3 attacks per child. Diarrhoea is more frequent in hot, dry periods—clearly associated with lack of water.

   Bottle-fed children: Due to lack of cleaning facilities, bottle-feeding is very dangerous in rural areas. Bottle-feeding is associated with a high mortality rate due to diarrhoea.

   Bottle-feeding is very popular amongst partially educated mothers. Grandmothers sometimes give bottle feeds because of

broken families, or working mothers.

Artificial feeding needs a lot of preparation, knowledge, and money which most rural and urban families do not have.

Travellers: Traveller's diarrhoea occurs in people who are exposed to a new environment. This situation can arise from natural disasters, man-made disasters (war refugees) or among migrants (*vijiji*, seasonal labourers) and holiday-makers. It is thought to be the gut's response to new intestinal flora, acquired through faeco-oral contact, but other factors such as changes in food may also contribute. This diarrhoea is self-limiting.

Acute *gastroenteritis* is endemic in all areas where sanitation is poor. From time to time the number of people with diarrhoea increases and so epidemics occur. The diarrhoeal diseases of children are closely related to this endemic diarrhoea in the general population.

3. *Epidemiology*

Many organisms can cause diarrhoea but it is difficult to prove that any particular organism is responsible. Even when sophisticated techniques are used, in half of the cases no organism can be found.

As will be described in the next chapters, bacillary dysentery, amoebic dysentery, paratyphoid fever, food poisoning, cholera, infectious hepatitis, and polio, may all present as gastroenteritis. In children diseases like malaria and otitis media may cause diarrhoea (parenteral diarrhoea). Other diarrhoeas are caused by the group of enteroviruses. In infants the diarrhoea may be caused by enteropathic E. coli (Escherichia coli), a commensal bacterium of the bowel of adults.

All these organisms are transmitted by the faeco-oral transmission route.

4. *Clinical picture*

The macroscopic aspect of the faeces differs with the cause. In E. coli diarrhoea there may be profuse watery diarrhoea with mucus and no blood. Fever is often absent. In weanling diar-

rhoea the onset is acute and the course may be rapidly progressive with frequent liquid or semi-liquid stools anything from 3 to 20 times daily (see also *Child Health* manual, Chapter 12).

Protein-calorie malnutrition is commonly associated with weanling diarrhoea, and frank kwashiorkor may follow an attack.

The fluid balance in children is more easily disturbed than in adults because of rapid fluid turnover. Dehydration readily occurs and may be severe.

The clinical picture of gastroenteritis in adults may vary from a mild abdominal disturbance (intestinal 'flu') to a dysentery-like disease.

5. *Management of the individual patient*

The drug of choice in the treatment of diarrhoea is rehydration fluid. (For the management of diarrhoea in children see *Child Health* manual, Chapter 12.)

6. *Control*

Infants: Prevent low birth weight and prematurity by improved antenatal care.

Weanlings: Prevent malnutrition in the weanling period by nutrition education and improved care for the under-fives.

Bottles: Discourage bottle-feeding (see Introduction).

7. *Action*

See bacillary dysentery.

8. *Summary*

Diarrhoeal diseases are very common in Tanzania. Children are especially at risk. Causative organisms are difficult to identify. Spread is by the faeco-oral transmission route. Correction of dehydration is the main priority in treatment. Control: improvement of sanitation.

BACILLARY DYSENTERY

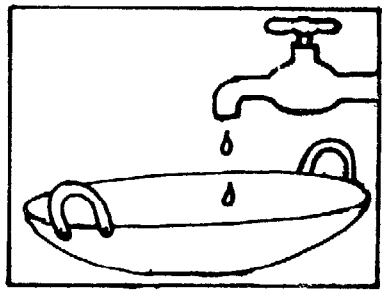
1. Bacillary dysentery is an acute bacterial disease of the intestine, characterized by diarrhoea, fever, vomiting, and abdominal cramps.

   Synonym: Shigellosis, *kuharisha damu.*

2. *Occurrence and importance*
   Bacillary dysentery is common throughout Tanzania, especially in areas where standards of hygiene are low. The main sanitary aspects are:

Rain washing faecal material into water.

   (a) *Faeces disposal*
   (b) *Availability of water*
   (c) *Fly population*
   (d) *Seasonal influences.*



Shortage of water for washing in dry season.

   (i) After the start of the rainy season much faecal material will be washed into ponds and rivers. This will result in heavy contamination of water sources and hence in a high incidence of diarrhoeal diseases.

   (ii) In the dry season the amount of water available for cleaning purposes decreases. Cleaning of eating utensils (and washing of hands) gets a low priority. The available water sources may be contaminated. Thus in the dry season an increase in dysentery cases can be expected.

   (iii) The number of house flies is dependent on seasonal influences. House flies are very likely to transmit dysentery. When the number of flies is high a peak in incidence of dysentery can also be seen.

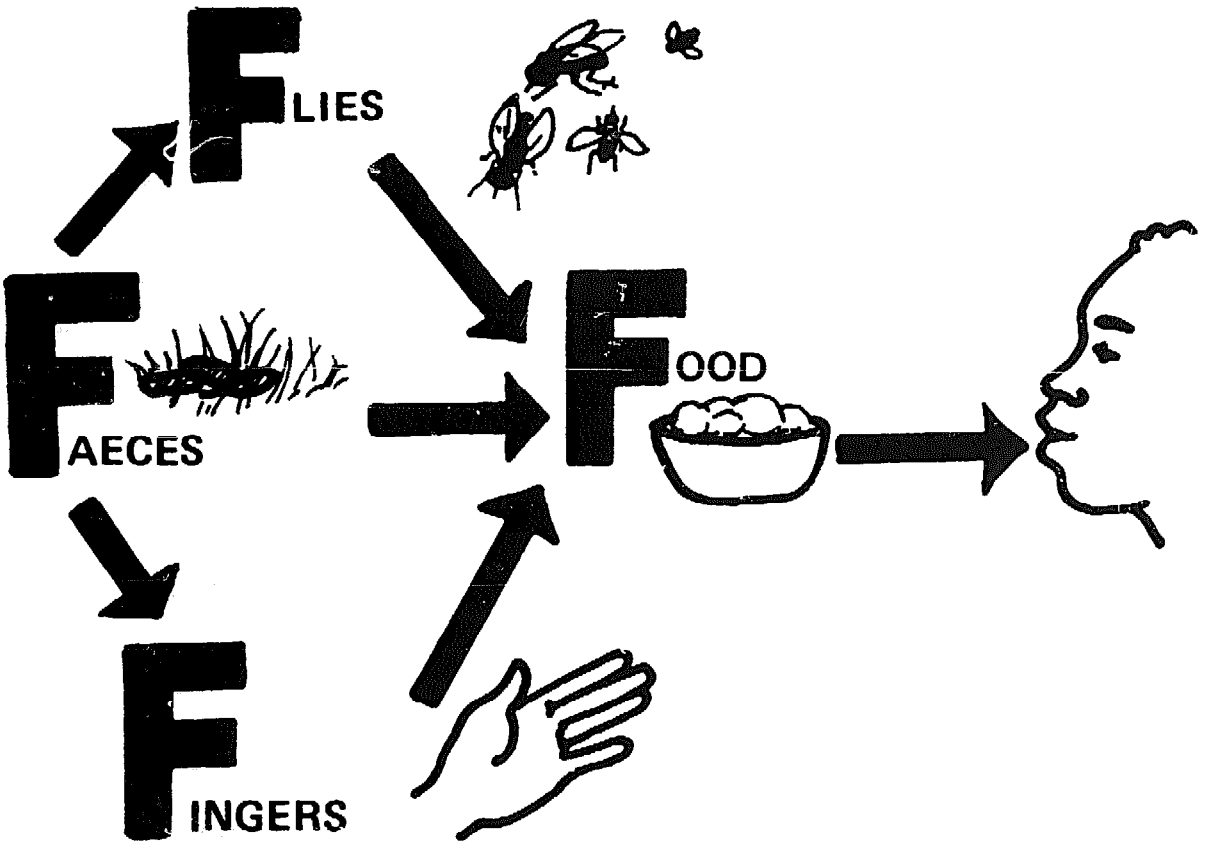
   (e) Other factors
   In malnutrition general resistance against dysentery is lowered. Infection with shigellae will more often result in

attacks of frank dysentery. Children and old people are therefore especially at risk. Other undernourished groups living in poor hygienic conditions, such as prisoners of war and refugees, are also at risk.

In children diarrhoea easily causes dehydration (see *Child Health* manual, Chapter 12.2). Dehydration is the main cause of death in diarrhoeal diseases.

3. *Epidemiology*

Bacillary dysentery is caused by non-motile gram-negative bacilli of the shigella group. Transmission is by faeco-oral contact.



The rule of F.

Shigellae multiply in food. When food is contaminated and is not served immediately, or dishes are not washed properly, the shigellae start to multiply. Ingestion of a single bacterium will not result in disease, a loading dose has to be ingested before

one gets dysentery. When shigellae get the opportunity to multiply the threshold for infection is easily passed.

Children are the main reservoir of infection. They defaecate in and around houses. As the bacilli are present in the stools of the child even several days after an attack, contamination of food and/or water can easily occur.

4. *Clinical picture*

The incubation time is short (1–4 days). In the well-nourished adult, infection with shigella may result only in an attack of mild diarrhoea. In the under-nourished child it may result in a fulminating toxic and fatal disease.

Mild cases are often not recognized and are regarded as non-specific gastroenteritis.

In a classical case the onset is sudden with fever, abdominal pains, and faecal diarrhoea.

After a few motions the diarrhoea stops and the dysenteric syndrome sets in. This is characterized by abdominal cramps (colicky pains) and tenesmus. Tenesmus is painful contractions of the sphincter ani, producing an almost continuous and irresistible urge to defaecate. However, no faecal matter is produced, only small quantities of purulent mucus and blood.

Toxaemia is present due to absorption of an exotoxin. The pulse is rapid, there is high fever, and vomiting. Convulsions occur in children. Dehydration is common and dangerous: it may cause muscular cramps, oliguria and shock.

Diagnosis:

Stools: macroscopic: dark red (blood) with much mucus
microscopic: numerous polymorph leucocytes and many erythrocytes. Macrophages which contain red blood cells (after phagocytosis) are easily mistaken for trophozoites of Entamoeba histolytica. The nucleus of a macrophage is clearly visible and is irregularly shaped. Macrophages move very much more slowly than trophozoites and do not go far in any particular direction.

Differential diagnosis	Bacillary dysentery	Amoebic dysentery
Incubation time	Short Less than one week	Long 3 weeks or more
Onset	Acute	Insidious
Occurrence	Epidemic	Endemic
Fever	Common	Only in complications
Clinical picture	'Lying-down dysentery'	'Walking dysentery'
tenderness	Whole abdomen	More local (sigmoid)
tenesmus	Very severe	Not usual
Stools: macroscopic	Mucus and blood only	Stools with blood and mucus
microscopic	Numerous red cells	Numerous red cells in clumps
	Numerous polymorphs	Polymorphs scanty
	Few bacteria	Many bacteria
	Macrophages	E. histolytica trophozoites with ingested red cells

5. *Management of the individual patient*

REHYDRATION IS THE FIRST PRIORITY

Treatment of the dehydration is all that is necessary in mild infections. In severe infection, the rehydration must be regarded as the first priority, because death will be largely due to dehydration (in combination with toxaemia).

Oral rehydration, can be started as soon as vomiting has stopped. Oral rehydration is always useful as an aid to parenteral rehydration and it carries less danger of disturbing electrolyte balance.

Morphia and other narcotics are not indicated, although you would like to use them for the painful cramps and tenesmus. But due to the poor circulation morphia is not well absorbed and it accumulates and may intoxicate during recovery from shock. Spasmolytics like belladonna will relieve most pain.

Antibiotics are only indicated in case of severe systemic symptoms. Antibiotics do not change the course of the disease and their routine use only results in rapid development of resistance.

Tetracycline is the antibiotic of choice because sulfonamide-resistant strains are very common.

Faeces of the patients are infective and should be handled with care.

—Rigid personal precautions by attendants.

—Protect faeces from flies, disinfect or discard in sewers.

—Concurrent disinfection, terminal cleaning.

Patients and contacts should not be employed as food handlers until danger of spread is no longer present.

6. *Control*

Prevention of bacillary dysentery depends on stopping the faeco-oral transmission.

7. *Action*

Patient —rehydrate (oral and parenteral)
—treat
—concurrent disinfection
—terminal cleaning
—exclude from food handling.

Outbreak of bacillary dysentery (or other diarrhoeal disease)
—check water supply
—can you recognize a localized pattern in the spread of the disease: families/village: is vehicle of transmission water? food? flies? check all points on the check list in the Introduction to this chapter (pp 125–7).

Direct your actions to possible cause:

—give health education on preparation of rehydration fluids; use of safe water (boiling); use of safe food (storage, fresh)

—proper refuse disposal

—proper faeces disposal.

Endemic bacillary dysentery

—give health education on use of latrines; safe water; safe food; refuse disposal

—stress the importance of prolonged breast-feeding

—give health education on personal hygiene

—stress in ward development committees the importance of improvement of water supply either with protected wells or piped water

—stress in the same committees the importance of latrines, that materials have to be made available

—inspect, with your HA, public eating houses, markets, boarding institutions (schools, camps); see check list pp 125–7.

8. *Summary*

Bacillary dysentery is an acute disease of the intestine varying from a mild diarrhoea to a severe toxic and fatal disease. It is widely spread in Tanzania and is common and severe in children, especially the malnourished. Rehydration is the main priority in treatment. Control depends fully on proper faeces disposal although improvement of water supply will greatly reduce incidence.

AMOEBIASIS

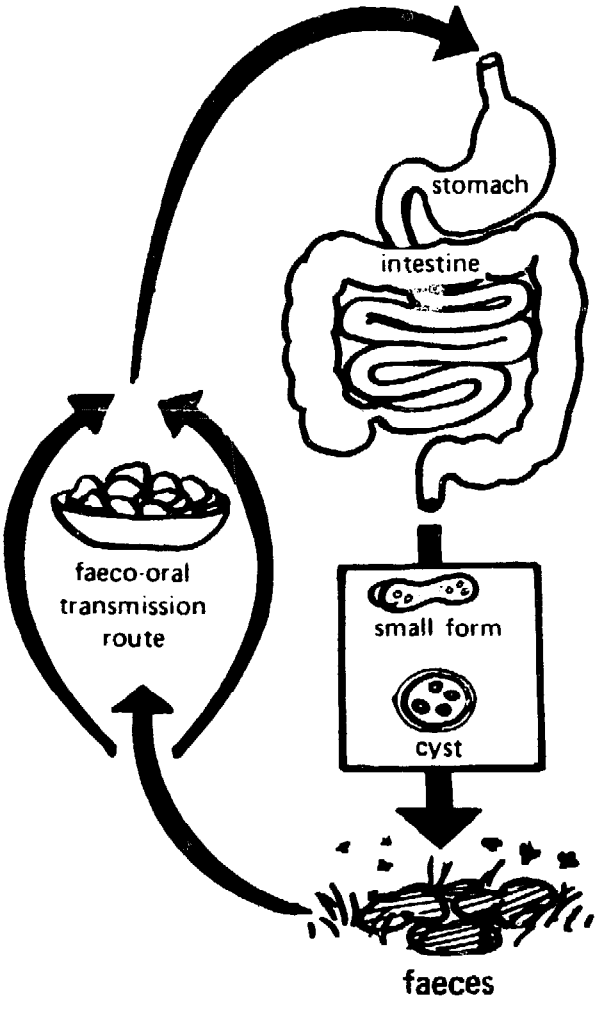
1. Amoebiasis is an infection with pathogenic amoebae. Infection with amoebae is in most cases symptomless but under certain circumstances the amoebae may change into tissue parasites and invade the bowel wall causing the disease amoebiasis. The infection can be spread to other organs, especially the liver.

2. *Occurrence and importance*

Clinical amoebiasis is endemic in areas where sanitary conditions are poor. In Tanzania liver amoebiasis is especially common in Arusha and Kilimanjaro region. The occurrence of liver abscess is strongly correlated with alcohol consumption. Formerly it was thought that amoebiasis was spread mainly through improperly prepared local brews. But males and females suffer equally from bowel amoebiasis. Liver amoebiasis is much more common in males who drink most of the brew. In females liver amoebiasis is found during pregnancy and the postnatal period. So immunity seems an important factor in the spread of the disease. Death from amoebic dysentery is rare (see table, p 121).

3. *Epidemiology*

Causative organism the protozoon Entamoeba histolytica. Normally this protozoon lives in the lumen of the large intestine as the 'minuta' (small) form. It feeds on the fluid intestinal contents of the bowel. As the contents of the bowel move on and become more solid, conditions are less favourable and the small forms encyst; these cysts are the infective form of the amoebae excreted in the faeces.



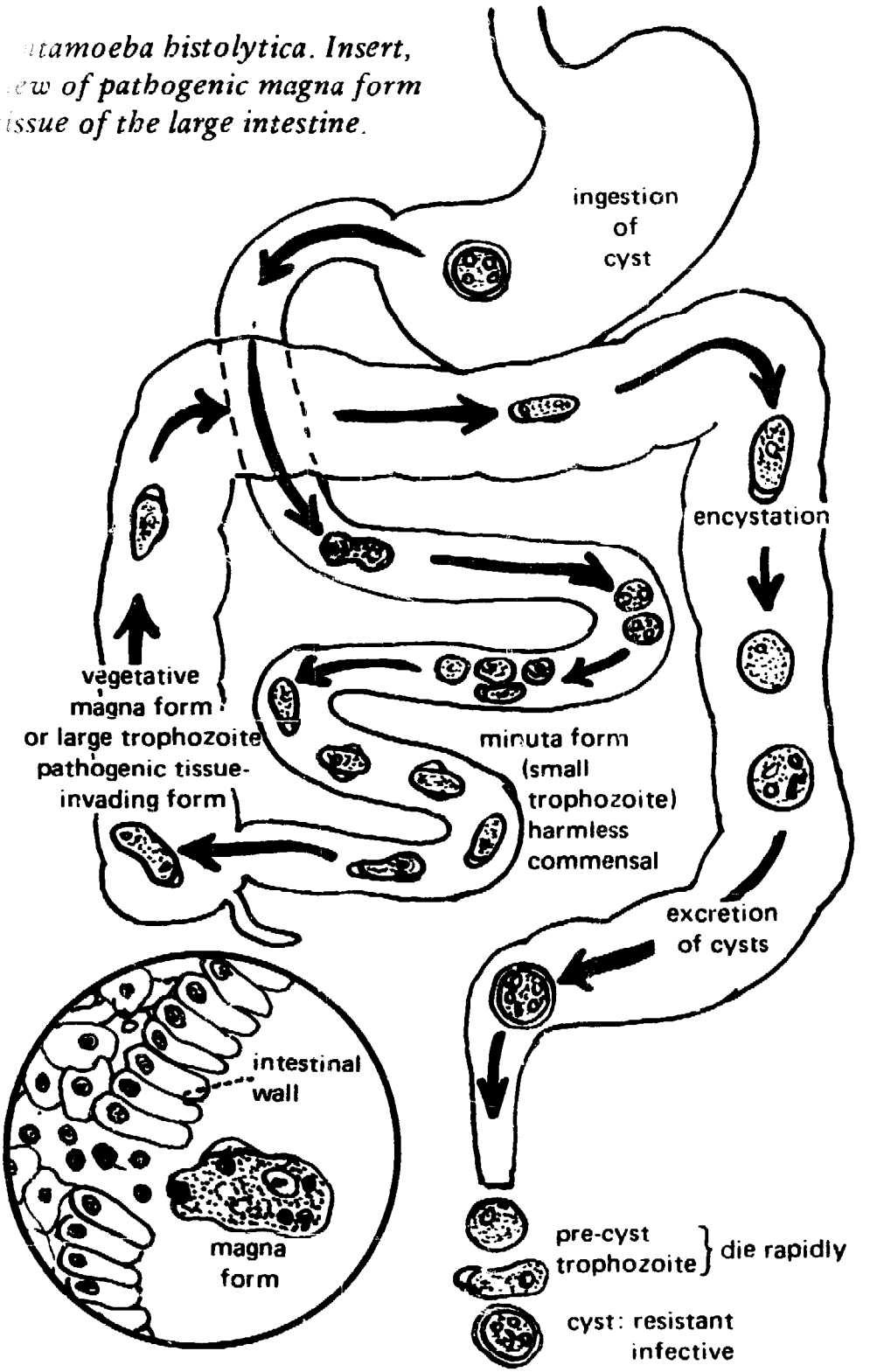
The transmission of amoebae.

In certain circumstances, which are not fully understood, the harmless small form changes into a tissue parasite. This tissue parasite, or 'magna' (large) form of the amoeba, invades the bowel wall. Here plenty of food is present

and the parasite grows in size and multiplies rapidly.

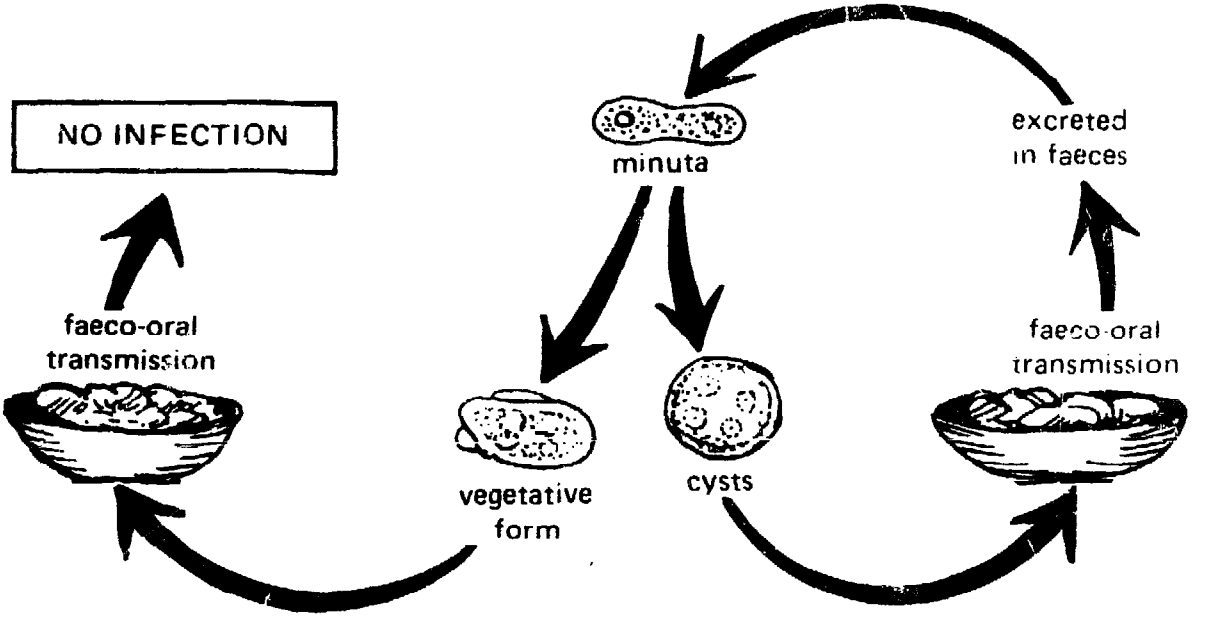
Small and large forms are both called trophozoites. The presence of ingested red blood cells in the large form is characteristic of E. histolytica.

Life cycle of Entamoeba histolytica. Insert, microscopic view of pathogenic magna form invading tissue of the large intestine.



Transmission: Cysts are excreted in the faeces of man. The cysts reach a new host by means of the faeco-oral transmission route.

As the cysts are formed from the small forms and not from the large forms the asymptomatic carriers of the small form are responsible for maintaining the infection in the community. Large forms are excreted in large numbers (by patients) during an attack of amoebic dysentery. These cannot infect another individual because of their fragility; even if they reach the mouth of another person they cannot withstand gastric acid and they are therefore a dead end of the cycle.



Infection with small amoebic forms (as shown by finding cysts in the stools) is very common in certain areas, but only relatively rarely do the amoebae invade the tissues and produce dysentery. The dysentery patient is not responsible for the spread of amoebiasis. Therefore amoebic dysentery cannot occur in epidemics like bacillary dysentery, but is endemic in a population in which many individuals are asymptomatic carriers and only a few get the disease. The reservoir of infection is formed by asymptomatic carriers and chronic patients excreting the infective cysts.

4. *Clinical picture*

INFECTION IS USUALLY ASYMPTOMATIC

When amoebae penetrate the intestinal wall they multiply in the submucosa, causing bottle-shaped ulcers. From these ulcers the amoebae may be transported to the liver. An amoeboma, or amoebic granuloma, may result from repeated invasion in the colon. An amoeboma may become very large, forming a hard swelling which is very difficult to differentiate clinically from carcinoma (try treatment for amoebae first in case of suspected colon cancer). Usually the onset of amoebic dysentery is insidious with abdominal discomfort. There may be a mild looseness of the bowels or frank diarrhoea with or without blood and mucus. Tenderness may develop over the caecum or sigmoid.

In severe cases the onset is more sudden, the patient is ill and toxic with fever and signs of dehydration. Hiccup is a bad sign. The faeces contain much dark and altered blood and blood-streaked mucus. Trophozoites are present in large numbers.

Chronic amoebiasis is difficult to diagnose. Alternating diarrhoea and constipation are often seen. The colon may be distended. Chronic amoebiasis may resemble duodenal ulcer, gall bladder disease, or carcinoma of the colon.

Amoebic liver abscess: This condition should be suspected in people who have lived in endemic areas.

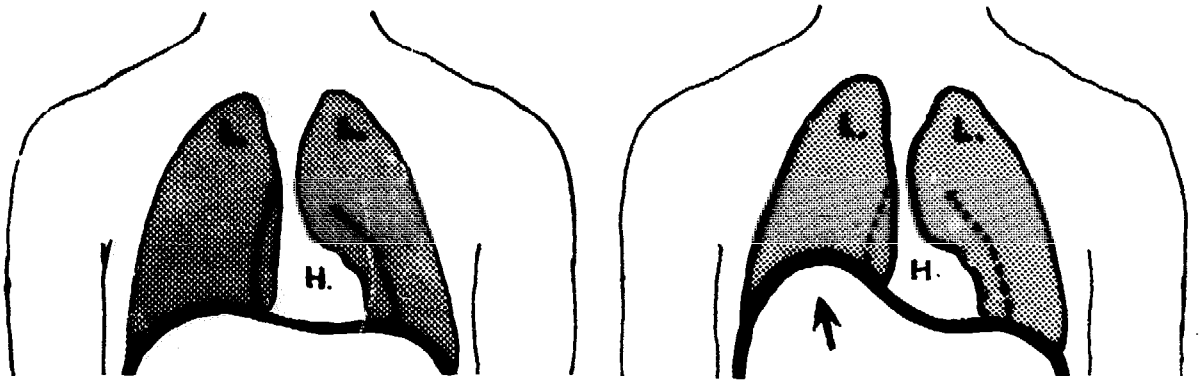
The most common findings are:

high ESR and leucocytosis; ESR ↑

enlarged and tender liver; WBC ↑

elevation and fixation of the right diaphragm (Xray). Fever is often mentioned as a frequent symptom, but is only present in bacterial superinfection, generally after aspiration.

An amoebic liver abscess may perforate into the chest cavity —see below; into the peritoneal cavity—acute abdomen; or



NORMAL ELEVATION

through the skin—cutaneous amoebiasis—see below.

Aspiration of a liver abscess should only be carried out where facilities for laparotomy are present, so not in a health centre.

REFER FOR ASPIRATION

Other extra-intestinal manifestations

—Skin: amoebiasis of the skin may occur where amoebae come into contact with the skin, that is:

(a) around the anus and perineum

(b) around incision wounds, e.g. after appendicectomy or drainage of a liver abscess

(c) around a fistula from a liver abscess which has perforated; this opening may be far away from the liver.

An amoebic skin ulcer is irregular and painful. The ulcer enlarges continuously because of necrosis of the edges.

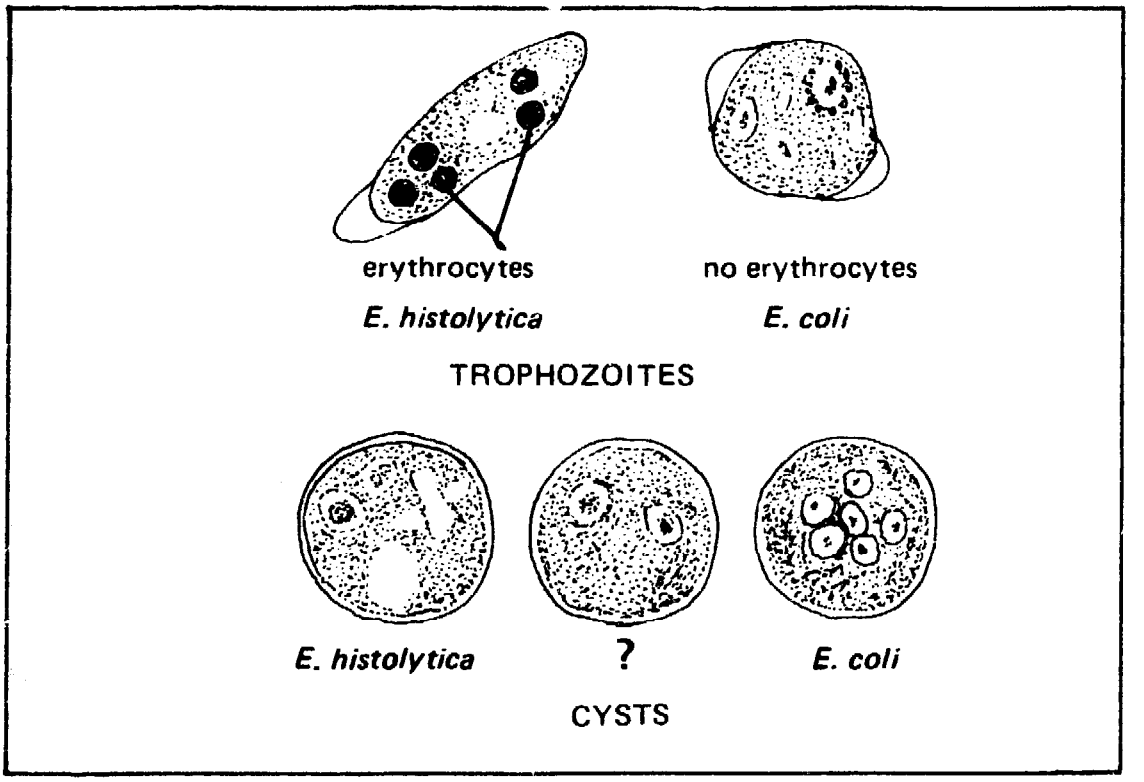
—Lungs: when a liver abscess breaks through into the chest cavity, a lung abscess (which may perforate into a bronchus) or empyema may occur. When a liver abscess is close to the diaphragm, the diaphragm will not move and the right lower lobe will not be well ventilated. As a result liver abscess is often accompanied or masked by bronchopneumonia in the right lower lobe. Pleurisy, dry or with effusion, may occur if the pleura covering the diaphragm is inflamed.

—Brain: rarely an amoebic brain abscess may complicate severe amoebiasis.

Diagnosis:

The presence of cysts in stools does not prove that symptoms are caused by entamoebae. Only large forms are proof of disease. Trophozoites of histolytica should be differentiated from those of Entamoeba coli. The main difference is the presence of ingested erythrocytes in the histolytica.

Cysts can be differentiated when a stool specimen is stained with Lugol's solution. Only then the nuclei will be seen. Coli cysts may have 2–8 nuclei, histolytica cysts have 1–4 nuclei. Differentiation is possible when cysts are seen with only one nucleus or with more than 4 nuclei.



Differentiation of E. histolytica and E. coli.

5 *Management of individual patients*

Amoebicidal drugs may act on either the large (tissue) form or on the small (bowel lumen) form. When a patient is treated both forms must be dealt with.

Schedules for treatment: (for dosages see table opposite)

Amoebic dysentery: First choice: metronidazole only.
Second choice: emetine + tetracycline followed by hydroxyquinolines.

Liver abscess: First choice: metronidazole + chloroquine.
Second choice: emetine + chloroquine followed by hydroxyquinolines.

Asymptomatic carriers: Do not treat in highly endemic areas except for food handlers who should be treated with hydroxyquinolines.

6. *Control*

The cyst-passers are responsible for the spread of amoebic dysentery in the community. They are usually asymptomatic and will not report to a health institution. People employed as food handlers, i.e. cooks, etc., should be screened before and during employment. Water cannot be made safe by ordinary chlorination. Superchlorination will kill the cysts. Boiling is safe.

As with bacillary dysentery the most important control method is proper faeces disposal.

7. *Action*

Search for carriers among food handlers.

8. *Summary*

Amoebiasis is an infection with potentially pathogenic amoebae. Infection occurs through the faeco-oral transmission route. Infection is usually symptomless but may result in attacks of dysentery and/or liver abscess. Treatment should cover both tissue parasites and parasites in the bowel lumen. Control depends on proper disposal of faeces. Asymptomatic carriers are difficult to trace.

Drugs used in the treatment of amoebiasis

Drug name & trade name	Dose and route	Dur-ation	Place of action	Particulars	Price of complete course in T Shs
Emetine hydrochloride	1 mg/kg im daily	5 days	Tissue	Not in ambu-lant patients; not in preg-nancy; not in patients with heart or kidney disease. Not more than 60 mg daily	8/-
Metronidazole Flagyl	800 mg tds (4 tabs) oral	5 days	Tissue and lumen	No alcohol	9/-
Tetracycline	250 mg qid oral	5 days	Lumen only	Indirect action through bacteria	14/-
Chloroquine	600 mg + 300 mg oral	2 days + 19 days	Liver only		1/15
Hydroxyquino-lines				Not longer than two weeks	2/50
Diodoquin (650 mg)	oral 1 tab tds	10 days	Lumen	caution: optic	
Embequin (300 mg)	2 tab tds	10 days	only	nerve damage	

CHOLERA

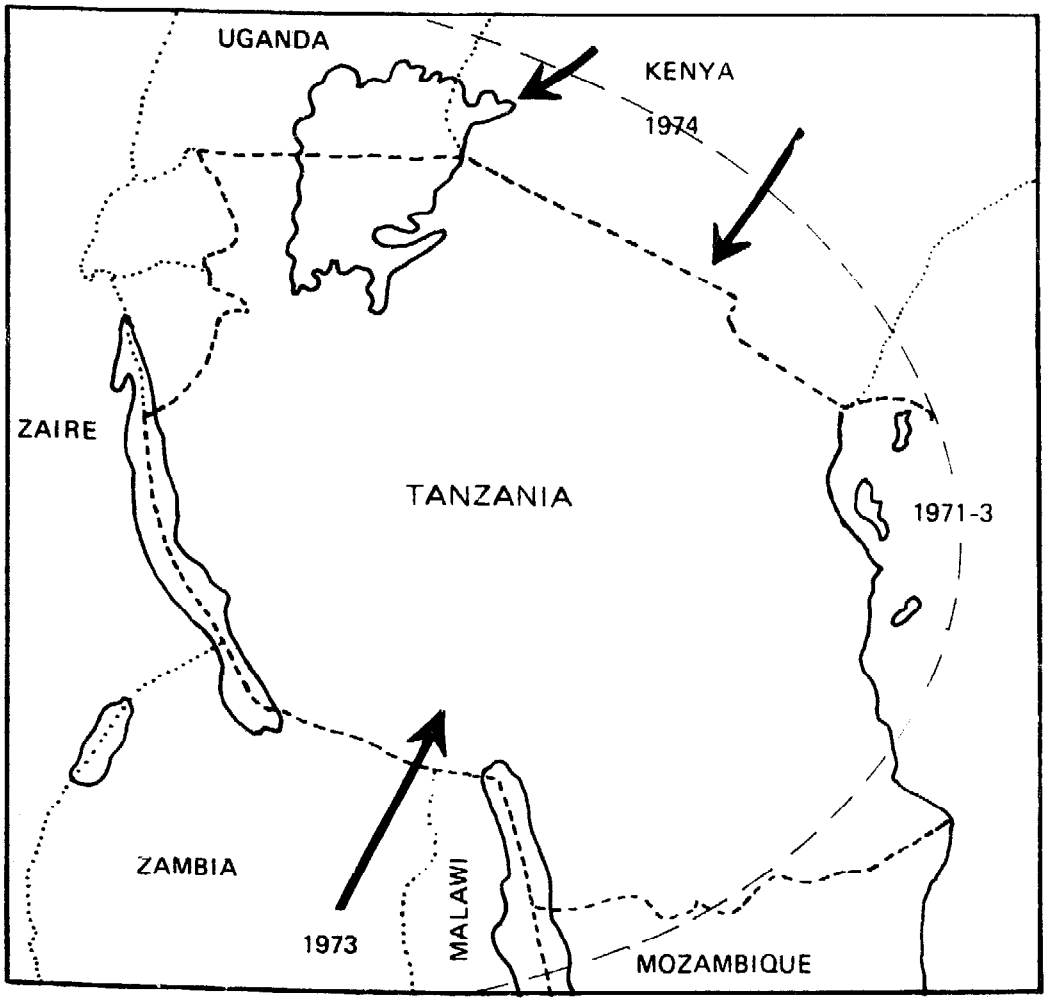
1. Cholera is an acute intestinal disease characterized by sudden onset, profuse watery stools, vomiting, rapid dehydration and circulatory collapse.

   Synonym: *Kipindupindu.*

2. *Occurrence and importance*

The current pandemic of cholera originates from Celebes (1961). In four years it reached 23 countries. By 1970 cholera appeared for the first time in the 20th century in Africa south of the Sahara. Cholera is spread overland, often by smugglers over uncontrolled routes.

In 1973 a few cases of cholera (El Tor) were seen in Rungwe district on the border with Malawi.



In 1974 the pandemic reached Nyanza province in Kenya. Due to the intensive communication between the lake regions in Tanzania and Kenya there is a continuous threat of importing a new epidemic.

Even in severe epidemics seldom more than 1–2% of the persons infected develop clinical signs of cholera. For every clinical case of cholera there may be 50–100 or more asymptomatic carriers.

Fatality rates of untreated classical cholera exceed 50%. But with adequate rehydration this can be below 1%.

Clinical cholera is usually seen in the lowest socio-economic groups. Other predisposing factors are: gastrectomy and treatment with broad-spectrum antibiotics (see Introduction to this chapter p 116).

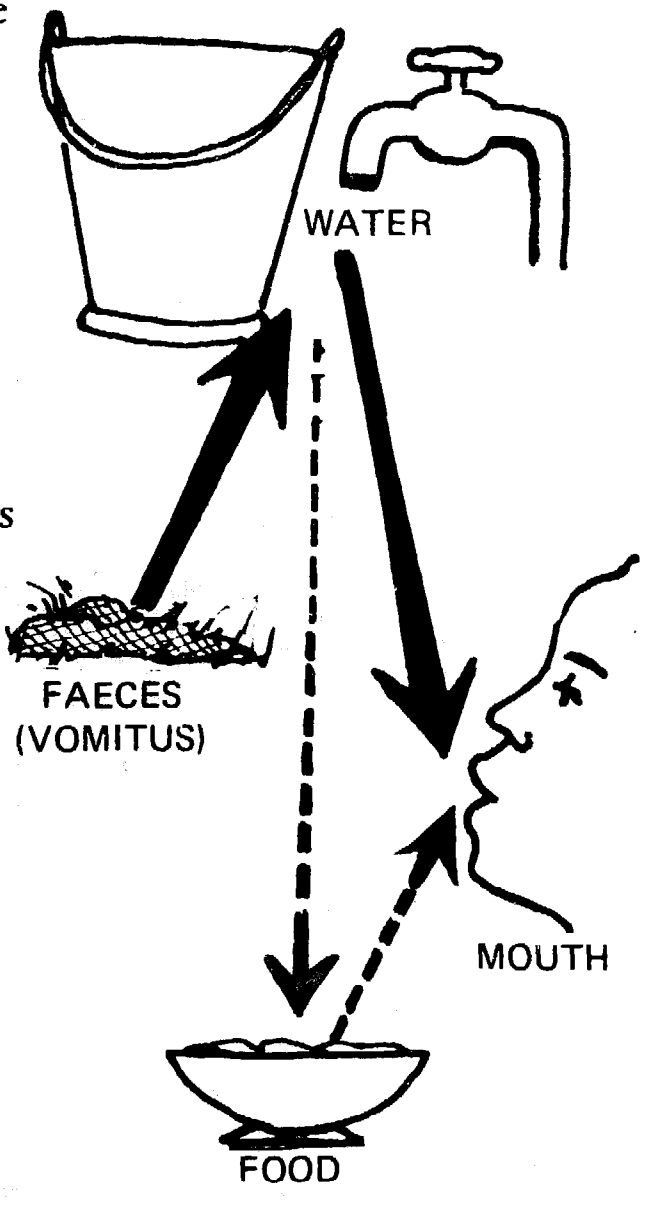
3. *Epidemiology*

Cholera is caused by Vibrio cholerae, the comma bacillus, a gram-negative, very small, curved, motile organism. The current pandemic is caused by a substrain: El Tor vibrio.

Because cholera vibrios are very sensitive to gastric acid a large number of vibrios have to be ingested in order to cause disease.

For this reason cholera is *not* a very infectious disease. Transmission is through the faeco-oral transmission route but almost all cholera infections are water-borne. Vibrios can live in water for two weeks. They prefer brackish (salty) water. In sea water they may survive for 8 months! Vibrios readily multiply in certain food such as milk and boiled rice especially when salt fish or meat is added.

The reservoir of infection is formed by the carriers. These carriers excrete vibrios in smaller numbers than the patients but because of their



Transmission is by the faeco-oral route.

freedom of movement and the fact that they far outnumber the patients they form the greatest danger to the community.

The present El Tor strain is generally clinically mild and therefore more easily spread by convalescents and carriers.

4. *Clinical picture*

The incubation time is usually 2–3 days.

MOST INFECTIONS ARE ASYMPTOMATIC OR CAUSE ONLY A SIMPLE SELF-LIMITING DIARRHOEA

Cholera is not a systemic infection. The vibrios are confined to the intestinal canal. The clinical syndrome of cholera is caused by water and electrolyte loss.

In a case of classical cholera the disease develops in three stages:

First stage: This lasts for 3–12 hours. Profuse watery stools pour from the patient. Soon faecal matter disappears from the stools, which become almost clear fluid with flakes of mucus giving them the classical rice-water appearance. Vomiting follows the diarrhoea. At first food is vomited but soon only rice-water is vomited. Severe cramps in the abdomen and limbs develop from salt loss.

Second stage: This is the stage of collapse from dehydration. The body becomes cold, the skin dry and inelastic. The blood pressure is low or unrecordable, the pulse rapid and feeble. Urine production stops and finally the patient may die of hypovolaemic shock.

Third stage: This is the stage of recovery, either spontaneously or with treatment. The diarrhoea decreases, the patient is able to take fluids, and the general condition rapidly improves.

Differential diagnosis	Cholera	Food poisoning
Diarrhoea	Precedes vomiting	Follows vomiting
Vomiting	Watery and projectile; causes no distress	Violent and distressing; vomit consists of food, is never watery
Nausea	Absent	Common
Abdominal pain	Not usually severe	Constant
Tenesmus	Absent	Common
Stools	Rice-water	Liquid, faecal, offensive, never colourless
Urination	May be completely suppressed	Never suppressed
Muscular cramps	Constant severe	In very severe cases, extremities only
Headache	Absent	Frequent

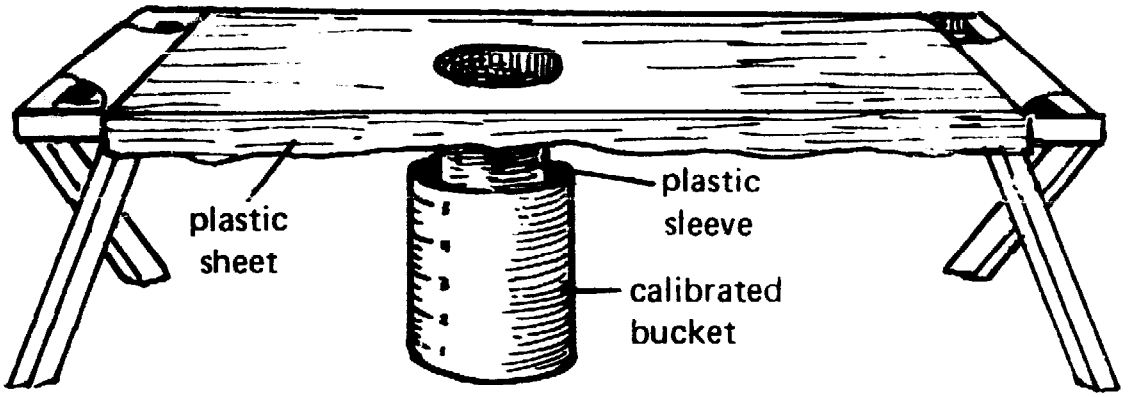
Diagnosis:
Cholera should be suspected in any outbreak of diarrhoeal disease. The diagnosis is made on clinical grounds. Do not refer patients to confirm diagnosis but send a piece of blotting paper immersed in the liquid stools of the patient for culture (inform DMO!).

DO NOT REFER A SUSPECTED CHOLERA CASE BUT INFORM YOUR DISTRICT MEDICAL OFFICER

5. *Management of individual patients*
Patients can be admitted to a temporary hospital (school, church). Strict isolation is not necessary as only the vomitus and stools are infective. These should be disinfected. Concur-

rent disinfection and terminal cleaning.

The patients are treated on 'cholera beds', beds with a central hole through which the continuous stools can pass into a bucket and be measured.



Cholera bed.

The essential cure of cholera is rehydration; this alone without any drug will save any cholera case if started in time.

Oral rehydration:

Patients of all ages who are strong enough to drink will voluntarily ingest the volume of glucose/electrolyte solution needed for rehydration and maintenance.

Patients in shock or too weak to drink require iv fluids for a few hours only and can then be given oral fluids. Vomiting is caused by acidosis and fluid loss. It may last for a few hours but the volume is usually small and able to be replaced by continuous drinking.

Oral rehydration should be given in frequent small amounts or by gastric tube (children). In oral rehydration, disturbances of electrolyte balance are less common and parenteral fluid is saved for those who really are in need of it.

A mother can give a child a teaspoon of fluid per minute and so 200–300 ml per hour.

A suitable oral rehydration fluid is:

NaCl 3.5 g
$NaHCO_3$ 2.5 g
KCl 1.5 g
Glucose 20 g
Water to 1000 ml

The absorption of glucose and sodium takes place in spite of the massive secretion of fluid by the intestine. The fluids can be administered by family members and non-professionals as well as by health workers.

Glucose has been shown to speed up the absorption of sodium and water in the small intestine. So glucose is an essential part of this oral rehydration fluid and cannot be replaced by other substances like sugar, except in emergency.

For children under 25 kg, use Ringer-lactate.

REHYDRATION WILL SAVE ALMOST ALL CASES

Tetracycline will speed up the cure and prevent the convalescent carrier stage.

It is given in a dose of 500 mg qid.

6. *Control*

Surveillance is the key to a successful cholera control programme.

Surveillance is the continuous watching of all aspects of a disease; it includes collection of morbidity and mortality reports, field investigations of epidemics or individual cases, laboratory investigations such as culturing.

Once an outbreak of a disease under surveillance is noted in a certain area immediate action must be taken in that area.

Surveillance depends on reporting suspected cases of cholera. It is obligatory to report any case immediately to the DMO by telegraph, telephone or any other rapid means.

CHOLERA IS AN INTERNATIONALLY NOTIFIABLE DISEASE

(See appendix B p 344.)

Three factors may influence reporting of new cases by the public:

(a) Reporting is greatly inhibited when repressive measures are taken once a case is identified. Repressive measures can be: quarantining a family, hospital, community or an area; imposing a military cordon or restricting movements of people or goods into and out of infected areas. Measures that limit the movements of people or restrict the shipment of goods and food are never necessary. Such measures will increase the hysteria among the people and will hinder surveillance by causing cases to be hidden or not reported.

(b) Among the first priorities in controlling cholera is saving lives. Fear and panic occur in the community when deaths occur. If cases are recognized and treatment given without delay fear will disappear and families will report their cases.

(c) Treatment centres should not be quarantined. Reluctance to report cases and fear of the disease can be overcome by allowing visits to the patients. A parent should stay with a paediatric case to assist in oral fluid therapy and nursing.

These measures are important because they stress the relatively benign nature of the disease and show it is not very infectious.

Water: As cholera is mainly a water-borne disease it cannot spread when water is made safe. This can be done by chlorination of public water supply or by boiling or treating supplies for individuals. In case of an emergency large quantities of water can be treated with bleaching powder (chloride of lime).

4 lbs of bleaching powder (= 1 lb of chlorine) in 100,000 gallons of water (or 2 kg powder in 500,000 litres) will give the required concentration of one part per million (1 PPM).

Food: Only certain foods can transmit cholera, under special circumstances and for a limited period of time. Milk products should be pasteurized. Uncooked food should be avoided or washed in safe water. Left-overs should be protected against contamination by flies.

Sanitation: Improvement of sanitation facilities will result in a lowered incidence of all diarrhoeal diseases including cholera. Emergency measures in case of epidemics are impracticable and not the first priority.

Chemoprophylaxis: Tetracycline can prevent cholera in households where there is a cholera case. But administration of tetracycline to the entire population is impracticable and will damage one of the natural defence mechanisms. Mass chemoprophylaxis can result in indiscriminate use of drugs because people may feel safer after having 10 doses of the protecting drug.

Quarantine: This can never be an efficient protection against the introduction of cholera into the community since convalescent patients and many carriers pass vibrios in their stools.

Vaccination: Cholera vaccine is of low potency. It gives little protection and this does not last long. The vaccine will give some individual protection against clinical cholera, but mass vaccination will increase the number of asymptomatic carriers. As a general measure to stop an epidemic from spreading, vaccination is of *no value*.

The public should be informed of the limited value of vaccination because in an epidemic they will demand protection and they may panic when they find that vaccine is not available. On the other hand, pressure may make the authorities start a vaccination programme, and so neglect the more important purification of the water supply. People who are vaccinated may have

a false feeling of security and may be encouraged to consume unsafe water or food.

PREVENT CHOLERA WITH CLEAN WATER SUPPLY

7. *Action*

If you suspect an outbreak of cholera in the catchment area of your health centre do the following:

—open a temporary hospital (in the school, church)
—notify your DMO and plan to inform the public
—treat the main water supply with bleaching powder or chlorine
—take stool specimens for culture
—do not refer suspected cases
—have large amounts of rehydration fluid prepared
—rehydrate as many patients as possible with oral fluids only
—give iv fluids for a short period, only to patients in shock
—have cholera beds prepared
—give health education on how cholera is spread; how water can be made safe (boiling).

8. *Summary*

Cholera is an acute intestinal disease characterized by rice-water stools, vomiting, rapid dehydration with shock. It is spread through contaminated water. Most cases are subclinical infections resulting in the carrier state. Control depends on surveillance (international). In an outbreak the first priority is to improve the water supply.

THE ENTERIC FEVERS

1. The enteric fevers include typhoid fever and paratyphoid fever. Typhoid fever is a systemic infectious disease characterized by high continuous fever, malaise, and involvement of lymphoid tissues and spleen.

Diarrhoea is *not* a common symptom in typhoid fever.

Synonym: *Homa ya matumbo.*

Paratyphoid fever may present like typhoid fever, but most cases present as gastroenteritis or transient diarrhoea.

2. *Occurrence and importance*

The enteric fevers are endemic in many regions of Tanzania. Epidemic outbreaks can occur when typhoid is introduced in a typhoid-free community.

Fatality rate can be 10% but with adequate antibiotic treatment this should be reduced to less than 3%.

Paratyphoid is not often diagnosed in Tanzania.

3. *Epidemiology*

The enteric fevers are caused by salmonellae.

Transmission is by the faeco-oral route. Salmonellae are passed out in faeces and urine. Main ways of spread are through contaminated water and food.

Contamination of food usually occurs from the hands of carriers or undiagnosed patients.

Reservoir: Man only; patients and carriers (family contacts may be transient carriers). After convalescence typhoid bacilli often settle in the gall bladder and are excreted in the faeces for a very long time.

Urinary carriers are seen particularly in areas where Schistosoma haematobium infections occur.

4. *Clinical picture*

The incubation time is 2–3 weeks.

The onset is gradual. Early symptoms and prodrome are severe headache, malaise, anorexia, body pains, and epistaxis. In the first week of the disease the temperature rises in steps. Most patients cough due to bronchitis. Usually the patient is constipated.

The pulse rate is relatively slow—that is slower than would be expected from the level of temperature. The spleen is usually enlarged and tender but this is of little help as a diagnostic aid

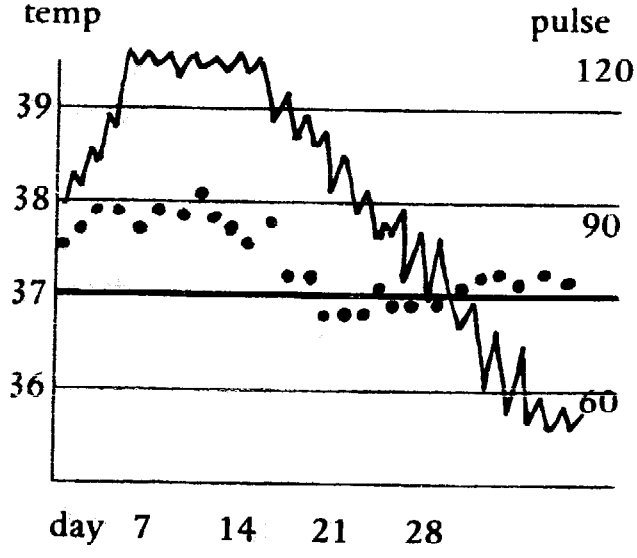
in malaria areas. During the first week of the disease the patient presents himself feeling sick and feverish. He may complain of cough and constipation.

Diagnosis:
The Widal test becomes positive on the 10th day of the disease. It must be repeated after one week. An *increase* in titre between the first and the second Widal tests is diagnostic of typhoid fever.

When someone vaccinated with typhoid vaccine gets fever, the Widal test titre rises. The same is true for someone who has had typhoid previously. Misinterpretation of the results of the Widal test often causes trouble.

Another way to prove typhoid fever is to make a blood culture. This may be positive during the first week and for a variable period after this. Stools are positive after the first week. Urine may be positive at any time.

Helpful laboratory tests are:
WBC: low (leukopenia) with a relative lymphocytosis.
Stools: in 100% of the cases occult blood is present in the stools.



Temperature and pulse rate in typhoid fever.

In the second week the temperature is continuously high. Half of the patients may develop diarrhoea. Typhoid patients may become confused and disorientated with hallucinations. The abdomen becomes distended and tender due to ulcers in the lymphatic tissues of the intestine.

These ulcers may cause bleeding and perforation in third week of the disease.

	Prodrome	First week	Second week	Third week
General	Headache Malaise	Apathy Meningism	Mental confusion Disorientation	
Temperature	Normal	Rising stepwise	Continuously high	Decreasing stepwise
Chest		Bronchitis	Eventually broncho-pneumonia	
Abdomen		Spleen enlargement	Abdominal distension and tenderness	Complications bleeding perforation
Stools		Constipation	'Pea soup' diarrhoea	

5. *Management of the individual patient*

Chloramphenicol is the drug of choice. Do not give a loading dose as it may cause a toxic crisis with circulatory failure (Herxheimer reaction). The dose is: 500 mg six-hourly for 10–11 days (if given for less than 10 days there may be a relapse). If a toxic crisis does occur corticosteroids can be tried (hydrocortisone 100 mg qid). Chloramphenicol will not bring down the fever immediately; usually it takes at least three days before the effect is seen. Alternatives to chloramphenicol are: ampicillin and co-trimoxazole (Septrin, Bactrim).

Patients should be isolated in a fly-proof room. Rigid personal precautions should be taken by attendants. Concurrent disinfection of faeces and urine and all contaminated articles is required before they can be disposed of directly into sewers, if they exist. Terminal cleaning of all contaminated articles must also be undertaken.

Perforation of the bowel needs surgical treatment. Patients

with signs of peritonitis should be referred as they may be about to perforate. Bleeding should be treated with blood transfusions. When treatment is started early it is usually not necessary to refer typhoid patients.

6. *Control*

General: As for diarrhoeal disease (see bacillary dysentery).

Carriers: Search for carriers is very impracticable in endemic areas of Tanzania. Repeated cultures are necessary and/or sophisticated immunological techniques. Carriers who work as food handlers are especially likely to transmit enteric fever. Patients and family contacts should not be employed as food handlers until danger of transmission is over.

Immunization: A combined vaccine against typhoid and paratyphoid is available—TAB vaccine. The usefulness of the addition of paratyphoid is doubtful but plain typhoid vaccine is not available in Tanzania.

The vaccine does not give complete protection, but the number of infecting organisms necessary to cause typhoid fever is higher in vaccinated than in non-vaccinated persons. Immunization should be repeated yearly. In the control of typhoid the vaccine is of little value but as individual prophylaxis, especially for health workers in endemic areas, it is useful.

7. *Action*

See Introduction faecal-borne diseases.

8. *Summary*

The enteric fevers are systemic diseases. Typhoid fever is characterized by high continuous fever and malaise. Paratyphoid fever may resemble typhoid fever or gastroenteritis. Both diseases are spread by infected water and contaminated food, through the faeco-oral transmission route.

Chloramphenicol is the drug of choice.

Control depends mainly on sanitary disposal of faeces and improvement of water supply.

FOOD POISONING

1. Food poisoning is a term applied to an acute intestinal disease acquired by the consumption of food or water.

   The cause may be an intoxication with chemicals (heavy metals, fluoride, and others), toxins produced by bacterial growth, and a variety of organic substances that may be present in natural food.

   Acute salmonellosis and intestinal anthrax are often regarded as food poisoning although these are more acute enteric infections than intoxications.

2. *Occurrence and importance*

   Rare; in small outbreaks; mortality is very low. Figures for Tanzania are not available. At least a part of all cases notified as gastroenteritis must be due to one or other form of food poisoning.

   Food poisoning is usually recognized when all members sharing the same food fall sick within a short time.

Food poisoning occurs among people who share the same meal.

3. *Epidemiology*

Food poisoning may be a real intoxication or an infection.

Intoxication may be caused by ingestion of food contaminated with toxin-producing staphylococci from purulent discharges of an infected person. The staphylococci multiply when food is allowed to stand for several hours before serving.

Infection may be caused by ingestion of salmonellae in food contaminated by infected faeces of Man or animals, or ingestion of meat containing anthrax bacilli.

Reservoir: staphylococci: Man only
salmonellae: domestic and wild animals
anthrax: cattle and game.

4. *Clinical picture*

Incubation time: short
—staphylococcal food poisoning: 1–6 hours
—salmonellal food poisoning: 12–14 hours.

Acute onset of vomiting and diarrhoea after ingestion of made-up food; usually a number of cases occur together.

There may be moderate fever, seldom over 38° (see also p 147—cholera, differential diagnosis). Complications are rare but children with sickle cell disease may develop a salmonellal osteomyelitis after salmonellal food poisoning.

Anthrax food poisoning may cause a similar disease (see p 213).

5. *Management of the individual patient*

Symptomatic treatment; correct dehydration; antibiotics are not indicated.

6. *Control*

(a) Serve meals immediately after preparing to avoid growth of staphylococci accidently introduced.

(b) Exclude people with pyogenic (staphylococcal) skin infections from food handling. Search for them in case of an outbreak.

(c) Cook foodstuffs derived from animals (salmonellae) thoroughly. Avoid the use of raw eggs.

Health education of food handlers (and housewives) about the necessity of refrigerating foods, washing hands, and maintaining a clean kitchen.

NB The toxin produced by staphylococci is heat-stable, so cooking of already prepared food will kill the staphylococci but will not break down the toxin unless the food is heated over 140° C. Thorough cooking of food will prevent all cases of salmonellal food poisoning.

7. *Action*

—Trace all participants of the infected meal and treat them.

—Try to establish the cause: staphylococci, salmonellae, or anthrax? Watch for neurological symptoms suggesting insecticide poisoning, mushrooms; rehydrate all patients, reassure or sedate the patients who panic.

8. *Summary*

Food poisoning occurs among people who share the same meal. Causes are intoxication or infection. Rehydration is the first priority. Reassure or sedate when necessary.

POLIOMYELITIS

1. Poliomyelitis is an acute viral disease with a wide range of severity, from symptomless infection to paralytic disease.

Synonym: Infantile paralysis, *ugonjwa wa kupooza*; abbreviated: polio

2. *Occurrence and importance*

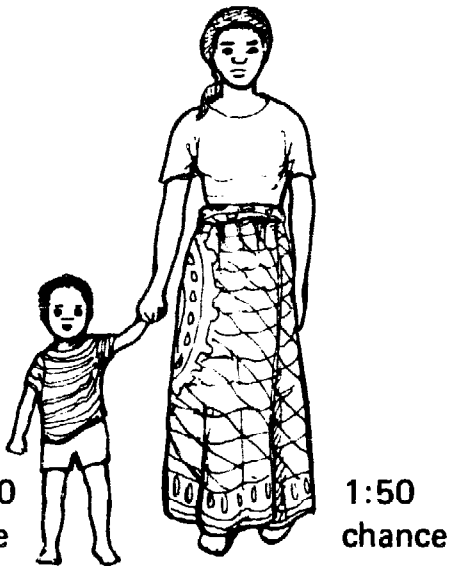
Polio infection is very common in Tanzania. It is so common that we can assume that all adults have had it, just as every adult has had measles when he was a child. Like measles, polio infection gives a life-long immunity. Infection below age 3

rarely results in paralysis, and because most people are infected at a very young age only a few develop the so-feared complication of polio paralysis with permanent disability.

In Tanzania most children are infected at a young age because of:

—poor hygiene
—large families
and overcrowding.

The possibility of paralysis increases with age. People who are infected after the age of 20 have a much greater chance of getting paralysis (one in fifty, 1:50).



Possibility of paralysis increases with age.

As standards of living rise, polio infection will occur at a later age and an increased number of paralytic cases can be expected. Therefore the children most at risk are not the children in rural areas but the unvaccinated children of the well-educated, the doctor, the teacher, the businessman, and the manager.

Paralysis is caused by the polio virus invading the CNS. This invasion is easier when nerve endings are damaged, e.g., by intramuscular injections, muscular exhaustion (sports, long walks), operations like tonsillectomy.

3. *Epidemiology*

There are three types of polio virus. The virus is detectable both in faeces and pharyngeal secretions but it is still not sure whether the faeco-oral route or the oral-oral route is most important.

Reservoir of infection: Persons with symptomless infections, mostly children.

4. *Clinical picture*

Infection with polio virus usually results in a mild general reaction; this is known as the 'minor illness'. There is fever, head-

ache, malaise, sore throat, and gastrointestinal disturbance lasting one or two days. Minor illness cannot be differentiated from other mild viral infections and is only recognizable during an epidemic of polio.

After the minor illness, the disease sometimes progresses into the 'major illness' with a recurrence of fever. The major illness can be divided into three stages.

Preparalytic stage: The temperature rises rapidly again to 39° –40° with headache and signs of meningeal irritation. The general symptoms return: malaise, vomiting, anorexia, diarrhoea. After one or two days the symptoms disappear. The patient may have recovered or may go on to the next, the paralytic stage. Physical activity at the time of temporary improvement may aggravate the degree of subsequent paralysis.

Paralytic stage: As the temperature goes down the paralysis appears. Paralysis can appear at any site of the body and is asymmetrical. The lower limbs are more often affected than the upper. Spread of paralysis is usually complete in 24 hours. The paralysed muscles are painful.

Post-paralysis stage: (Or stage of residual disability.) Paralysis or weakness of muscles will lead to deformity and contractures. A severely affected limb will show effects of abnormalities in blood circulation such as coldness and cyanosis. There may be retardation of bone growth, resulting in shortening of the affected limb.

5. *Management of the individual patient*

No specific drug is available as poliomyelitis is a viral infection.

Absolute bed rest is necessary in the preparalytic stage until it is sure that a patient will not develop paralysis. In the paralytic stage mobility by passive movements of the affected limb must be maintained to prevent contractures. In the post-paralysis stage of the disease active physiotherapy should be started. Physiotherapists can help in assessment of disability and deformity, with active exercises and passive stretching of contractures, and by measuring for braces (calipers) and gait training.

Patients may be isolated but this will not stop the spread of an epidemic as for every paralytic case there are about 1000 infected non-paralytic cases in the community. It is better to vaccinate all contacts.

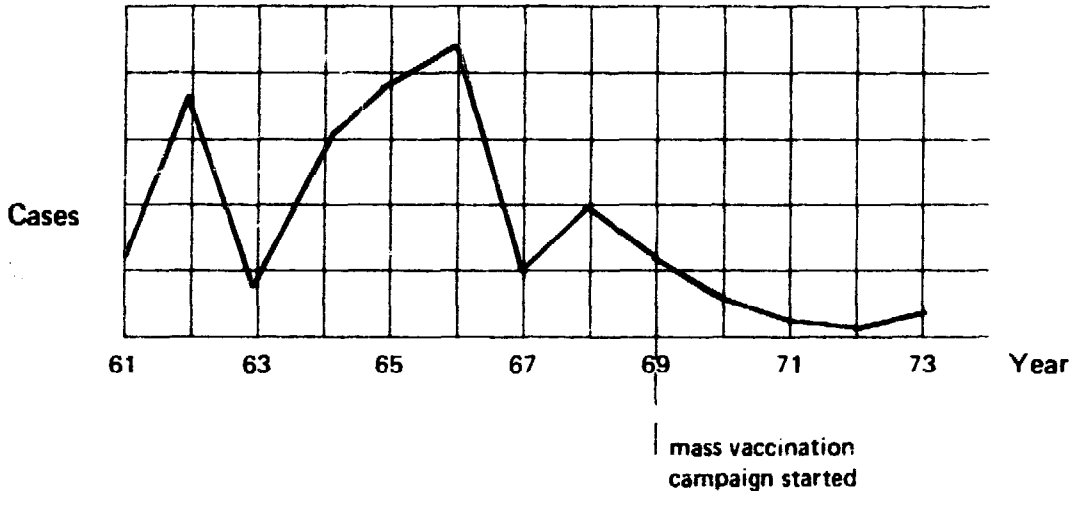
6. *Control*

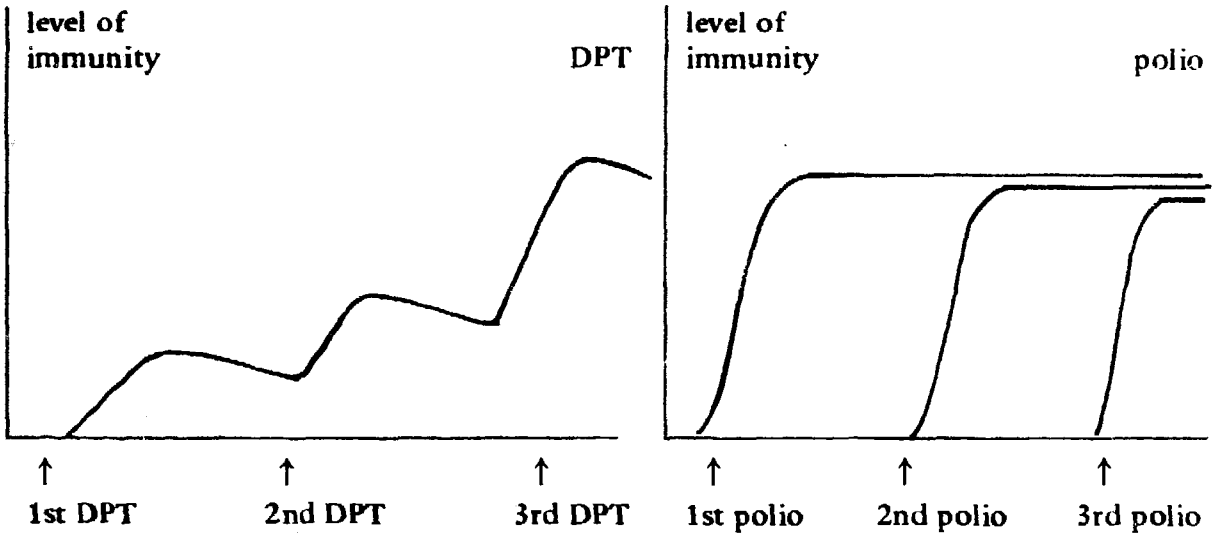
Immunization: The vaccine available is Sabin, a live attenuated vaccine given orally. The disadvantage of the Sabin vaccine is that when another enterovirus is present in the bowel it can compete with vaccine. For this reason it should not be given when there is diarrhoea.

Polio vaccine contains three types of virus. When it is given, usually one strain becomes dominant and the antibody response will be mainly against that type.

Therefore the vaccine has to be given three times, at intervals of at least a month.

Incidence of poliomyelitis in Tanzania 1961-73





Note: The second and/or third injections of DPT are needed to boost the response to the vaccine as a whole. The second and/or third polio feedings are to make sure all three strains get a good chance to 'take', as probably only one will 'take' at each feeding.

Polio vaccine is given routinely to all under-fives, starting the vaccination at the first visit to the clinic. It also can be given to schoolchildren, even to adults in case of an epidemic.

Since 1969 the incidence of polio has fallen in Tanzania. In that year a mass vaccination campaign was started. There is a danger, however, if not all children are routinely immunized against polio; a nation-wide epidemic can break out again. This will not happen if 80% of all individuals have immunity (high herd immunity). This can be achieved by continuous immunization of the under-fives, and schoolchildren.

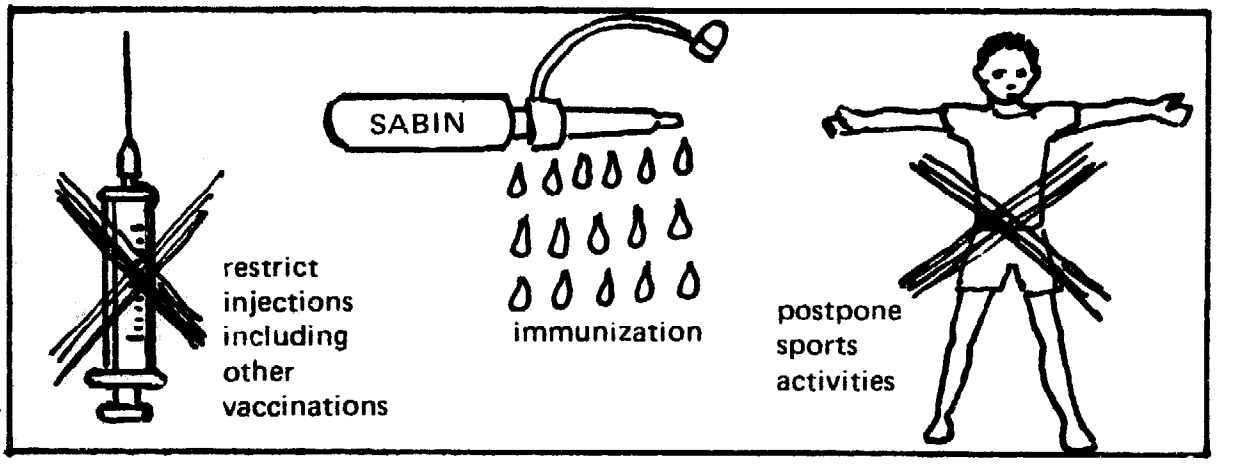
Notification: Poliomyelitis is a notifiable disease. Inform your DMO when you suspect an outbreak or when you are confronted with a new paralytic case (see Appendix B p 344).

Other measures: In case of an outbreak of poliomyelitis:

—Prescriptions for injections must be minimized. DPT vaccinations should be postponed.

—Individuals should not tire themselves; should avoid games, swimming, and heavy work, especially if they feel a little unwell ('minor illness').

—Mass vaccination with the oral vaccine should be started immediately.



7. *Action*

General—check your MCH clinic. Are polio vaccinations carried out routinely? Is the paraffin fridge working as it should?

Outbreak of polio:

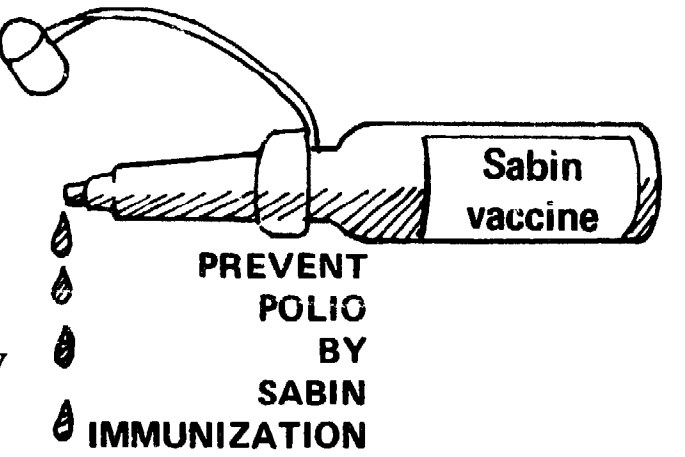
—inform your DMO

—start mass vaccination of all under-fives

—postpone other vaccinations (injections)

—restrict use of all injections

—advise people, especially teachers, to postpone sports activities and other exercises



—refer paralytic patients for physiotherapy as soon as the acute stage (pain and fever) is over.

8. *Summary*

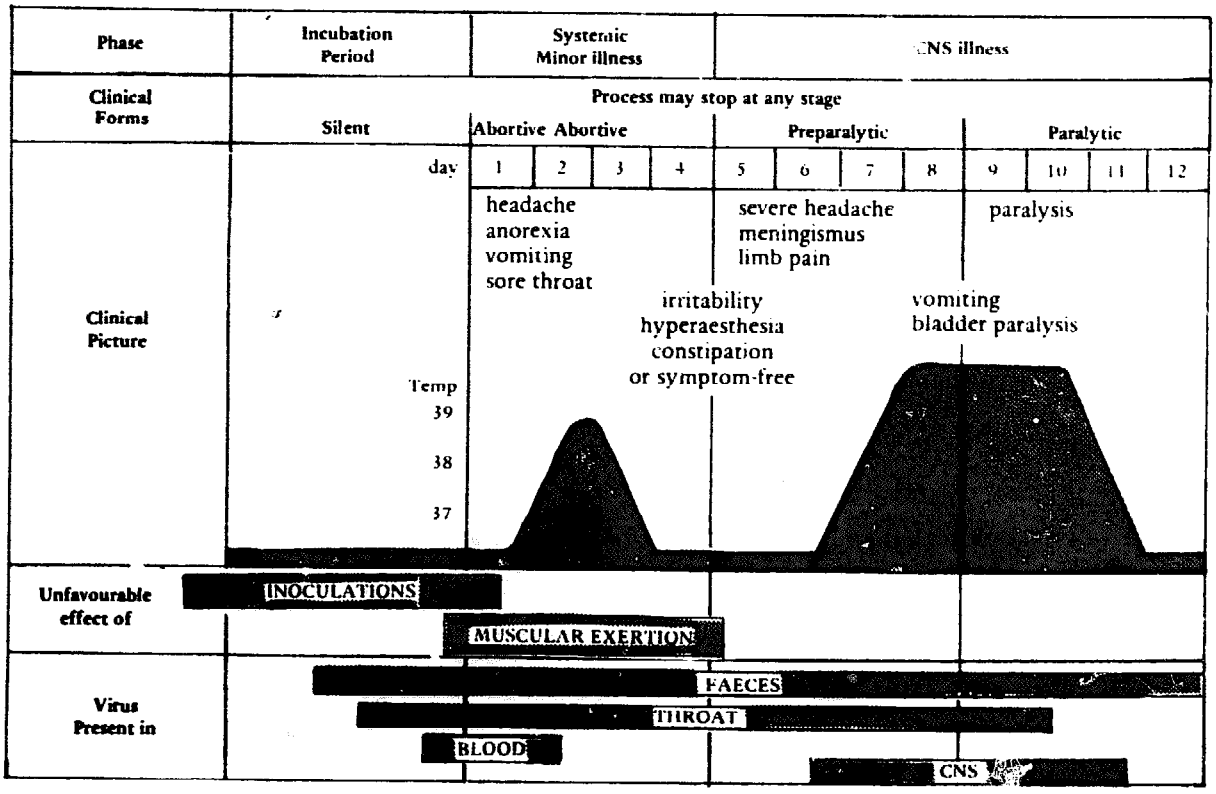
Poliomyelitis is an acute viral disease which can be complicated by CNS involvement.

Transmission is by close contact. Up to now most children

have been infected at a young age resulting in relatively few cases of paralysis.

Paralytic cases need physiotherapy. Polio can be prevented by vaccination.

CONSEQUENCES OF EXPOSURE



VIRAL HEPATITIS

1. Viral hepatitis is characterized by abdominal discomfort, malaise, usually followed by jaundice. Viral hepatitis is split into two entities:

   Infectious hepatitis. Synonym: Hepatitis A or short-incubation hepatitis

   Serum hepatitis. Synonym: Hepatitis B or long-incubation hepatitis

2. *Occurrence and importance*

   Occurrence and importance of each of the diseases is difficult to

estimate as on clinical grounds it is usually impossible to be sure which of the two diseases is being dealt with.

In general the case fatality rate of infectious hepatitis is below 1%. For serum hepatitis it is between 6 and 12%.

Infectious hepatitis occurs in slowly spreading epidemics and more in rural areas than in cities. Epidemics are associated with low standards of hygiene. In areas of poor sanitation children are infected at a young age.

The disease in children is very mild, like a gastroenteritis, without jaundice. The resulting immunity is long-lasting. So the epidemiology is comparable with poliomyelitis. When children escape infection at an early age they are more likely to develop jaundice when they are infected later in life. Most cases of infectious hepatitis are therefore diagnosed in older children and young adults.

Serum hepatitis does not occur in epidemics and is more common in adults. It is associated with later liver cirrhosis and hepatocellular carcinoma. Up to now, no such relationship has been shown for infectious hepatitis.

3. *Epidemiology*

In the blood of patients with hepatitis B the Australia antigen can be demonstrated. The type of this structure is not yet established. It seems to be a virus particle and it is held responsible for the disease. Recently a similar particle has been demonstrated in the urine of patients suffering from hepatitis A.

Hepatitis A (infectious hepatitis)

The infectious agent is excreted in faeces and urine. Most probably it is also present in nasal and pharyngeal discharges. The main way of transmission is by faecal contamination of water. Some epidemics suggest airborne spread. The infectious agent is also circulating in the blood before the jaundice appears, so parenteral spread through infected blood and syringes is possible.

Summarizing: mode of transmission unknown, probably mainly faeco-oral, but also airborne and parenteral.

Hepatitis B (serum hepatitis)

It is transmitted by parenteral inoculation of human blood and human blood products through infected needles and syringes. The antigen has been isolated from mosquitoes so a vector-borne spread is also possible. Other evidence suggests faeco-oral, airborne, and sexual transmission also occur.

Summarizing: mode of transmission mainly parenteral inoculation with human blood, but also by other routes and possibly by vectors.

Note: It is now certain that there is at least one more hepatitis virus besides A and B.

4. *Clinical picture*

Incubation period: Hepatitis A about three weeks
Hepatitis B about three months

Hepatitis A

Pre-icteric phase, presents like a gastroenteritis with sudden onset, fever, malaise, anorexia, nausea, and abdominal discomfort. Children often do not go into the next phase.

Icteric phase: after a few days jaundice appears. The jaundice is partly of the hepato-cellular type and partly obstructive, because the swollen liver cells block the bile flow in the bile capillaries. Itching is not usually present.

Complete recovery in about two weeks is the rule but a long period of extreme tiredness with depression may occur after the jaundice has disappeared.

Hepatitis B

The onset is more insidious, there is usually no fever and prodromal symptoms such as joint pain and urticaria can occur. Complications, such as progress into a chronic hepatitis ending in liver failure or cirrhosis are more common in this type of hepatitis. The persistent presence of the antigen is also associated with the occurrence of primary liver (hepato-cellular) carcinoma.

Diagnosis:

Stools are usually pale. The urine is dark and contains bilirubin. WBC total and differential are normal. There is no albuminuria. In the blood, both direct and indirect bilirubin are raised. A blood slide should be taken to exclude other causes of jaundice such as malaria and relapsing fever.

Differential diagnosis of jaundice and fever	**Blood slide**	**WBC total**	**Albuminuria**
Viral hepatitis	Negative	Normal	No
Yellow fever	Negative	Low	Yes
Relapsing fever	Positive	Raised	Yes
Malaria	Positive	Low	No

Differential diagnosis of viral hepatitis

	Hepatitis A	**Hepatitis B**
Synonyms	infectious hepatitis short-incubation hepatitis	serum hepatitis long-incubation hepatitis
Causative agent	virus A	virus B
Epidemiology fatality age group spread	1% children, young adults epidemic	6–12% all age groups sporadic
Transmission	faeco-oral contact (also direct contact and parenteral)	parenteral (also contact; vectors?)
Clinical onset fever prodrome course	sudden frequent none usually complete recovery	insidious mild or absent yes may progress to chronic hepatitis

5. *Management of the individual patient*

No specific treatment is available, therefore there is no strict reason for admission to hospital. Bed rest at home is best and will prevent spread of the disease. If admission for one or other reason is indicated, isolate the patient. Patients should be advised to eat what they like. Fat can be restricted when there is nausea. Vitamin B is often prescribed but has no proven value. No alcohol. Corticosteroids are not of any value.

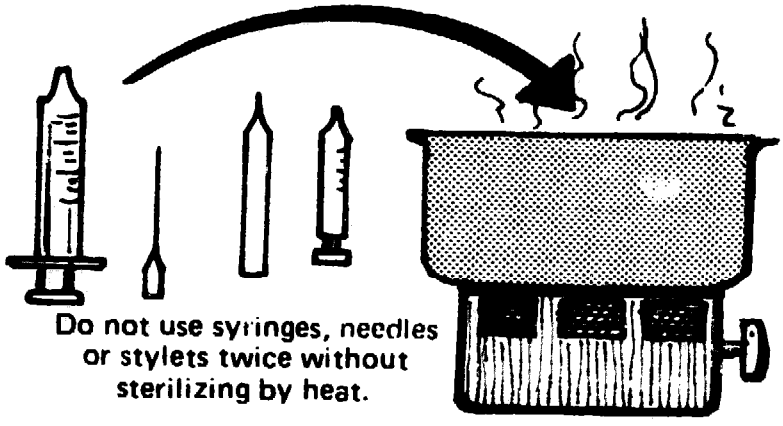
6. *Control*

As long as the mode of transmission is not fully understood, prevention is difficult. Improvement of sanitation will diminish transmission of infectious hepatitis.

Blood for transfusion should be screened in the future for the presence of hepatitis antigens. Donors with a history of jaundice should be excluded.

Thoroughly heat sterilize all syringes, needles, stylets for finger puncture.

Do not use needles or syringes twice without sterilizing. Discourage tattooing.



Control of serum hepatitis.

Do not use syringes, needles or stylets twice without sterilizing by heat. Boil for *40 minutes*, especially surgical instruments which have come into contact with blood.

7. *Action*

—Isolate patients with hepatitis.

—Check your sterilizing equipment.

—Introduce a rule that no needle, stylet or syringe is used twice without proper heat sterilizing.

—Exclude donors with a history of jaundice.

8. *Summary*

Viral hepatitis is characterized by abdominal discomfort and malaise, most often followed by jaundice. Two types are recognized with a different clinical pattern and epidemiology. Modes of transmission are not fully established. No specific treatment is available. Control is difficult since epidemiology is not fully understood.

Chapter five

HELMINTHIC DISEASES

Introduction

The worms of medical importance in Tanzania can be divided into three groups according to their form.

Group	Name	Particulars
Nematodes or Roundworms	Ascaris lumbricoides Stronglyoides stercoralis	Lung passage
	Ankylostoma duodenale Necator americanus	Hookworms
Cylindrical and elongated	Trichuris trichiura Enterobius vermicularis	
	The filarial worms	Insect vector see p 82
Cestodes or Tapeworms	Taenia saginata	Have an intermediate host (cow) rare in Tanzania
Flat and segmented	Echinococcus	dog tapeworm (causing hydatid disease)
Trematodes or Flukes	Schistosoma mansoni	Snail intermediate host
	Schistosoma haematobium	
Leaf-like or cylindrical		see p 107

Filarial disease and schistosomiasis are dealt with in the chapter on vector-borne diseases. The final host of all worms in the table is Man except for the dog tapeworm for which Man is an accidental intermediate host. The eggs of all the intestinal worms are excreted in the stools. Sanitary disposal of faeces is the preventive measure of choice because it will control all these worm diseases (except hydatid disease).

INTESTINAL WORMS ARE A PROBLEM OF POOR SANITATION

Proper disposal of faeces is one of the most difficult preventive measures to achieve, because the co-operation of every member of the community including children is necessary. When even one person does not dispose of his stools properly, transmission may continue. Building of latrines is of no use if no health education is given in an attempt to change the attitudes and behaviour of the people. Because of the lack of proper latrines and the attitudes of people it will take a long time before worm diseases are controlled in Tanzania; it *is* possible to control them, however.

Incidence of intestinal worms as reported by Tanzania hospitals (1973)

Ascaris	128,000
Hookworm	177,000
Tapeworm	20,000

Enterobius, Trichuris, and Strongyloides are not reported.

These figures are derived from returns of hospital in- and outpatients. As most worm infections are symptomless or treated at HC and dispensary level these figures cannot be regarded as a real reflection of the incidence of worm diseases.

ASCARIASIS

1. A chronic nematode infection of the small intestine. Symptoms are usually vague or absent.

   Synonym: *Michango*

2. *Occurrence and importance*

   Ascariasis is especially common in hot, humid areas of Tanzania (coastal belt). Children are more frequently and more heavily infected than adults because of their habit of putting all kinds of things in their mouths. Adults may develop some immunity.

3. *Epidemiology*

   Ascariasis is caused by Ascaris lumbricoides, a large intestinal roundworm. The roundworm lives in the small intestine. A female may produce up to 200,000 eggs daily. The eggs are passed out in the faeces. They have to be embryonated in soil before they are infective.

   This takes 8–50 days. The soil must be loose and not too dry. Oxygen must be available and the temperature over 15° C. But eggs can survive adverse circumstances for a long time, and embryonated eggs can be carried away from the defaecating place into houses by feet, footwear or in dust by wind. They can also pass through the gastrointestinal tract of animals and remain infective.

   Only when an egg is swallowed by a human being do the eggs hatch in the intestinal canal.

   The usual vehicle is salad or other food eaten raw, especially when the field has been manured with human faeces.

   To reach maturity the larvae need to pass through the lungs. The larvae penetrate the intestinal wall and reach the liver via the portal system. From the liver they are carried through the right side of the heart into the lungs. Here they penetrate into the airways and pass via the bronchioli, bronchi, and trachea to the pharynx. Then they are swallowed, return to the gastro-intestinal tract, and settle in the jejunum.

First passage through gastro-intestinal tract.

Liver, heart and lung passage.

Second passage through gastro-intestinal tract.

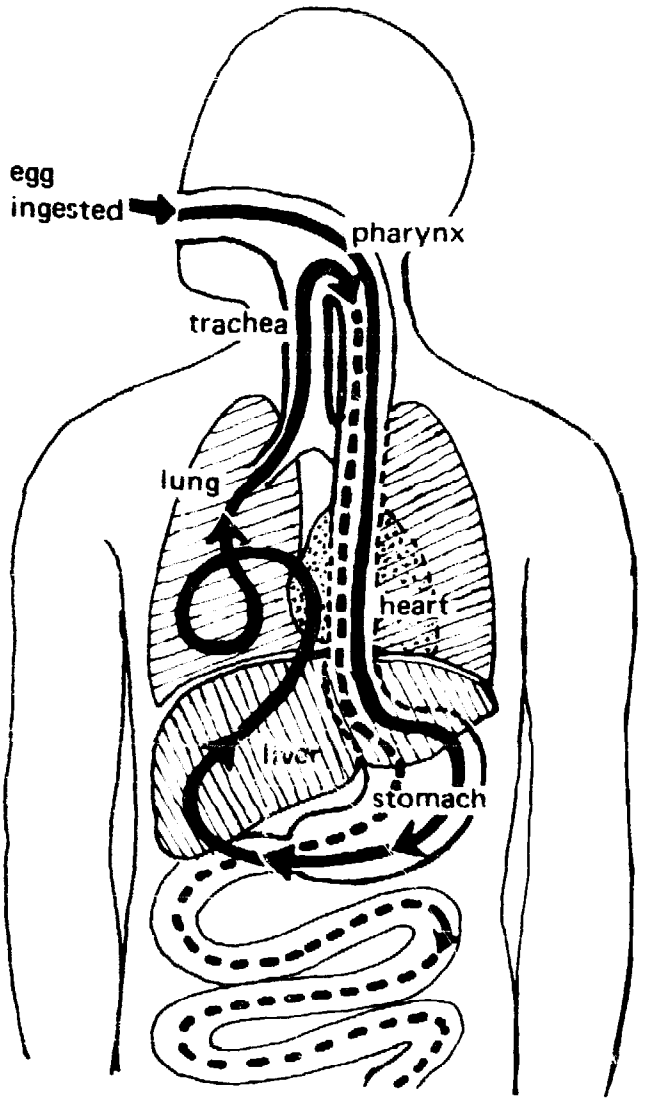


Diagram of lung passage of ascaris larva.

During the lung passage eosinophilia develops. This eosinophilia is temporary if no new infection occurs. The migration phase is associated with fever, cough, and allergic dermatitis. Migration may also cause pneumonitis or pneumonia.

4. *Clinical picture*

Except for the temporary symptoms during the lung passage, infection with ascaris is symptomless or symptoms are not characteristic.

There may be vague abdominal discomfort, restlessness and insomnia. Occasionally a worm may leave the body (in vomitus or stools) upsetting the patient and his family.

Complications may occur in very heavy infections or due to wandering worms.

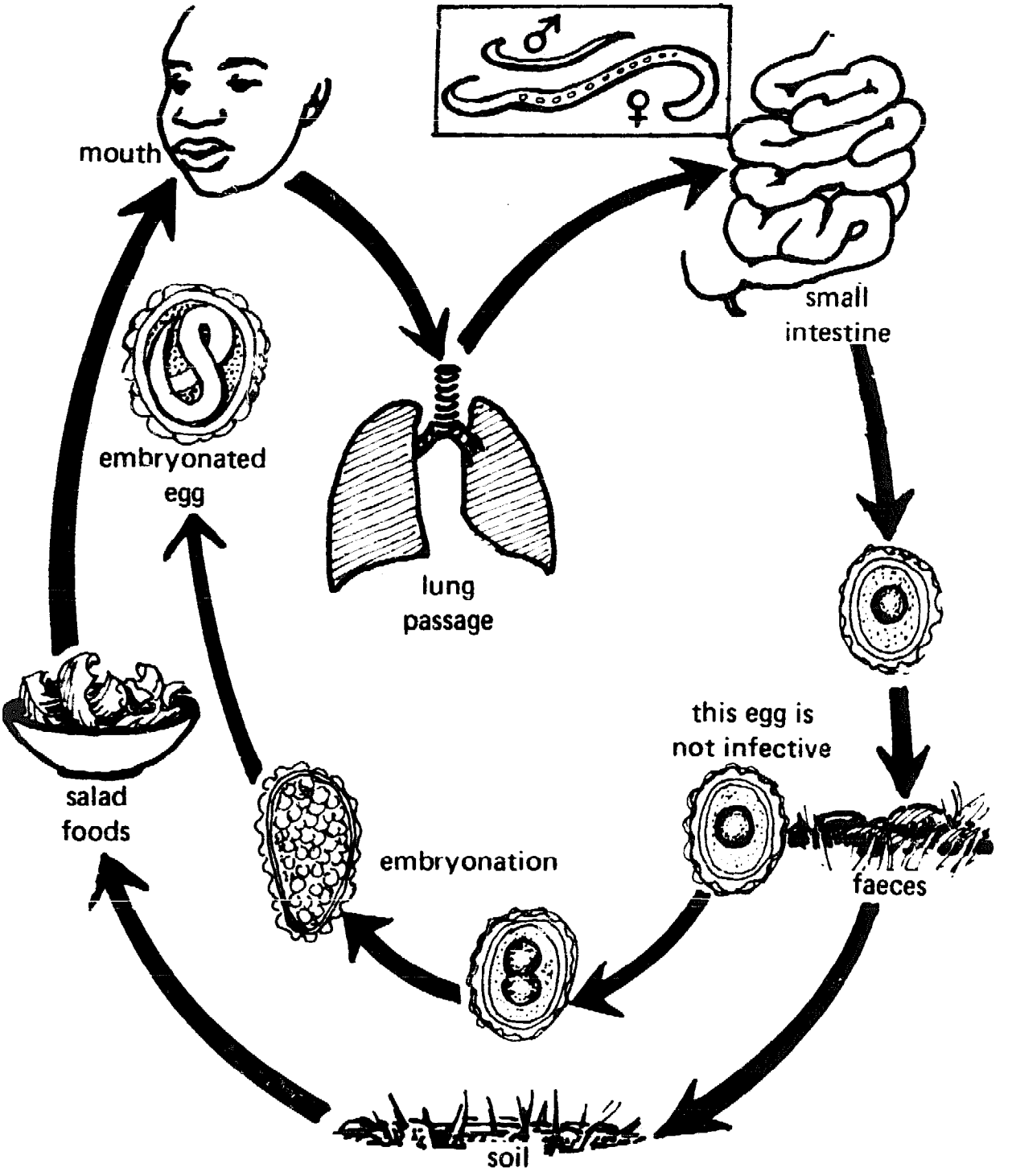


Diagram of life cycle of Ascaris lumbricoides.

Intestinal obstruction may occur at the ileo-caecal junction by a tangled ball of worms. Obstruction may be partial or complete.

Wandering of the worms is provoked by TCE (hookworm

treatment). It can result in vomiting worms, worms in the larynx causing difficulties in breathing, obstructive jaundice.

Malnutrition: Treat the underlying cause, the worm may be a minor contributory cause.

Diagnosis: By stool examination.

5. *Management of the individual patient*

Treatment (a) levamisole (Ketrax) three tablets as a single dose
or (b) piperazine (Antepar) syrup 150 mg/kg single dose.

Intestinal obstruction REFER, surgery might be necessary; pass gastric tube; no piperazine.

6. *Control*

Faeces disposal: Provision of adequate facilities for faeces disposal combined with health education on their use.

Faeces use: Discourage use of fresh human faeces for manuring. Composting for 6 months will kill ascaris eggs. After this time the compost can safely be used as fertilizer.

Health education: On use of toilets, washing hands before handling food, not to eat food which has dropped on the floor.

Treatment of infected persons: Mass treatment and screening of individuals is of no use as long as faeces are not disposed of safely.

7. *Action*

—Inspect your health centre. If there are pit latrines, encourage their use, ensure they are clean, make one person responsible for them. If there is no health centre pit latrine, build one.

—Give health education on the use of latrines.

—Inspect the latrines built at the primary school, the market; check on their existence, use and cleanliness. Report your findings in the ward development committee.

—Stress in the ward development committee the importance of latrines for individuals.

—Ask the committee to make materials available for constructing latrines.

—Build, in co-operation with the villagers, a demonstration pit latrine in a suitable place as an example of proper construction.

—Try to find out why latrines are not being used. Find out about the local taboos. Concentrate your health education on changing any unhygienic traditional behaviour.

—Discuss with the *bwana shamba* the use of human manure.

8. *Summary*
Ascariasis is a mild nematode infection. If common it shows a problem of sanitation and the need for action. Control is mainly by improving faeces disposal.

ENTEROBIASIS

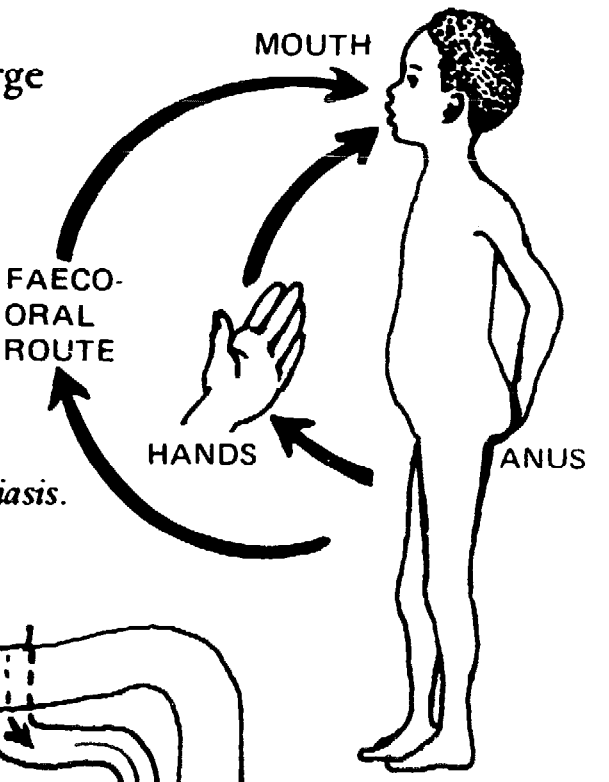
1. A benign intestinal disease with mild non-specific symptoms (pruritus ani is common).

2. *Occurrence and importance*
Most common in temperate zones, especially in school children. Usually a whole family is infected. Common in boarding institutions.

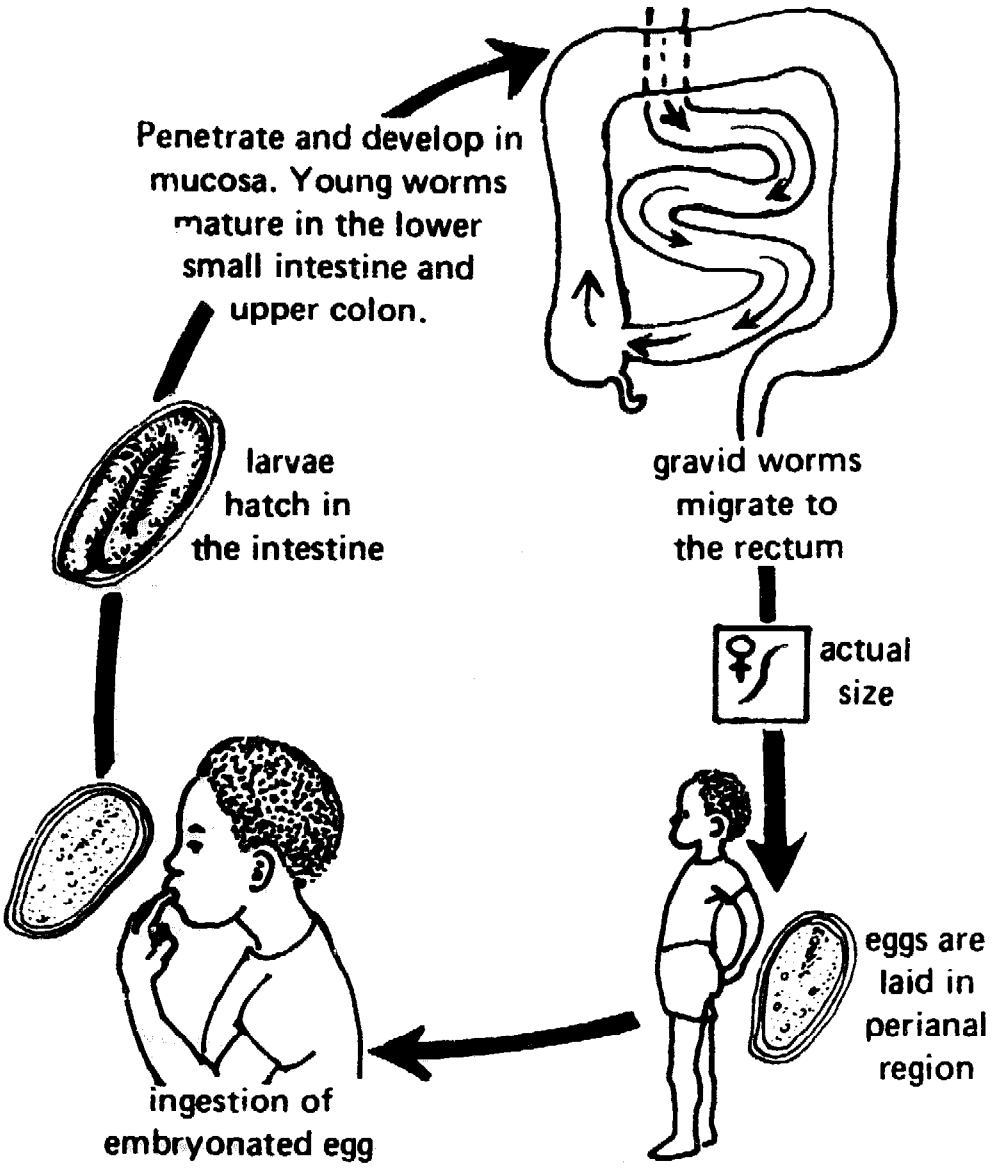
3. *Epidemiology*
Enterobiasis is caused by Enterobius vermicularis, the thread- or pinworm. Initial infection occurs by the faeco-oral route. Infection is maintained by direct transfer of infective eggs from the anus to the mouth (auto-infection) or indirect faeco-oral contact through clothing, bedding, food, or other articles. Airborne infection through inhalation of dust containing eggs and consequent swallowing is possible.

After ingestion, the eggs hatch in the stomach and small intestine. Young worms mature in the lower small intestine and

upper colon. Gravid worms migrate to the rectum to discharge eggs on the perianal skin, especially during the night. This causes itching and consequent scratching. Auto-infection is then easily established. One life cycle takes 3-6 weeks.



Transmission of enterobiasis.



Life cycle of Enterobius vermicularis.

4. *Clinical picture*

There is no lung passage, and no intensive tissue contact with the worm, therefore no eosinophilia. Main symptoms are: pruritus ani (itching), scratching effects, and disturbance of sleep.

Diagnosis: Apply transparent adhesive tape over the anus in the early morning, stick the tape onto a slide, and search for typical eggs under the microscope.

5. *Management of the patient*

Piperazine: 75 mg/kg/day for one week. Treat the whole family. During treatment strict precautions should be taken to avoid auto-reinfection (see below: personal hygiene).

6. *Control*

—Personal hygiene: bathing and handwashing, cut nails short, clean underclothes, night clothes and bedclothes.

—Correct overcrowding.

—Proper faeces disposal.

7. *Action*

—Treat whole families.

—Give health education to infected individuals to prevent reinfection.

8. *Summary*

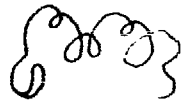
Enterobiasis is benign intestinal nematode disease. Auto-reinfection is common. Control: personal hygiene + treatment.

TRICHURIASIS

1. Trichuriasis is a nematode infection of the large intestine, usually asymptomatic.

Synonym: Whipworm.

2. *Occurrence and importance*
Common in hot, humid climates.

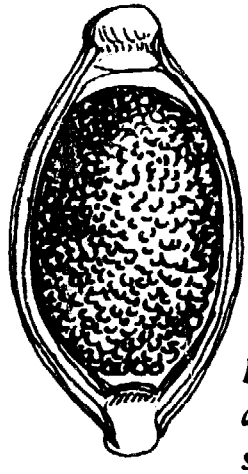


Trichuris trichiura, actual size.

3. *Epidemiology*
Trichuriasis is caused by Trichuris trichiura. Transmission is indirect, eggs passed in the faeces require embryonation in soil (as ascaris), so auto-infection is not possible. Infection occurs in a similar way to ascariasis. When embryonated eggs are ingested they hatch. The mature worms attach themselves to the mucosa of caecum and colon. There is no lung passage.

4. *Clinical picture*
Eosinophilia is present only in heavy infections, when the worms enter the mucosa of the rectum. Mild infections are symptomless, but heavy infections may result in abdominal discomfort, bloody diarrhoea, loss of weight, anaemia, and prolapse of the rectum.

Diagnosis: By stool examination. More than 200 eggs in an ordinary faecal smear indicates heavy infection.



Egg of T. trichiura as seen in faecal smear.

5. *Management of the individual patient*
Thiabendazole (Mintezol) 50 mg/kg/day for three days. Slight infections do not need treatment (no auto-infection).

6. *Control* and *Action*
As for ascariasis (sanitary disposal of faeces, personal hygiene).

7. *Summary*
Trichuriasis is a mild nematode infection transmitted indirectly

by the faeco-oral route.

HOOKWORM

1. Infection with hookworm may vary from symptomless infection to a chronic debilitating disease with a variety of symptoms. It depends on the extent of the infection, the nutritional state of the patient, and the degree of anaemia caused.

   Synonyms: Ankylostomiasis, hookworm disease, or *safura.*

2. *Occurrence and importance*

   For their development hookworms need a hot humid climate. A minimum temperature of 18° C is required, though the eggs can withstand lower temperatures (10° C). Hookworm infection is most common in the hot humid areas of Tanzania.

   Many people harbour hookworms without any ill effect (hookworm carriers). The nutrition, the daily iron intake, and the total worm load determine whether a carrier is going to have ill effects from the parasite and become a hookworm sufferer.

   Hookworm anaemia is one of the main causes of anaemia in the community. Anaemia has a profound effect on the working capacity and the feeling of well-being of the individual. The economic loss caused by anaemia is enormous but difficult to calculate (e.g. it is difficult to assess how many more acres an adult man would be able to cultivate if he were not suffering from hookworm anaemia).

   In theory a healthy adult male should have an Hb of 14.6 g/100 ml. An Hb of 13 g is acceptable. An Hb of 10 g/100 ml is a sign of disease and needs action (see table on next page).

3. *Epidemiology*

   There are two types of hookworm, both nematodes, Necator americanus and Ankylostoma duodenale.

   The eggs are already embryonated when passed out with the

WHO standards for lower limits of normal haemoglobin. (Standards at sea level, in g/100 ml. Increase for altitude: 0.4 g/300 m.)

Adult males	13
Non-pregnant females	12
Pregnant females	11
Children—at birth	16–20
—below ½ year	10
—½ y to 6 y	11
—4 y to 14 y	12

Females and children do have a slightly lower Hb than adult males. They also have fewer iron reserves and therefore suffer more easily from anaemia.

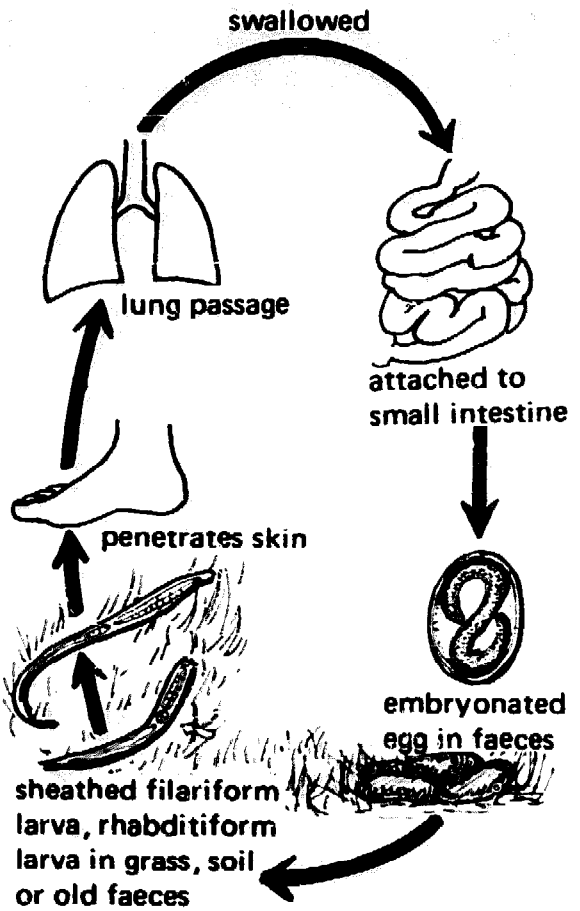
ANY Hb BELOW THESE LEVELS INDICATES DISEASE

faeces. When faeces stand for a long time before examination the free larvae can be found.

The larvae leave the faeces and bury themselves in moist damp soil. These larvae are called rhabditiform and are not infective before they have changed into the sheathed filariform stage (about 5 days). The filariform larvae may attach themselves to grass or hide in the soil. As soon as they are touched by something they attach themselves to it. When this happens to be a human leg or foot they penetrate actively through the skin, and reach the lungs via the venous system and the right side of the heart.

In the lungs they penetrate into the alveoli and are then carried up passively through bronchioli, bronchi, and trachea to the larynx-pharynx. Next they are swallowed and reach the duodenum, 3–5 days after penetrating the skin.

The worms are attached to the mucosa with hook-like teeth, present in their buccal cavity. The whole cycle is complete in about 40 days.



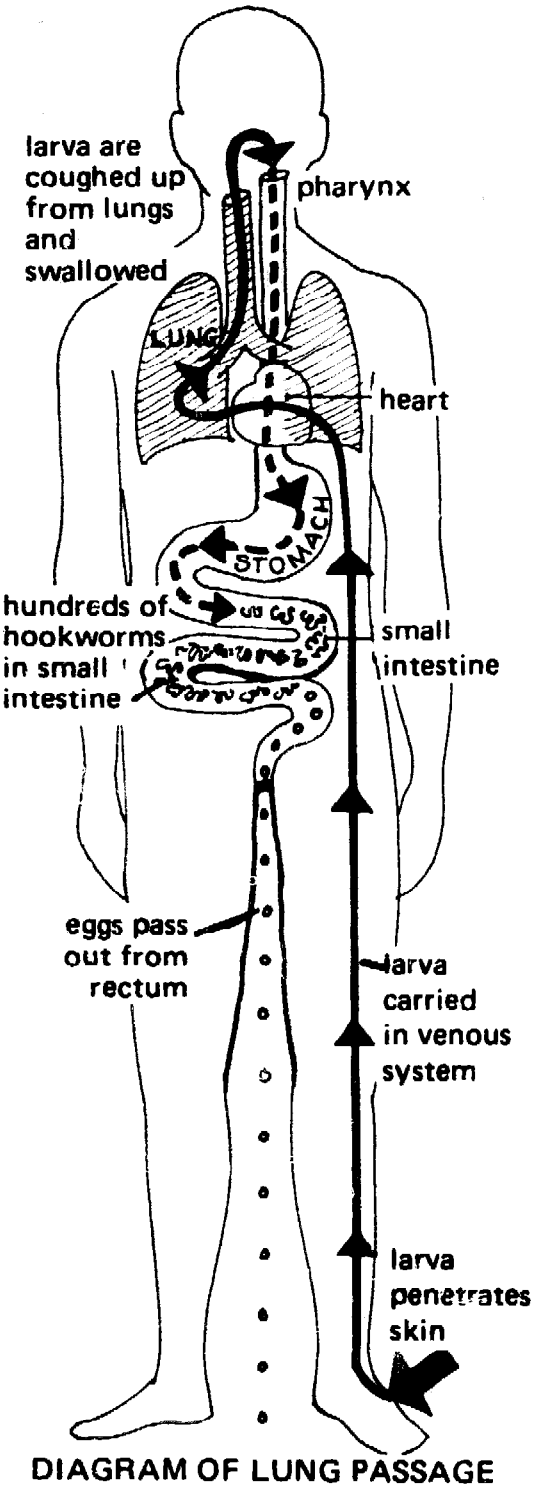


Diagram of life cycle of hookworm.

4. *Clinical picture*

The main sign of hookworm disease is *anaemia*. This is due to the sucking of blood by the worm. Most of this blood is then excreted by the worm which continues to suck and destroy

more blood.

The proteins of the blood are broken down in the bowel and the aminoacids are reabsorbed. The iron of the blood is not reabsorbed and is lost.

The anaemia is typically an iron deficiency anaemia and responds very well to iron therapy.

HOOKWORM CAUSES LOSS OF IRON

A. duodenale causes a daily blood loss of 0.2 ml. One N. americanus causes a daily blood loss of 0.03 ml. A worm load of more than 100 A. duodenale or more than 500 N. americanus always causes anaemia.

In hookworm disease firstly the body reserve of iron is used up, then gradually the Hb drops, sometimes to very low levels, with all the signs and symptoms of severe anaemia (fatigue, pallor, oedema, tachycardia, heart dilatation with murmurs, eventually CCF).

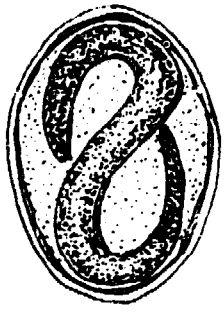
Other signs: *Ground itch* on the place where the larvae penetrate the skin some erythema, papules, and itching is seen. Most common between the toes and on the back of the feet.

Lung passage: some coughing, eosinophilia.

Gastrointestinal tract: dyspepsia, abdominal pain, distension, sometimes diarrhoea. In heavy infections, diarrhoea is mixed with blood. The complaints resemble very much the complaints of a duodenal or gastric ulcer. But these ulcers are not so frequent in Africans.

Diagnosis: Hookworm eggs in the faeces. Differentiation of necator and ankylostoma is not possible from the eggs but the adult worms can be distinguished. More than 100 eggs in an ordinary faecal smear indicates a heavy infection.

If the faeces have been standing for a long time the eggs hatch.



Embryonated egg of hookworm in faecal smear.

the larvae then seen in the faeces are easily mistaken for larvae of strongyloides.

5. *Management of the individual patient*

Not all hookworm infections need treatment. Reinfection is very likely if the community as a whole does not change its way of faeces disposal. The first priority in treatment should be treatment of the anaemia.

Iron deficiency: Anaemia is treated with iron orally (ferrous sulphate for two months). A high protein diet is necessary to replace protein loss. De-worming is indicated when the worm load is so severe that treating the anaemia only will not give results *or* when reinfection is unlikely to occur.

De-worming

(i) TCE or tetrachlorethylene, dose: 4 ml orally on an empty stomach. Advantage: cheap (T Shs 0.08). Disadvantage: toxic, cannot be given in severe anaemia and when there is a mixed infection with ascaris (risk of intestinal obstruction due to activation of the roundworms).

(ii) Levamisole (Ketrax) dose: 3 tablets single dose. Advantage: can be used in mixed infections, not toxic. Disadvantage: expensive (T Shs 0.30). Not very effective against Necator, the more common hookworm.

(iii) Bephenium (Alcopar), dose: 1 sachet, single dose. More expensive than levamisole (T Shs 1.80).

6. *Control*

Shoes: Wearing of shoes will prevent infection, but usually shoes are not worn during shamba work. It is in the shamba that the infection is acquired.

Wearing of shoes will only protect the individual who is able to buy shoes. The transmission of hookworm in the community will continue.

Faeces disposal: Proper disposal is the only way to eradicate hookworm infections (see ascariasis).

De-worming campaigns or mass treatment are only effective after the campaign for proper faeces disposal has been effectively implemented.

7. *Action*
   - –Faeces disposal: as for ascariasis.
   - –Health education on personal protection by wearing shoes.
   - –Health education on a balanced diet to prevent anaemia when iron intake is borderline.
   - –Mass treatment campaign only when everyone has and uses latrines.

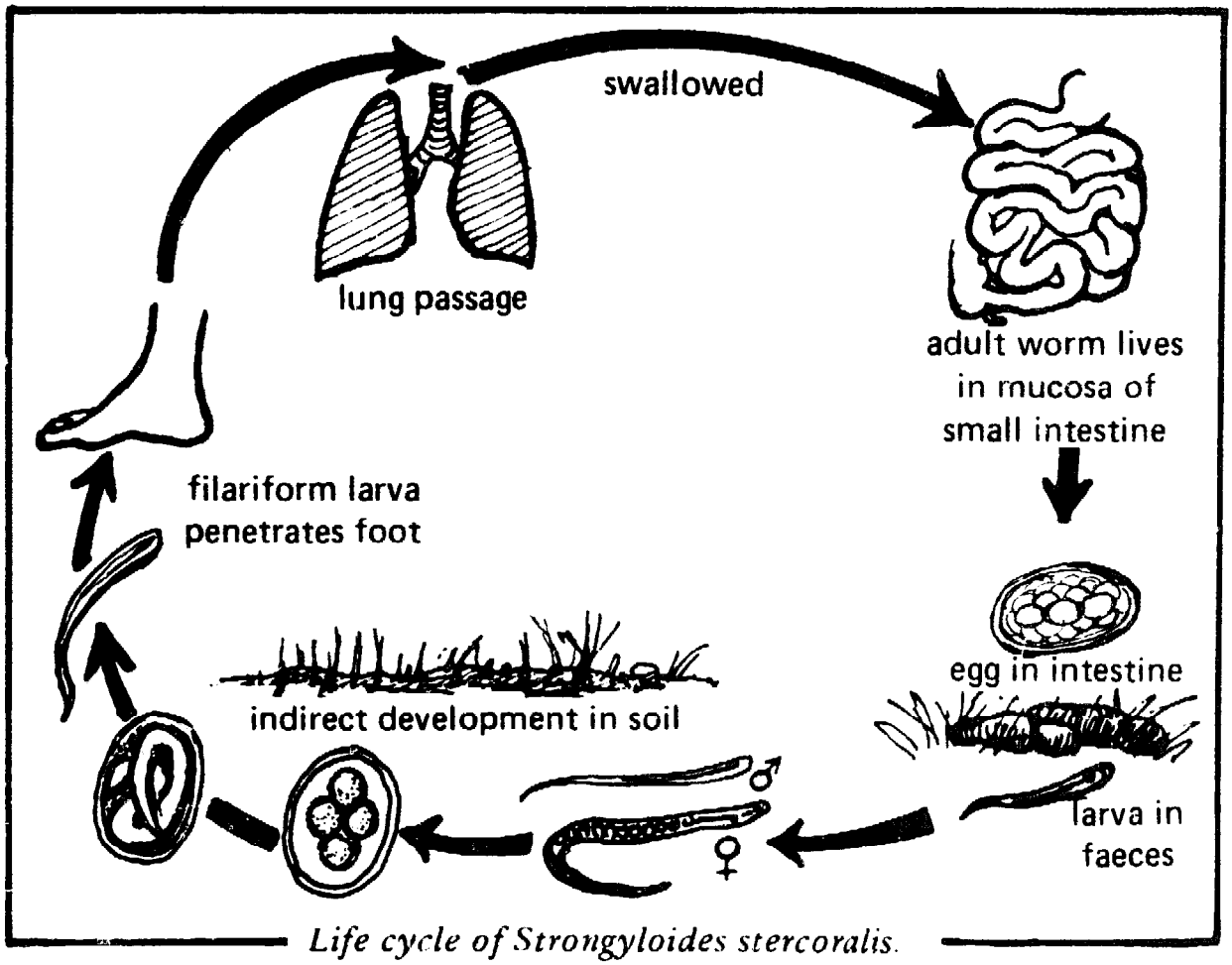
8. *Summary*
   Hookworm infection is caused by nematodes. Hookworm disease occurs when continuing loss of iron results in anaemia. Anaemia occurs more rapidly when dietary iron intake is borderline. Treatment should concentrate on the anaemia. Prevention in the individual is by wearing shoes. Control in the community is possible only through improvement of sanitation.

STRONGYLOIDIASIS

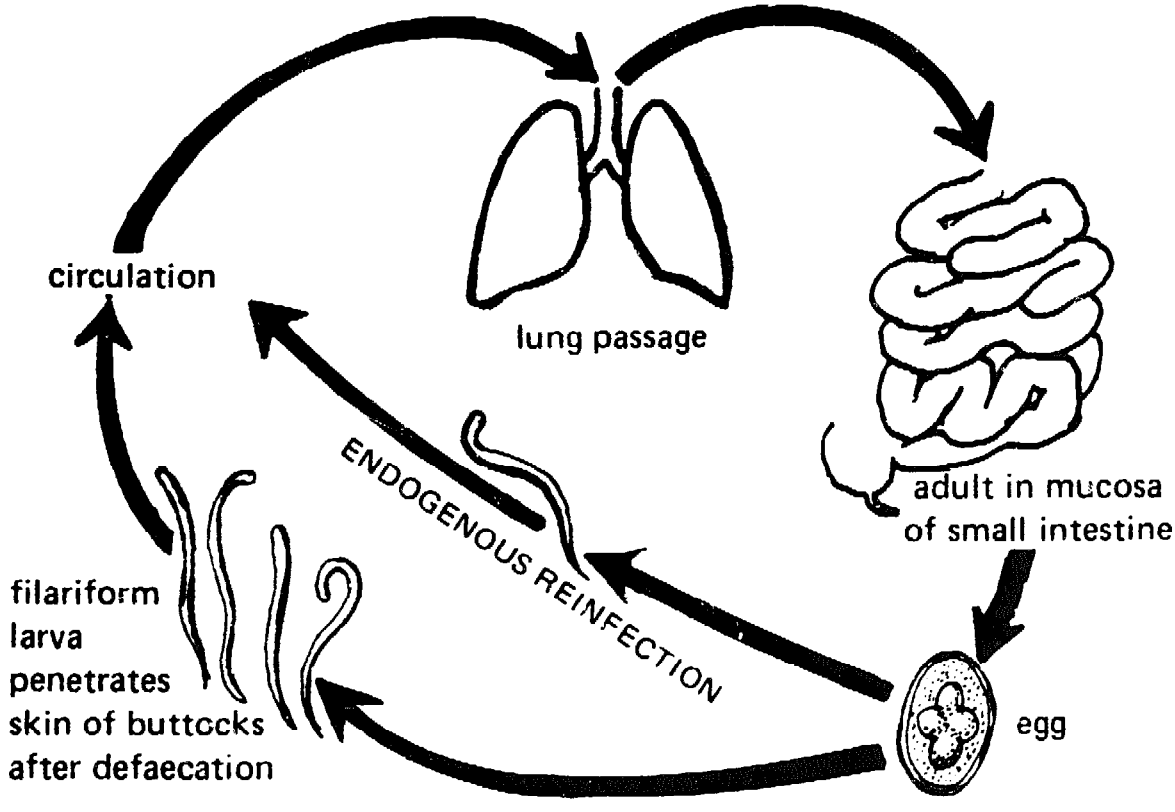
1. Strongyloidiasis is an infection by Strongyloides stercoralis, a nematode worm. The female adult worms live in the mucosa of the duodenum and jejunum. Most infections are without symptoms and signs.

2. *Occurrence and importance*
   Geographical distribution is the same as in hookworm infection. Infections are not usually very severe, but extremely heavy infections (e.g. after immunosuppressive drugs) may result in death because of malabsorption syndrome.

3. *Epidemiology*
   Strongyloides stercoralis resembles the hookworms in appear-



Life cycle of Strongyloides stercoralis.



Auto-reinfection cycle.

ance of adults, eggs, and larvae. In strongyloides infections larvae are usually found in the stools. These larvae may develop either into free-living adults which produce a next-generation infective stage outside the body, *or* the larvae may develop directly into infective filariform larvae which penetrate the skin. The rest of the cycle is similar to hookworm infection. Because of direct development into the infective stage *auto-infection* is common. Even within the bowel the larvae may become infective and penetrate the bowel wall. This is called *endogenous reinfection.*

4. *Clinical picture*
Usually infection is symptomless. In very heavy infections the number of worms in the mucosa may interfere with the normal function of the bowel resulting in malabsorption and diarrhoea. The continuous reinfection may cause urticaria and other hypersensitivity reactions. A high eosinophilia can be explained by continuous reinfection by strongyloides.

Diagnosis: Larvae in fresh stool specimen.

5. *Management of individual patient*
Drug of choice is thiabendazole (Mintezol). This drug is usually not available in health centres: dose 25 mg/kg/day in divided doses for 3/7.

Levamisole (Ketrax) is effective in about 50% of the cases: dose as for hookworm.

6. *Control*
Same as in hookworm infections.

7. *Action*
See ascariasis p 176.

8. *Summary*
Strongyloidiasis is a nematode infection of the duodenum and jejunum. Usually symptomless, occasionally resulting in a mal-

absorption syndrome. Treatment thiabendazole or levamisole. Control as for other intestinal helminths.

TAENIASIS

1. Taeniasis is an infection with the adult Taenia.

   Synonym: *Tegu*, tapeworm.

2. *Occurrence and importance*

   All cases of taeniasis reported in Tanzania are caused by Taenia saginata (beef tapeworm). Tapeworm is especially common in areas where beef is eaten raw or only lightly cooked. Incidence in Tanzania is quite low. Effects on patients are mild. At present Taenia solium (the pork tapeworm) is not reported in Tanzania.

3. *Epidemiology*

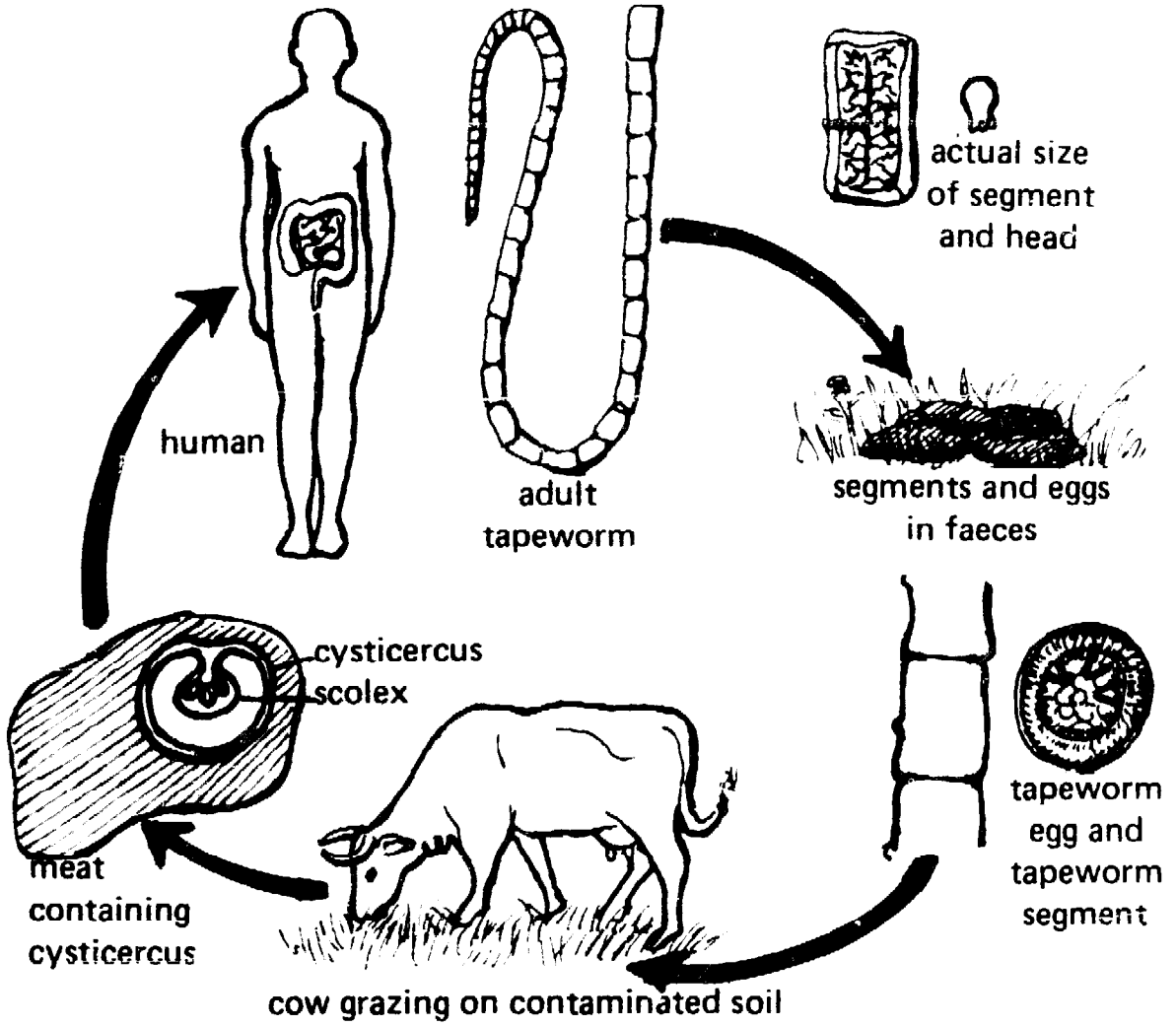
   The adult tapeworms live in the small intestine of Man. Mobile segments of the worm containing gravid uterus and/or the eggs are passed in the stools. The eggs have to be ingested by cows. In the gastrointestinal tract of the animals the embryos hatch and penetrate the bowel wall and are carried via the blood-stream to striated muscles. Here the larvae invaginate, grow, and form the infective cysts, called cysticerci.

   When beef containing a cysticercus is ingested the cyst is dissolved by the gastric juice and in the small intestine the scolex (head of tapeworm) evaginates like the finger of a glove and the worm attaches itself to the bowel wall.

4. *Clinical picture*

   Most infections with taenia cause no signs or symptoms. In some people it causes loss of weight, abdominal discomfort, and pruritus ani (itching). There is no eosinophilia.

   Diagnosis: Macroscopic appearance of worm segments in the



Life cycle of Taenia saginata. The cycle is completed in 8–10 weeks.

stools (or rarely by microscopic examination finding the eggs). Eggs are not laid singly and appear only accidentally in the stools.

5. *Management of the individual patient*
Niclosamide (Yomesan) 4 tablets as one dose is the treatment of choice. Alternative treatment: dichlorophen (Antiphen) 60 mg/kg.

6. *Control*
Faeces disposal: See ascariasis.
Meat inspection: Condemn infected meat.
Health education about the dangers of eating beef which is not cooked thoroughly. Deep freezing of beef will kill all

cysticerci in 24 hours and it can then be eaten raw or partly raw. (Deep freezing will not give protection against other diseases which may be transmitted by infected meat, such as salmonellosis, brucellosis, and anthrax.)

7. *Action*

—See ascariasis for faeces disposal.

—Give health education on danger of eating raw meat.

8. *Summary*

Taeniasis is an infection with adult tapeworms. Infection is usually symptomless. Infection is exclusively acquired by eating partly raw meat. Treatment is with niclosamide. Control depends on meat inspection, proper faeces disposal.

HYDATIDOSIS

1. Hydatidosis is a disease caused by the cysts of the dog tapeworm.

   Synonym: Echinococcosis.

2. *Occurrence and importance*

   Hydatidosis is rare in Tanzania but a major problem in other parts of E. Africa, e.g. in Turkana, Kenya.

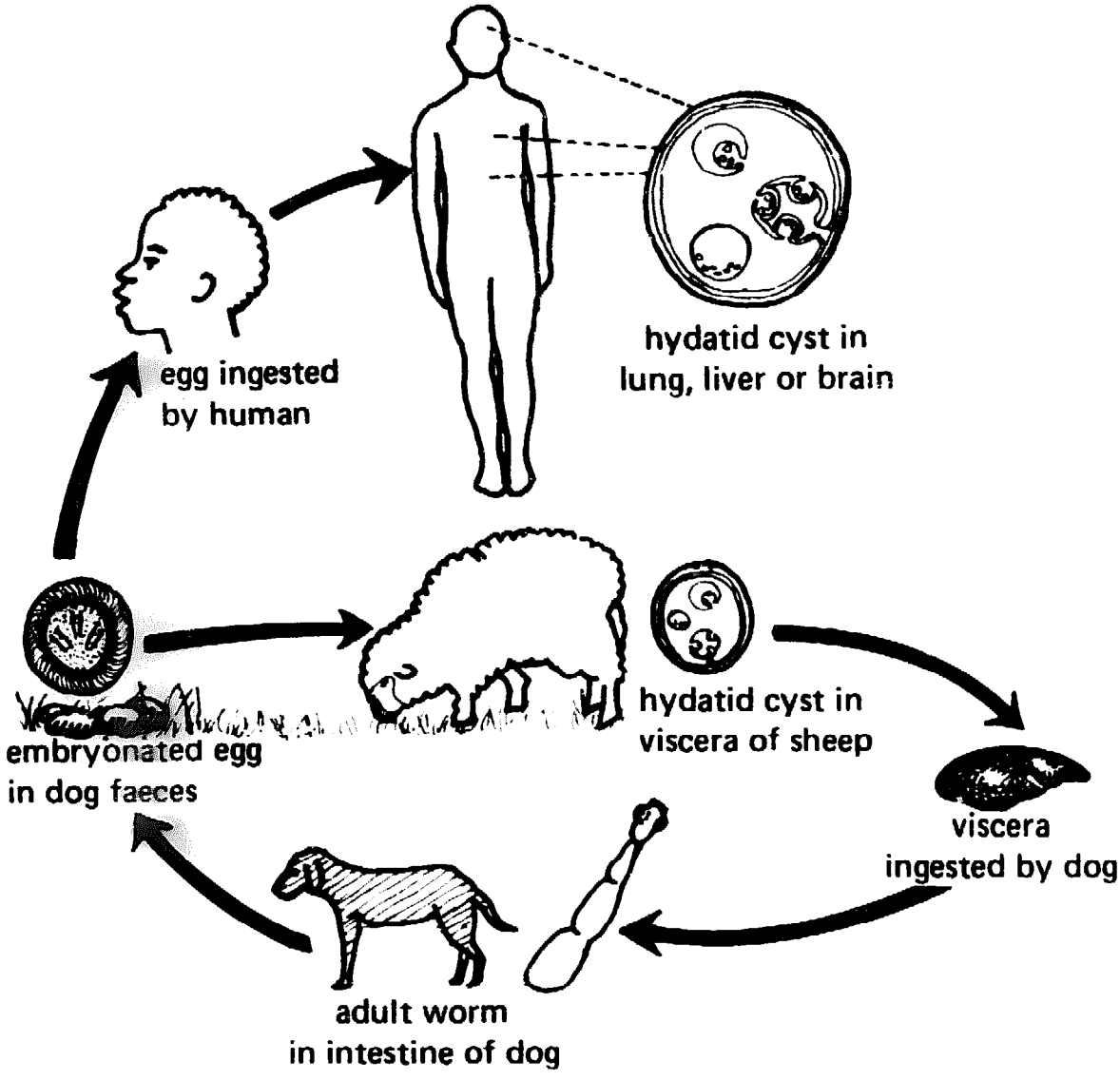
3. *Epidemiology*

   Dogs and other carnivores* are the final host of the dog tapeworm. Eggs are passed in the dogs' faeces and ingested by sheep or goats.

   The eggs hatch in the sheep's intestine and larvae penetrate the intestinal wall to form cysts with many daughter cysts in the liver. These cysts are the infective stage for the dog and develop into mature worms when they are ingested. Similar to the cow-man cycle in taeniasis, but in taeniasis Man is the final host.

*Carnivores: flesh-eating animals, e.g. hyena, jackal.

Man becomes infected with the cysts when he accidentally swallows eggs from dogs' faeces.



Life cycle of Echinococcus granulosus.

4. *Clinical picture*

As in sheep, a slowly growing cyst develops, usually in the liver but almost any tissue in the body can be affected, the lung being the next commonest site. With the liver enlargement the disease may suggest amoebic liver abscess but the general condition of the patient remains good and there is a high eosinophilia.

An Xray may be taken in hospital, and serological tests can

be done to assist in making the diagnosis. Refer all suspected cases to a hospital.

5. *Management of the individual patient*
The cyst should not be punctured. This may provoke a severe anaphylactic response. The condition has to be differentiated from amoebic liver abscess. Treatment is only by surgical removal of the cyst. Chemotherapy is not yet available.*

6. *Control*
—Meat inspection, condemn infected meat. Do not feed it to dogs. De-worm dogs regularly (take care where they then defaecate).

—Health education, especially to children, in endemic areas on the dangers of close contact with dogs (licking).

7. *Action*
Health education

—on the dangers of close contact with dogs

—on the necessity of de-worming dogs regularly

—on the dangers of feeding dogs with uncooked viscera of sheep especially if condemned.

8. *Summary*
Hydatidosis is caused by the growing cysts of the dog tapeworm. Treatment is surgical. Control is by avoiding contact with dogs.

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*A new anthelmintic drug, mebendazole, related to thiabendazole, is under investigation and may turn out to be effective alone or in combination with surgery.

Chapter six

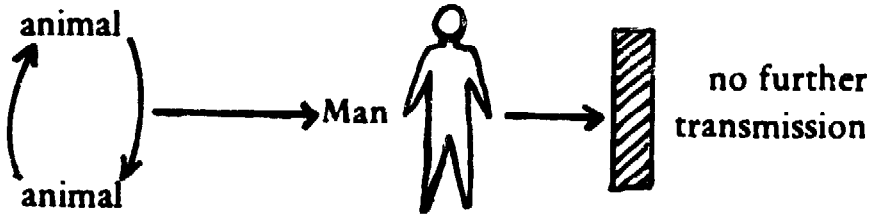
DISEASES THROUGH CONTACT WITH ANIMALS OR THEIR PRODUCTS

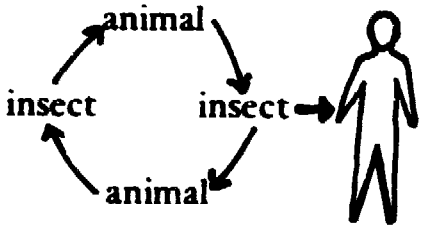
Introduction

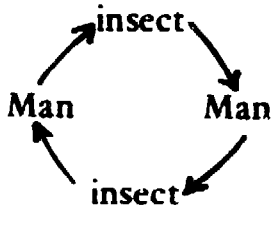
Infectious diseases transmitted under natural conditions between vertebrate animals and Man are called *zoonoses.*

Of most of these diseases Man is a dead end of the transmission cycle. This means under normal conditions Man will not infect other human beings.

Examples:
rabies,
brucellosis,
anthrax

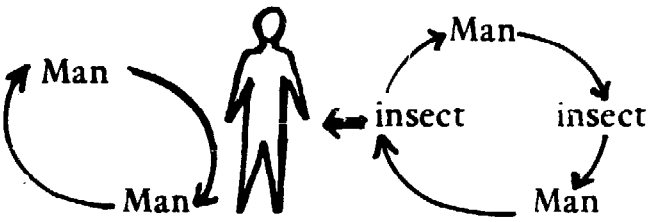


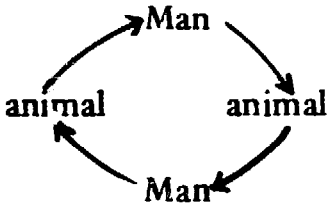




In some diseases Man can transmit the disease to other human beings.
Examples: yellow fever, rhodesiense trypanosomiasis.

In yellow fever and trypanosomiasis the transmission is always through vectors. In plague transmission may be airborne.





In some parasitic diseases Man acts as the final host, and animals are the intermediate host. In Tanzania there is only one disease in this group: infection with tapeworm. In hydatidosis Man may act as intermediate host: the final host is the dog.

Tetanus bacilli are commensals of cattle (and Man). They can cause disease only when the normal transmission cycle is interrupted and infection is established through wounds.

Tetanus cannot be transmitted to others so forms a dead end of the cycle. For Tanzania important diseases acquired through contact with animals are summed up in the table on the next page.

RABIES

1. Rabies is a disease of a animals (a zoonosis) incidentally transmitted to human beings by the bite of a rabid animal.

   Synonym: *Kichaa cha mbwa.*

	Disease	Reservoir animals affected	Transmission to Man	See
ZOONOSIS	plague	rodents	vector	Chapter 3
	sleeping sickness	cattle and game animals	vector	Chapter 3
	yellow fever	monkeys	vector	Chapter 3
	anthrax	cattle (and game)	ingestion inhalation contact	Chapter 6
	brucellosis	cattle	(ingestion) contact	Chapter 6
	rabies	wild animals	bite wound	Chapter 6
Final host	taeniasis	cattle	ingestion	Chapter 5
Intermediate host	hydatidosis	dogs with dogs	close contact with dogs	Chapter 5
Commensals	tetanus	cattle	contaminated wounds	Chapter 6

2. *Occurence and importance in Tanzania*

Rabies is spreading fast in Tanzania. Before 1971 the disease was confined to Iringa, Kigoma, Dodoma (Mpwapwa) and West Lake (Ngara).

In 1977 the disease had spread to the coast, central plateau, northern and southern highlands and lake areas.

Rabies is a feared and dreadful disease because:

(a) When no immediate action is taken after the bite by an infectious animal, the disease will be irreversible once clinical signs have appeared (case fatality rate is 100%).

(b) Tanzania has a large potential reservoir for the disease in wild animals.

(c) The transmission between wild animals and domestic animals is difficult to control under present conditions.

3. *Epidemiology*

Rabies is caused by a virus. The virus has preference for the salivary glands and nervous tissue, causing finally an encephalitis leading to death.

The main reservoir for disease is wild animals such as jackals, mongooses, foxes. These animals do not live in close contact with Man. The domestic animals most affected are dogs and cats. Cattle (oxen) may also become infected.

Man becomes infected when he is bitten by a rabid animal, usually a dog. The saliva left behind in the wound contains the virus. By way of nervous tissue the virus reaches the brain.

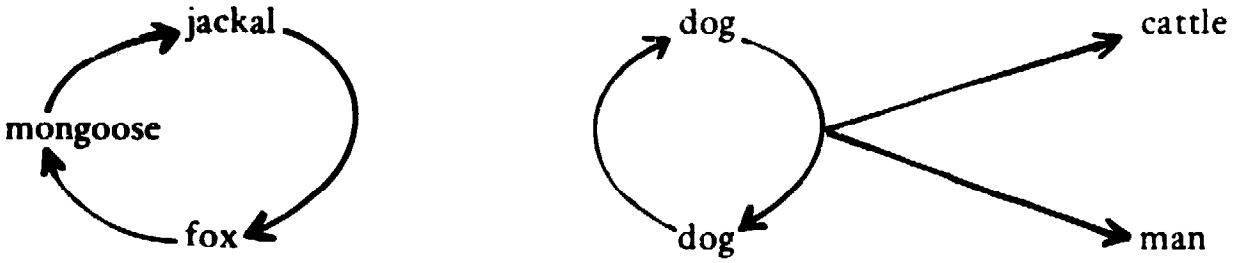


Diagram of transmission

4. *Clinical picture*

In human beings: The incubation time depends on:

(a) The site of the bite, whether it is far or near to the brain.

(b) The kind of wound (abrasion, small wound, wound with extensive tissue damage).

(c) The amount of virus deposited in the wound.

Possible incubation time 2 weeks to 1 year. Average 2–3 months.

Early symptoms and signs: more than normal pain in the

wound, later development in two different pictures.

Furious rabid
—convulsions
—spasms
—intense anxiety

Paralytic rabid
—depression
—paralysis of limbs
—spasms of pharyngeal muscles resulting in hydrophobia: because of the pain it causes the patient avoids swallowing.

In animals:
—abnormal behaviour
—biting without, or after the slightest, provocation
—death within 10 days after first appearance of disease.

Note: Animals may also be depressed and curl up in a corner.

5. *Treatment of individual patient*

No cure is possible once the disease has actually developed. But PREVENTION IS POSSIBLE before it has reached that stage. Although proper first aid will reduce the chance of the virus entering the body, it does not replace anti-rabies vaccination.

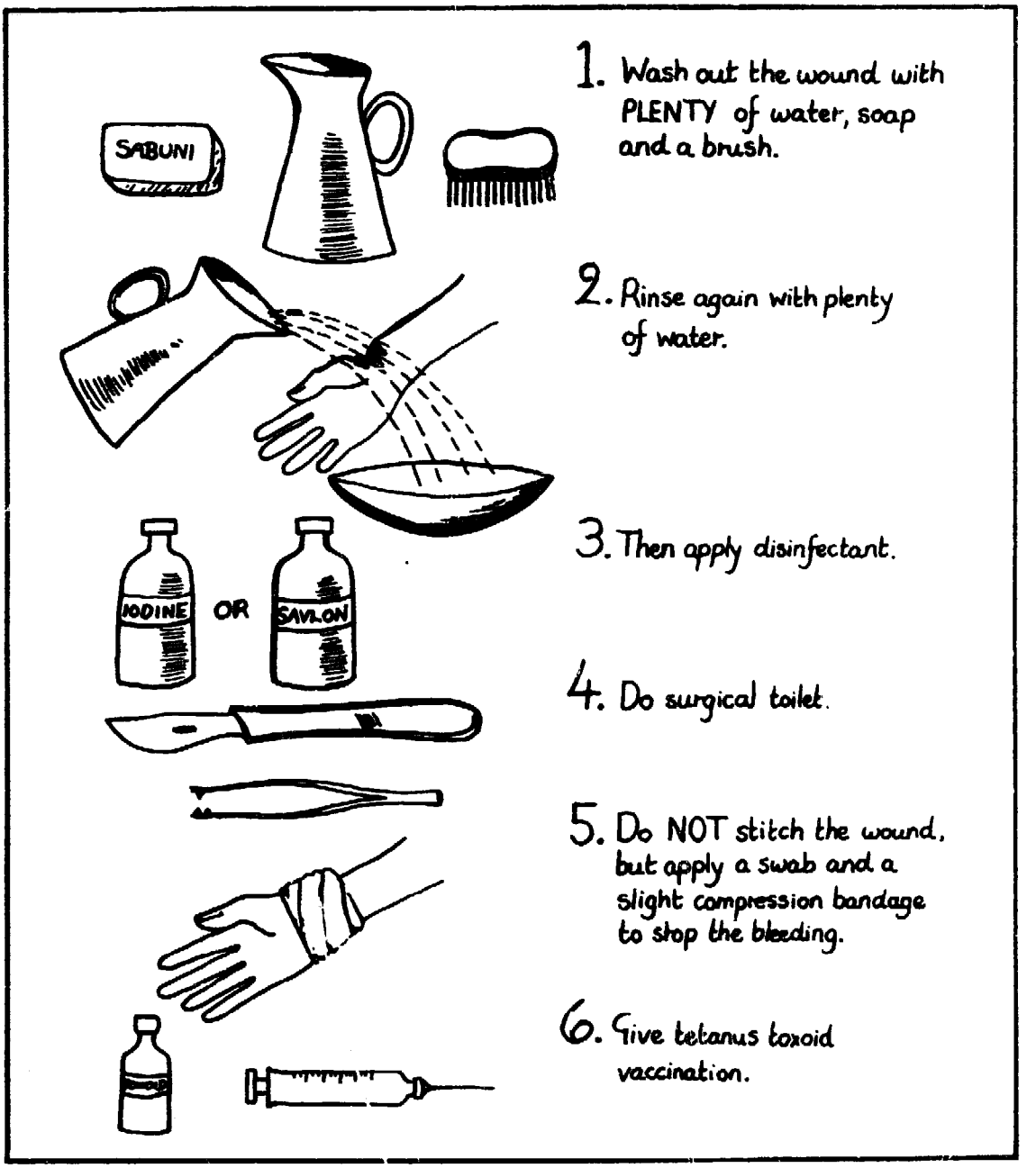
Prevention of rabies is based on proper first aid in all cases of animal bites and immunization.

How to handle a case of animal bite—situations possible:

(a) The animal is available and looks healthy. Write down name, *balozi*, and living place of the patient. Ask him to come back for a check-up after 10 days. Note date of appointment in diary.

The animal must be observed for 10 days. If it remains healthy for 10 days, there is no danger of rabies. If the animal shows signs of rabies, trace the patient immediately and refer him with a proper referral letter to the district hospital for vaccination.

(b) The animal has disappeared: refer the person for vaccination.



Rabies prevention: proper first aid for all animal bites, and immunization.

(c) The animal looks rabid and has been caught: lock up the animal carefully; inform the DMO of your suspicion of rabies, and refer the patient for immunization.

(d) The dead animal is brought to your dispensary. Bury the animal taking care that there is no danger of being infected (*unless* you have made arrangements for investigation of the dog with the local Veterinary Department and they have given you detailed instructions how and where to send the

infectious material). *Do not* send heads of rabid dogs in plastic bags or wrapped in papers by bus or post to a Veterinary Department which does not have the equipment for laboratory confirmation of the diagnosis.

Refer the patient for vaccination.

6. *Control*

The control of rabies is the full responsibility of the Veterinary Department. You must play an important role in health education about prevention, however.

Active vaccination of domestic animals is possible and gives protection for 3 years. Stray dogs must be killed.

General vaccination of humans with the vaccine presently available in Tanzania has too many complications. It is reserved for those who have been exposed, or those with a high risk of exposure, e.g. veterinary officers.

Rabies is a notifiable disease.

7. *Action*

In all cases of animal bites:

—give first aid (see p 199)
—give tetanus prophylaxis
—decide on rabies immunization
—observe biting animal for 10 days if available
—refer all patients for whom rabies immunization is indicated with a proper referral letter indicating why you refer the patient.

If you are working in an area where rabies is a problem:

—make arrangements with the Veterinary Department for investigation of suspected dogs and specimens (*NB* staff working with such material should of course be immunized)
—if you are seeing many people with bites from suspected rabid animals, discuss with your DMO what the health centre and its staff can do
—where there are such facilities, encourage people to have their dogs (and, ideally, cats as well) vaccinated every 3 years.

8. *Summary*

Rabies, once manifested in human beings, is a 100% fatal disease. Prevention is possible, but only when *immediate* action is taken for exposed persons.

The control of domestic animals (i.e. by vaccination) is the responsibility of the Veterinary Department.

Notes. (1) Types of anti-rabies vaccine

There are several types of anti-rabies vaccine available. Most of them are made from brains of infected animals and contain traces of neural tissue. These vaccines often cause local side effects, and occasionally serious nervous system reactions due to their neural tissue content. Immunization courses involve multiple injections (varying from 7 to 21). The most suitable site is the subcutaneous layer of the abdomen.

Recently a new type of vaccine has been produced which is grown in human (non-neural) tissue culture; side effects (local and general) are said to be so rare that it is suitable for general use, but unfortunately the price is so high that it cannot yet be made generally available in Tanzania.

Always read carefully the instructions included with whichever type of vaccine you have to use.

(2) Indications for vaccine and serum

Immunization against rabies can be done with anti-rabies serum and anti-rabies vaccine.

Serum gives passive immunity. It has an immediate effect but the immunity is short-lived. Anti-rabies serum is expensive and not available at the moment in Tanzania.

Vaccine gives active immunity. It takes about two weeks before antibodies appear. After active immunization the level of antibodies is high and immunity is long-lasting.

Indications for use of anti-rabies serum and anti-rabies vaccine are tabled on the next page.

Nature of exposure	Irrespective of previous vaccination		Recommended treatment
	at time of exposure	during 10 days	
1. Contact, but no lesions; indirect contact; no contact	Rabid	–	None
2. Licks on the skin; scratches or abrasions, minor bites (covered areas of arms, trunk and legs)	(a) suspected as rabid	Healthy	Start vaccine, stop treatment if animal remains healthy for 5 days (a,c)
	(b) rabid: wild animal or animal unavailable for observation		Serum + vaccine
3. Licks of mucosa: major bites (multiple or on face, head, finger, or neck)	Suspect or rabid, domestic or wild animal or animal unavailable for observation		Serum + vaccine. Stop treatment if animal remains healthy for 5 days (a, c)

a. Observation period in this chart applies only to dogs and cats.
b. All unprovoked bites in endemic areas should be considered suspect unless proved negative by laboratory examination.
c. Or if brain is found negative by laboratory examination.
d. In general, exposure to rodents and rabbits seldom, if ever, require specific antirabies treatment.

TETANUS

1. Tetanus is an acute disease caused by a toxin produced by tetanus bacilli and is characterized by painful contractions of voluntary muscles.

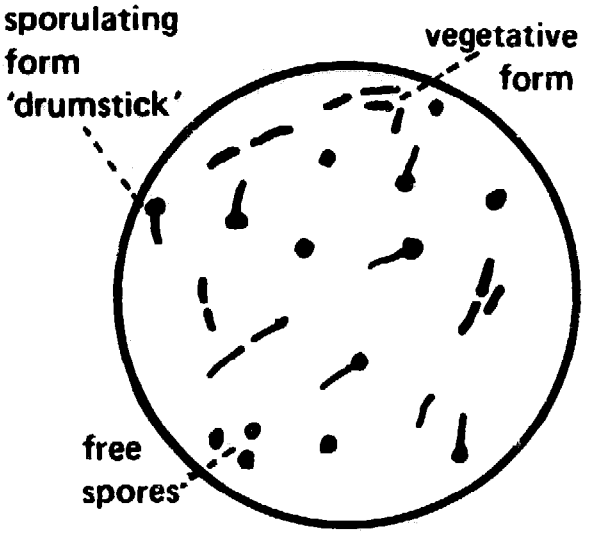
   Synonym: Lockjaw, *pepo punda*.

2. *Occurrence and importance*

Tetanus occurs in sporadic cases all over Tanzania. It is more common in rural areas than in towns. Tetanus is an important disease because of its high case fatality rate of close to 100% in untreated cases. Groups specially at risk are farmers, soldiers, newborns and, in general, anyone with a dirty wound. (Many of the newborns with clinical tetanus will die—50% even when the nursing care is excellent.)

3. *Epidemiology*

Tetanus is caused by the toxin produced by Clostridium tetani, a gram-positive rod which forms terminal spores, giving it a characteristic drumstick shape.



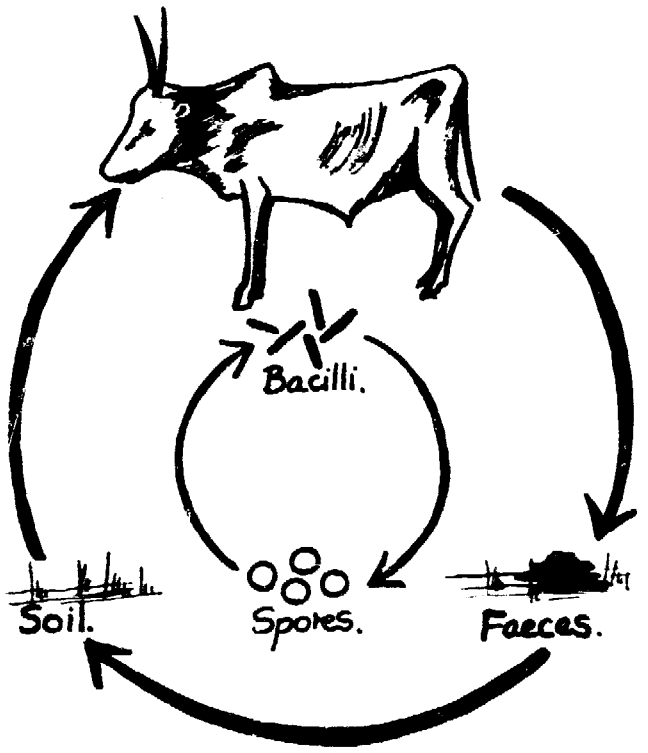
The organism is an obligate anaerobe, so it cannot live and multiply in the presence of oxygen.

The spores are highly resistant to drying and high temperatures; they even withstand boiling for short periods.

Normally tetanus bacilli live in the bowels of animals (and Man). When bacilli are passed out in faeces, spores are formed in order to survive the bad living conditions of the outside world. Therefore tetanus spores may be present in any soil contaminated by faeces, particularly faeces of cows. Tetanus bacilli continue their life cycle when the spores are swallowed by animals (and Man). Disease in Man occurs only when spores enter the body through a contaminated wound.

The source of the infection is soil, street dust, or faeces with spores of tetanus bacilli.

Because tetanus bacilli can only live and multiply in the absence of oxygen, tetanus is most likely to develop in deep penetrating wounds with tissue necrosis.



Tissue necrosis is a clear sign of impaired blood supply, as it is the blood that brings the oxygen to the tissues. In a mixed infection aerobic bacteria can consume so much oxygen that conditions may become favourable for Clostridium tetani.

Wounds which favour tetanus are:

(a) Umbilical stump in newborn (necrosis).

(b) Crush wounds (necrosis, poor blood supply).

(c) Stab wounds (deep).

(d) Wounds with foreign bodies (always infected), human (!) and animal bites.

(e) Burns (necrosis, blood supply).

(f) Endometritis (after abortion or childbirth, from the use of poorly sterilized instruments).

(g) Surgical wounds (from dressings or instruments).

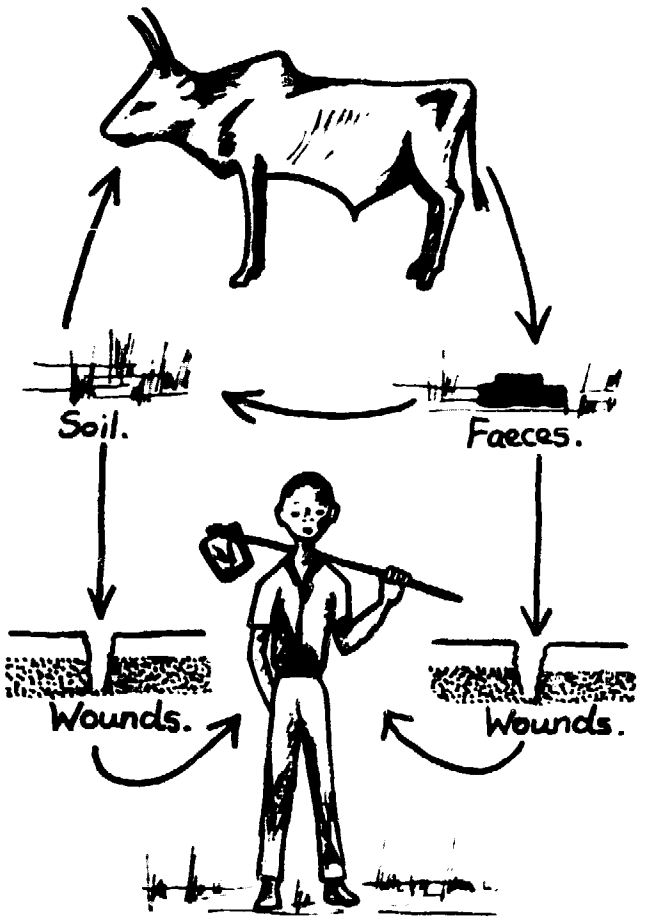
(h) Chronic ulcers (like jiggers) and chronic discharging ears.

(i) Endogenous infection may occur when intestinal tetanus bacilli are introduced into the tissues (during bowel surgery).

In 10–15% of all cases of tetanus the site of entry remains undetected. Tetanus bacilli are harmful only when they are lodged in the tissue because from there the toxin can be transported to the central nervous system. When tetanus spores or bacilli are

swallowed the bacilli can live and multiply in the bowel but no harm will be done as the toxin is not absorbed from the bowel.

A special form of tetanus is seen in the newborn. After birth there are many changes in the blood circulation of the newborn with the result that blood no longer flows through the umbilicus.



Transmission of tetanus infection.

The cord becomes necrotic and falls off in a few days. It is clear that the umbilicus is a wound and necrotic tissue is present, an ideal place for tetanus bacilli to enter the body, especially if they are directly applied to this wound in the form of cow dung, as has been the custom in some tribes in Tanzania, or by using unsterile instruments for cutting the umbilical cord. When a wound is infected with tetanus bacilli the normal transmission cycle is interrupted. So a patient with tetanus is not infectious to others.

4. *Clinical picture*

The usual incubation time is 5 days to 3 weeks (but it can be from 3 days to 3 months). The time between the first signs and the first generalized spasms is called the *onset time*. The shorter the incubation time and the onset time the more fulminating is the disease.

The disease starts with increased tone in the jaw muscles —trismus and risus sardonicus ('devil's grin'), later painful spasms occur in all muscles, with disturbance of swallowing and respir-

ation. The spasms of the neck muscles may resemble the neck stiffness of meningitis, but Brudzinski and Kernig signs are negative. There is no drowsiness or change in consciousness as in meningitis. There may be fever. Death occurs due to asphyxia because of either:

(a) Spasm of glottis, thoracic muscles, diaphragm.

(b) Chronic hypoventilation because of muscle stiffness.

(c) Periods of apnoea.

(d) Aspiration and subsequent suffocation.

In the newborn the first sign of tetanus is *inability to suck in a baby who was first doing well*; later spasms appear accompanied by severe apnoea and cyanosis.

5. *Management of individual patients*

Patients must be referred to hospital as quickly as possible.

Spasms: Even at a dispensary or health centre an attempt must be made to control the spasms before referring the patient. The ideal drug to use is diazepam (Valium) in high doses. Start with 10–40 mg iv and give the same dose at the same time in the form of crushed tablets through a gastric tube. Maintain the sedation by giving the drug every 3 hours through the tube.

Further doses of diazepam should be given according to the condition of the patient. When spasms continue more diazepam should be given. Diazepam is usually very well tolerated and the maximum dose is very high (500 mg daily). As the success of tetanus treatment depends entirely on the control of spasms, do not hesitate to increase the dose of this drug until the patient is very well sedated.

But watch for signs of over-sedation. Reduce the dose when patients do not react at all when touched. A properly sedated patient gets spasms lasting only one second when awakened by touching or shaking.

RESERVE YOUR DIAZEPAM FOR TETANUS CASES ONLY

Chloral hydrate, phenobarbitone, or chlorpromazine will sustain the effect of diazepam.

Children: chloral hydrate.

Adults: phenobarbitone 100 mg 4 hourly.

Secondary infection: To combat secondary infection and the tetanus bacilli themselves crystalline penicillin 1 mU and PPF 1.2 mU are given on admission. PPF 1.2 mU is continued daily for 5 days.

Antibiotics do not alter the severity or the duration of the spasms. Spasms are not caused by bacilli but by the toxin already fixed to the neurones.

ATS (anti-tetanus serum): The effect of anti-serum in the *treatment* of tetanus is still doubtful. Dose: adults and children 10,000 units im or iv once; give test dose first, keeping adrenaline at hand because allergic reactions are common and dangerous.

Surgical treatment: Look for any wound, and clean with spirit or Savlon. Operative procedures in a patient with established tetanus are not recommended because severe, uncontrolled spasms commonly accompany surgery performed in the early stages of the illness. Tracheostomy with artificial respiration can only be performed in specialized centres.

Intensive care: Careful nursing is the most important part of treatment. Formerly tetanus patients were frequently nursed in dark and isolated rooms. This resulted in the nurses forgetting to observe the patients. With the sedatives available at present the patient will not be disturbed by light or noise. Most important is that the patient be kept under constant observation by the nurse in charge. If he develops breathing arrest due to spasms, immediate mouth to mouth respiration should be performed (tetanus is not infectious). When he is spastic, immediate sedation should be given. Observation from minute to

minute will save the life of a tetanus patient.

ISOLATING TETANUS PATIENTS RUNS THE RISK OF FORGETTING THEM

The patient should be nursed in a semiprone position (never on his back). Every two hours his position must be changed. Raise the foot of the bed to stimulate lung drainage. This will help to prevent pneumonia.

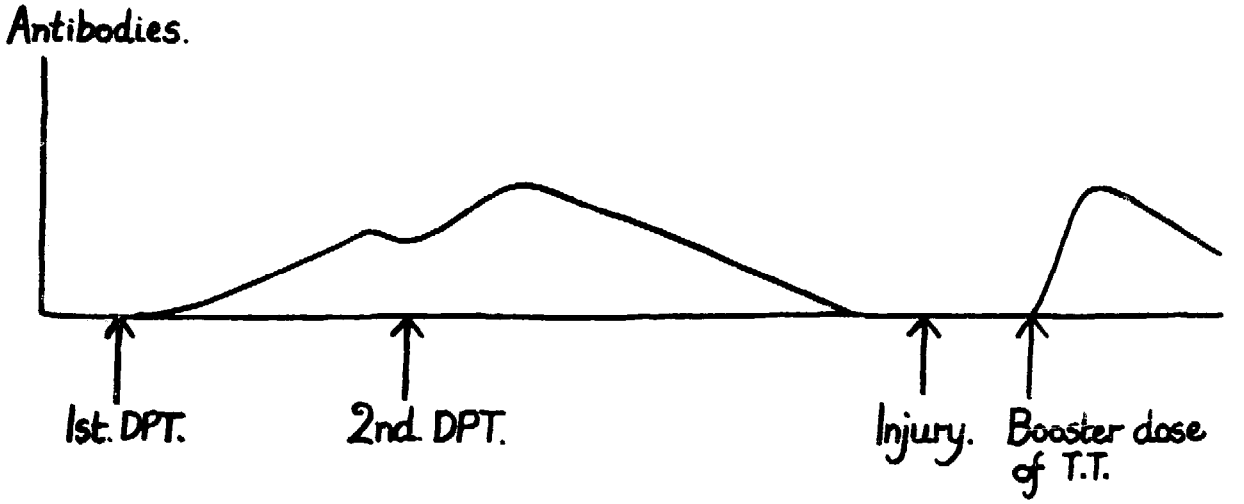
Every patient must have an observation chart on which medication, fluid input, spasms, and position are recorded.

Toxoid (TT): Patients with tetanus do not develop immunity against tetanus. They still have to be vaccinated with tetanus toxoid to avoid recurrence.

6. *Control*

Proper surgical treatment of wounds such as removal of foreign bodies and excision of necrotic tissue will diminish the risk of tetanus. Active immunization with tetanus toxoid gives solid protection. The first inoculations should be given in childhood together with diphtheria toxoid and pertussis vaccine (DPT).

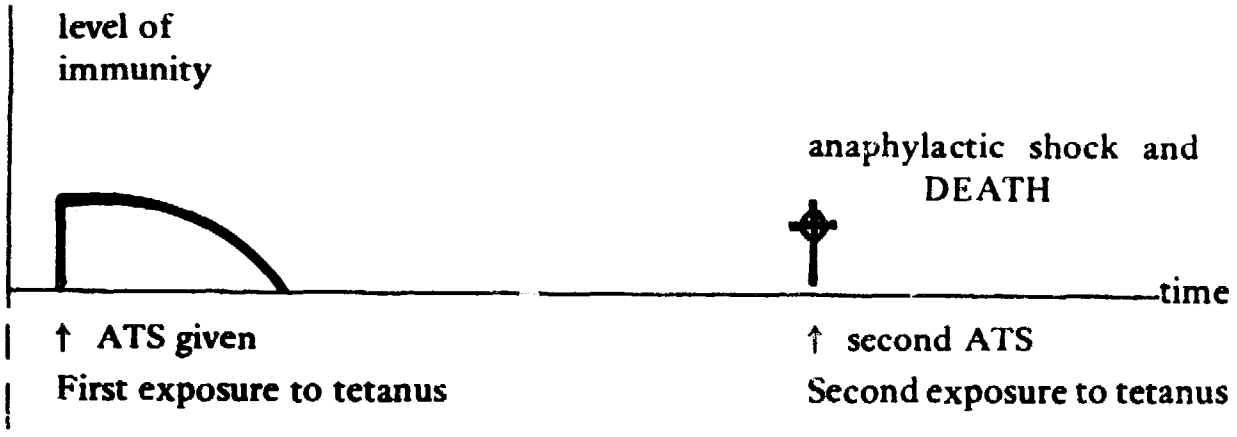
In case of injury later in life, if primary vaccination was complete, a single booster injection will result in a rapid and high rise in antibody levels which are protective for 7–10 years.



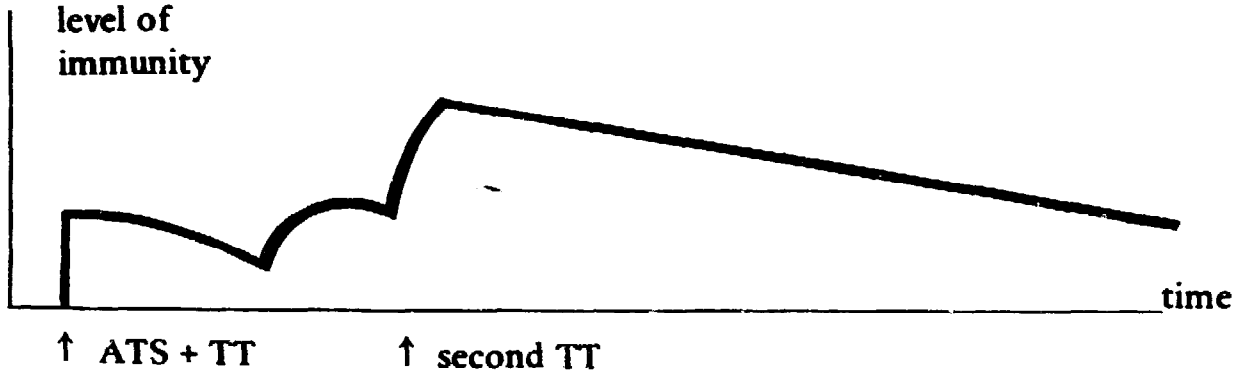
When no previous active immunization has been performed, the patient presenting with an injury needs prompt protection

as the incubation time of tetanus is shorter than the time the body needs to produce active antibodies. This protection can be given in the form of anti-tetanus serum (ATS).

Passive protection lasts only for a very short time (10 days) and therefore ATS should *never* be given without active immunization with tetanus toxoid. The routine of prescribing ATS and PPF to all injured patients is an example of bad doctoring which should be stopped. ATS is a serum and therefore a potentially dangerous drug, especially on second administration. This is expressed in the diagram below:



When both immunizations are combined the person at risk is fully protected as illustrated in the graph below:



Conclusion:

NEVER GIVE ATS WITHOUT TOXOID

Tetanus of the newborn can be prevented by active immunization of the mother during pregnancy. Ideally three injections of tetanus toxoid are given 4 weeks apart. The first one must be given at the first visit to the antenatal clinic. There is no need to wait until 28 weeks of pregnancy. The last one must be given during the last month before the expected date of delivery.

After the administration of tetanus toxoid, antibodies are circulating in the maternal blood and are transferred to the fetus via the placenta and cord. The newborn is therefore getting antibodies (passively) from the mother which will protect him during the healing of his umbilical wound after delivery.

DPT should never be given to adults as the pertussis part of the vaccine may cause severe reactions; DPT cannot replace tetanus toxoid. (Do not use DPT for pregnant mothers when tetanus toxoid is out of stock.)

There are no contraindications for tetanus toxoid and side effects are minimal (pain at the injection site for 12–24 hours).

7. *Action*

Patient:

—Control spasms with either diazepam iv 10–40 mg or phenobarbitone 100 mg plus chlorpromazine 50 mg.

—Pass gastric tube.

—Repeat same dose of diazepam immediately by mouth.

—Give PPF 1.2 mU daily for 5 days.

—Write letter of referral and put patient on transport.

—Give tetanus toxoid to your patient after recovery.

Community:

—Give TT to all pregnant mothers.

—Give DPT three times to all under-fives.

—Encourage health centre and hospital deliveries.

—Train and equip village midwives and MCH workers.

—Give health education on proper clean treatment of wounds and umbilical cord.

—Vaccinate all schoolchildren with TT.

DPT FOR ALL UNDER-FIVES
TT FOR ALL PREGNANT MOTHERS
TT FOR ALL CONTAMINATED WOUNDS
TT FOR PATIENTS RECOVERING FROM TETANUS
ATS ONLY WHEN INDICATED, AND ALWAYS WITH TT

8. *Summary*

Tetanus is a severe complication of wound infections. It can be prevented by vaccination.

When routine DPT vaccinations were given during childhood only a booster is required, this will result in a rapid and high rise of immunity.

TETANUS CAN BE CONTROLLED BY VACCINATION

ANTHRAX

1. Anthrax is an acute bacterial disease, primarily of grass-eating animals. Occasionally it infects Man.

The clinical picture in Man depends on the port of entry.

Synonyms: Hide-porter's disease, wool-sorter's disease, malignant pustule, splenic fever, *kimeta.*

2. *Occurrence and importance*

Anthrax is a disease of cattle, goats and sheep. It can occur suddenly in big numbers (epizootic) especially in times when cattle are moved from one place to another, e.g. after droughts

or flooding.

The clinical picture and the severity depend on the port of entry in Man.

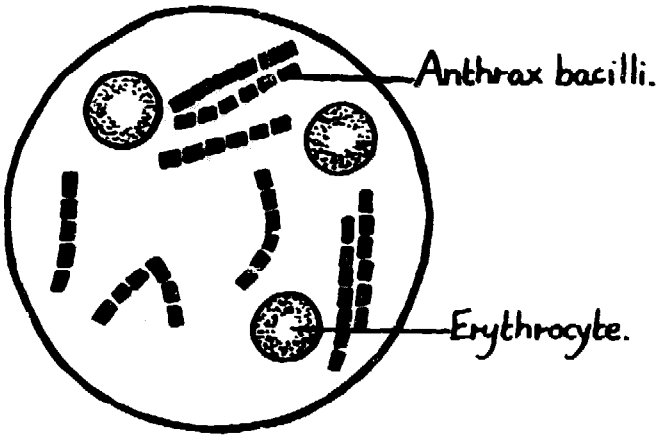
Anthrax of the skin occurs mostly in persons handling cattle: farmers, butchers, workers in the hide-processing industry.

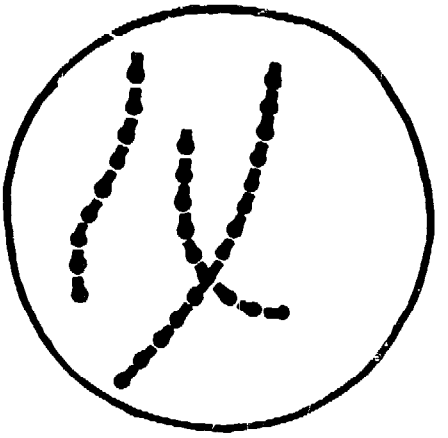
Anthrax of the lungs occurs in persons working with infected wool.

Anthrax of the bowel occurs in families who eat meat of sick or dead animals.

3. *Epidemiology*

Anthrax is caused by a spore-forming bacillus. The spores are formed when the bacillus is exposed to air. Spores are extremely resistant to climatic conditions and can survive for years in the soil. Vultures may spread the spores. The spores enter the animals orally.





Anthrax bacilli containing spores.

Anthrax in animals is characterized by a short fever followed by septicaemia and death.

Animals which have died of anthrax show:

—jelly-like oedema of the spleen
—dark unclotting blood
—enlarged lymph glands
—bloody oedema of subcutaneous tissue
—haemorrhagic enteritis.

The body of the sick or dead animal is swarming with bacilli which are easily spread via their faeces, urine, saliva, and blood.

4. *Clinical picture in Man*

Depends on portal of entry.

Table: Clinical picture in Man

Portal of entry	Cracks of skin	Respiratory tract	Digestive tract
Early signs	papule	mild symptoms of URTI	–vomiting –watery diarrhoea
	blister		
	malignant pustule		
Full picture	–black central necrosis –extensive oedema –painless swelling	–serious respiratory distress (pneumonia) –shock	–fever –sepsis
Case fatality	low	high	moderate
Laboratory diagnosis from	fluid of vesicles	sputum	faeces
Treatment	penicillin	oxygen/penicillin	rehydration

Diagnosis is confirmed by finding gram-positive rods in a specimen (fluid from vesicle, sputum, faeces).

5. *Management of the individual patient*

Any antibiotic can be used. The drug of choice is penicillin. Isolation is unnecessary.

6. *Control*

The major role in control is played by the Veterinary Department. Control measures are:

(a) Proper disposal of infected animals. The carcasses must be

burnt or buried at least 2 m deep in the ground in quicklime.

(b) Inspection of all meat offered for sale.

(c) Vaccination of all susceptible animals every year.

(d) Strict rules for disinfecting skins and hides in the leather-producing industry.

(e) People at risk may be protected with *human* vaccine.

7. *Action*

—In time of famine people will eat diseased or dead animals. Whole families will come to the dispensary, all with gastro-intestinal complaints. In this case rule out anthrax.

—During your regular health talks about nutrition, explain the dangers of eating sick or dead animals, unless the meat inspector has approved certain parts of the meat.

—Meet with the veterinary officer. Ask him about the incidence of animal diseases which are dangerous for human beings. Then you know which diseases you can expect in your area.

—In case of epidemics among animals, use your authority among the peasants; convince them that their cattle must be vaccinated.

—Explain why skins of diseased animals can be dangerous if untreated.

8. *Summary*

Anthrax is a disease of grass-eating animals occasionally transmitted to Man. In Man it may cause disease in the skin, respiratory tract or digestive tract. All antibiotics are effective; penicillin is the drug of choice.

Control in animals is possible by vaccination and proper disposal of affected animals.

Control in Man involves meat inspection and disinfection procedures in handling skins.

BRUCELLOSIS

1. Brucellosis is a disease of animals. It may be transmitted to human beings and is characterized by fever, headache, weakness, sweating, joint pains, and generalized aching.

   Synonyms: Undulant fever, Malta fever.

2. *Occurrence and importance*

   Brucellosis is an occupational health hazard of farmers, veterinarians, and butchers, therefore more common in males than in females. The incidence in Tanzania is unknown because many cases may be missed due to lack of laboratory facilities. The infection in animals causes repeated abortion presenting an economic problem for cattle owners. Case fatality rate is less than 2%. Death can be caused by complications of the disease.

3. *Mode of transmission*

   Close contact with infected cattle provides opportunity for human infection. The disease is transmitted by contact with tissues, blood, urine, vaginal discharges, aborted fetuses or, especially, placentas of infected animals. The disease can be transmitted by ingestion of milk or milk products from infected animals, but this seems to be uncommon.

   Causative organism: Brucella abortus (cattle)
   Brucella melitensis (goats, sheep)
   Brucella suis (pigs).

4. *Clinical picture*

   The patient usually presents with fever of unknown origin (PUO). Onset is usually insidious. A common complaint is pain in the lumbosacral region or hip joint. Pain may be severe.

   The diagnosis is difficult on clinical grounds. The occupation of the patient may raise suspicion. Splenomegaly is common but of little help in malaria areas. Serological diagnosis is possible but many patients come to hospital when the titres are already falling.

   An intradermal test can only prove prior contact; a negative

test excludes brucellosis.

5. *Management of the individual patient*
Tetracycline for 2–6 weeks. Addition of streptomycin will reduce the relapse rate but watch for side effects (ototoxicity) in long-term use.

6. *Control*
Boil milk before use and/or use only boiled milk for preparing cheese and butter. Slaughter infected animals and vaccinate calves in endemic areas.

Health education on the dangers of handling placenta and fetus of an aborted animal.

7. *Action*
If brucellosis is a problem among farmers in your area contact the veterinary officer and discuss the problem. If he confirms the high prevalence of the disease arrange for serological diagnostic procedures in your health centre and test everyone with fever not responding to antimalarials. Give health education to the farmers.

8. *Summary*
Brucellosis is an occupational health problem of cattle and goat farmers. Patients present with fever. Diagnosis is difficult. Treat with tetracycline.

Chapter seven

AIRBORNE DISEASES

Introduction

The group of airborne diseases is of the utmost importance in Tanzania. It includes the two most common causes of death (pneumonia and measles) which together cause a quarter of all deaths in Tanzanian hospitals.

Table 1: Most common causes of death in Tanzanian hospitals in 1973.

1973		No. of deaths	% of total number of deaths	
Pneumonia	(1)	2,073	14.1%	25%
Measles	(2)	1,628	11.1%	
Gastroenteritis	(3)	1,317	9.0%	

The organisms causing the diseases in the airborne group enter the body via the respiratory tract. When a patient or carrier of pathogens talks, coughs, laughs, or sneezes, he discharges fluid droplets. The smallest of these remain up in the air for some time and may be inhaled by a new host. Droplets with a size of 1–5 microns are quite easily drawn into the lungs and retained there.

Droplets which are bigger in size will not remain airborne for long but will fall to the ground. Here, however, they will dry and mix with dust. When they contain pathogens which are able to survive drying these may become airborne again by wind or something stirring up the dust, and they can then be inhaled.

Only the small droplets and the partly dried droplets (droplet nuclei) which remain airborne for a long time can spread disease far (more than 3 metres). When the droplets dry most of the micro-organisms die.

Airborne diseases, obviously, will spread more easily when there is overcrowding as in overcrowded classrooms, public transport, canteens, dance halls, and cinemas.

Good ventilation can do much to counteract the effects of overcrowding. Airborne diseases are mostly acquired through the respiratory tract, but not all of them are primary respiratory tract infections (Table 3).

The respiratory infections are caused by a wide variety of organisms and are classified according to the clinical picture as follows:

Respiratory tract infection

1. *Acute respiratory infections*
   Upper respiratory tract infections (URTI) such as coryza, pharyngitis, tonsillitis, laryngotracheitis, or bronchitis.
   Lower respiratory tract infections (LRTI) such as bronchiolitis, lobar and/or bronchopneumonia.

2. *Chronic respiratory infections*
   Whooping cough.
   Tuberculosis.
   (see Chapter 8).

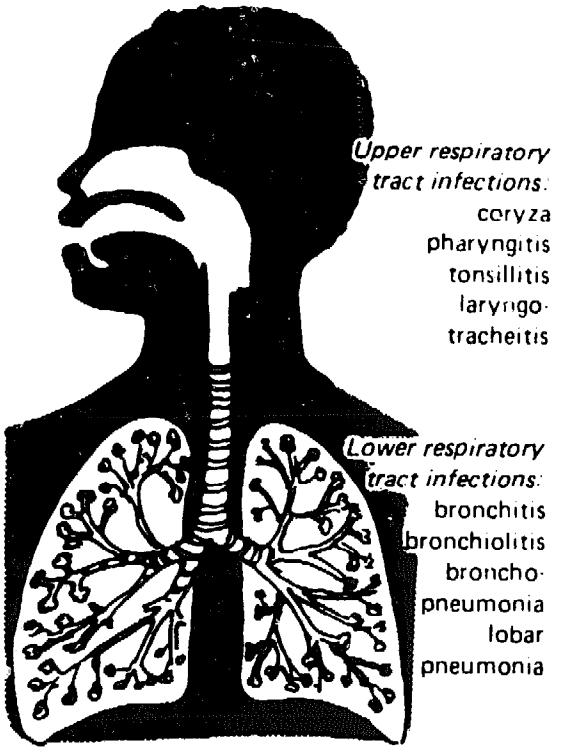


Table 2: Acute respiratory tract infections

Acute respiratory tract infections	Common name	Main sign or symptom	Causative organisms and incubation time	Remarks
Coryza	common cold (*mafua*)	running nose	virus 3 days	
Pharyngeal syndrome	sore throat	sore throat	virus streptococci 2–5 days	differentiate! treat accordingly
Influenza	flu	systemic disturbances	virus 1–3 days	
Herpangina		vesicles in mouth	virus 3–5 days	
Acute laryngo-tracheitis	croup	inspiratory stridor	virus +secondary infection 3–5 days	refer
Bronchitis		cough no dyspnoea	virus bacterial dust (smoke)	
Acute bronchiolitis of infancy		dyspnoea expiratory wheezing	virus 2–4 days	refer
Pneumonia	(*kichomi*)	dyspnoea fever	pneumococci and others virus 1–3 days	admit

The danger of the respiratory tract infections is their effect on the gaseous exchange in the lungs.

The main sign of disturbed gas exchange is dyspnoea. Dyspnoea is always a sign of severe disease. All patients with dyspnoea, whatever the cause, need admission and intensive treatment because

life is at stake.

From Table 2 it can be seen that the diseases above the double line do not have dyspnoea as their main symptom. But they can all be complicated by one of the diseases below the double line. Note that the last two diseases disrupt the gaseous exchange because of damage lower down in the respiratory tract.

The respiratory tract infections are important due to their frequent occurrence and their tendency to be complicated by secondary bacterial infections.

Other diseases which enter through the respiratory tract are:

Table 3

Diseases	Causative organism	Main way of transmission	Complicated by LRTI	Occurrence in Tanzania	Approximate fatality rate
Measles	virus	airborne droplet	++	++++	± 5%
Chickenpox	virus	airborne droplet	+	++	1%
Smallpox	virus	airborne droplet and contact	+	-	40%
Mumps	virus	contaminated articles (eating utensils)	-	+	1%
Rubella	virus	airborne droplet	-	+	0
Meningitis	viruses and bacteria	airborne droplet or endogenous (depending on organism)	-	+++	± 20%
Leprosy	mycobacterium	airborne	-	++	0

Control of airborne diseases

Since the infective particles are spread by droplets from the respiratory tract of patients or carriers, an important part of the control of these diseases is based on preventing droplets from

being inhaled by others; in other words, people must not inhale 'second-hand' air.

Ventilation

Ventilation removes used air and replaces it with clean air. As soon as droplets are taken outside the sun sterilizes them. Increased ventilation can be achieved by building houses with windows, thus allowing natural ventilation. Cinemas and meeting halls should make use of artificial ventilation by means of fans.

Overcrowding

Having too many people in the same room should be avoided. This is especially important in prisons, garrison barracks, dormitories in boarding schools, on ships, and in urban housing where many people may be forced to live in a single room. Building houses with adequate space (even using local materials) is of value.

Isolation

This was a method formerly used to prevent the spread of disease. All people known to be suffering from airborne diseases were isolated.

Examples: isolating Tb patients in sanatoria (*kibongoto*); isolating measles children in infectious disease wards.

It is known now that most diseases are infective before they are recognized. Children with measles are most infective before the measles rash is apparent. Tb patients present themselves for treatment only when serious complications like haemoptysis occur. In the meantime they have infected all the people they live with.

Nobody likes to be isolated. Isolation can be frightening. Because of this people who may be suffering from 'isolatable' diseases tend to delay reporting to medical facilities. At present isolation is not regarded as important.

Health education about personal hygiene

—To cover the mouth when coughing and sneezing.

—To use a handkerchief or paper for disposal of nasal secretions

and sputum.

—Not to spit on the ground in or outside the house.

—Not to share cigarettes, drinking bowls in pombe shops, or eating utensils.

Wearing of masks in hospitals

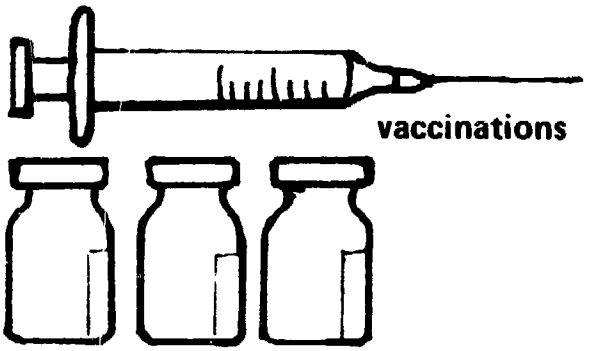
A number of pathogenic organisms are present without symptoms or signs in the throats and noses of many people, including hospital staff. To avoid spreading infection to susceptible patients such as premature babies and patients for operation, masks covering nose *and* mouth should be worn when dealing with these high risk groups. The masks should be frequently washed and dried in the sun.

Spread of airborne diseases. Correct these:

—influenza
—meningococcal meningitis
—rubella
—mumps.

The vaccines for the last four diseases are not essential, are expensive, and not very effective. These vaccines are not available for general use in Tanzania.

Action



—Stress in the ward development committee the importance of building spacious and well-ventilated houses.
—Vaccinate all children attending the MCH clinics (the healthy as well as the sick).
—Inspect the conditions in the local prison and discuss the possibilities of avoiding overcrowding by building more prison wards.
—Give health education about personal hygiene and explain why.

COMMON COLD

1. Synonym: *Mafua.*

2. *Occurrence and importance*

The common cold is the most frequent cause for seeking medical attention throughout the world. Each individual can expect to have one or more colds every year. Factors in the occurrence of common colds are: overcrowding and inadequate clothing. In the highlands, where these conditions prevail, a greater incidence can be expected. Incidence is highest in children under 5, gradually declining with increasing age. Its main im-

portance in the community is that it is the cause of many days of disability and time off work or school.

People who suffer from colds often have a predisposition to serious bacterial complications such as sinusitis, otitis media, laryngitis, tracheitis and bronchitis.

Prevention of colds—these children in cool mountain areas need clothes.

3. *Epidemiology*

The common cold is caused by a number of viruses all giving the same clinical picture. The virus is spread by droplets and by indirect transmission such as freshly infected articles (handkerchiefs). Virus may be discharged in faeces and these may be involved in transmission.

4. *Clinical picture*

—Nasal discharge: first watery, then mucoid and purulent.

—Sneezing.

—Some fever may be present.

—Headache. The headache is caused by obstruction of the canals leading to the air-containing structures in the skull, sinuses, and middle ear. Blockage of these contributes to secondary bacterial infections like sinusitis and otitis media.

—Dry or painful throat.

—conjunctivitis.

Differential diagnosis:

- early measles
- early whooping cough
- hay fever.

5. *Management of the individual patient*

Since the common cold is caused by a virus, no effective treatment can be given. The headache may be relieved by Aspirin. Ephedrine nose drops delay the battle fought in the nasal mucosa between the virus and the body defence system. They give temporary relief but delay cure. After their effect is finished the complaints are often worse than before (rebound effect). So prescribe ephedrine nose drops (¼%) only to breast-fed children with breathing difficulties and to children with otitis media.

Antibiotics are of course not indicated. When they are given to prevent secondary bacterial infections, they do not reduce the number of complications but select the bacilli causing the complications.

6. *Control*

See Introduction URTI.

7. *Action*

Use the opportunity to VACCINATE all children brought to you for treatment of common cold. A common cold is not a contra-indication for any of the vaccinations given at the Under-Fives Clinic.

8. *Summary*

Common cold is an acute infection of the upper respiratory tract. It is characterized by coryza and malaise. Fever is not common. Important because of high attack rate, secondary bacterial infections. Especially common in children. Treatment is symptomatic.

SORE THROAT

1. Synonyms: Tonsillitis, pharyngitis.

2. *Occurrence and importance*

Equally as common as the common cold and may accompany

it. Streptococcal tonsillitis may be followed by allergic reaction in some cases which may result in:

—acute glomerulonephritis
—acute rheumatic fever
—rheumatic heart disease.

3. *Epidemiology*

Most sore throats are caused by viruses. In some cases the causative organism may be streptococci.

4. *Clinical picture*

—Painful swallowing.

—Pharynx, tonsils, and adenoids may be red and swollen.

—Differentiation between streptococcal and viral throat can be made with the aid of the table below:

	Streptococcal tonsillitis	Viral tonsillitis or pharyngitis
High fever	+	±
Pus on tonsil	+	—
Marked cervical lymph gland swelling	+	—

—When the sore throat is caused by herpangina, small painful blister are present on the mucosa of the throat and the cheeks.

5. *Management of the individual patient*

Streptococcal:	Penicillin (PPF 0.8 mU for 5 days)
Viral:	Gargle with salt water, Aspirin. Antiseptic lozenges (Dequadin) are not of any use.

Herpangina: No treatment possible—self-healing in a few days.

6. *Control*
See Introduction.

7. *Action*
—Check the use of penicillin in your health centre.
—Discuss with your staff the (strict) indications for antibiotics.
—Examine routinely all patients complaining of sore throat.

8. *Summary*
Sore throat is a common condition caused by viruses or streptococci. Treat according to cause.

INFLUENZA

1. Influenza is an acute respiratory tract infection of specific viral origin characterized by sudden onset of headache, myalgia, fever, prostration and cough.

   Synonym: 'flu.

2. *Occurrence and importance*
Influenza is important because of potential rapid spread to epidemic proportions, the high attack rate, and the seriousness of complications. Epidemics are associated with a rise in general mortality (excess mortality).

   Deaths occur especially in the elderly and in those with chronic diseases.

   Influenza can occur in pandemics (1889 – 1918 – 1957 – 1968 – 1974) and in major epidemics with intervals of 2–3 years for influenza A, and 4–6 years for type B. Type C occurs sporadically and in minor epidemics.

3. *Epidemiology*
The incubation period is between 1 and 3 days.

4. *Clinical manifestations*
Sudden onset of – headache
– muscle pains
– fever
– prostration
– cough.

Complications – bronchopneumonia caused by Haemophilus influenzae or staphylococci
– sinusitis
–when incorrectly treated with pencillin, 'flu may be complicated by a pseudomonas pneumonia which cannot then be treated with more penicillin.

Differential diagnosis: Malaria; relapsing fever; other viral URTI infections.

5. *Management of individual patients*
–Bed rest.
–Aspirin may relieve pain and diminish fever.

6. *Control*
Vaccination is possible, but due to its expense and the limited protection it gives it is not considered useful in Tanzania.

7. *Action*
See Introduction.

8. *Summary*
Influenza is an acute infection of the upper respiratory tract

characterized by general symptoms. Important because of rapid spread, high attack rate in all age groups, and secondary bacterial infections leading to excess mortality. Control by vaccination is not a priority.

ACUTE LARYNGO-TRACHEITIS

1. An acute bacterial or viral infection of the larynx and trachea, resulting in narrowing of the airways, which may easily lead to obstruction especially in children.

   Synonym: Croup.

2. *Occurrence and importance*

   The disease usually develops as a complication of measles. In children the larynx and trachea are narrow. Swelling of these structures, due to infection, together with sticky mucus may easily obstruct the airways. This can cause death. In adults the larynx and trachea are so wide that they permit some swelling without causing severe distress and death.

3. *Epidemiology*

   The laryngitis may be part of the viral infection. It may be complicated by bacterial infection like Haemophilus influenza.

4. *Clinical picture*

   The first sign is a hoarse voice. Rather suddenly the child may develop a barking cough and dyspnoea. In children the dyspnoea is ***inspiratory***. The inspiration is noisy (stridor). When the obstruction is severe, retraction of intercostal and supraclavicular spaces can be seen during inspiration. The child is restless and is fighting for air.

5. *Management of the individual patient*

   (a) Humidification of the air: all infections of the upper respiratory tract cause an increase of mucus secretion. This mucus

is sticky and difficult to remove, and it easily dries up and forms crusts which are the main cause of the obstruction.

To remove these crusts the secretion must be made liquid by humid air. This is a main part of the treatment.

How to humidify the air:

Steaming: Electric kettle or charcoal stove with kettle —boiling water gives off hot vapour.

Put the child at one end of cot far from the hot steam to avoid burns.

Cover the cot with bed sheets. The child will inhale vapour which will reduce crust formation.

[Covering the cot with wet blankets is not as good as steaming but is better than nothing.]

['Cold steam' machines are not advisable since they break down very easily.]

(b) Hot drinks: heat makes the secretions thin; frequent small amounts should be given.

(c) Antibiotics: use broad-spectrum antibiotics (chloramphenicol).

(d) Corticosteroids: the effect of these drugs in these acute conditions is still doubtful.

(e) Intubation: When you have small (children's size) tracheal tubes intubation may be life-saving.

(f) Tracheostomy: This procedure needs continuous experienced nursing care. If this is not available, tracheostomy will surely cause the death of the child.

6. *Control*

See introduction; measles as an underlying cause of laryngotracheitis is preventable by vaccination.

7. *Action*

—Check your MCH services, ensure that eligible children receive

measles vaccine.

—Allocate a charcoal stove and a kettle to the children's ward. Find ways to guarantee a regular supply of charcoal to make sure that steaming is possible whenever it is indicated.

8. *Summary*

Acute laryngo-tracheitis is characterized by inspiratory stridor. It may occur as complication of other respiratory tract infections, especially measles. Treatment is by humidification of air. Control is concentrated on prevention of measles.

PNEUMONIA

1. Pneumonia is an acute respiratory infection with fever, cough, and dyspnoea. It is recognized above all by the severity of the dyspnoea rather than Xray changes or signs discovered by auscultation.

   Synonym: *Kichomi.*

2. *Occurrence and importance*

Pneumonia is a common disease of infancy and old age. Pneumonia is by far the commonest cause of death in patients admitted to Tanzanian hospitals.

Before the time of antibiotics the case fatality rate was between 20 and 40%. At present the fatality rate is about 5%.

Factors predisposing to pneumonia are upper respiratory tract infections, especially influenza in the elderly and measles and whooping cough in children.

These infections cause damage to the epithelium of the lungs and so clear the way for bacterial superinfection. Smallpox, typhoid, acute and chronic bronchitis, and sinusitis can all be complicated by pneumonia. Other conditions associated with pneumonia are aspiration of a foreign body, and lung oedema.

Patients who are chronically ill and bedridden do not ventilate the lower parts of the lungs very well. Therefore they easily

develop pneumonia of the lower lobes (hypostatic pneumonia).

3. *Epidemiology*

Pneumonia can be caused by several organisms:

—pneumococci
—streptococci
—staphylococci
—klebsiellae
—Haemophilus influenzae
—viruses which cause upper respiratory tract infections can also cause pneumonitis.

(See sections on upper respiratory tract infections, measles and chickenpox.)

The specific lung infections that may occur in anthrax, plague, and tuberculosis are not considered here.

Pneumonia is also associated with the lung passage of larvae of ascaris, strongyloides, and hookworm. A chemical pneumonia is the main danger resulting from kerosene poisoning, especially if, wrongly, attempts are made to wash out the stomach.

Because of its close association with measles and influenza, the incidence of pneumonia follows the pattern of these diseases.

Epidemic pneumococcal pneumonia can occur in institutions, barracks and on board ship where people are crowded in their living and sleeping quarters.

Many healthy people are carriers of the organisms which can cause pneumonia. Transmission is by droplet spread, direct oral contact, or indirectly through freshly infected articles; it results in the pneumococci colonizing the nasopharynx but not necessarily in disease.

4. *Clinical picture*

The key symptoms of pneumonia are cough, fever, and dyspnoea. The key sign is crepitations originating from the alveoli (fine crepitations).

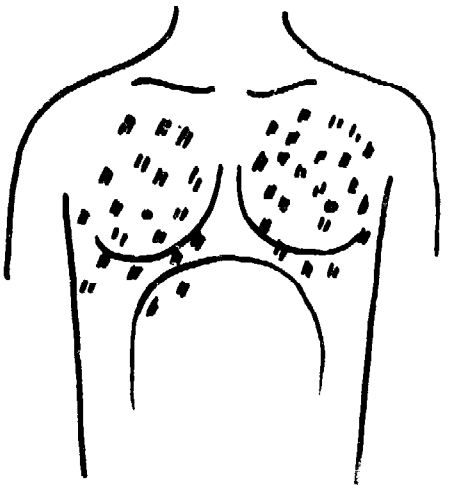
Bronchopneumonia: This is the most common form of pneu-

monia. It is very often a complication of an existing disease: whooping cough, measles, smallpox, typhoid, influenza, acute or chronic bronchitis, sinusitis.

Bronchopneumonia also occurs in chronically ill patients (hypostatic pneumonia), after aspiration, in lung oedema, in bronchostenosis (bronchial carcinoma) and after lung infarction.

In bronchopneumonia the onset is *not* abrupt; there may be remittent, intermittent, or continuous fever; there is rarely herpes. The sputum is purulent but there is no brick-coloured (rusty) sputum.

Findings: variable fine crepitations, especially over the lower lobes, bilateral areas of impaired resonance, bronchial breathing with bronchophony and increased vocal fremitus. WBC high, with many polymorphs.



Bronchopneumonia.

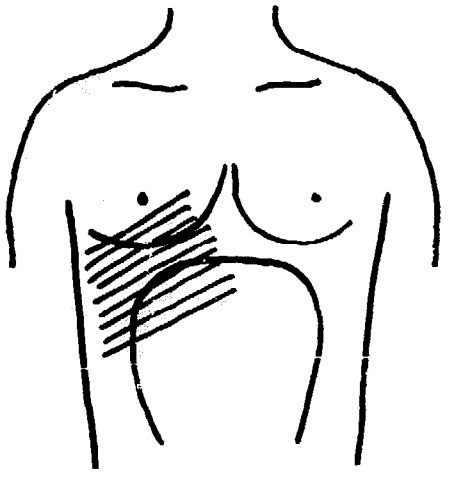
Bilateral signs:
percussion—impaired resonance
auscultation—bronchial breathing, variable fine crepitations
palpation—increased vocal fremitus.

Lobar pneumonia: The onset is often very sudden in completely healthy people, with chills, stabbing pains in the chest and dyspnoea.

Fever is high and continuous (39°–40° C); general symptoms are prostration, anorexia, headache, insomnia and vomiting.

The cough is dry on the first day but later becomes productive. On the third day typical sputum is produced which is glassy, sticky and with a rusty-red colour (brick-coloured sputum). About half of the patients have herpes labialis.

Chest examination will show signs of consolidation over one lobe, that is, dullness with bronchial breathing, increased vocal



Lobar pneumonia.

fremitus and fine crepitations.

In the very old there may be little fever. Diagnosis depends on finding signs of consolidation. WBC high, many polymorphs.

Consolidation in one lobe:
percussion—dullness
auscultation—bronchial breathing, fine crepitations
palpation—increased vocal fremitus.

Differential diagnosis:

—Malaria can mimic pneumonia—exclude by doing a blood slide.

—Typhoid also presents with fever and cough. There are no fine crepitations on chest examination.

—Bronchitis. Acute bronchitis presents with fever and cough, but gaseous exchange is not disturbed and there is no dyspnoea and/or cyanosis. Crepitations or rales originate from the bronchi and are coarse. Coarse crepitations should be differentiated from fine crepitations originating in the alveoli. Bronchitis is often wrongly diagnosed as pneumonia, resulting in over-treatment with penicillin. If antibiotics are indicated in the treatment of bronchitis, sulfa or broad-spectrum antibiotic should be used (H. influenzae).

Complications of pneumonia:

(a) Delayed resolution; watch for Tb, foreign body in lungs.

(b) Pleural effusion.

(c) Empyema, lung abscess.

(d) Heart failure and atrial fibrillation (give digitalis).

(e) Pneumococcal meningitis, pericarditis, or arthritis.

5. *Management of the individual patient*

Frequently a patient with mild pneumonia can be treated on an outpatient basis, particularly if he is living near the health unit.

But any patient with dyspnoea should be admitted for initial treatment with crystalline penicillin 0.5 mU 6-hourly, oxygen may be indicated when there is cyanosis or restlessness or delirium (indicating cerebral hypoxia).

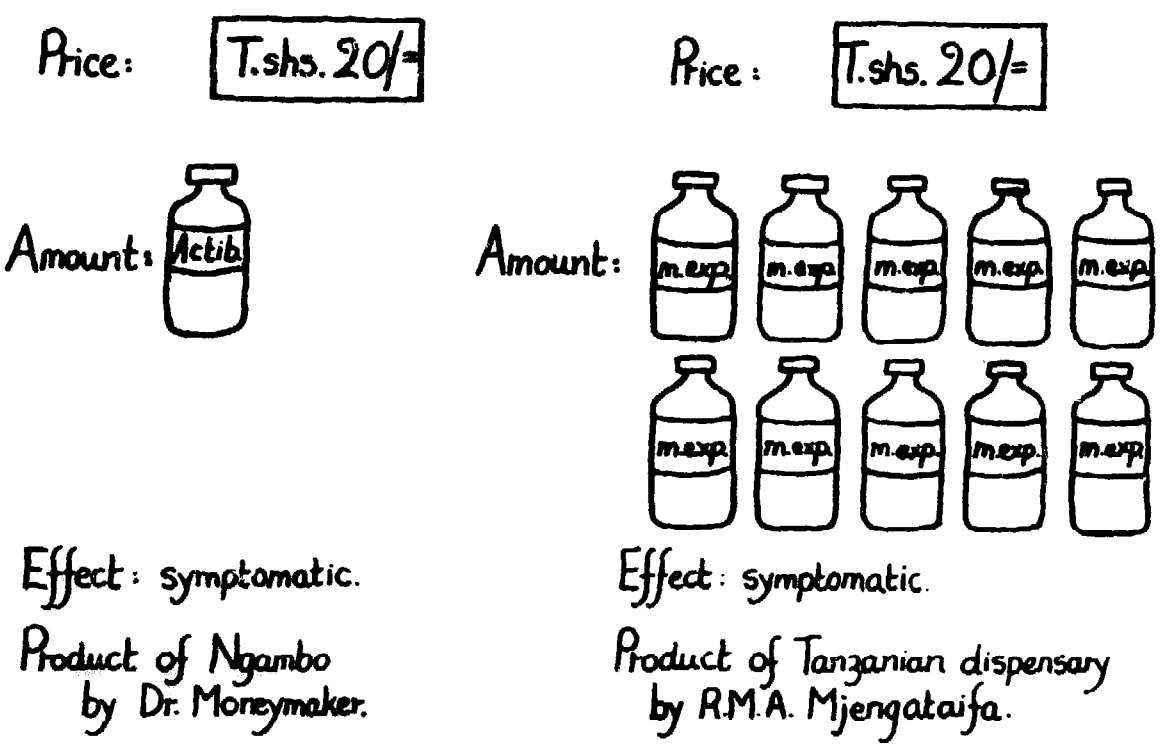
If there is no improvement within 48 hours, the causative organism is most likely not sensitive to penicillin (H. influenzae, staphylococci, klebsiellae); change to tetracycline (adults) or chloramphenicol. H. influenzae is often sensitive to sulfadimidine. If the patient responds well to the treatment and/or can be treated on an outpatient basis give PPF 1.2 mU daily.

Dehydration should be prevented by ample fluid or be corrected if it has already occurred.

Adequate humidity is important, especially in dry climates. Humidity can be raised by hanging wet bed clothes (wrung out in water) with a fan blowing on them, if electricity is available.

Expectorants are not of use. These products have no proven

Comparison of different cough mixtures.



pharmacological action. Cough suppressants are often added to cough mixtures. These drugs will prevent mucus being coughed up and so will prolong the duration of the disease (compare anti-diarrhoeals and duration of infection). Cough suppressants are only of help in giving a very tired patient the rest he needs at night. They act by sedation. All cough mixtures are placebos. Cough mixtures which contain many ingredients like the special prepacked preparations will benefit only the purse of the producer.

WHEN A PATIENT WITH PNEUMONIA DOES NOT IMPROVE ON TREATMENT, EXAMINE THE SPUTUM FOR Tb

6. *Control of pneumonia*

—The control of pneumonia is based on the general principles governing control of the airborne diseases.

—Vaccination to prevent diseases frequently complicated by pneumonia, such as measles and whooping cough, is of utmost importance—watch for signs of pneumonia.

—Mortality can be greatly reduced by early diagnosis and treatment, especially in children and the elderly; diagnosis should be made at dispensary and health centre level; prompt adequate therapy should be started in these units.

EARLY TREATMENT REDUCES MORTALITY

—Maintenance of proper drainage and ventilation in bedridden patients, by raising the foot of the bed, and changing position two-hourly. Ventilation exercises in postoperative and elderly patients.

7. *Action*

—Treat vigorously all patients with dyspnoea diagnosed as pneu-

monia.

—If you insist on using cough mixture as placebo, make one yourself to save expense.

—Make sputum gram stains to assist in the choice of the most appropriate antibiotics.

8. *Summary*

Pneumonia is characterized by cough, fever, and dyspnoea. It often occurs as a complication of an upper respiratory tract infection. The drug of choice is penicillin. According to the degree of dyspnoea, patients are treated as in- or outpatients.

Control depends on control of primary disease.

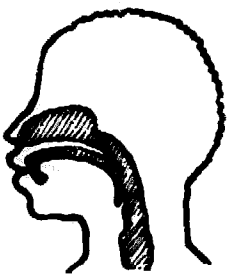
Mortality can be greatly reduced by early diagnosis and treatment.

HAEMOLYTIC STREPTOCOCCAL DISEASE

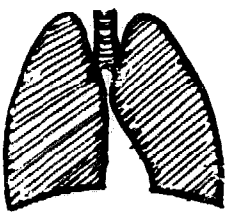
1. Streptococci are part of the bacterial flora present on the skin, and in the nose and throat of healthy people. Some types, e.g. the haemolytic streptococci, may cause disease.

Skin
erysipelas
impetigo
wound infection

Subcutaneous
cellulitis
lymphadenitis



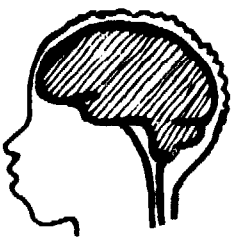
Throat
tonsillitis
streptococcal
sore throat



Lungs
pneumonia

Ears
otitis media
mastoiditis

Brain
meningitis



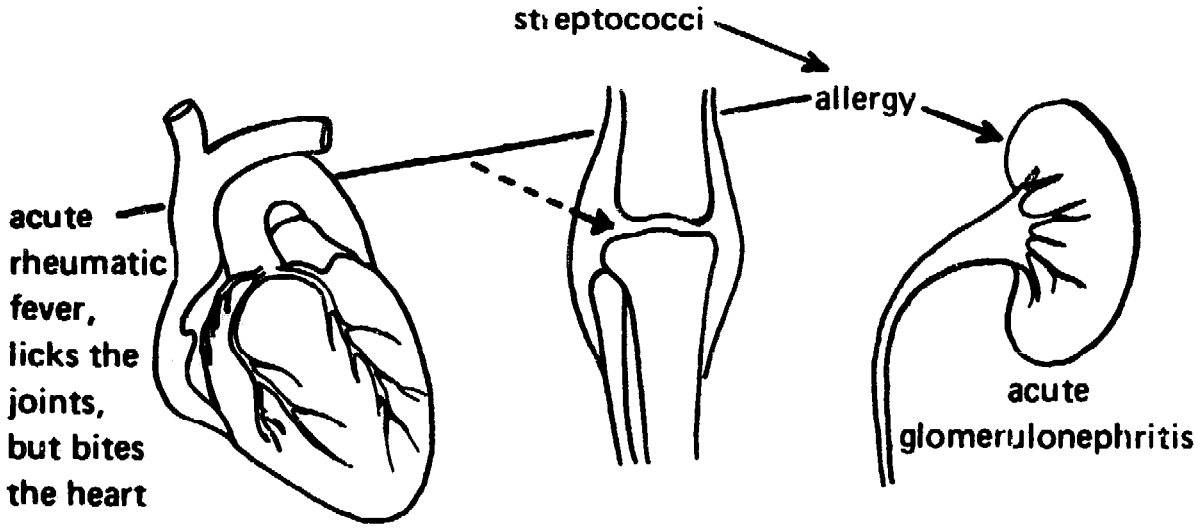
Uterus
puerperal
sepsis

2. *Occurrence and importance*

Diseases caused by haemolytic streptococci are common. Scabies lesions are often secondarily infected with streptococci.

In some individuals streptococci provoke an allergic reaction. This reaction may cause serious damage to the heart and kidneys. In the kidney it causes acute glomerulonephritis. In the heart it damages the valves, resulting in mitral stenosis. In heart disease the joints are also involved: acute rheumatic fever.

Acute glomerulonephritis and acute rheumatic fever occur mostly in children.



Allergic complications after infection with streptococci.

3. *Epidemiology*

Streptococci are spread by skin-to-skin contact or by droplet infection. Healthy carriers may harbour streptococci in the nasopharynx.

Explosive outbreaks of streptococcal sore throat may follow ingestion of contaminated milk or other food.

4. *Clinical picture*

This depends on the place of infection.

Throat: Tonsillitis and sore throat, see acute respiratory tract infections.

Skin: Erysipelas is a rapidly spreading infection of the skin

with systemic disturbance. The onset is acute with high fever, headache and vomiting. The skin lesion is a painful, spreading swelling with a well-defined raised edge.

Impetigo consists of superficial vesiculopustular lesions in the epidermis. The lesions rapidly burst and the resulting skin defects are covered with crusts. Lesions heal without scarring. Impetigo is very often secondary to other skin diseases, e.g. impetigo of head and neck in pediculosis capitis (head louse). Impetigo may also be caused by staphylococci.

Subcutaneous: Cellulitis—inflammation of skin and subcutaneous tissue. The skin area is oedematous, shiny and hot. The edges of the inflamed area are sharply marked. Regional lymph glands may be inflamed and the infection may be complicated by septicaemia. When cellulitis occurs in the hands or feet, tendon sheaths and bones may become involved.

Uterus: Puerperal sepsis—invasion of the uterus by streptococci happens mostly after childbirth and abortion. This results in endometritis, showing as foul-smelling lochia, with slight fever. When the Fallopian tubes are invaded there is high fever, and severe low abdominal pain.

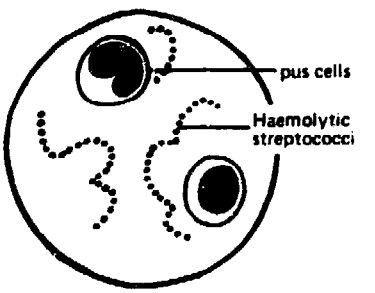
Ears: Otitis media occurs as a complication of upper respiratory tract infections. Blockage of the Eustachian tubes prevents drainage of the middle ear. As a result pus accumulates until the drum perforates. Fever and pain are the main symptoms (see also *Child Health* manual, Chapter 10.6).

Mastoiditis is a complication of otitis media. There is tenderness and swelling over the mastoid often associated with fever and vomiting. Meningitis may follow mastoiditis.

Lungs: Pneumonia—see acute respiratory tract infections.

Brain: Meningitis—see p 245.

Diagnosis: Gram stain of pus, sputum, CSF, will show gram-positive cocci in chains.



Cocci in chains.

5. *Management of the individual patient*

Superficial skin infections like impetigo and wound infection do not need treatment with antibiotics. Impetigo can be treated with an antiseptic such as sulphur ointment* or gentian violet paint 1%.

All other conditions and infections of the newborn are potentially dangerous and should be treated with antibiotics. Penicillin is the drug of choice.

6. *Control*

General hygiene will reduce predisposing infections like scabies and louse infestation.

Aseptic techniques in hospitals will reduce wound infections and puerperal sepsis. For control of tonsillitis and pneumonia, see acute respiratory tract infections. For control of meningitis, see p 250.

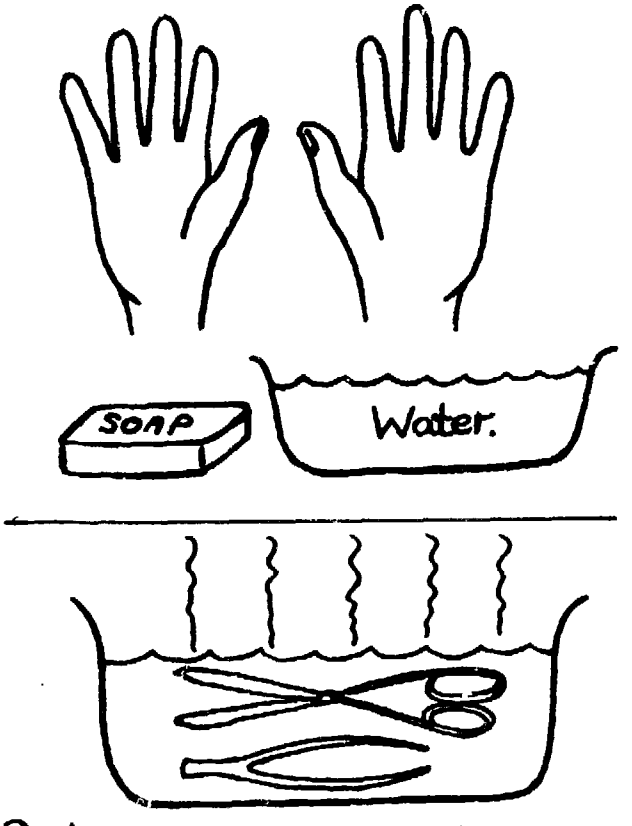
7. *Action*

—Isolate septic patients in a septic ward. If you do not have a separate ward, make a septic corner in the ward.

—Place a basin with water and soap in each ward, to make it easy for everyone to wash their hands regularly.

—Allocate enough forceps and scissors for changing dressings for each individual patient.

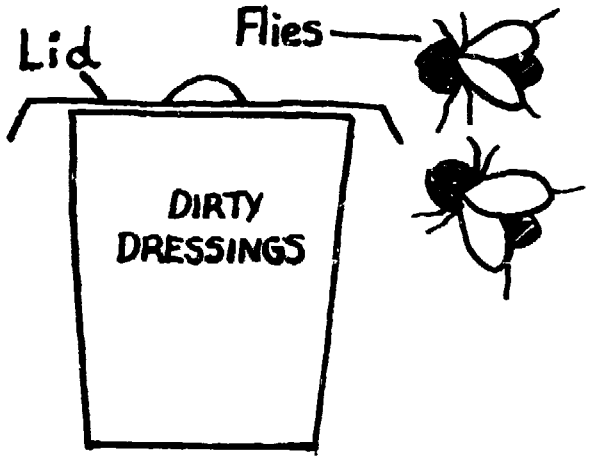
Note: Wiping forceps with an an antiseptic solution does



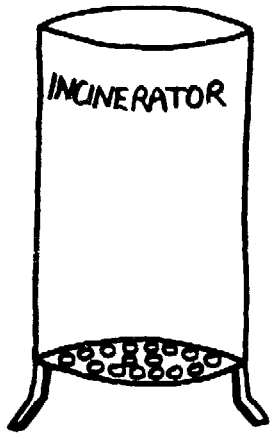
*Sulphur ointment. Do not confuse with *'sulfa'* as in sulfadimidine.

not kill the bacteria immediately. The quickest way to kill bacteria is to boil the instruments.

Do not dispose of dirty dressings in a forgotten corner near the health centre within flying distance of flies.



Dispose of dirty dressings by wrapping them in used paper (used stationery or newspapers) and burning them daily in your incinerator.



It is useless to give health education on environmental sanitation unless your health centre is a good example itself.

—Make the wards, dressing rooms, and theatre fly-proof. It is better for the patients, and in the long run for your drug vote, to invest some money in screening material rather than 'covering' all patients with antibiotics.

8. *Summary*

Haemolytic streptococcal disease may occur in skin and inner organs. Dangerous complications to the heart and kidney may arise due to an allergic response to streptococci in some individuals. The drug of choice is penicillin.

STAPHYLOCOCCAL DISEASES

1. Staphylococci are bacteria which produce different clinical

pictures depending on where they are. They often produce pus.

Skin: impetigo
boils
infection of lacerations.

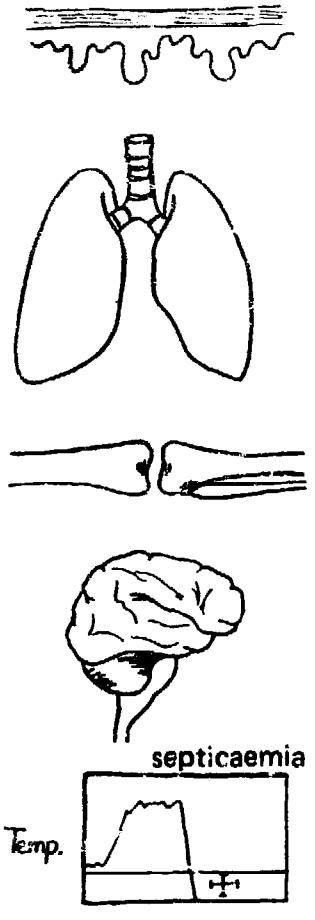
Lungs: pneumonia
abscess.

Bones: osteomyelitis
arthritis

Brains: meningitis
abscess

Blood: septicaemia

Food: food poisoning
(see p 157)



2. *Occurrence and importance*

Diseases caused by staphylococci are common.

Infections of the skin are of minor importance, but they may be the portal of entry for the bacteria into the inner organs.

Staphylococcal infections of the internal organs are dangerous: they are especially likely in people with a weakened defence system; after viral infection; in the chronically ill; and in patients undergoing major surgery.

3. *Epidemiology*

There are many different types of staphylococci. Only a few of these cause disease. Staphylococci are a normal part of the bacteria (the flora) living on the skin, and in the nose or in the throat. 30–40% of healthy persons are carriers of harmful staphylococci. They may produce staphylococci in their nasal secretions and infect others (this is the reason why face masks must be worn during operations). Due to the overuse of anti-

biotics in hospitals some types of staphylococci have developed resistance to most antibiotics.

These types may cause the dreaded infections with what are called 'hospital staphylococci'. Patients with purulent wounds may infect all operation patients. The staphylococci may be spread by flies, by the fingers of the nursing and medical staff, and by invisible droplets containing bacteria.

4. *Clinical picture*

This depends on the site of the infection. When the bacteria are only causing superficial infection of the skin toxins are not absorbed into the blood circulation and no signs of general infection appear.

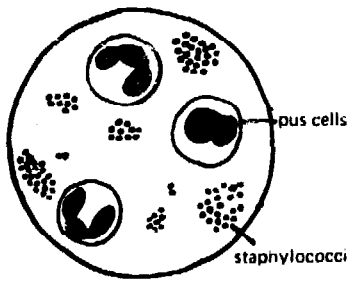
If an unripe abscess is incised, or if boils are squeezed, bacteria may enter the blood-stream and give rise to a septicaemia with fever, malaise, and headache.

When the bacteria are localized in the internal organs, the clinical picture is related to the disturbance of these organs.

Impetigo, see Streptococcal Disease. Pneumonia, see Acute Respiratory Tract Infection; Meningitis, (p 245).

Diagnosis:

Gram stain of pus, sputum, or CSF will show gram-positive cocci in clusters.



Gram positive cocci in clusters.

5. *Management of individual patients*

(a) Superficial skin infections: no systemic antibiotic treatment is needed. Local application of antiseptics, e.g. flavine, eusol, Gentian Violet 1%, hibitane, and frequent changes of dressings will do.

(b) In case of involvement of internal organs: quick treatment with high doses of broad-spectrum antibiotics is indicated—

tetracycline, chloramphenicol.

(c) In a newborn with generalized skin infection systemic penicillin treatment is indicated.

6. *Control*

—Known patients with purulent lesions must be isolated.

—Dressings from purulent wounds must be handled with care.

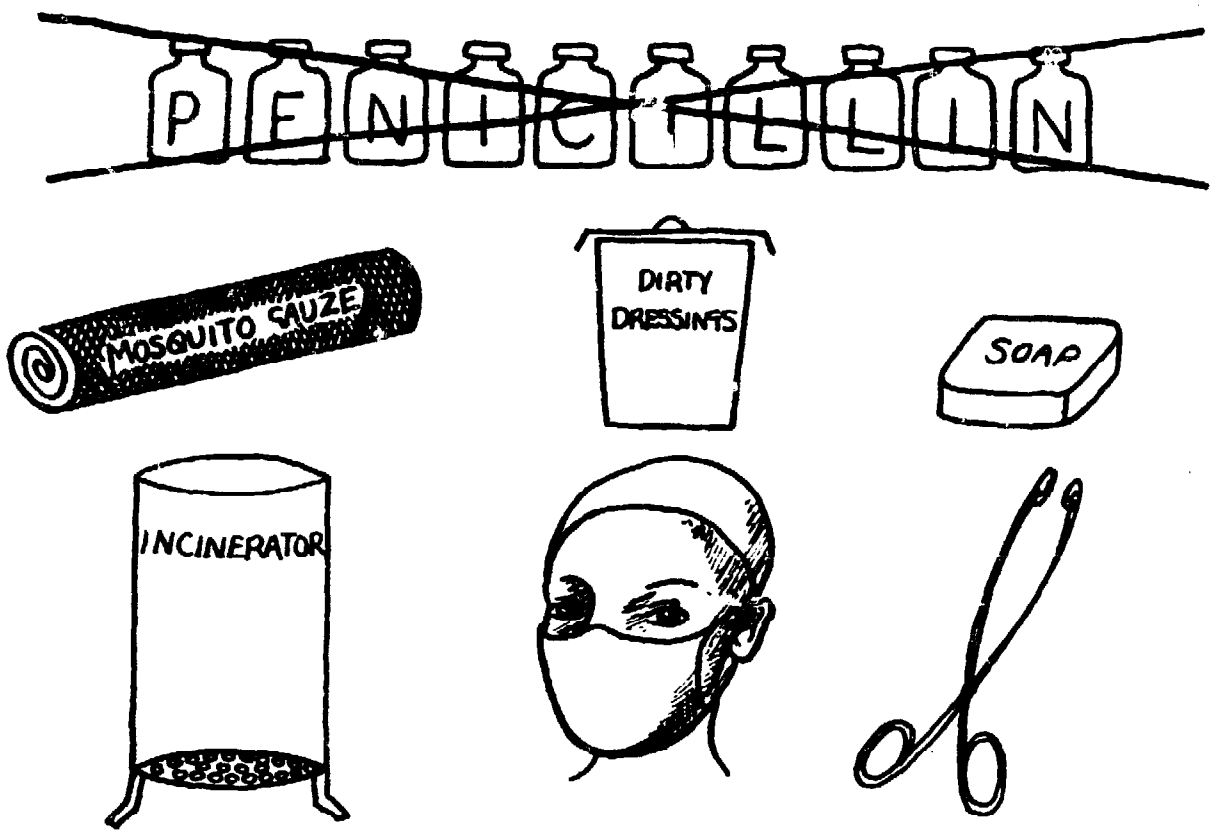
—Aseptic techniques should be taught to all nursing staff.

7. *Action*

See Streptococcal Diseases (p 237).

8. *Summary*

Staphylococcal infections are common. They are dangerous when they occur in internal organs. Sources of infection are patients with purulent wounds, or healthy carriers. Control consists of strict application of antiseptic measures.



Prevention is better than cure.

MENINGITIS

1. Meningitis is an inflammation of the pia and arachnoid membranes; it may be either acute or chronic, and sterile (as in subarachnoid haemorrhage) or infective.

2. *Occurrence and importance*

All forms of meningitis occur in Tanzania. Meningococcal meningitis can occur sporadically or in the form of epidemic outbreaks especially in crowded institutions such as barracks, camps, and prisons.

Predisposing factors in meningitis.

In the cold dry season, especially in the highlands, the temperatures drop to very low levels at night. This makes people crowd together in badly ventilated rooms. Their mucous membranes are also irritated by dust and by the smoke of firewood. Under such conditions the meningococci spread easily.

Other forms of meningitis always occur sporadically as they are usually complications of other diseases. All forms can obstruct normal CSF flow and so cause hydrocephalus, and can damage cranial nerves proximally by adhesions which can result in paralysis or loss of senses (deafness, blindness). If meningitis is properly treated, mortality should not exceed 5% but if treatment is delayed, mortality may be over 30% and commonly those who do survive have permanent damage.

Table of predisposing factors

Causative organism	Age group most at risk	Predisposing condition
Pneumococci Streptococci	Adults	Mastoiditis, otitis media, sinusitis, pneumonia, head injury, puerperium, pregnancy
H. influenzae	Children	Respiratory tract infection, otitis media, mastoiditis
Salmonellae	< 2 years	Diarrhoea or septicaemia
Meningococcus	Children, young adults	Overcrowding
M. tuberculosis	Children	Malnutrition
Virus	Children	Epidemics of mumps, measles, polio, chicken-pox, and other viruses

3. *Epidemiology*

A wide variety of organisms may cause meningitis. Meningitis should be differentiated from meningism or meningeal irritation as is seen for example in malaria, by doing a lumbar puncture. Chronic meningitis may be caused by Tb.

The acute forms of meningitis may be caused by several viruses and bacteria. Aseptic or viral meningitis differs from bacterial meningitis because it is usually a self-limiting disease or associated with other clinical entities such as mumps, poliomyelitis, measles and others.

Acute bacterial meningitis may be caused by (in order of frequency):

—pneumococci (Diplococcus pneumoniae)
—H. influenza (Haemophilus influenza)
—meningococci (Neisseria meningitides)
—E. coli (Escherichia coli)

–Salmonella
–staphylococci (Staphylococcus aureus)
–streptococci (Streptococcus pyogenus)

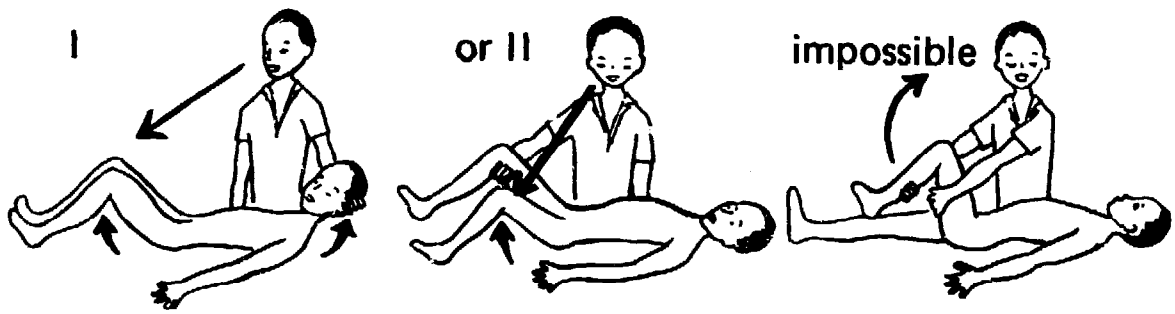
25% of healthy people may carry meningococci and the other organisms mentioned above; therefore healthy carriers are common.

Transmission occurs by direct contact and droplet spread of discharges from nose and throat of infected persons (mostly carriers).

4. *Clinical picture*

The onset is acute with headache, fever and often rigors. The headache becomes severe and spreads down the neck. There may be pain in the back and limbs. In children convulsions are common at the onset. The patient is irritable, becomes confused and drowsy, and later comatose.

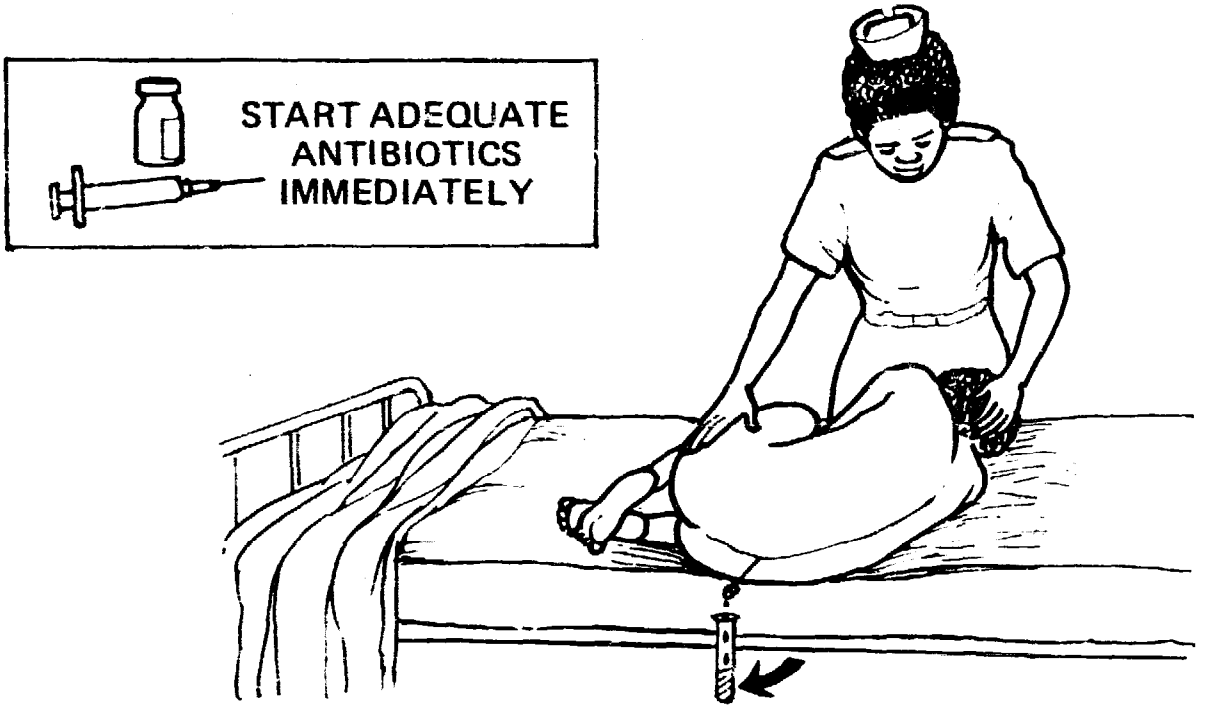
Signs of meningeal irritation are clear and present at an early stage: neck rigidity, Kernig's sign and Brudzinski's signs are positive (in babies the anterior fontanelle may bulge).



Brudzinski I | Brudzinski II | Kernig's sign

When signs of meningeal irritation are present a lumbar puncture must be done without delay to confirm the diagnosis and start appropriate therapy immediately (see next page).

WHEN TO DO AN LP
IF IN DOUBT, DO IT



If you think of LP do it.

In meningitis the CSF is under pressure. When it is an aseptic (viral) meningitis the fluid is most often clear, though it may be turbid due to the presence of many lymphocytes (protein is then slightly increased and sugar is normal). Differentiation between such an aseptic meningitis and tuberculous meningitis may be extremely difficult (history). In bacterial meningitis the CSF is always turbid and contains hundreds of polymorph leucocytes (the protein content is greatly raised and glucose content markedly lowered).

Disease	CSF	Cells	Protein	Glucose
Acute bacterial	Always turbid	Increased polymorphs	Raised	Low
Tb	Opalescent or clear	Lymphocytes increased	Raised	Low
Aseptic or viral	Opalescent or clear	Lymphocytes increased	Raised	Normal
Meningism	Clear	Normal	Normal	Normal

5. *Management of the individual patient*

The following situations may occur:

(a) LP not possible, patient can reach a hospital within 3 hours therefore make a blood slide, give chloroquine, and refer the patient for LP.

(b) LP not possible, hospital cannot be reached within 3 hours therefore examine blood slide, give chloroquine, put the patient on chloramphenicol and crystalline penicillin, and refer to hospital.

(c) LP possible therefore do LP without delay, examine blood slide. Put patient on chloramphenicol and crystalline penicillin after doing LP, wait for results of LP and continue treatment according to results (see table), then refer the patient.

Organism	Stained form	Arrangement	Drug of choice	Price/day
Pneumococci	gram +ve cocci	diplococci	crystalline penicillin 6 mU stat 3 mU 6-hourly	12/-
H. influenza	gram -ve rods	different shapes	chloramphenicol 1 g 6-hourly	1/20
Meningococci	gram -ve cocci	diplococci	6 mU stat 3 mU 6-hourly	12/-
E. coli	gram -ve rods		chloramphenicol	
Salmonellae	gram -ve rods		chloramphenicol 1 g 6-hourly	1/20
Staphylococci	gram +ve cocci	clusters	chloramphenicol	
Streptococci	gram +ve cocci	chains	crystalline penicillin 6 mU stat 3 mU 6-hourly	12/-

REFER ALL CASES OF PURULENT MENINGITIS TO HOSPITAL

Additional drugs to be given are: Aspirin to reduce fever, phenobarbitone to prevent convulsions. Rehydration is also very important, feed by gastric tube if necessary.

To prevent complications like hydrocephalus and death, it is essential that antibiotic treatment is started *early* and is given in proper doses. Thus the most important task for the health centre is to start adequate treatment before referral to hospital. This will have a profound effect on the prognosis of all cases of meningitis.

TREAT PURULENT MENINGITIS FOR AT LEAST TWO WEEKS

6. *Control*

When an outbreak of meningococcal meningitis occurs, emphasis must be placed on careful surveillance, aiming at early diagnosis and immediate treatment of suspected cases. Health education on the dangers of sleeping in crowded badly ventilated houses will help to reduce transmission.

It has been shown that when overcrowding is reduced and people sleep at least 3–5 ft apart, both the infection rate and the number of carriers fall.

Since there are many sulfonamide-resistant strains of meningococci, the use of sulfa 1 g bd for 2 days as a chemoprophylactic has become less effective. Alternative: single dose PPF 0.6 mU. Chemoprophylaxis is only indicated for household members.

7. *Action*

—Order lumbar puncture needles for your health centre (Central Medical Store, Class 3, Item 60, 5/- each).

—Do LP in all cases of neck stiffness.

—Suspect meningitis in children who are drowsy or who have a fit.

—Start immediate treatment with crystalline penicillin and chloramphenicol when the CSF is cloudy.

—Refer all cases of purulent meningitis to hospital.

—Report meningococcal meningitis immediately to the DMO and discuss what other investigations or measures, such as surveillance, should be undertaken.

—Give penicillin prophylaxis to household contacts in cases of meningococcal meningitis.

—Stress the dangers of overcrowding.

—Stress the importance of early reporting and treatment.

8. *Summary*

Meningitis occurs all over Tanzania in sporadic and epidemic forms. The disease is spread by droplet infection, the reservoir is the asymptomatic carrier. Treatment should start early with very high doses of penicillin and chloramphenicol. Report to DMO. Correct overcrowding.

MEASLES

1. Measles is an acute general infection caused by a virus. The clinical features are the results of the reactions of the defence mechanisms of the body against the virus.

The disease is very infectious. In general it is a disease affecting children. It has been dealt with in the *Child Health* manual Chapter 11.1.

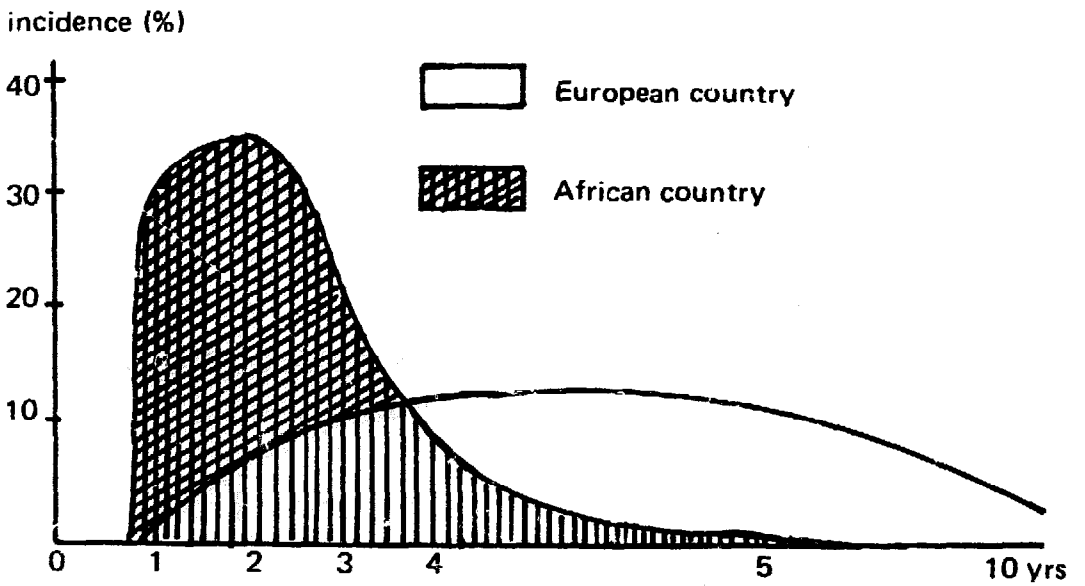
Synonyms: Morbilli, rubeola*, *surua*.

2. *Occurrence and importance*

Measles is found all over Tanzania. The high fatality rate of

*Rubella is German measles.

measles in Tanzania is caused by malnutrition and undernutrition. In malnourished children the defence mechanisms are slow and weak, so the virus has more opportunity to do damage before it is arrested. Children are most likely to suffer from undernutrition in the weaning period. This is also the time when African children get measles. For children in good nutritional condition measles is a mild disease.



Comparison of age distribution in a European and an African country. In Africa most children develop measles before the age of 3 years. In Europe many children escape measles until after the age of 5.

The overall fatality rate of measles in Tanzania is at least 5%. That is about 400 times higher than the fatality rate of measles in Europe, and means that in total at least 20,000 children die in Tanzania each year of measles.

3. *Epidemiology*

Measles is spread by small invisible droplets containing virus particles. The virus particles come from the secretions of the respiratory tract of patients suffering from measles. The disease spreads very easily. Before it becomes clear that a child is suffering from measles, the disease has already spread to its contacts.

The disease gives a life-long immunity. Nearly everybody

gets the disease once in his life, if he is not vaccinated.

ONE OUT OF EVERY 20 CHILDREN DIES FROM MEASLES
PREVENT MEASLES BY VACCINATION

4. *Clinical picture*	5. *Management of the individual patient*
Uncomplicated measles; Well nourished child or slightly underweight	Treat as an outpatient
Fever Conjunctivitis Rhinitis Cough Koplik's spots (or stomatitis) Skin rash	Ensure proper fluid intake and good food, tepid sponging, Aspirin, daily follow-up, single dose Vitamin A 200,000 u orally

WEIGH ALL CHILDREN WITH MEASLES

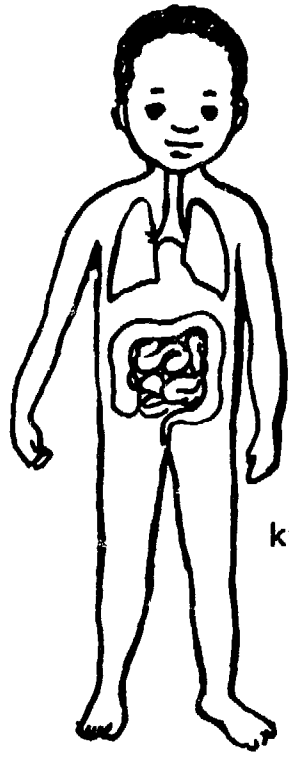
Complicated measles; child very underweight or with other signs of malnutrition.	Admit; give balanced diet with protein and energy-rich foods.
Dyspnoea, nasal flaring	Pneumonia (see p 231)
Hoarseness, barking cough, inspiratory stridor	Laryngo-tracheo-bronchitis (see p 229)
Sore mouth inability to suck	Stomatitis (clean mouth 4–6-hourly)

(continued on next page)

clinical picture (ctd)	*Management (ctd)*
Diarrhoea, vomiting	Gastroenteritis (see p 128) or *Child Health* manual, Chapter 12)
Vomiting, convulsions	Exclude malaria, meningitis give phenobarbitone
Dryness of eyes, photophobia, hazy cornea	Xerophthalmia, give Vitamin A, 200,000 units/day orally, and chloramphenicol eye ointment, atropine eye ointment.
Red eardrum or discharge from ear	Otitis media, treat with PPF
Persistent pneumonia	Suspect Tb (see Chapter 8)

NO ROUTINE ANTIBIOTICS

UNDER-NUTRITION CAUSES SEVERE MEASLES: SEVERE MEASLES CAUSES KWASHIORKOR



6. *Control*

The only successful way of preventing serious side effects of measles is by vaccinating all children in Tanzania. Measles vaccine is not cheap but the damage done by the disease justifies the expense of an overall vaccination coverage.

Measles is infectious before it is evident that the child is suffering from the disease. During the incubation period a child will have a high fever. This is a frequent reason for seeking medical attention. Many children are admitted during the prodromal stage. The result is that under-fives clinics and children's wards are centres of measles infections. Most children attending clinics or admitted to hospitals are going to be infected with the measles virus.

The incubation time of the weakened measles virus of the measles vaccine is 48 hours less than the incubation time of the strong measles virus. Measles infection taking place at under-fives clinics and children's wards can be prevented by vaccinating *all* children attending under-fives clinics and *all* children admitted to hospitals without delay.

In the prevailing circumstances it is safer to give children the weakened vaccine than to expose them to the strong measles virus spread by children already incubating the disease. So there are no contra-indications to measles vaccination.

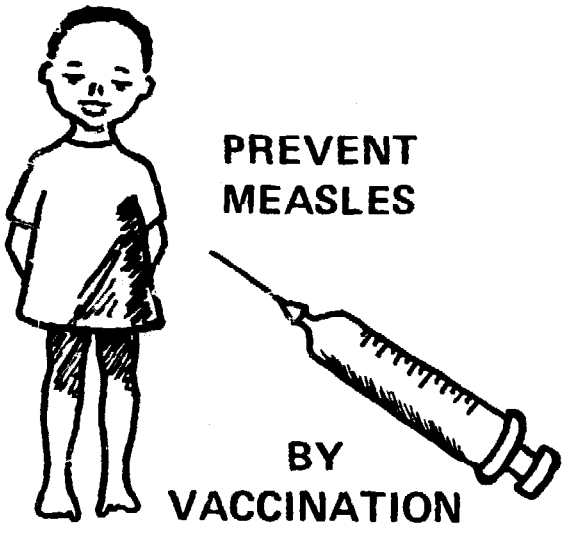
GIVE MEASLES VACCINATION TO ALL CHILDREN BOTH THE HEALTHY AND THE SICK

The vaccine is made from live attenuated virus. It is difficult to preserve. Exposure to heat or light, or contact with antiseptics may destroy it.

Vaccination can be given to children when the passive immunity inherited from the mother has worn off—that is from the age of six months onwards.

If the vaccination is given between the ages of 6 and 9 months, it should be repeated at the age of 1 year in order to

be sure that the protection is strong.



MEASLES VACCINATION IS AN ESSENTIAL PART OF THE MCH CLINIC

Another task of the MCH clinic is to prevent and treat undernutrition. This is done through nutrition education to mothers and by weighing the children and plotting the weights on the growth card. When this is efficiently done mortality of measles will go down because of better nutrition.

REDUCE MEASLES MORTALITY BY BETTER NUTRITION

7. *Action*

—Arrange with your DMO to get a regular supply of measles vaccine.

—If your health centre has a refrigerator be sure it is functioning properly.

—If your health centre does not have a refrigerator make arrangements with an institution or individual with a refrigerator to store your vaccine there. (Even if the health centre has a refrigerator it might be advisable to make prior arrangements in case your refrigerator breaks down.)

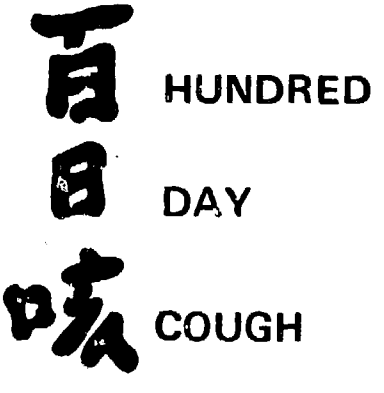
—Make a small cold box. This is a wooden box lined with polystyrene. Polystyrene has an excellent insulating capacity. It is commonly used for packing breakable goods (radios etc.). Arrange with the storekeeper to save any left-over polystyrene

(see *Community Health* manual for how to make a cold box). This box is useful for transporting vaccines.

- –Immunize all children over 6 months who are admitted to the children's ward.
- –Immunize all infants who come to the health centre with other complaints. Explain to the mothers the importance of vaccination and the possible side effects.
- –Routinely immunize all eligible children, sick or healthy, coming to the MCH clinic. Discuss indications and contra-indications with the staff.
- –Ensure that your MCH clinic weighs all children under 3 years and that the weights are plotted on each child's growth chart.
- –Teach your staff to take action when a child is underweight.

8. *Summary*
Measles is an important cause of under-five mortality in Tanzania. It can be prevented by vaccination. For details see *Child Health* manual, Chapters 11.1 and 6.9.

WHOOPING COUGH



1. Whooping cough is caused by bacteria. The bacteria damage the cells of the respiratory tract. The damage produces a clear, very sticky mucus which blocks the lumen of the bronchioli. Children suffering from whooping cough try to remove the mucus by coughing. The cough is present in typical attacks which end in the characteristic whoop.

The cough may last for many weeks. The Chinese name for

whooping cough is 'hundred-day cough'.

Synonyms: Pertussis, *kifaduro*.

2. *Occurrence and importance*

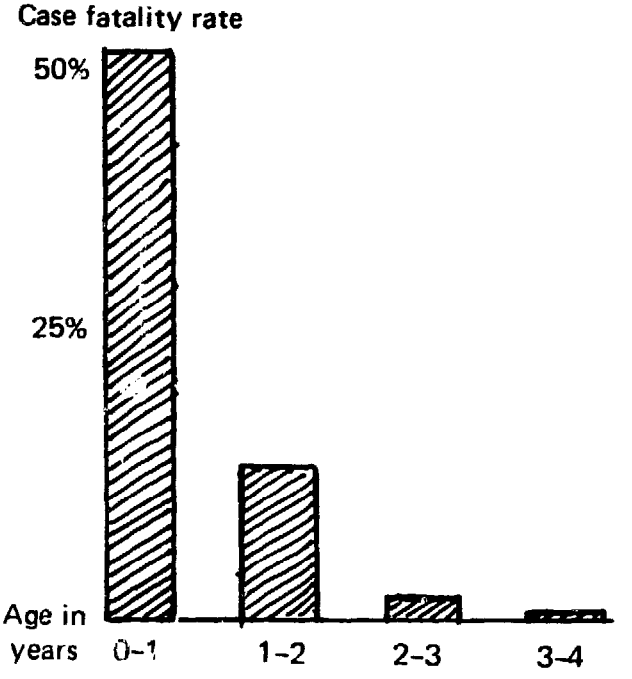
Whooping cough is a very common disease of children in Tanzania. The case fatality rate is highest in children under 6 months. Newborns get little or no protective antibodies from their mothers. When they are exposed, they may develop an atypical illness which is seldom recognized as whooping cough.

Early infection: whooping cough.

The Tanzanian child will be exposed to whooping cough early in his life. He goes on his mother's back when she visits the market, and visits other families.

Due to the 'extended family' system, he will always be in contact with other children.

Because of early exposure, the case fatality rate of whooping cough is high in Tanzania.



Distribution of deaths in each age group.

The persistent cough and accompanying sticky mucus may prevent the child from sucking well. The supply of breast milk may then be reduced due to incomplete emptying of the breast. Whooping cough can lead to malnutrition. The complications of whooping cough are serious. These facts together make whooping cough an important disease.

3. *Epidemiology*

Whooping cough is spread by droplets. The droplets come from secretions of the upper respiratory tract. The disease is already infectious before the typical attacks appear.

4. *Clinical picture*

The incubation time is short (about 6–12 days). The clinical picture of whooping cough in children under 3 months old is quite different from that in older children.

Clinical picture	Children under 3 months old	Children over 3 months old	Remarks
Catarrhal period duration 14 days	–little cough –nasal discharge	–cough –nasal discharge	like the common cold
Paroxysmal period duration about 6 weeks	–attacks of breathing arrest –cyanosis	–typical attacks of cough with whooping followed by vomiting –sticky secretion –subconjunctival haemorrhages due to the violence of the cough.	in older children diagnosis is made from the typical whoop. In young children diagnosis is frequently missed.

YOUNG BABIES DON'T WHOOP

Slowly the cough may diminish or, as stated before, it may

last a very long time. After improvement the disease may recur, 'recrudescence'.

Complications are: encephalitis, pneumonia, and otitis media.

Diagnosis: WBC over 30,000 with marked lymphocytosis.

LYMPHOCYTOSIS SUGGESTS WHOOPING COUGH

5. *Management of the individual patient*

Although the cough is distressing for the child and the mother, do not give cough suppressants to the child. Over-treatment is very likely to occur and if cough is suppressed the sticky mucus will stay behind in the bronchi. Proper ventilation is diminished and secondary infection can get a hold. Cough suppressants are only indicated when the child gets too weak to drink and eat and cannot sleep. The suppressant can be given for a night to give some rest.

Sedatives for a very worried mother are sometimes a very effective treatment for the child.

Chloramphenicol and tetracycline are only effective when given in the first week of the disease. It is most important that the child gets enough fluid. Without adequate fluid he will become dehydrated; dehydration also follows feeding difficulties and vomiting. The mother should be encouraged to breast-feed the child immediately after an attack as there will then be a quiet period during which the child will not cough or vomit.

Isolate known cases of whooping cough.

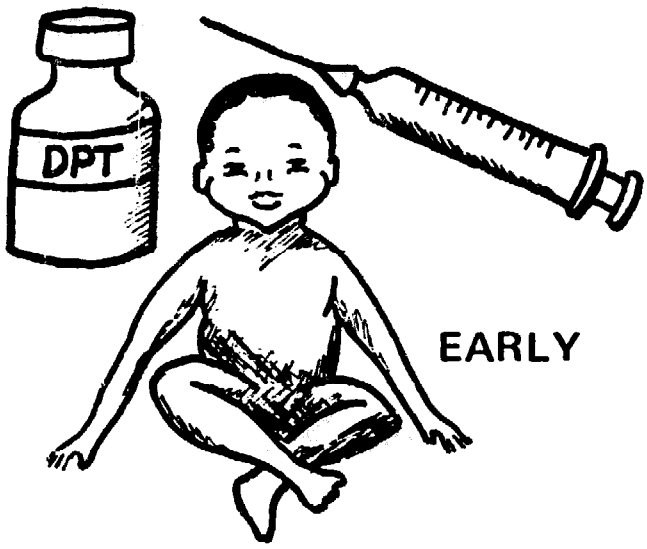
6. *Control*

The only way to control and eradicate whooping cough is by mass vaccination with triple vaccine.

Start vaccination against whooping cough early, preferably at the age of one month. This is suggested for the following two reasons:

(i) Very little or no passive immunity is inherited from the mother.

(ii) It is in the first three months of life that whooping cough has a very high mortality.



Whooping cough vaccine is part of triple DPT vaccine. It is made of dead bacilli so the immune response is not as effective as after vaccination with a live attenuated virus, therefore, give DPT three times.

Infants or young children who are contacts of known pertussis cases may be given chloramphenicol as chemo-prophylaxis together with vaccination.

PREVENT WHOOPING COUGH BY DPT VACCINATION

7. *Action*

—Make arrangements to get a regular supply of DPT.

—Start your DPT vaccinations at the age of one month as a routine.

—Compare the number of vaccinations monthly with the number of children attending the OPD for treatment. Make it a routine to check the vaccination status of every child you see. Advise mothers to bring their children for vaccination.

—Give contacts chloramphenicol together with DPT vaccination.

—Check the storage conditions of your vaccines. Make a habit of checking every day that the refrigerator is functioning properly (e.g. freezing in the ice tray compartment). Defrost the refrigerator weekly.

8. *Summary*

Whooping cough is an important airborne disease. Mortality is highest in very young children. The clinical picture in young children is quite different from that in older ones. Treatment is difficult. The most important control measure is DPT vaccination.

Comparison of chickenpox and smallpox.

Chickenpox	***Smallpox***
1. Chickenpox is a mild viral disease, characterized by fever followed by a typical skin rash. Synonyms: Varicella, *tetekwanga.*	1. Smallpox is a dangerous viral disease, characterized by sudden onset of high fever, general malaise, and prostration 2–4 days after the onset of the prodromal symptoms a skin eruption appears. Synonyms: Variola, *ndui.*
2. *Occurrence and importance* Chickenpox is a very common children's disease. It can occur in very young babies. It occurs all over Tanzania. It is usually a very mild disease. Case fatality rate is below 1%.	2. *Occurrence and importance* Smallpox used to be a continuous health hazard in Tanzania. From time to time epidemics occurred. In 1968–69 a big smallpox campaign was organized. Millions of people were vaccinated. As a result of this no smallpox has been reported in Tanzania since 1970. It continued in some other countries including Ethiopia, Somalia and Kenya until 1977 since when no cases have been reported. It is therefore hoped that the disease has been eradicated from the world. Smallpox is a killing disease. Case fatality rate is about 40%.

Chickenpox

3. *Epidemiology*

Chickenpox is caused by varicella-zoster virus. The virus is spread by droplet infections containing virus particles from the respiratory tract. The scabs from the skin are not infectious. The disease leaves immunity against chickenpox, but the virus stays within the body and may reappear as herpes zoster when the immunity of the person is weakened, e.g. in leukaemia, diabetes, or old age.

4. *Clinical picture*

Incubation time 13–21 days.

Prodrome: minor fever.

Clinical phase: mucosal lesions in mouth and throat.

Skin:

A maculopapular rash which become vesicles within a few hours. Vesicles are oval and superficial, after 3–4 days pustules, which collapse and dry leaving no scar.

Distribution:

Mostly on the trunk; armpits involved; seldom on palms and soles.

Smallpox

3. *Epidemiology*

Smallpox is caused by the variola virus. The disease is spread by droplet infections containing virus particles from the respiratory tract of patients. Skin-to-skin contact and contact with clothing worn by patients may also lead to infection. The disease is highly infectious. In overcrowded areas with lack of basic hygiene, explosive outbreaks of the disease may occur. Scabs contain virus and are infectious. The disease leaves a long-lasting immunity.

4. *Clinical picture*

Incubation time 8–17 days.

Prodrome: headache, high fever, severe toxaemia.

Clinical phase: mucosal lesions in mouth and throat.

Skin:

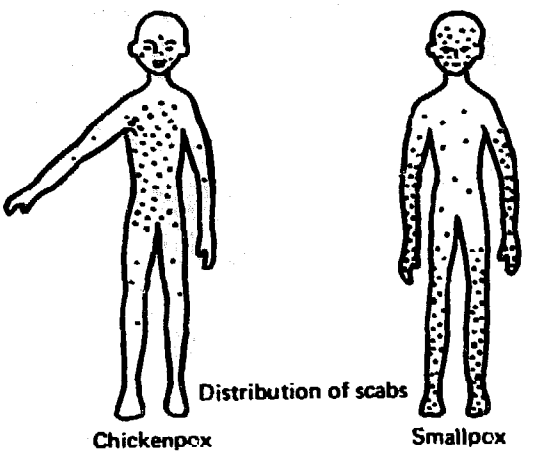
First macules (flat), after twenty-four hours papules (raised), 3 days later deep, round vesicles. Coalescence of pustules, oval as well as round shapes are suspect for smallpox. Secondary infection of vesicles leads to pustules. When the scabs fall off deep scars are left.

Distribution:

Mostly on extremities and face; armpits usually free; often on palms and soles.

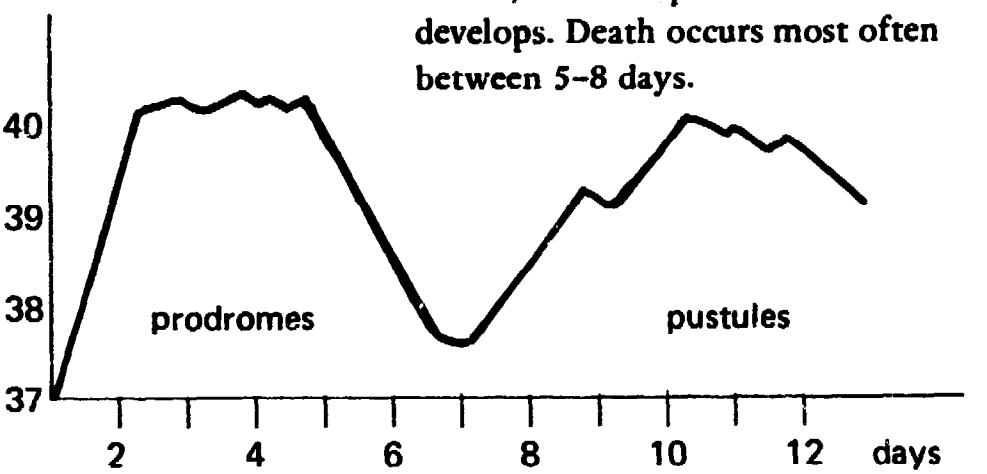
(Diagrams on next page)

Chickenpox ***Smallpox***



Course:
Groups of new pocks will appear over many days, therefore, pocks at different stages of development can be found.

Course:
All pocks are in the same stage of development. Pocks appear only on the first two days of the disease. After the prodromal stage the fever comes down; when the vesicles become secondarily infected, a second period of fever develops. Death occurs most often between 5–8 days.

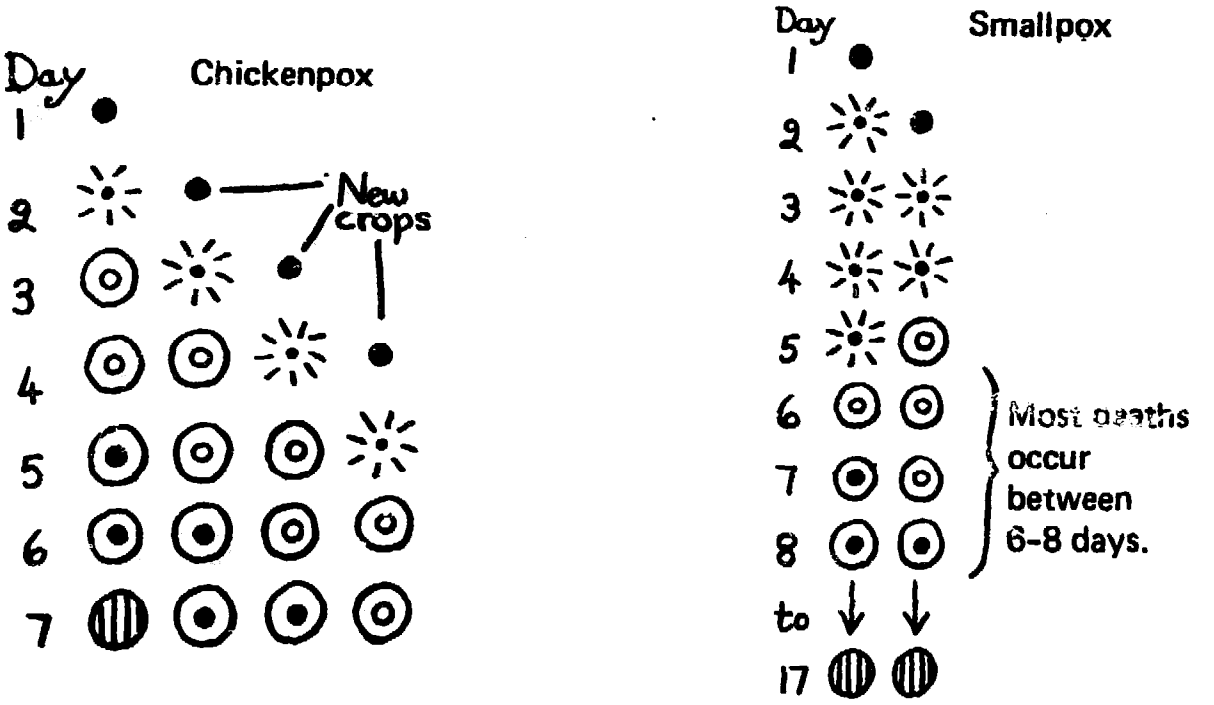


Fever pattern of smallpox.

Complications:
Unusual
Secondary infection

Complications:
Bronchopneumonia
Otitis media
Corneal ulceration → blindness
Encephalitis.

Development of pocks from day to day, showing the difference between chickenpox and smallpox.



Key.

Macule	●	Non-elevated discolouration of the skin, less than 1 cm in diameter.
Papule	✳	Small, solid, clearly defined elevation of the skin, less than 1 cm in diameter.
Vesicle	◎	Small collection of fluid superficially in the dermis, less than 1 cm in diameter.
Pustule	◉	Vesicle containing pus.
Crust	▥	Or scab, irregular masses of dried exudate, usually mixed with bacterial debris and epithelial cells.

Chickenpox

5. *Management of individual patient*
Symptomatic—calamine lotion may relieve itching. In case of doubt about diagnosis, isolate until you are sure.

Smallpox

5. *Management of individual patient*
Isolate patients until all scabs have fallen off. The phase of secondary infection may be shortened by antibiotics. Daily cleaning of eyes.

Chickenpox

6. *Control*

Since chickenpox is already infectious in the incubation period isolation will not help in controlling the disease.

7. *Action*

Not relevant.

Smallpox

6. *Control*

—In case you find a patient suspected of smallpox, isolate him in your dispensary. Notify your DMO immediately. Do not refer the patient. This could spread the disease.

—Trace the place of origin of the patient. Find out if more cases are there with a similar pattern.

—To avoid the possibility of smallpox reappearing in Tanzania, vaccinate as much as possible. (For details see the chapter on immunization in the *Community Health* manual.)

7. *Action*

What can be done to maintain the eradication of smallpox?

What you can do at the health centre:

—Find out the estimated number of births in the catchment area of your health centre

$$\frac{\text{birth rate x number of inhabitants}}{1000}$$

and compare this with the number of smallpox vaccinations given annually in your health centre. Now you have an idea of how many children are not yet vaccinated. To interrupt the transmission of smallpox at least 80% of the population should be vaccinated.

—Order the WHO bifurcated

Chickenpox

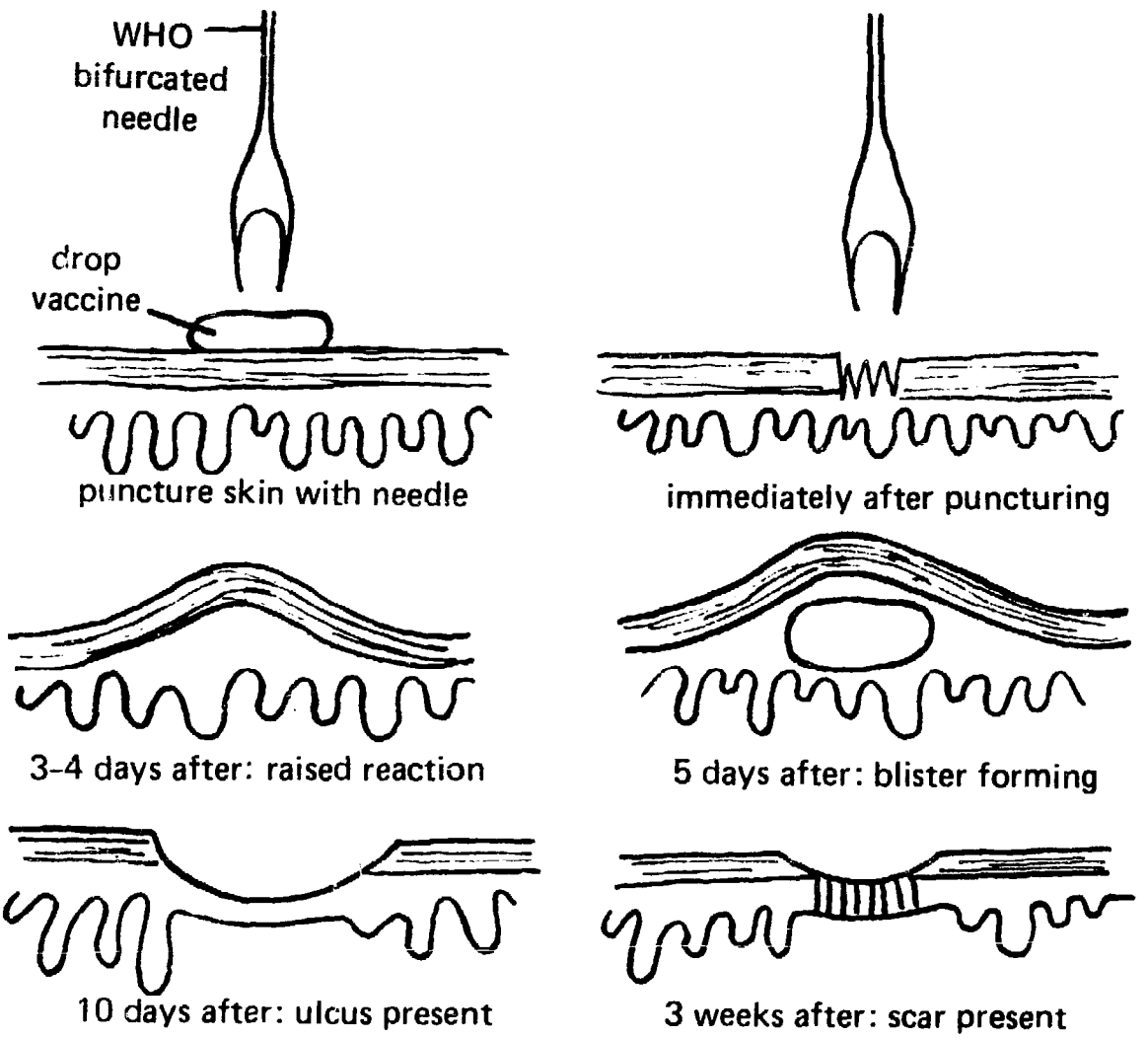
Smallpox

needles advised for smallpox vaccinations from the district health officer.

—Check your stock of smallpox vaccine. Is it enough for the vaccination needs of your health centre?

—If the attendance at your MCH clinic is very low, give smallpox and BCG vaccination to all children born in your health centre.

—Arrange with the headmaster a suitable day for (re-) vaccinating all children.



Chickenpox

Clinical variant of chickenpox: Herpes zoster.

Synonym: Shingles.

Herpes zoster is a vesicular eruption of the skin confined to the area served by one nerve. The vesicles dry up into a crust in a week or so. The condition may be very painful. Herpes zoster is caused by the same virus as chickenpox, and may break out many years after the original infection for reasons which are not known.

In general herpes zoster occurs only in old age or when immunity is impaired, e.g. after measles or in Hodgkin's disease or other reticuloses. The prognosis is good. There is no specific treatment. To prevent secondary infection the skin should be kept clean and dry. Treatment of pain is very difficult. Do not use narcotics (for fear of addiction!).

Smallpox

Clinical variant of smallpox: Alastrim.

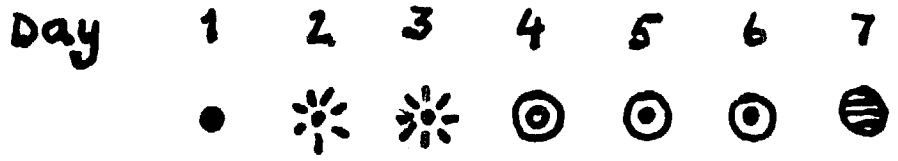
Synonym: Variola minor.

This is caused by another virus, but similar to the common smallpox virus. The disease is less dangerous, case fatality rate never exceeds 5%. The development of the pocks is more rapid. Within one week all pustules are dry. This form of smallpox occurs in Ethiopia, Kenya, and Somalia.

Smallpox in vaccinated persons: when the immunity of a person is declining he may develop a weakened form of smallpox.

Two forms exist: in one form the lesions do not develop further than the papular stage. In the second form the lesions look like chickenpox, but still have the distribution of smallpox. This makes the differentiation possible. Both forms are seen mostly when a smallpox epidemic is occurring.

Alastrim (variola minor)



Development of pocks.

Chickenpox

8. *Summary*

Chickenpox is a common infection of children. It is a mild disease. Its importance lies in differentiating it from smallpox.

Herpes zoster is a late manifestation of infection with varicella virus.

Smallpox

8. *Summary*

Smallpox is a dangerous disease. Due to country-wide vaccination campaign which was part of the WHO eradication campaign, the disease has not been seen in Tanzania since 1970. Surveillance is indicated. To avoid recurrence great emphasis should be placed on continued vaccinations of all newborns.

Table: Comparing chickenpox and smallpox

	Chickenpox	Smallpox
Occurrence in Tanzania	very common	None since 1970
Case fatality rate	very low	high (40–50%)
Prodromal symptoms	none	severe
Distribution	trunk	extremities, face
Fever	rises as rash appears	settles as rash appears
Pocks	all stages of development present	all in same stage of development
Prevention by vaccination	no	yes
Immediate notification necessary	no	yes
Isolation essential	no	yes

RUBELLA

1. Rubella is a mild febrile disease with a 3-day rash resembling the rash of measles and difficult to see on a dark skin. Often there is enlargement of the lymph nodes, typically post-auricular, sub-occipital, or posterior cervical.

   Synonym: German measles.

2. *Occurrence and importance*

   Primarily a childhood disease. For children it is a harmless disease. Important only when infection occurs in females during the first few months of pregnancy. Then it can result in extensive fetal infection with congenital defects in the infant: congenital rubella syndrome.

   In East Africa this congenital syndrome is not often diagnosed, probably because most people contract the disease during childhood and develop life-long immunity. When people live further apart and living standards go up infection during childhood will be reduced and congenital rubella syndrome could become a problem.

3. *Epidemiology*

   Transmission is by droplet infection.

4. *Clinical picture*

   Rubella is a very mild disease and is often not recognized. It is easily confused with measles (history of second measles infection). Congenital rubella may result in one or more of the following defects:

   Eyes: Cataract or microphthalmia (too-small eye).
   Ears: Partial or complete deafness.
   Heart: Patent ductus and other abnormalities.
   CNS: Microcephaly (small head); mental retardation.
   Hepatosplenomegaly with jaundice.
   Thrombocytopenia with purpura.

RUBELLA IS MILD AND HARMLESS EXCEPT IN PREGNANCY

Differential diagnosis	Rubella	Measles
Koplik's spots	No	Yes
Suboccipital lymph nodes	Yes	No
Cough and conjunctivitis	Rare	Usual
Fever	Minimal	High

5. *Management of the individual patient*

No treatment is available or necessary for patients suffering from rubella. Pregnant women should be kept away from known cases of rubella.

KEEP PREGNANT WOMEN AWAY FROM RUBELLA

6. *Control*

Prevention not necessary as long as congenital rubella is not a problem. Avoidance of infection is hazardous because it may result in infection during pregnancy; the reverse—artificial exposure of girls during childhood—is advisable and has been practised. Immunization is possible with a live attenuated vaccine but is of little value in Tanzania where most children acquire natural immunity before puberty.

It is better to encourage people to expose themselves to the natural disease in childhood. This will result in natural active immunity and will prevent rubella syndrome later. Since the disease is mild in itself, natural immunity is preferable to artificial immunization.

When a pregnant mother has been exposed to a known case of rubella, passive immunization with human gammaglobulin is

possible. It is not always successful.

7. *Action*
Bring young girls into contact with a rubella patient.

8. *Summary*
Rubella is a viral infection, harmless except in pregnancy.

MUMPS

1. Mumps is an acute viral disease characterized by fever, and swelling and tenderness of at first one of the salivary glands, usually the parotid, sometimes the sublingual or submaxillary.

2. *Occurrence and importance*
Death from mumps is extremely rare but can occur as the result of a complicating encephalitis. Mumps is a relatively trivial illness in young children but if contracted after puberty it can have serious complications.

These are: orchitis in 20–35% of the males; oophoritis in about 5% of females.

Sterility may follow bilateral orchitis but this is rare.

3. *Epidemiology*
Transmission is by droplet spread and direct contact with saliva of an infected person, or indirect through freshly infected articles.

4. *Clinical picture*
Uncomplicated: fever, difficulty in swallowing, swelling and tenderness of salivary gland, at first unilateral, later both sides are affected. There is no rash and the fever and swelling subside in a few days. The complications are:
Orchitis: Swelling and severe pain, usually one-sided.
Oophoritis: Severe lower abdominal pain, vomiting.
Prostatitis: Unexplained fever, dysuria.

Mastitis: Pain and swelling (can occur in both sexes).
Pancreatitis: Severe upper abdominal pain, vomiting, and fever.
Meningitis: Common. Headache, fever, vomiting, neck rigidity; prognosis is excellent, subsides in a few days.
Encephalitis: Rare. Severe headache, fever, vomiting, cranial nerve palsies, drowsiness, coma, mortality about 50%.

Diagnosis: Mumps must be differentiated from:

—Cervical lymphadenitis, in which the ear lobe is not lifted upwards and outwards (exclude Tb).

—Kwashiorkor may cause swelling of parotid glands but no tenderness and fever.

—Burkitt's tumour: swelling is unilateral, firm, fixed to the jaw.

5. *Management of individual patient*

No specific treatment is available, only bed rest and symptomatic treatment. Ensure proper fluid intake.

Patients with orchitis can be given analgesics, a scrotal support and bed rest. The sterility due to mumps orchitis is caused by the oedema of the testes. The testes are surrounded by the tense fibrous tunica vaginalis. This does not give way, so oedema will result in diminished blood supply followed by atrophy of the testes.

If the testes are tense and very tender on palpation, refer to hospital for surgical decompression by slitting the tunica.

6. *Control*

Because mumps is such a minor disease during childhood, there are undoubtedly advantages in getting over it before puberty, particularly as immunity is lifelong. There is a live attenuated vaccine available but protection lasts only for about 4 years. It is not in use in Tanzania because there is very little priority for prevention of such a mild childhood disease.

7. *Action*

Not relevant.

8. *Summary*

Mumps is a mild childhood disease. Specific treatment is not available. Immunization is not a priority in Tanzania.

Chapter eight

TUBERCULOSIS AND LEPROSY

Introduction

Tb and leprosy are dealt with in this separate chapter because both diseases have much in common and both differ in the same way from other communicable diseases.

1. *Mycobacteria*

Both tb and leprosy are chronic diseases caused by mycobacteria, Mycobacterium tuberculosis and Mycobacterium leprae respectively.

Mycobacteria are capsulated. The capsule takes up carbol fuchsin easily but mycobacteria in contrast to all other bacteria and cells cannot be decolorized even by a combination of acid and alcohol. Mycobacteria are therefore called Acid/Alcohol-Fast Bacilli or AAFB (or more commonly Acid-Fast Bacilli or AFB). Mycobacteria divide very slowly. This accounts for the long incubation time of tuberculosis and leprosy. Mycobacteria have two phases:

—active, metabolizing and multiplying

—inactive (dormant), not metabolizing and not multiplying.

When mycobacteria are inactive they do not metabolize and so will not take up toxic products which might kill them; in other words antibiotics will have little effect on them in this phase. Inactive bacteria can become active later. Therefore antibiotic treatment in leprosy and tb has to be continued for a long time.

2. *Common*

Tb and leprosy are both very common infections, but most people infected with tb or leprosy do not actually suffer from the disease; they are able to overcome contact with tb and leprosy bacilli without ill effects. In only a small number of people are the bacilli able to settle or to multiply and cause disease. In other words both diseases are caused by a failure of the host to deal adequately with the infection. So there is a difference between infection and disease.

3. *Long-term treatment*

Long-term treatment is difficult for most people to keep up. Very often patients feel better after a few months of treatment. If treatment is discontinued or taken irregularly resistance against the commonly used drugs may develop. Then the disease process will continue and the patient will again become infectious.

4. *Control*

Control of leprosy and tb is based on the principle of treating as many infectious cases as possible. Only when 75% of all cases take their drugs regularly is transmission interrupted in such a way that the incidence will go down.

Therefore the control of tb and leprosy is based on:

—case finding, that is diagnosing as many cases as possible

—case holding, that is ensuring that every diagnosed case gets and takes his treatment as long as indicated.

5. *Health centre*

Case finding and case holding of leprosy patients have been the task of several projects dealing with leprosy only.

In some regions schemes to control leprosy or tb have been in operation. It is now widely recognized that schemes designed to combat a single disease are expensive and the impact they have had on the incidence is far less than expected.

It is also realized that if we want to diagnose and treat as

many cases as possible, facilities for diagnosis and treatment must be available as close to the homes of the patients as possible—that is in every health centre and every dispensary.

This can only be achieved when all health workers in the rural health units:

—understand the problems related to leprosy and tb control
—have experience in diagnosing and treating tb and leprosy
—realize that they are the front-line workers in the battle against leprosy and tb.

6. *National policy*

The above facts are recognized by the Ministry of Health. The proposed national tuberculosis prevention scheme will be co-ordinated with the present leprosy control scheme under one director, and will be integrated into the basic health services. Emphasis will be put on:

—training personnel and making every medical worker aware of treatment methods
—the use of the microscope as the main means of diagnosis. It is planned that each health centre and each dispensary with an RMA will have a microscope
—involvement of local leaders in active case finding and case holding
—principal method of treatment will be as out-patients
—district co-ordinators will be RMAs or health officers.

7. *Summary*

(i) Tb and leprosy are chronic diseases caused by mycobacteria.

(ii) Tb and leprosy infections are very common. Both diseases are related to failure of host resistance.

(iii) Only long-term treatment can cure tb and leprosy.

(iv) Tb and leprosy control is based on case finding and case holding.

(v) Case finding and case holding is only successful if health

workers are motivated, co-operation from the public is achieved, and facilities are present in all rural health institutions.

(vi) At the national level tb control and leprosy control are to be co-ordinated under one director. Emphasis will be on training and involvement of local leaders. Treatment should be as out-patients with close follow-up.

TUBERCULOSIS

1. Tuberculosis is a general systemic disease. In most cases the lungs are involved. The disease is caused by a failing resistance of the person to tuberculosis.

   Synonyms: Tb, *kifua kikuu.*

2. *Occurrence and importance*

   Tuberculosis is very common in Tanzania. Its prevalence is estimated at about 2/1000 or 0.2% of the total population. Thus in Tanzania there are 30,000 people with tb. It is found throughout the country.

   A health centre is supposed to serve about 50,000 people. This means that each health centre *should* be caring for about 100 tb patients.

   The case fatality of tb is 14% per year. Thus it causes 4,000 deaths annually in the country. The incidence rate is 100 per 100,000. This means 15,000 new cases per year.

   Conclusion: the disease is not yet under control. Patients suffering from the disease are very disabled. This, and the high prevalence, make tuberculosis one of the diseases of major public health importance in Tanzania.

3. *Epidemiology*

   Tb is caused by M. tuberculosis. All mycobacteria have a wax-like skin. This skin gives them excellent protection against diffi-

cult conditions. The bacteria are spread by the sputum particles of patients with open-lung tuberculosis.

Tubercle bacilli have two different life phases:

—an active phase in which in three weeks one bacillus can multiply to become one million

—a dormant phase in which there is no division, no metabolism, but bacilli are able to survive for a long time.

Nobody is born with ready-made immunity against tb bacilli. Nearly everyone in Tanzania will harbour them in his body at least once during his life.

How is tb spread? By patients coughing and spitting their sputum on the ground.

Tb bacilli are resistant to drought, but soon die when exposed to bright sunlight. In a dark house they are able to survive for months in the dust on the floor. When a person sleeps in that house he may inhale the bacilli sticking to the dust particles in the air.

The normal process in 99 out of 100 *infected* people is this: the bacilli enter the lungs; they start multiplying in the primary focus in the lung; they spread to the draining lymph nodes. Then general dissemination of bacilli throughout the body follows. Within a few weeks host resistance develops and most bacteria throughout the body are destroyed. A few bacteria in certain preferential places persist in the inactive dormant phase. The resistance of the host prevents the bacteria from multiplying further.

This is tb *infection*: it is the harbouring of inactive living tubercle bacilli in the body. Nearly every adult in Tanzania has had a tb infection. In most people the primary infection passes unnoticed, or is diagnosed as 'flu'. Tb *infection* should be clearly distinguished from tb disease. Tb disease is associated with active bacilli. In tb disease the bacteria are in the active phase. Disease may develop straight from the primary focus, but it may also develop from formerly inactive bacteria hidden away, e.g. in the upper parts of the lungs, or in the vertebrae. These bacteria have been sleeping for a long time but become active

when the resistance of the body fails. Then disease develops.

Factors in contracting tb *disease*:

(a) The younger the age at the time of primary infection the greater the risk of developing the disease.

(b) Bad housing conditions; dark houses; overcrowding.

(c) Poor nutrition/pregnancy/ other diseases, e.g. diabetes.

(d) Close contact with tb patients.

(e) Use of drugs which suppress the body defence system: corticosteroids.

Crowded places should be well-ventilated.

The amount of tb in Europe started to decline steadily long before the bacilli were discovered and suitable medicines found. This shows clearly that other factors are more important than treatment in the control of the disease. When environmental conditions improve, the health of the population will improve and tb will decrease.

TB IS A DISEASE OF POOR PEOPLE

4. *Clinical picture*

Mode of presentation:

The majority of people present as pulmonary tb with cough for over 2 months.

—productive cough
—haemoptysis
—loss of weight
—fever
—general weakness

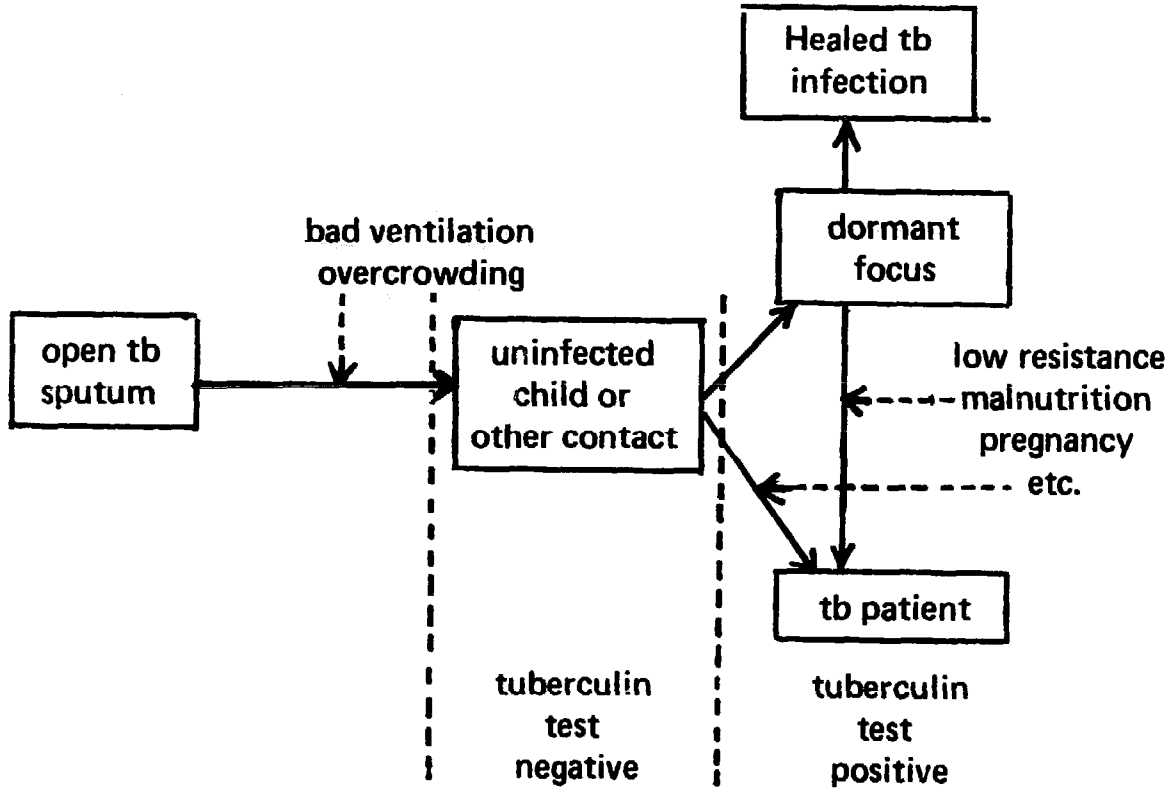
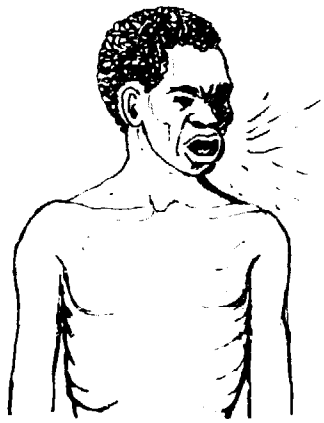


Diagram of the course of tb infection.

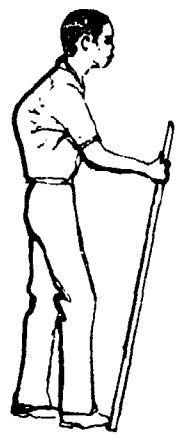
Extra-pulmonary forms of tb

–pleurisy	–tb of genital organs
–peritonitis	–tb meningitis
–tb of kidneys	–miliary tb
–glandular tb	–tb of spine.



Cough and cachexia.

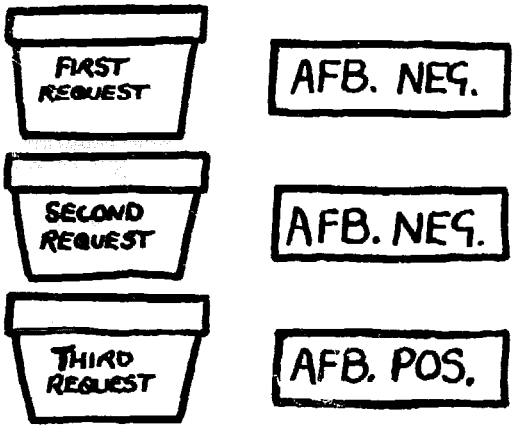
Enlarged glands are felt beneath the skin.



Tb of spine.

Diagnosis:

(i) Demonstration of AFB or Ziehl-Neelsen-pos. bacteria in sputum. A negative sputum should be repeated twice. Also possible but less probable, demonstration of AFB in urine, CSF, pus, or fluid aspirate. A chest Xray is useful in estimating the progress of the disease but not essential for making the diagnosis.



Repeated examination of sputum is the key to diagnosis.

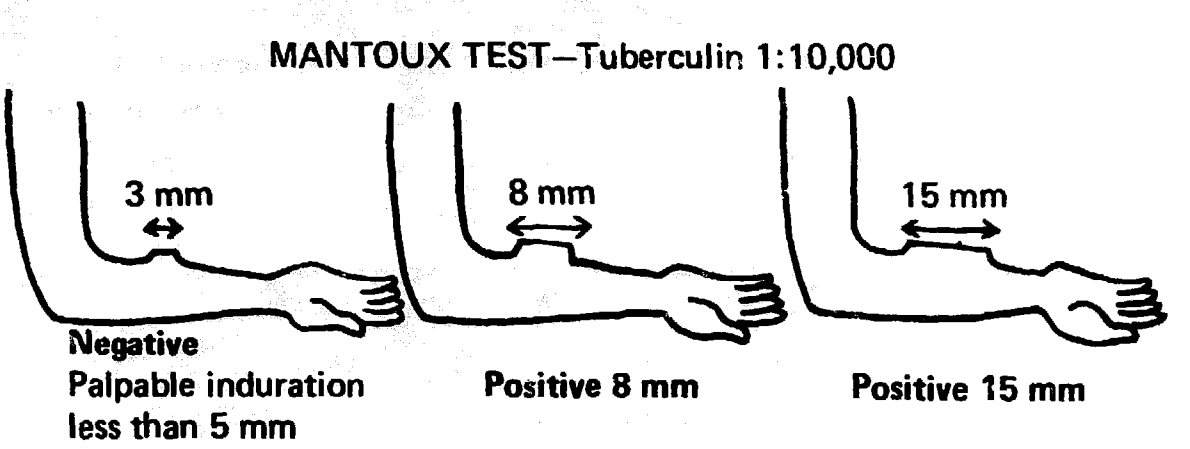
KEY TO DIAGNOSIS:
REPEATED EXAMINATION OF SPUTUM

Use wide-bore jars for collecting sputum (in a small-bore jar —like a penicillin bottle—the patient will deposit saliva only). When wide-bore jars are not available use empty matchboxes or banana leaves. Burn them afterwards.

(ii) There are two types of tuberculin tests
(a) Mantoux test
(b) Heaf test.

(a) *Mantoux test*

A small amount of diluted tuberculin (0.1 ml of 1:10,000) is injected intradermally, resulting in a weal resembling an insect bite. If no weal appears the injection was subcutaneous and the result is unreliable. The day after the injection there may be some redness in the area. Pay no attention to this. The test should be read after 2–3 days. Measure palpable induration only.

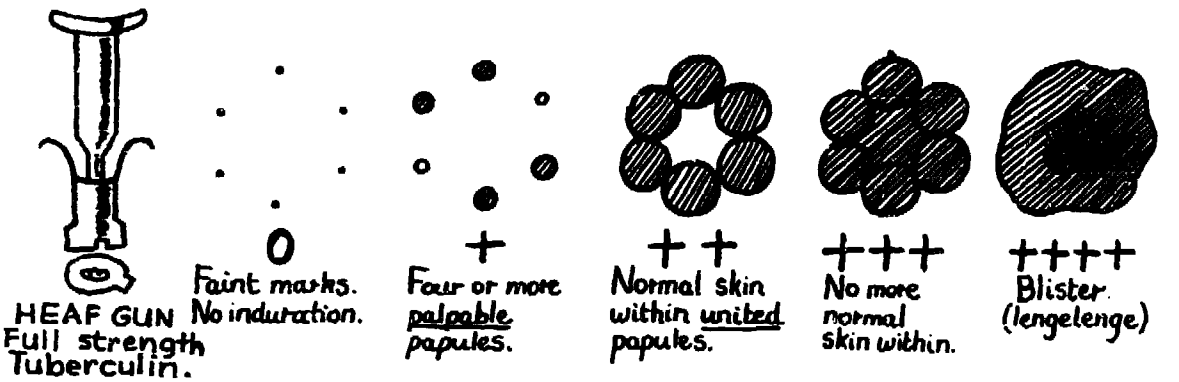


Results of tuberculin test.

Mantoux test.

(b) *Heaf test*

The Heaf test is performed with a special instrument, the 'Heaf gun', which has six small raised points to inject tuberculin into the skin. A drop of *undiluted* tuberculin is put on the skin. Then the gun is placed over the drop and fired. After three to seven days the reaction is read.



Heaf test.

Tuberculin test

A positive tuberculin test means the person is harbouring tb bacilli. The tuberculin test does not distinguish between people with tb infection (dormant) and patients suffering from tb disease (active multiplying bacteria). Positive tuberculin tests occur in most adults in Tanzania as well as those who have been (successfully) immunized with BCG.

A negative tuberculin test usually means that the person has

no infection. However, a person suffering from measles, wasting disease, or malnutrition may have a negative tuberculin test even while suffering from active tb disease (false negative results). Another common cause of false negative tuberculin test is the use of spoilt tuberculin or a wrong technique (e.g. subcutaneous injection).

Diluted tuberculin is absorbed by the glass of the bottles. To minimize this problem, special dark-coloured tuberculin bottles are used for storage in a refrigerator. They bear an expiry date on their label after which the the test material must not be used. Always test the efficacy of the tuberculin by performing a Mantoux test on a person known to be tuberculin-positive (yourself if you are positive, or a tb patient).

When the tuberculin bottles are empty they must be returned to the central pathology laboratory before new ones can be issued.

5. *Management of the individual patient*

The backbone of the treatment of tb is thiazina. Thiazina is a combination of two different drugs. One tablet of thiazina contains INH 100 mg and thiacetazone 50 mg.

When patients are put on thiazina alone and they take their drug regularly 80% are cured after one year. This cure rate can be improved a little if streptomycin is added in the first month of treatment. The cure rate will be 90% provided the patients continue to take their thiazina tablets regularly. Health workers, like other workers, easily form habits. One such habit is writing tds after all tablets which are prescribed. There is an important difference between '1 tds' and '3 od', as is shown by cure rates of patients on different regimes. Only when 3 tablets of thiazina are taken *at one time* ('3 od') does the blood level reach a peak high enough to be efficient.

The cure rates mentioned above cannot be raised by:

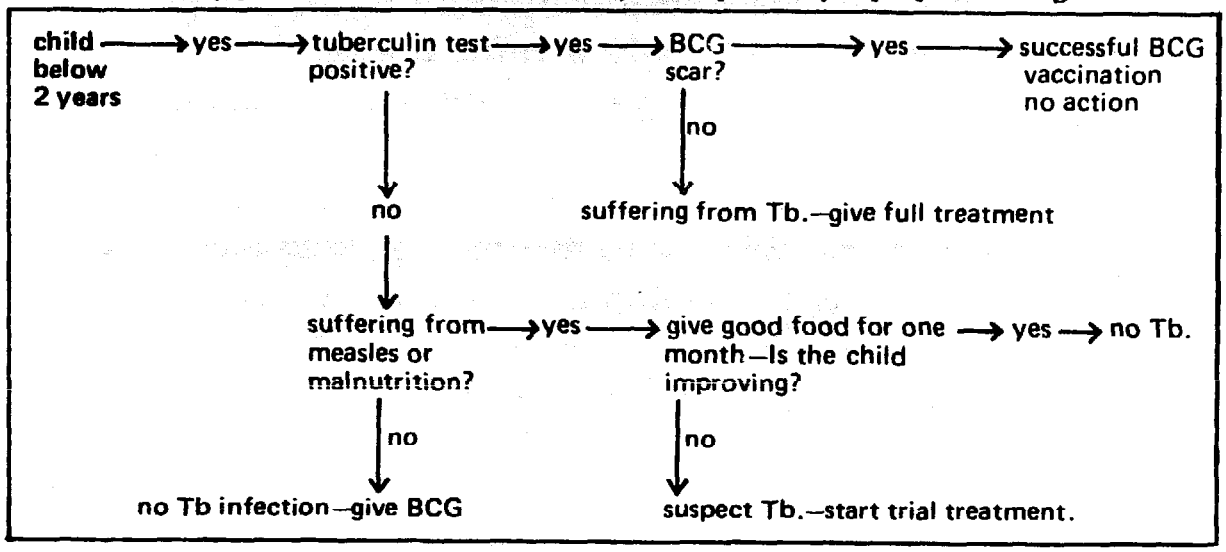
—hospitalization

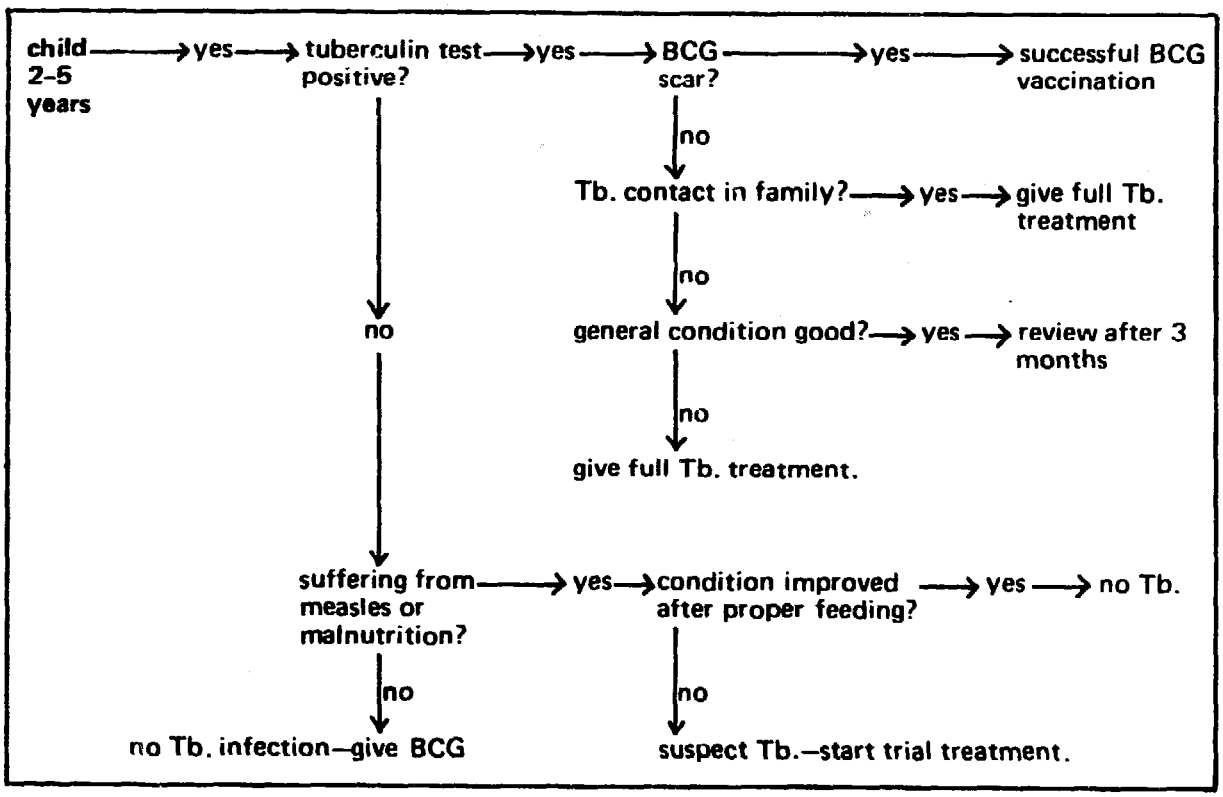
—increased dosage of thiazina

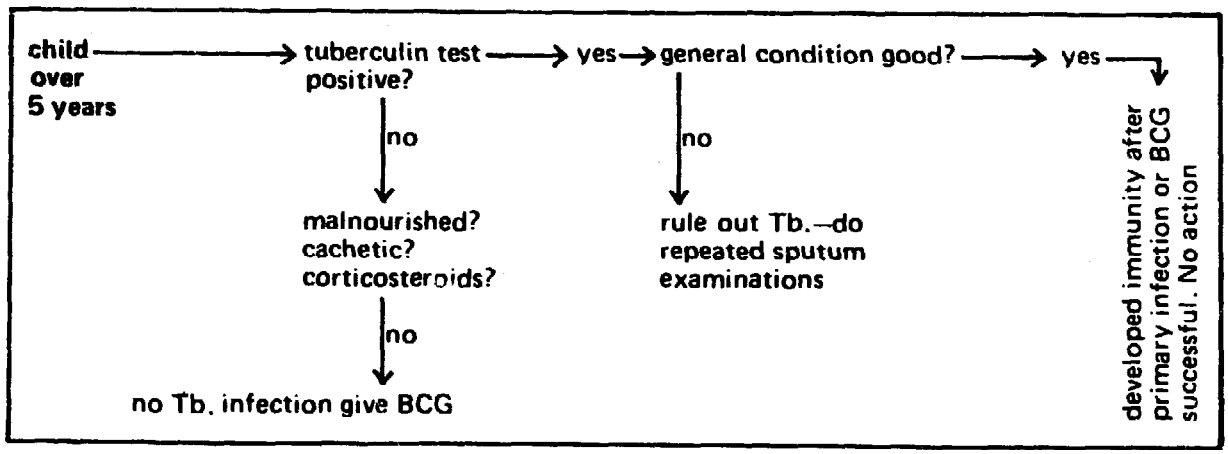
—longer period of streptomycin.

Diagram showing interpretation of the results of properly performed tuberculin tests.

In case of problems, do not hesitate; refer the patient for proper investigation







Therefore the standard treatment for tb is:

(i) Thiazina 3 tabs od for 12 months at least.

(ii) Initially streptomycin 1 g daily for one month on an out-patient basis.

Cure is mostly dependent on the regular intake of thiazina. One of the main problems of tb treatment is resistance. Resistance is most likely to develop in patients who take their thiazina irregularly (or intermittently).

Therefore it is essential to find out from a patient whether or not he has been treated before.

Tb is a disease with social consequences for the patient. Tb patients may fear that their social contacts will be lost if it is known that they are suffering from it. This may make the patient move from one place to another to hide his disease.

After initial treatment he will feel better and may discontinue treatment. When his condition gets worse again, he may come to your hospital/health centre. Very often he will not then admit spontaneously that he has been treated before. Most probably he was told to continue his treatment until he was found to have been cured. So the patient is ashamed and will present himself as a new case. Because of his irregular treatment he may have developed resistance to thiazina. If you treat him as a new case, not knowing about his previous treatment, you will put him on a streptomycin/thiazina regimen. Since the bacilli of this patient are resistant to thiazina, this is like putting him on streptomycin alone.

In tb treatment always use a combination of two or three drugs, otherwise resistance will develop rapidly. And this will happen to the patient resistant to thiazina who is put on treatment as if he were a new case. He will rapidly develop resistance to streptomycin as well.

Therefore patients who have been treated before, or patients who are attending irregularly, or patients who are lost sight of for a few months should *never* be put on streptomycin.

When a patient is resistant to both streptomycin and thiazina it is very difficult to treat him. When streptomycin can still be

used, quite simple regimens are available. If you are in doubt about whether a patient is resistant to thiazina or not there is no harm in putting him on thiazina alone. If he is resistant he will not improve. If his bacilli are still sensitive he will improve and will not develop resistance if he takes the drugs regularly.

To find out whether a patient is resistant to tb drugs his sputum has to be sent for culture and sensitivity testing. A tb culture takes six weeks and the sensitivity test another six weeks (tb bacilli divide very slowly). Postal delay has to be added. When such a patient is referred, the same procedure is followed. Almost no patients are put on second-line drugs before the culture results are known.

DAILY THIAZINA TABLETS FOR AT LEAST ONE YEAR

Admission to hospital?

Before the introduction of efficient drugs, tb was treated by extensive bed rest in special tb hospitals.

Admitting patients to hospitals is a heavy burden on the hospital services without benefit for most of the patients. Admission for a few days only may be advantageous, providing enough time to instruct and educate the patient. This can best be done by a good auxiliary or nurse.

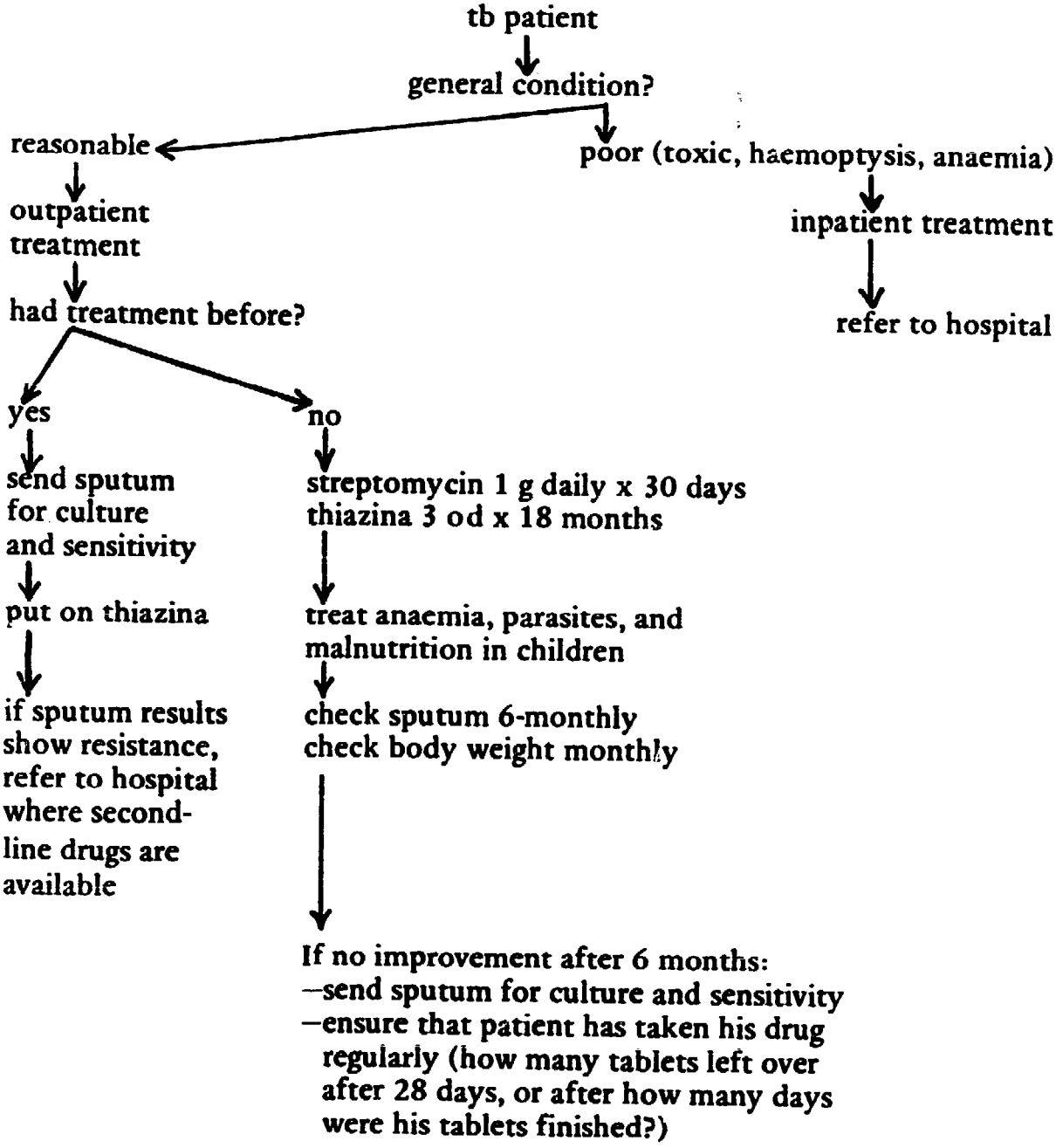
Only patients who are severely ill should be admitted to hospital—and even then they are not admitted because they have tb, but on account of their poor general condition.

When the patient cannot come for daily streptomycin injections and you have enough beds you may admit him for one month for the injections. Remember, however, that this is not essential. The most essential part of tb control is to make the patient take thiazina for at least one whole year.

SUCCESSFUL TB TREATMENT
SUCCESSFUL CASE HOLDING

Tb treatment is more effective when supportive measures are taken against anaemia and parasites.

Treatment schedule



Side effects of common tb drugs

Streptomycin	–allergic reaction (rash, fever) –numbness around mouth –dizziness (mild)	reduce dose
	–hearing strange noises –very giddy –ataxic gait –deafness	stop immediately
INH	–peripheral neuritis –dermatitis-like pellagra	prevent with vitamin B complex
Thiacetazone	–nausea and vomiting	tablets to be taken with meal or in divided doses temporarily
	–persistent vomiting	refer for desensitization

REFER ALL CASES OF SEVERE REACTIONS

6. *Control*

Tb is a disease of humans only. Treatment of all people suffering from tb would result in eradication of the disease. In practice this is difficult. The available drugs for treatment need to be taken for a long period. This involves the co-operation of the patients themselves. At present only a small proportion of tb patients are under treatment. For every diagnosed and registered patient there remain two undiagnosed and untreated.

The disease spreads because of low social and economic conditions. To improve these conditions is the task of the whole nation, not a task for doctors only.

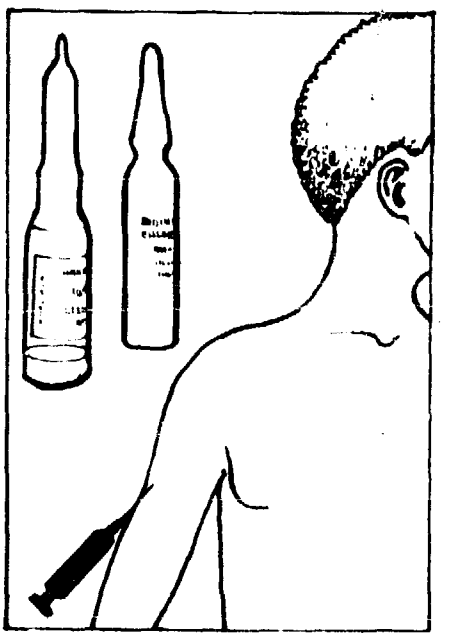
The present ways of fighting the disease are:

(i) Vaccination of population with BCG.
(ii) Case finding: continuous searching for new patients.
(iii) Case holding: continuous treatment of known cases.
(iv) Health education about transmission of tb.
(v) Prevention of malnutrition in children.

GIVE BCG AT BIRTH OR AT MCH CLINICS
REPEAT ON SCHOOL ENTRY

(i) *BCG*

This is a vaccination by means of artificially weakened tubercle bacilli resulting in good protection against tb (and, to some extent, leprosy). For technical details see *Community Health* manual (Immunization). BCG should be given on the right shoulder, where it will leave a small scar.



All babies born in medical institutions must be vaccinated before returning home.

Even without a refrigerator you can start BCG vaccination in your health centre if you get a monthly supply. The price of one dose is 0/75 cents. Vials containing 10 doses will soon be available.

Open one vial daily for newborns and children attending the MCH clinic.

(ii) *Case finding*

Continuous searching for new patients. Remember that at present more tb patients are walking around untreated than are under treatment. Making the diagnosis of tb starts by suspecting it. Suspect tb in every person complaining of cough for more than one month. Suspect tb in malnourished children who do not improve on a good diet. Examine all contacts, especially children, of known tb

cases.

Adults: if coughing, examine three sputum specimens.
Children; do tuberculin test, if positive, treat them as tb.

(iii) *Case holding*

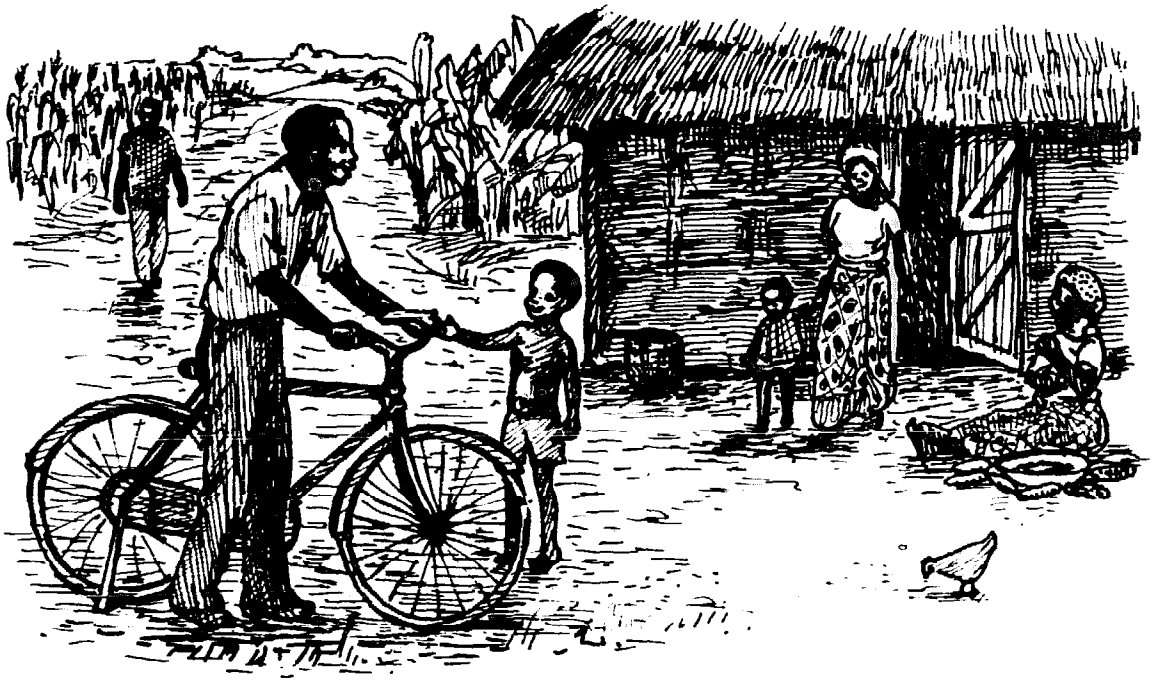
Efficient treatment and control of known tb patients. Research has shown that the most important factor for success in the treatment is *daily* taking of drugs. Irregular treatment results in the spread of bacteria resistant to the first-line treatment. This causes treatment-resistant tuberculosis. A patient not taking his drugs for a week is considered irregular. A patient not taking his drugs for one month is regarded as a non-complier. How can you avoid a high number of non-compliers?

(a) Take time to explain to the patient at the time of his first visit the nature of his disease. Explain that he *can* be cured but *only* if he takes the drugs *regularly* and *until* he is considered cured. Explain that after a few months he will feel so much better that he will think he is completely cured, but that the disease will relapse if he does not completely finish the prescribed treatment.

(b) Have a special day, weekly or monthly, for a chest clinic (it is best to co-ordinate this with, say, market day in your village). On this day tb patients should be given priority. Keep a special tb register book in your health centre, as without proper registration it is impossible to check on whether all patients turn up. Tell the patient how many weeks his drugs are supplied for, and tell him the date he is to report back.

(c) Always inform an influential member of the patient's family about the importance of the patient taking his drug daily and attending the clinics.

(d) Explain to the patient that you will write a letter of referral if he wants to move to another area, so he can continue his treatment at another health unit.



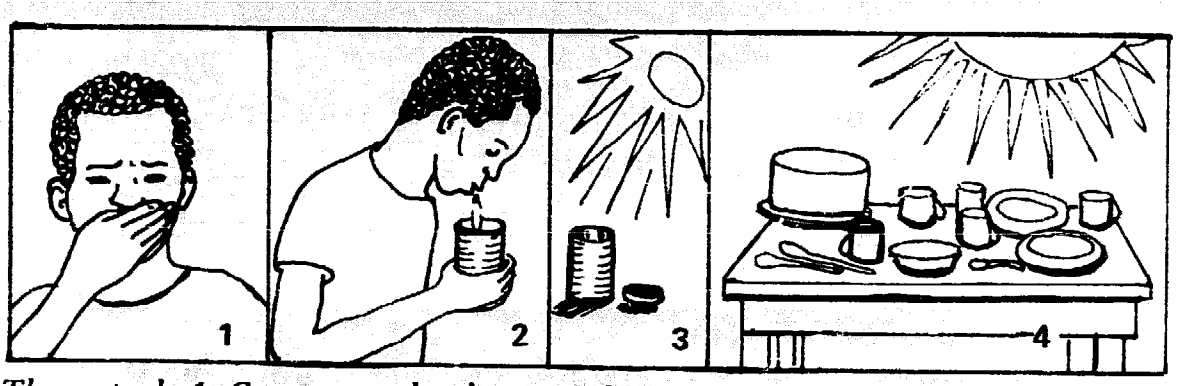
Home visiting.

If a patient does not turn up for one week send the health auxiliary from your health centre to contact the patient and to convince him of the importance of regular attendance. If a patient is regarded as a non-complier, contact the CCM chairman and the ten-cell leader; ask for their co-operation.

(iv) *Health education*

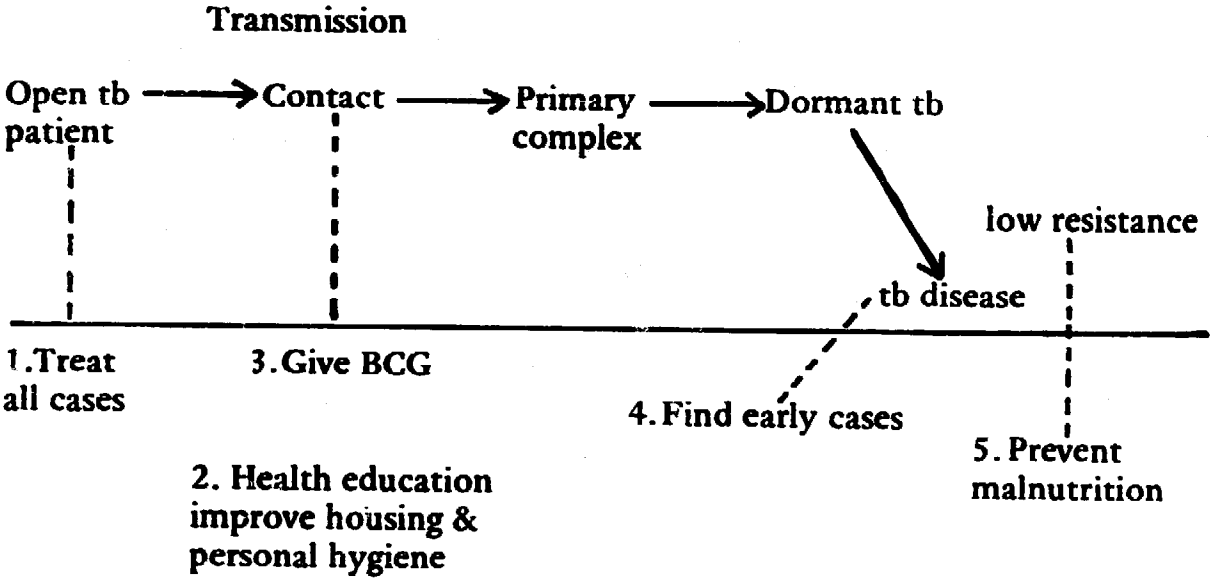
It is most important to explain to the community the dangers of having untreated tb patients among them. Families and friends should encourage patients to go for treatment and to continue the treatment as long as necessary. When new houses are built emphasis should be laid on the importance of windows for light and ventilation. Light destroys tb bacilli. Ventilation reduces the risk of inhaling infective bacteria. Explain to patients how tb is spread.

Tell the patient to cover his mouth when coughing, not to spit on the floor but to spit into a jar with a lid, which is cleaned daily with hot water and left drying in the sun. Eating utensils should also be dried in the sun.



Tb control: 1. Cover mouth when coughing. 2. Spit into a container with cover. 3. Wash and sterilize container in the sun. 4. Eating utensils should be dried in the sun.

Diagram of tb control.



At Kibongoto on the slopes of Mt Kilimanjaro is the national tb hospital. This is the central teaching hospital and referral centre for complicated cases of tb.

The East African Tuberculosis Investigation Centre is in Nairobi. It directs all research on tb within East Africa. Schedules for treatment are tested and results of treatment campaigns evaluated.

7. *Action*

—Do three sputa for AFB in all patients complaining of cough

for longer than two weeks.

—Check the equipment of the laboratory of your health centre and satellite dispensaries. Do they have:

a microscope
slides
immersion oil
carbol fuchsin 3%, acid alcohol 3%, methylene blue 0.3%
spirit lamp?

—Check the microscopist; if the results of his sputum examinations are doubtful, arrange a refresher course in the district hospital with your DMO.

—Check your MCH services. Ensure that all children are routinely weighed and that the weights are plotted on the growth card. Teach your staff to take action when a child's weight is between 60 and 80%.

—Be sure you get a monthly supply of dry-frozen BCG vaccine.

—Give BCG vaccination to all newborns in your maternity ward before they go home.

—Use the rest of the doses left in the vial that same morning for vaccination at the MCH clinic.

—Arrange with the teachers of the schools for a day to vaccinate all schoolchildren in the catchment area of your health centre.

—Compare the number of registered patients in your health centre with the estimated number of tb patients in its catchment area (0.2% of the total population). Now you know the number of patients who are not under treatment.

—Check every month the number of non-compliers. Find out how many have been traced by the health auxiliary. What were the explanations for not attending? Try to find ways to overcome the problems of your patients.

—Stress in the ward development committee the importance of building houses with windows.

8. *Summary*

Tb disease is caused by failure of host resistance against Mycobacterium tuberculosis. The main symptoms are chronic cough and loss of weight. Diagnosis is made by sputum examination. Standard treatment is thiazina 3 tabs od for one-and-a-half years on an outpatient basis. Control is based on case finding, case holding, and immunization with BCG.

LEPROSY

1. Leprosy is an infectious chronic disease of man caused by the leprosy bacillus and by tissue reaction against this bacillus. It affects mainly the peripheral nerves and skin, but also the mucous membranes of the upper respiratory tract, and some internal organs such as testes, kidney, and liver.

   Synonyms: Hansen's disease, *ukoma**.

2. *Occurrence and importance*

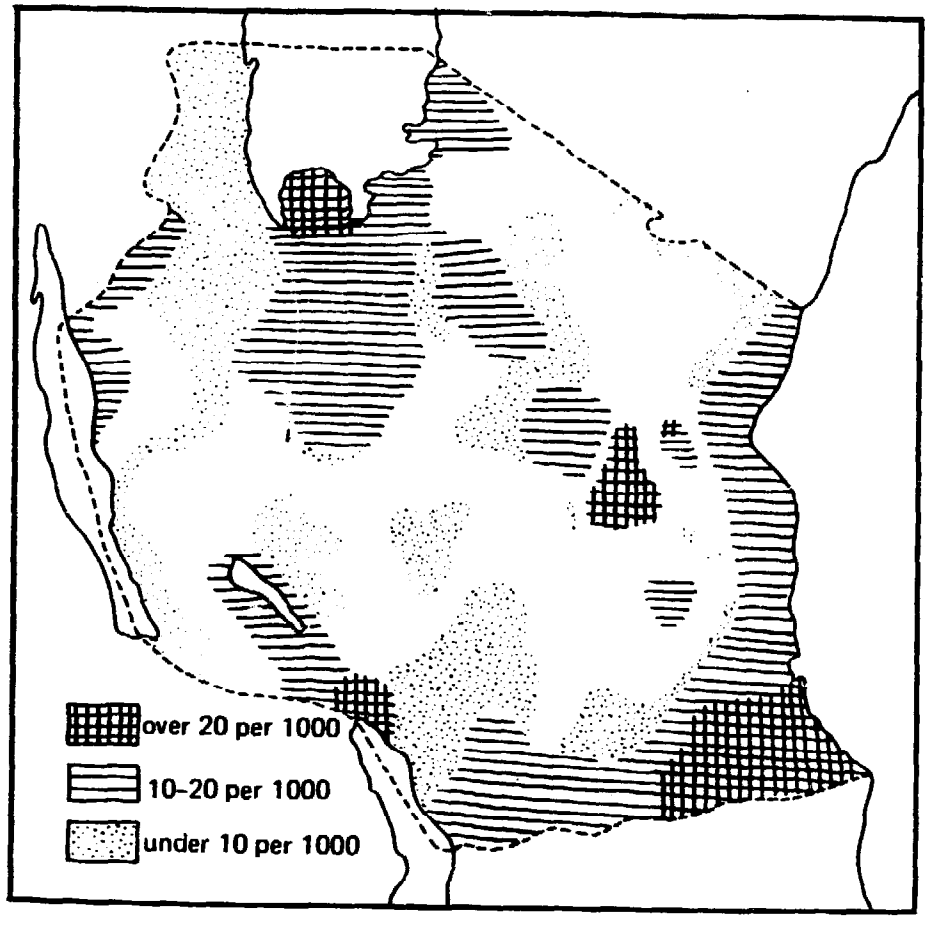
   Leprosy is not a killing disease: it is a crippling disease. The life-expectancy of an individual with leprosy is not much less than the life expectancy of a healthy person, but leprosy is a very disabling and deforming disease.

   Deformities occur in hands, feet, and face. Eye involvement may cause blindness. Testicular atrophy may result in impotence, sterility, and gynaecomastia. Leprosy does not only harm the patient physically: the deformities mark the patient as suffering from leprosy and prevent a normal life by making labour and farming difficult. Often the patient suffering from leprosy finds that other members of the community avoid him. This is because of traditional beliefs about its cause and even incorrect modern ideas about its contagiousness. No other disease causes such a reaction in the community and so much

**Ukoma* is not completely equivalent to leprosy. It refers more to the deformities associated with leprosy, and the word cannot be used for early leprosy patients or patients with one or two patches who do not have any deformity.

distress and unhappiness to the patient and his family as does leprosy. This anxiety may follow leprosy patients throughout their lives and bring a permanent shadow over their families and their professional and social activities. Without justification, leprosy patients can lose their jobs. Leprosy patients are often divorced or the community may prevent them from marrying. They frequently have to leave their villages either because they are known to have leprosy or because they fear that their disease will become known when people see them going for treatment. Some patients are even deserted by their families.

According to WHO's definition leprosy is hyperendemic when the prevalence is over 10 per thousand. In Tanzania there are about 150,000 people suffering from leprosy. This is a prevalence rate of 10 per thousand. So leprosy is hyperendemic in Tanzania. The highest number of patients is found in the border regions (with the exception of Kilimanjaro).



Approximate distribution of leprosy.

Leprosy is an important disease from the public health point of view because of its (a) high prevalence; (b) severe disability and deformities and chronic course; and (c) social aspects and stigma.

A lot of research has been done to determine the risk factors for getting leprosy. Results:

Race: No racial immunity, but black races have less severe forms of leprosy.

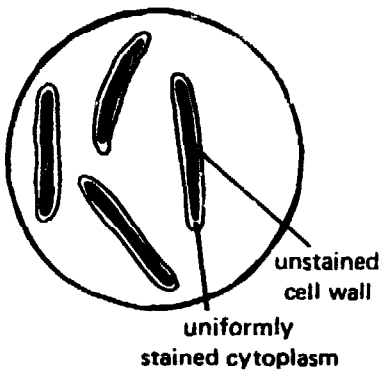
Sex: Infectious forms are more frequent in males.

Age: No age group is more susceptible than another. Most new cases are diagnosed between 15 and 25 years of age. This is not due to susceptibility but to opportunities for contact and length of incubation period.

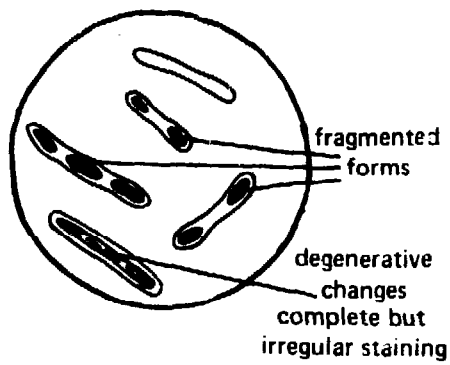
Heredity: The incidence of leprosy among children of lepromatous parents is 10 times higher than that found in the population at large. It is not fully understood whether this is due to severe exposure or to hereditary (genetic) factors. Leprosy should not be regarded as a hereditary disease.

3. *Epidemiology*

Leprosy is caused by the leprosy bacillus or Mycobacterium leprae. It resembles Mycobacterium tuberculosis in staining properties. Not all patients suffering from leprosy are equally infectious to others. Those in whom the skin smears are negative are not regarded as 'open' (infectious) cases and will not spread the disease. Patients with positive skin smears may infect others if they are not under regular treatment. When



Mycobacteria in a positive smear.



Non-viable forms of mycobacteria after start of treatment.

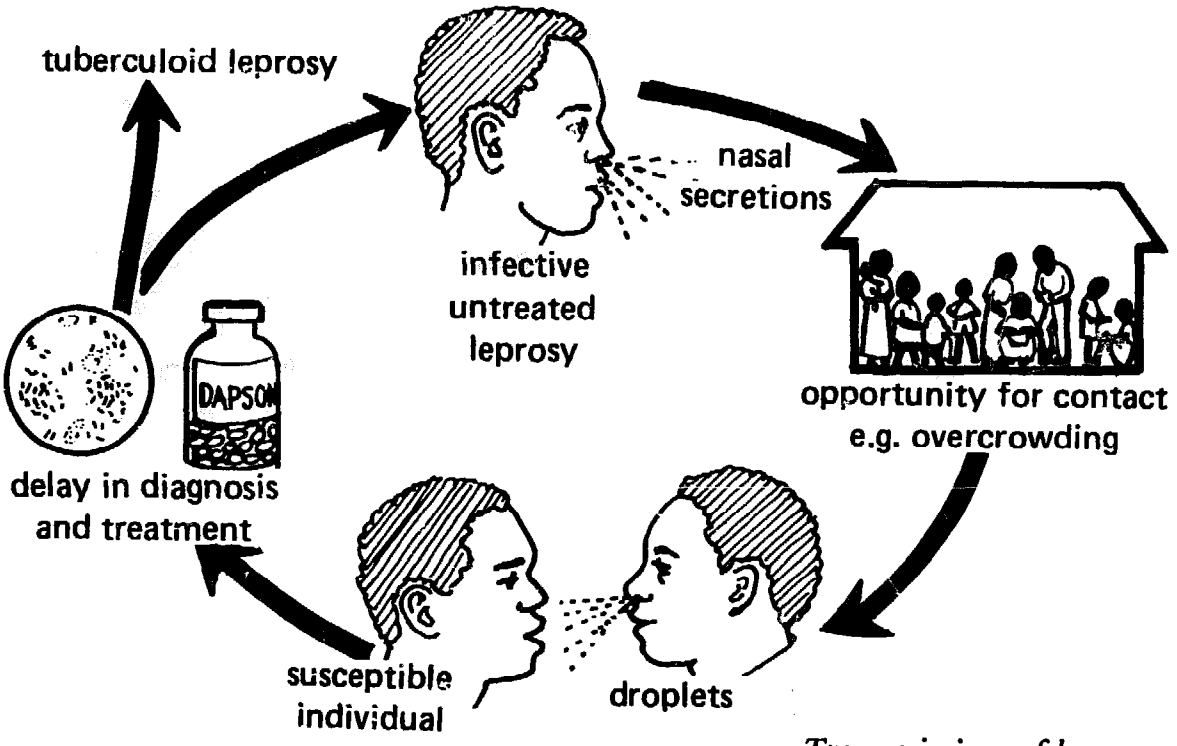
treatment is started and continued regularly more of the bacilli will stain irregularly and take on abnormal forms. This shows that the bacilli have died and the patient is no longer infectious.

Epidemics occur when leprosy is introduced in a community for the first time. When the epidemic has passed the disease settles down into an endemic state. In Tanzania leprosy occurs in the endemic form.

There are, broadly speaking, two forms of leprosy: the tuberculoid form and the lepromatous form (for sub-divisions see *Clinical picture*). In lepromatous leprosy many more bacteria are present than in tuberculoid leprosy. The most heavily infected sites in patients with lepromatous leprosy are the skin, the nose, and the upper respiratory tract. In the nose bacteria are often found even before skin lesions are evident, and again in the nose the leprosy bacteria are found very near to the surface, while in the skin lesions there is usually a clear zone at the surface. In the nasal secretions large numbers of viable bacteria can be found: up to 1000 million can be discharged daily by a patient.

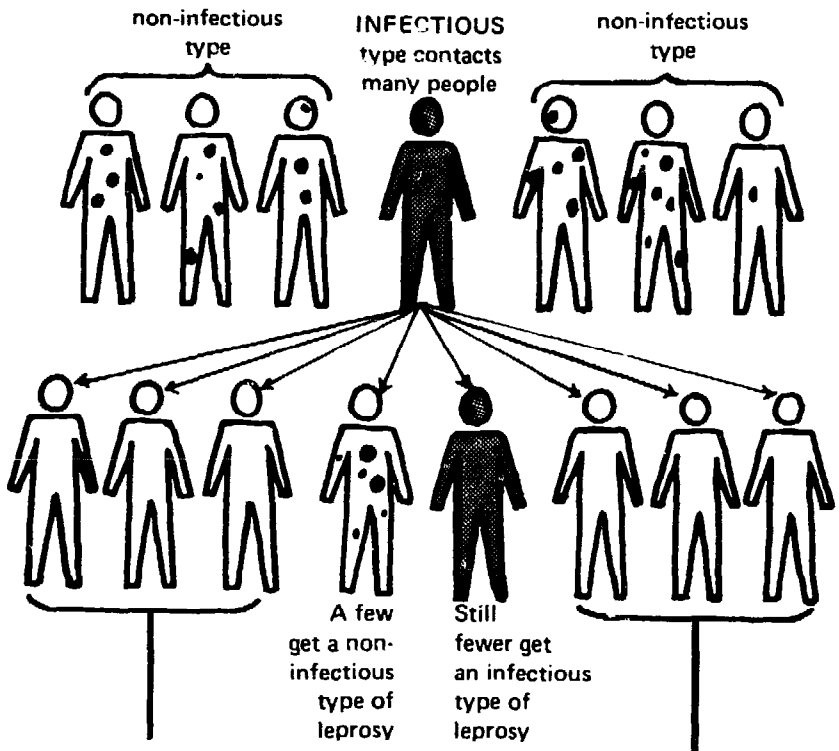
It is very likely that leprosy is transmitted by sneezing, coughing, spitting and unhygienic nose-cleaning habits. This is probably by way of droplets, as in tuberculosis. It is very unlikely that leprosy bacilli are able to penetrate the intact skin. It may be that they are able to enter through small wounds but this is probably not the normal route. It is therefore still uncertain whether the portal of entry for leprosy bacilli is the skin or the respiratory tract. In any case leprosy is most common among household contacts (as is the case with tb).

Only patients who discharge live leprosy bacilli can infect other people. Most people who come into contact with leprosy bacilli do not develop leprosy. They have a high resistance to M. leprae and their bodies are able to kill any live M. leprae which might enter them. ONLY A VERY FEW PEOPLE that lack high resistance will get leprosy from an infectious patient. Most of these people, fortunately, will develop a non-infectious



Transmission of leprosy.

type of leprosy. This shows that leprosy is not easily caught by other people; this fact is also demonstrated by the incidence of leprosy among leprosy health workers not being higher than the incidence of leprosy among the general population.



Most people have high resistance and do not get leprosy.

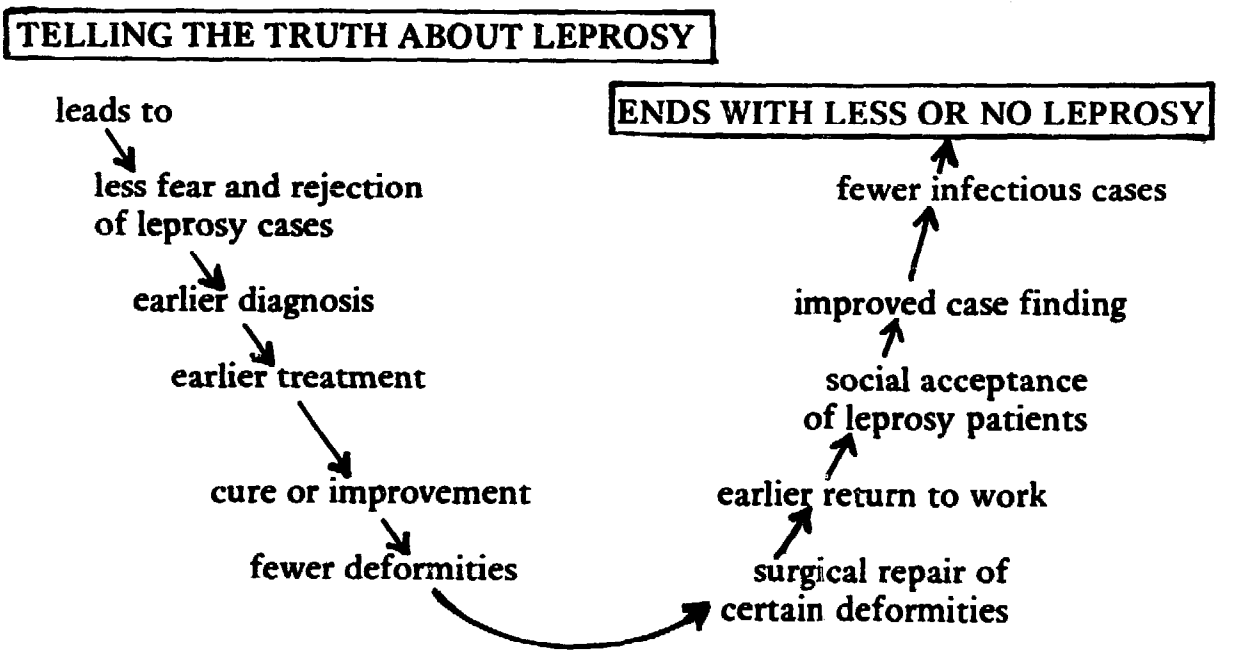
Most people have high resistance and do not get leprosy. From the diagram can be seen that leprosy may increase in a community:

(a) When there are many infectious untreated cases.

(b) When social conditions provide an opportunity for contact: overcrowding, social customs, poor houses.

(c) When there are many susceptible newcomers in a given area, or when leprosy is introduced into a previously leprosy-free area.

(d) When patients tend to hide their disease; this will delay early diagnosis; when people do not hide their disease early diagnosis and treatment will decrease the incidence of leprosy in the community (see diagram).



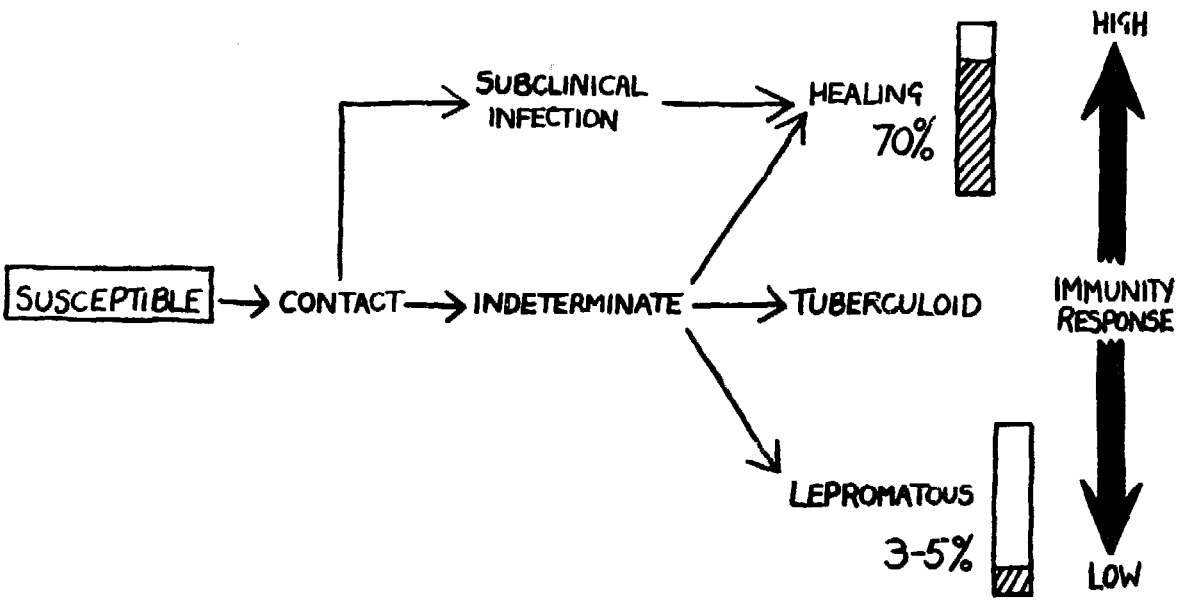
4. *Clinical picture*

Inbubation time: M. leprae have a long multiplication time. They divide once every two weeks. This accounts for the very long incubation time and the slow course of the disease. Average incubation time is 6 years (range 1–12 years).

Pathology: The lepra bacillus multiplies in macrophages of the skin and nerve fibres (Schwann cells). The organism multi-

plies best in the cooler parts of the body so that the skin of the face and limbs and their more superficial nerves are invaded first.

As a result of the invasion of bacilli there is an inflammatory response. Clinically a small vague hypopigmented macule appears. The lesion is called indeterminate* as there is no indication as to how it will develop. Most indeterminate lesions heal spontaneously, many are never noticed. Lesions which do not heal may develop into tuberculoid, borderline, or lepromatous leprosy. This depends on the ability of the host to develop (cellular) immunity against M. leprae.



According to the degree of cellular response by the host, the following can occur. In **tuberculoid** leprosy there is a strong focal reaction to the bacteria. The intracutaneous nerves are infiltrated by lymphocytes and completely destroyed resulting in anaesthesia. Acid-fast bacilli are usually few or absent. Invasion of peripheral nerve trunks is early.

In **lepromatous** leprosy there is little response, very few lymphocytes are found, nerve twigs are not infiltrated by lymphocytes, and masses of bacilli can be seen. Damage to peripheral nerve trunks is late.

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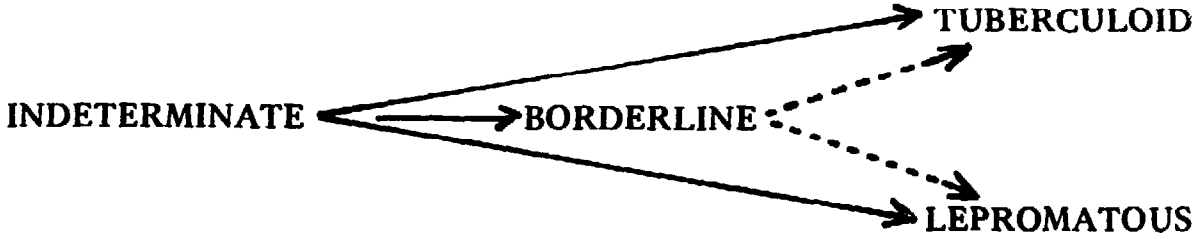
*Do not confuse 'indeterminate' with 'intermediate'.
Indeterminate: it cannot be determined or defined.
Intermediate: in between.

The body reaction, however, is not always clear as in these tuberculoid or lepromatous types. Borderline leprosy shows features of both the tuberculoid type and the lepromatous type. Lymphocytes are present but there is only slight infiltration of the fine nerve twigs; acid-fast bacilli occur in moderate numbers. Borderline leprosy is unstable and can change into one of the definite forms (but this is very unusual). Because of the instability widespread damage to peripheral nerves may occur. According to the above the clinical forms of leprosy are divided into four as follows:



Unstable forms — Polar stable forms

The clinical characteristics of the different types of leprosy are an expression of the cellular immunity or natural resistance of the patient.

	Signs of low resistance	*Signs of high resistance*
Number of lesions	Many	Few
Size of lesions	Small	Large
Distribution	Symmetrical	Asymmetrical
Definition of lesions	Ill-defined	Clearly defined
Hypopigmentation	Slight or none	Marked
Central healing	None	Marked
Progression	Progressive	Self-healing
Loss of sensation	None	Clear
Skin smear	Positive	Negative

TUBERCULOID LEPROSY

Lesion

The skin patches in tuberculoid leprosy have the following

characteristics:

—usually few in number
—patches are raised or flat
—the edge is well-defined
—there is loss of sweating (dry surface)
—there is loss of hairs
—there is loss of sensation
—often there is central healing
—hypopigmentation is marked
—the patches are large and asymmetrically distributed.

Nerves

In the area of the patches the cutaneous nerves may be enlarged. The main nerve trunks may be enlarged over a part of their length. Nerves are always involved early in the course of the disease. Paralysis is common.

Smear

Skin smear is negative.

Tuberculoid leprosy is sometimes divided into two subgroups: TT and T.

Patients with the highest resistance have only a few self-healing patches (less than five). If resistance is lower there will be more patches with a tendency to a more symmetrical distribution especially on the knees and elbows. The patches may show typical features of tuberculoid leprosy mentioned below:

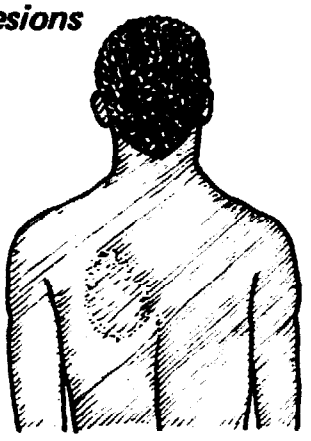
—small patches near larger ones (satellites)
—spreading of the edge of a patch in an irregular way, like oil spreading on the skin or water running out of a puddle (streaming)
—two or more patches joining together to form one major patch (confluence).

LEPROMATOUS LEPROSY

Lesions

Early stage: innumerable, small, ill-defined, slightly or not

Tuberculoid leprosy

Skin lesions 	usually few raised or flat, large or small well-defined edges, regular or irregular surface dry always some loss of feeling, sometimes quite marked often central healing some colour change in patch, red or pale asymmetrical
Nerve trunks	sometimes one or two trunks involved early
Smears	negative

raised, patches, macules, or nodules. When the whole skin is involved little abnormality can be detected except that the skin, on palpation, feels thicker than normal. This is most prominent on the face and ears. There is no loss of hair, no loss of sweating, no loss of sensation.

Nerves

The nerves are involved late. Long sections of nerve trunks are symmetrically enlarged. Anaesthesia of glove-and-stocking type occurs, and leads more commonly to disability than paralysis.

Smear

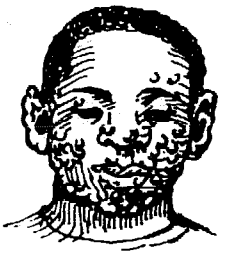
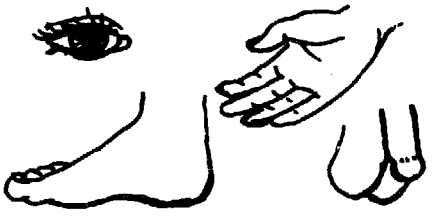
Positive skin smear.

Other organs

Nose: saddle-nose deformity, because of destruction of nasal cartilage; ulceration of nasal mucosa; blocked nose; epistaxis.

Eye: iritis and damage to ciliary body; hazy cornea; cataract; glaucoma.

Lepromatous leprosy

Skin lesions Too many to count	Many different kinds of lesions, e.g. —nodules —raised patches —flat red patches —areas of thick skin	
Other organs involved	—eyes —hands and feet —testicles	
Nerve trunks	Many involved but late in the disease.	
Smears	Strongly positive	

Larynx: hoarseness of the voice.

Testis: atrophy resulting in impotence, sterility, and gynaecomastia.

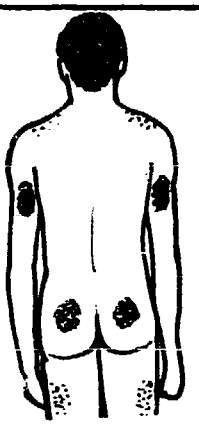
Bones: leprous osteitis resulting in 'banana' fingers.

Teeth: loss of upper central incisors due to damage to the maxilla.

Lepromatous leprosy is sometimes divided into two subgroups: L and LL.

If resistance is almost completely absent the onset of lepromatous leprosy is insidious with vague slightly erythematous macules which are very difficult to see. Loss of eyebrows (madurosis) may be the only clear sign. When resistance is higher the lesions are more obvious because they are usually slightly elevated in their centres. The lesions are shiny and smooth with vague edges. There is no loss of sensation and because of spread through the blood-stream the lesions are distributed symmetrically over the body.

Borderline leprosy

Skin lesions		very many edges may be well-defined outline often irregular raised or flat some loss of feeling no healing centre symmetrical
Nerve trunks		often many involved fairly early
Smears		range from negative to positive

BORDERLINE LEPROSY

Borderline leprosy has features of both tuberculoid leprosy and lepromatous leprosy. It is an unstable form and may change from more tuberculoid signs to more lepromatous signs and vice versa. Borderline leprosy is divided into three sub-groups: BT or borderline tuberculoid; BB or typical borderline; and BL or borderline lepromatous.

In most cases either the tuberculoid or the lepromatous element predominates and BB is seldom diagnosed by expert leprologists. There should not be too much worry about finer points in distinguishing these varieties and all should be considered 'borderline'.

Lesions

The lesions are not so clearly defined. There may be a slight loss of sensation only. The borders of the lesions are not raised very much and the lesions tend to be symmetrically distributed but not as much as in lepromatous. The centre of the lesions is often more raised than the edge (especially in BB and BL).

Nerves

The nerves are very often affected in the beginning as well as at the end of the disease. Nerve involvement is more generalized

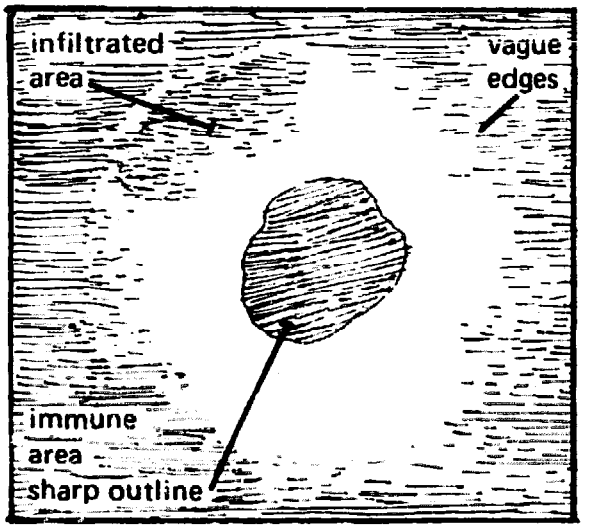
than in T leprosy.

Smear

Often positive in BB and BL. The number of bacilli found is not as high as in L leprosy.

Other organs

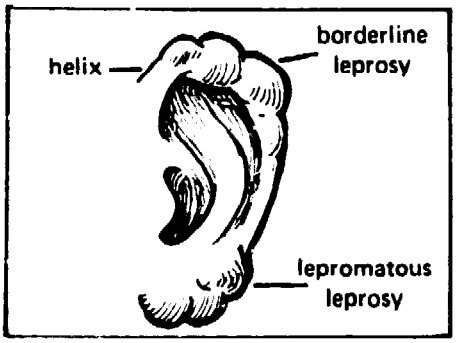
These are not involved in uncomplicated cases. Other typical signs of borderline leprosy are:



—A patient may show clear patches (as in T leprosy) together with nodules (as in L leprosy).

—Lesions may show immune areas. These look like areas of central healing but are in fact normal skin in the centre of an infiltrating patch separated from it by a sharp boundary. They are a characteristic feature of borderline leprosy.

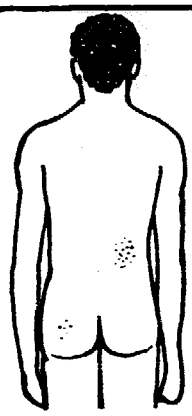
—Lepromatous nodules occur on the ear lobes. The nodules in borderline leprosy occur on the upper part (the helix) of the ear.



INDETERMINATE LEPROSY

The onset of leprosy is frequently visible as a flat patch or macule, ill-defined, with a slight loss of pigment. There is no loss of sensation and no bacilli are found in smears. Organs and nerves are not involved.

Indeterminate leprosy

Case history (length of time, signs or symptoms have been present)	Short, 3–6 months maximum.
Skin lesions 	patch only, few, 2–3 at most edges not well defined surface flat loss of feeling slight
Nerve trunks	not involved
Smears	negative

Reactions

Everything in leprosy tends to be slow and chronic, but sudden changes do occur in all types of patients. Such a sudden change is called a reaction. Reactions are the result of a change in the balance between the immunity of a patient and the bacilli. There are two main types of reaction.

Type I reaction or reversal reaction

This is a change in cellular immunity. Usually the immunity increases suddenly resulting in a rapidly increased response of the body to the leprosy bacilli. This 'fight' causes sudden inflammation in places where the leprosy bacilli are present. Type I reactions are especially common in the unstable forms of leprosy, that is, in borderline leprosy, and are mostly seen in the first year of treatment or earlier. (Often they are the reason the patient comes to hospital where he is diagnosed for the first time.)

Type II reaction or ENL or erythema nodosum leprosum

This is caused by a reaction between dead leprosy bacilli and circulating antibodies. In L leprosy the cellular immune response is low but there are many circulating antibodies. These are ineffective and do not help the patient very much. Type II reactions occur especially in BL and L leprosy and are mostly seen 6 months or more after the start of treatment. They continue for years.

Reactions are provoked by:

—other diseases, e.g. malnutrition, malaria, worm diseases, anaemia and especially tb

—extra stress or strains, physical or emotional, e.g. hard work, worries, cold weather, etc.

—hormonal changes, menstruation, pregnancy, childbirth, abortion, puberty

—other drugs, iodine (orally), traditional *dawa*

—vaccinations, smallpox, BCG

—infected wounds, osteomyelitis.

Formerly it was believed that treatment could provoke a reaction. To avoid reactions the treatment was started with low doses and increased slowly. Leprosy specialists then found out that reactions do not occur due to treatment. We now start treatment right away with the full dose.

Reactions are characterized by local inflammation with all its typical signs:

—redness
—hotness
—swelling
—pain
—loss of function.

In Type I reaction the inflammation is seen in the skin lesion and nerves.

Lesions

The lesions become warm, tender, reddish, and raised.

Nerves

The nerves become tender and thickened. Acute paralysis is common. Pain is severe and widespread. There may be generalized oedema especially of the hands, the feet, and face. Oedema readily causes nerve damage.

Type I reactions are important because the acute paralysis is a major cause of permanent disability and deformity. Type I reactions may be extensive with many patches and nerves involved.

In Type II reactions (L and B) the inflammation is seen in the form of newly appearing painful red nodules called erythema nodosum leprosum (ENL). ENL may be mild or severe. Mild ENL: not many lumps, mainly on face, outer sides of arms, and front of legs. Patient does not feel very ill. Severe ENL: patient feels ill with fever and pain everywhere. Many lumps which may be spread all over the body. Complications are common. Look for:

—lymphadenitis
—iritis
—arthritis
—epistaxis or blocked nose
—hoarseness of the voice, sometimes stridor
—periosteitis (hands, feet, or long bones)
—nephritis (proteinuria)
—orchitis
—ulcerating nodules.

Summary of reaction types

Type	Classification	Nerve signs	Skin Signs	Management
Type I or Reversal	in BT BB BL	Nerve damage common and obvious	Existing lesions become red raised	Refer to hospital immediately
Type II or ENL	in L and BL	Nerve damage not common eyes, joints, testes may be involved	Fresh red lumps appear	Treat mild cases refer severe and non-responding cases

Relapse or worsening of leprosy must be differentiated from reaction. The main difference is the speed and severity of the events occurring and usually differentiation is not difficult (see table).

Differences between worsening or relapse, and reaction

Signs	Relapse	Reaction
Onset	Slow, weeks or months	Sudden, hours
Damage	Slow	Rapid, especially in nerves
Sites	New patches anywhere on body	Usually only in patches or infiltration already present
Pain	Usually painless	Nerves often painful
General condition	Not affected	Often fever, joint pains
Response to treatment	Slow	Rapid
Treatment	Anti-leprosy, e.g. DDS	Anti-inflammatory, e.g. rest, Aspirin, chloroquine

Nerve damage and disability

The primary cause of disability is the destruction of nerves.

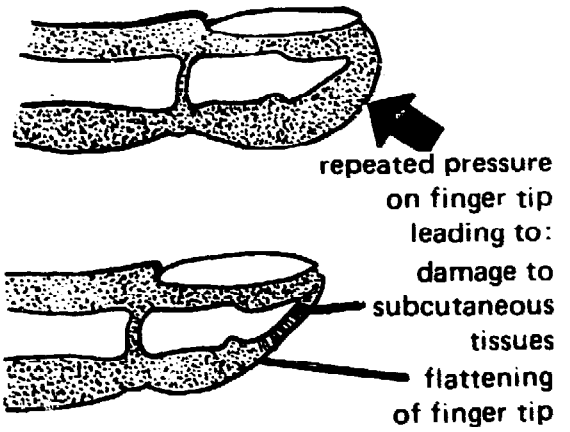
1. Damage to sensory nerves → anaesthesia.
2. Damage to motor nerves → paralysis.
3. Damage to autonomic nerves → impaired circulation, skin atrophy, loss of sweating.

1. *Anaesthesia*

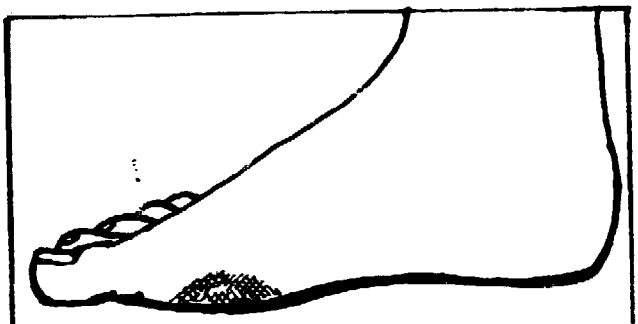
   Minor injuries are not noticed. Wounds fill with dirt and with every movement infection is driven deeper into the tissues. Only advanced infection will alarm the patient (fever, a painful lymphadenitis or a grossly swollen foot or hand).

Pressure is not noticed. Normally a person will shift his weight from one foot to the other or from toes to heel. When a foot or a hand is anaesthetic prolonged pressure on one place is not noticed, resulting in ischaemia (too little blood flow) to the tissues and injury. This happens especially on those places where the skin is near to the underlying bones.

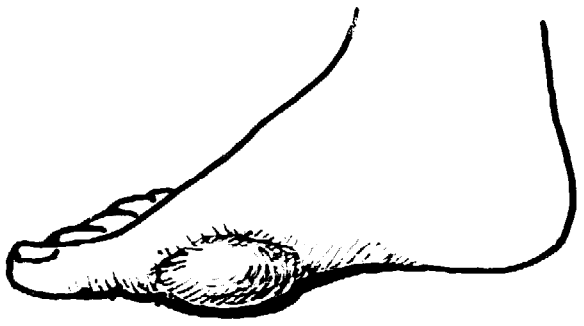
A normal person feels when a joint is being strained. His leg will give way and he will fall down or he will strain other muscles supporting the joint.



Mechanism of damage due to anaesthesia.



First injury only a bruise.



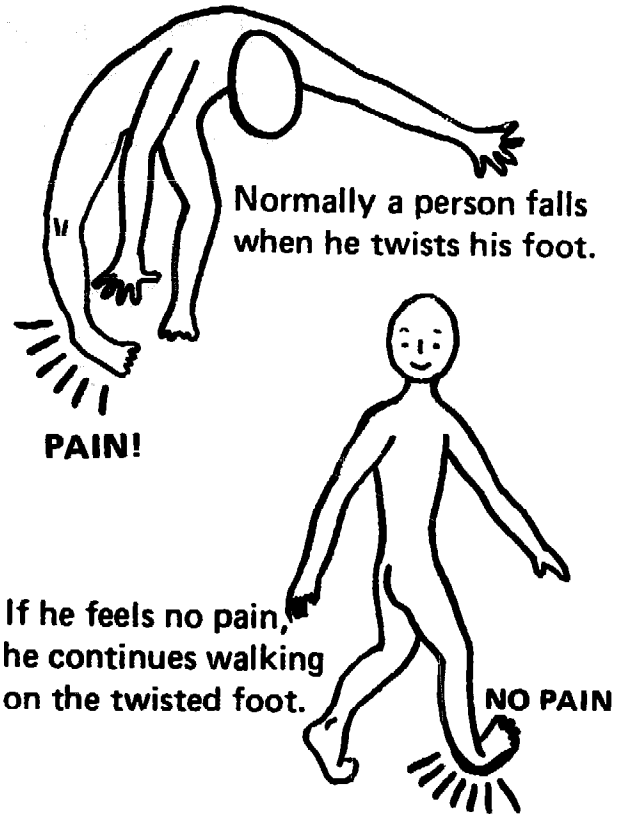
The bruise causes swelling, and the swelling bears all the weight, so multiplying the danger of further deterioration.

An anaesthetic joint will not feel this and a patient may put all his weight on a turned ankle and tear ligaments without realizing there is an injury.

Anaesthesia will delay recognition of infection just as it delays recognition of injury. As a result infection is advanced when it is recognized (osteomyelitis). When the bones are inflamed osteoporosis occurs and there is increased risk of fractures and further deformity.

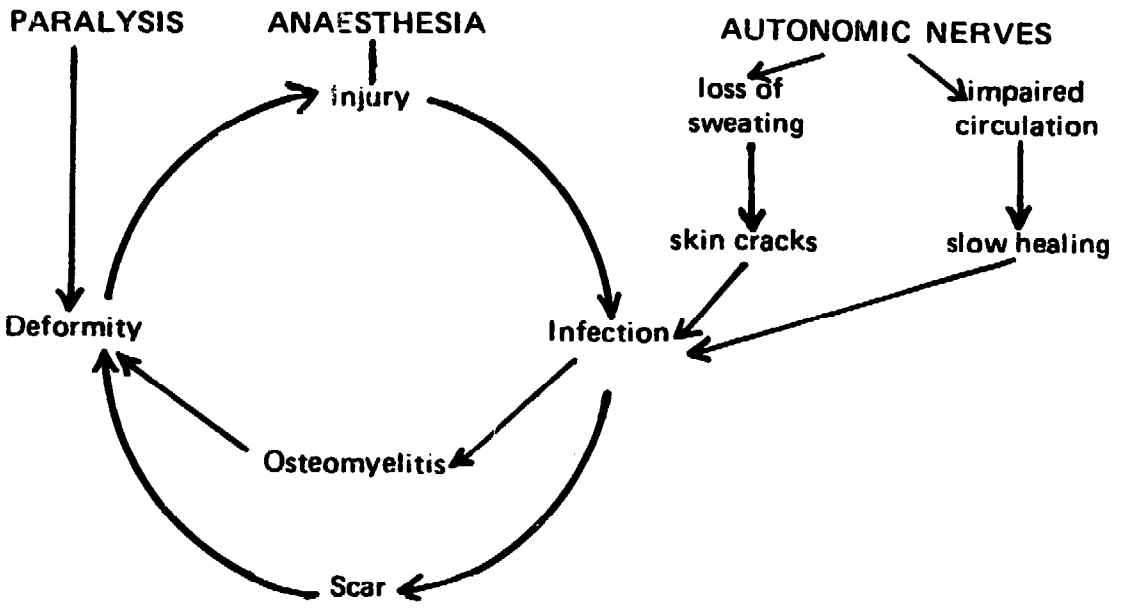
In chronic osteomyelitis pieces of dead bone are expelled by the body causing shortening of the fingers and toes.

Scar formation: through the injuries and infection scar tissue will form. Scar tissue is weaker than normal tissue and has poorer blood supply. It is more liable to new injuries resulting in more scar formation.



2. *Paralysis*
Paralysis causes muscular imbalance and leads to abnormal position of fingers and toes. These deformities result in abnormal pressure points, with increased risk of injury. Therefore paralysis increases the effects of anaesthesia.

3. *Autonomic nerves*
The autonomic nerves control the blood flow in small blood vessels and the sweat glands. Damage results in impaired circu-



lation and wounds will heal slowly. Loss of sweating results in dryness of skin and cracks easily occur. Cracks will become infected and increase the damage already done.

Leprosy patients are likely to get three types of ulcers on their feet:

1. Those due to injuries and burns, usually unnoticed until secondarily infected (because of anaesthesia).
2. Cracks of the heel and/or lateral border of the foot due to absence of normal sweating.
3. The specific plantar ulcer which is due to walking on an anaesthetic foot.

Plantar ulcers are of two types:

1. *Primary:* occurring at certain elective sites, over bony points exposed to maximum pressure and friction during walking.
2. *Secondary:* occurring over bony points exposed to maximum pressure and friction *after* the feet have become deformed by previous osteomyelitis.

In the primary ulcer the order of events is:

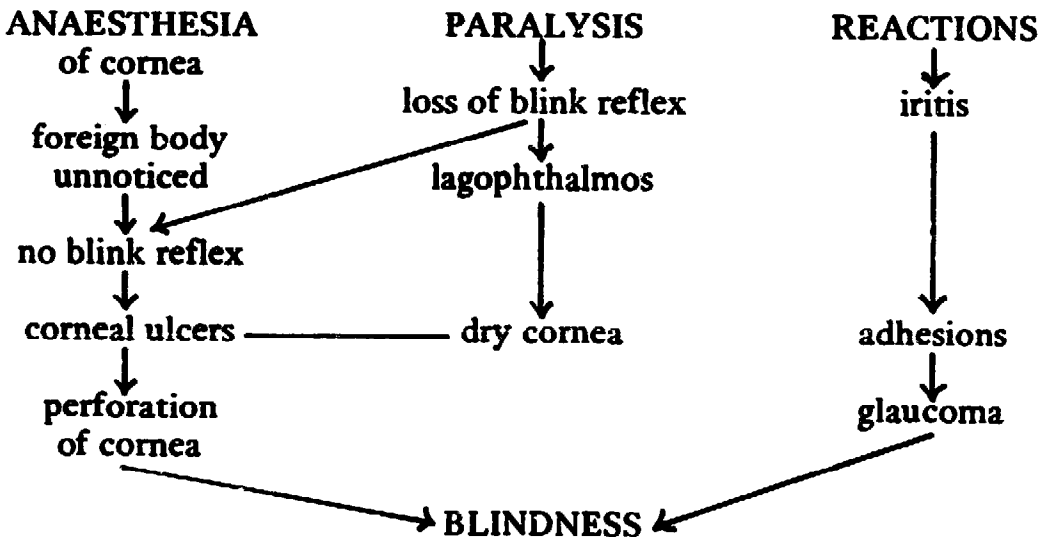
(a) An area of tender swelling, boggy but not fluctuant.
(b) A necrotic blister.
(c) An ulcer going down to the bone.
(d) Secondary infection not involving the bone.
(e) Osteomyelitis.
(f) Sequestrum formation with persistent ulceration.
(g) Further ulceration elsewhere on the plantar surface of the foot, consequent upon deformity following bone absorption or destruction (followed by secondary ulcers).

Eye problems

Eye conditions are important in leprosy because many leprosy patients have insensitive hands and feet. They are unable to use their feeling for examining objects or for walking and they need

their eyes to avoid injuries. Impairment of vision means an extra, severe, handicap. There are three main ways in which vision can be impaired.

1. Anaesthesia of the cornea—foreign bodies in the eye, not noticed, leading to corneal ulcers.
2. Paralysis of the eyelids may result in loss of blink reflex or in lagophthalmos (the inability to close the eye), dryness of the cornea, and corneal ulcers.
3. Leprosy reactions resulting in inflammation of the iris (iritis), adhesions, glaucoma, and blindness.



Diagnosis of leprosy

The diagnosis of leprosy depends on the demonstration of one or more of the following cardinal signs.

1. Hypopigmented skin patches with loss of sensation.
2. Abnormally large peripheral nerves at predilection sites with skin patches.
3. Acid-fast bacilli in skin smear.

1. *Loss of sensation*

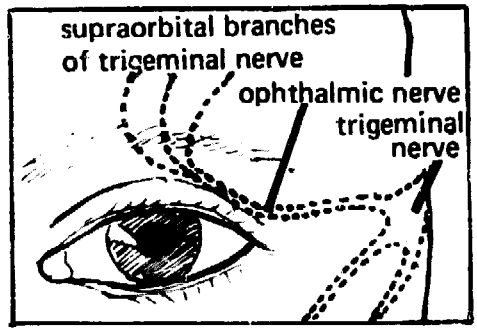
   Loss of sensation is tested with cotton wool, rolled at one end to a thin point. Explain to the patient what you are doing. Ask

the patient to undress. Have a trial on normal skin to show the patient the procedure. Ask the patient to point to the place where you touch him. Start the examination on his back, touching normal skin, and try only to touch the patches when you are satisfied that he understands the procedure. Cover his eyes when you touch parts of the body which can be seen, but reserve this till the end of the examination when he has got used to it. If a patient points more than 2 cm away from the spot you examined, we call this *mislocation.* Mislocation is the earliest sign of loss of sensation.

2. *Enlargement of the peripheral nerves*
Involvement of the peripheral nerves is most easily diagnosed at the so-called sites of predilection. There the nerves are lying superficially and enlarged nerves are most easily palpable. Always compare left and right sides of the body before you decide that a nerve is enlarged.

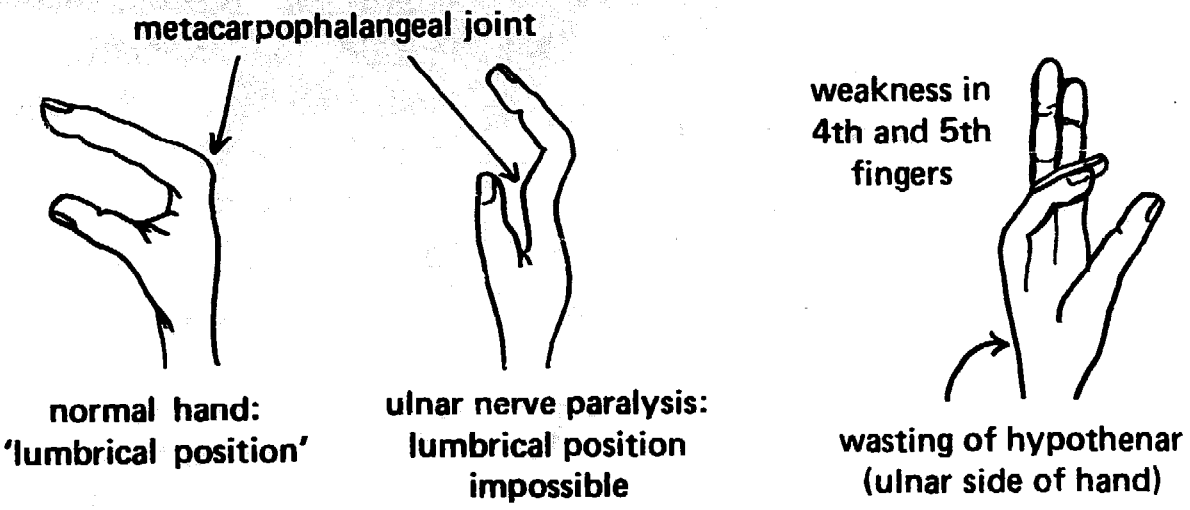
Facial nerve: Predilection site over jaw joint. If affected: lagophthalmos and/or dropped mouth. If severely damaged: palsy of all muscles of facial expression.

Trigeminal nerve:
Predilection site over eyebrows. If affected: anaesthesia of cornea.



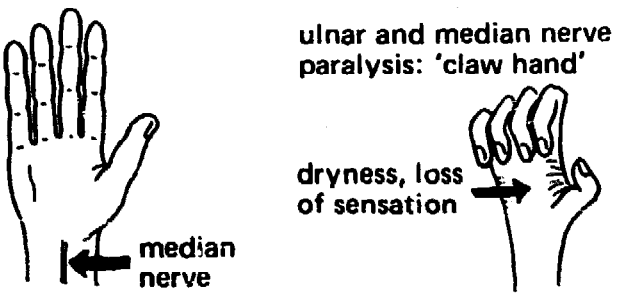
Greater auricular nerve:
Predilection site posterior border of the sternomastoid muscle halfway between origin and insertion.

Ulnar nerve: Predilection site medial side of elbow just behind the epicondyle of humerus. If affected: weakness of the 4th and 5th fingers and lumbrical position (see diagram) of these fingers impossible; wasting of hypothenar muscle; hand in PV examination position; ulnar side of the hand dry and insensitive.



Ulnar nerve damage.

Median nerve: Predilection site at the middle of the inner side of the wrist. If affected: claw hand (always in combination with ulnar palsy); dryness; insensitivity of inner hand.

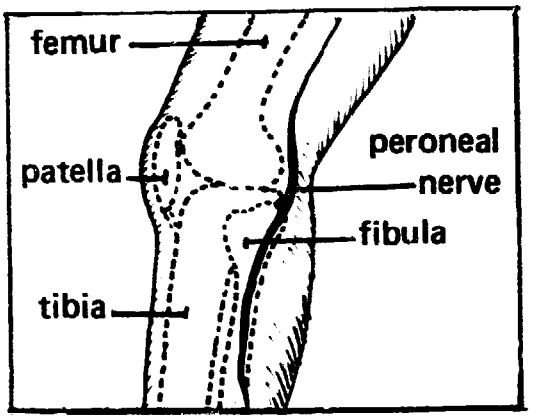


Median nerve damage.

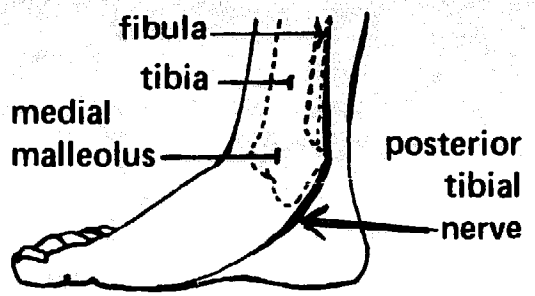
Radial nerve: Predilection site radial side of wrist; upper arm underneath biceps. If affected: drop wrist; ioss of sensation in radial part of hand.

Radial nerve damage.

Peroneal nerve: Predilection site outer side of knee joint just behind neck of fibula. If affected: drop foot; cock's gait.



Side view of the knee area showing peroneal nerve.

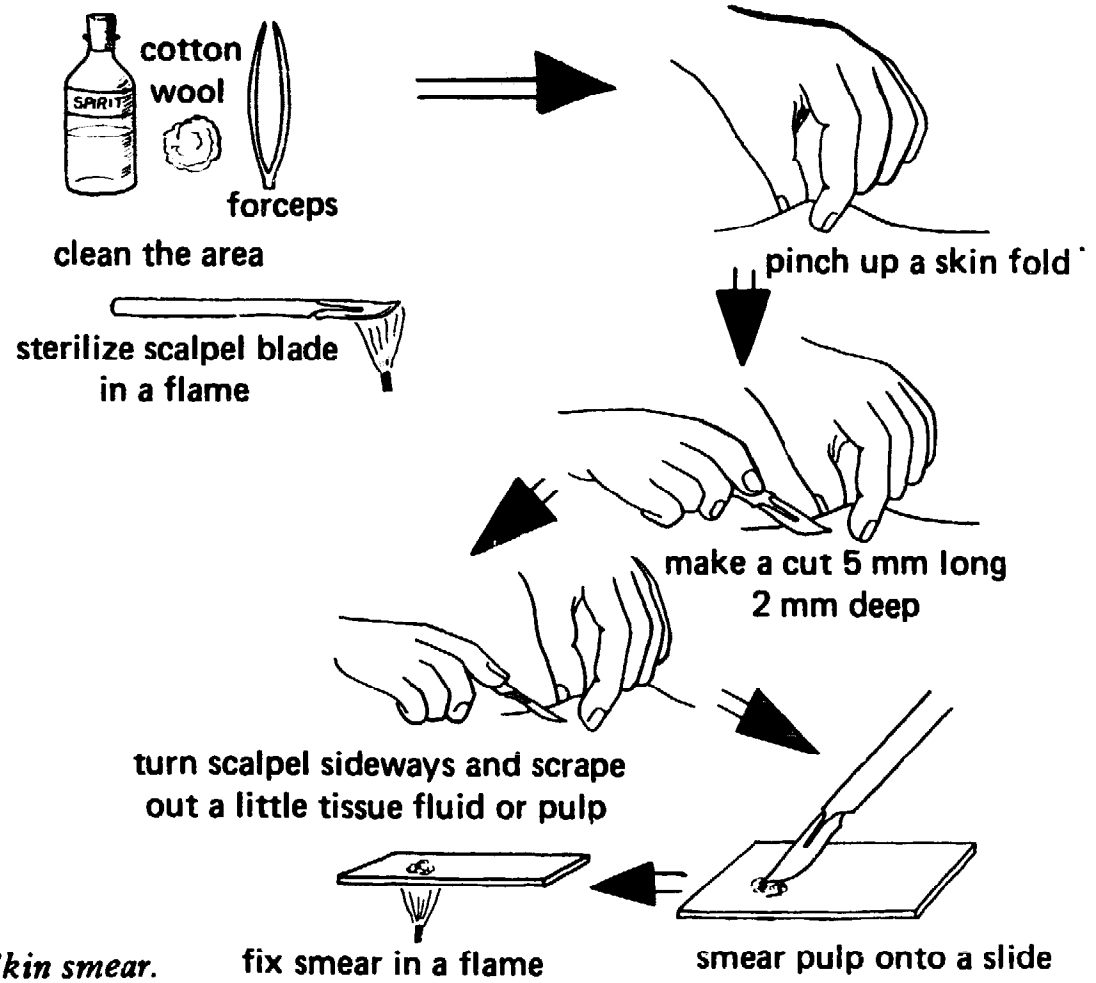


Posterior tibial nerve: Predilection site behind medial malleolus of ankle joint. If affected: clawing of toes; anaesthesia of foot.

If affected: clawing of toes, anaesthesia of foot.

3. *Skin smear*

Bacilli are most likely to be found in the edge of an active lesion (active means the lesion is spreading, raised, or red). In tuberculoid patients the chance of finding bacilli is small. For detailed description of the technique advised see: King, *A Medical Laboratory for Developing Countries*, Chapter 11.11 or King, *Medical Care in Developing Countries*, Chapter 24:53.



Skin smear.

In advanced cases diagnosis is easily made if you find loss of sensation and enlarged nerves. Differential diagnosis may be difficult in

(a) indeterminate macules
(b) early tuberculoid macules
(c) early diffuse lepromatous leprosy.

Never make the diagnosis by exclusion or by therapeutic trial. If the diagnosis is in doubt a skin biopsy should be taken (refer). In early lesions without nerve enlargement it is safe to wait some months and see the patient again. Never start treating a patient for leprosy when the diagnosis is not sure.

Classification

Classification of leprosy patients into T, B, L, or I will help you to serve your patients better. Classification helps you to know:

–Probable length of treatment.

–Complications you can expect, e.g. reversal reaction in borderline patients.

–Infectivity: BL and L cases are more likely to have infected their household members. It is more important to look for non-attending BL and L cases than to look for non-attending T cases.

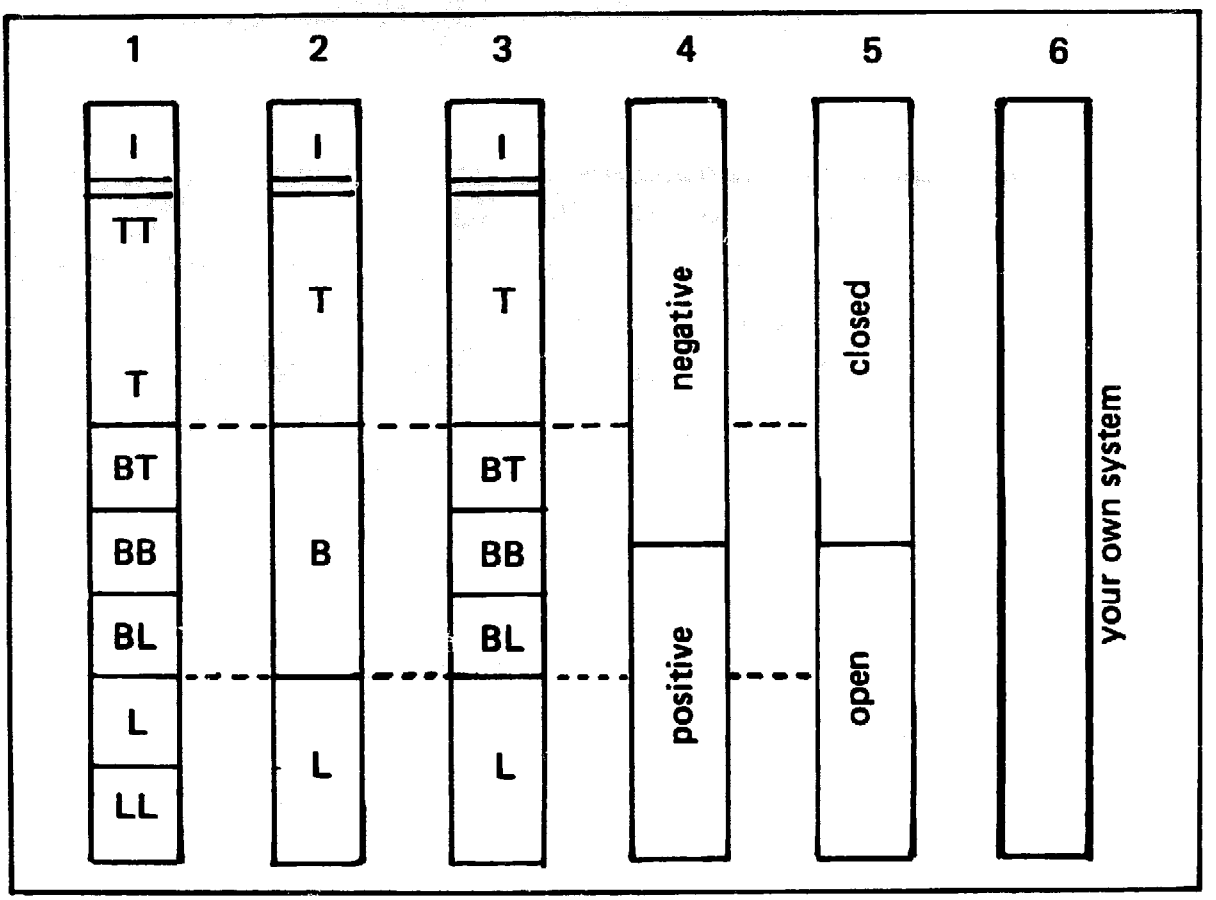
Sometimes very detailed classification systems are used. In rural areas or without a specialist knowledge it may be very difficult to classify a patient in such a system. In the table on the next page several classifications are compared.

5. *Management of the individual patient*

The one priority in the management of the leprosy patient is prevention:

–prevent nerve damage
–prevent deformity
–prevent blindness
–prevent defaulting.

Methods of classifying leprosy patients



Prevent nerve damage

Most nerve damage is caused by reactions. But anaesthesia and paralysis should not be considered as inevitable consequences of leprosy. Early diagnosis and treatment plus *adequate management of reactions* should prevent all disabilities. Once a leprosy patient is diagnosed, the possibility of reactions should be explained to him. There are the reactions which cause much preventable nerve damage, and there are often reactions which make the patient think that he is getting worse under treatment—he may therefore not report with his reactional state. The patient must be warned that reactions will occur and that immediate adequate treatment is essential.

Prevent deformity

All deformity in leprosy is either preventable or correctable. Early treatment is the essence of prevention but even patients with established nerve damage can be helped. It is essential to break

through the circle of injury-infection-deformity by:

—proper care of skin
—proper care of minor wounds and skin infections
—provision of shoes
—physiotherapy
—health education.

Skin care: If the skin is dry (loss of sweat), teach general skin care. A dry skin is likely to crack and to allow easy entry by bacteria.

1. Soak in soapy water, either cool or warm. Hot water burns the skin (anaesthesia). Soak until tissue is soft (10–15 minutes).
2. Try to remove the thick skin with a blunt object like a key, rough towel or fingernail. Never remove skin with sharp objects like a knife. Thick skin forms callus and callus gives pressure on the tissues below. Because of anaesthesia this pressure is not felt by the patient.
3. Cover the damp skin with cooking oil. In this way the water is kept in the skin.

Heel cracks:

1. Soak for some hours in water.
2. Rub with Vaseline.
3. Put on a sock.
4. Do this daily before going to bed.

The above skin care should be repeated whenever the skin seems dry but at least three times daily. Advise the patient to keep oil near the washing bowl and to apply a little after washing the hands and feet.

The care of minor wounds and sepsis of hands and feet:

A. Uninfected wounds

All patients must be constantly reminded to report *immediately* all minor wounds or burns of their hands and feet. These should be treated as soon as possible in the following manner:

(i) Wash with soap and water and nailbrush.

(ii) Soak in soapy water for 20 minutes.

(iii) Cover with a simple dressing (preferably not flavine, which delays healing).

(iv) Splint in the position of function.

Minor burns (uninfected blisters) should merely be covered with adhesive plaster and *not* opened.

B. Infected wounds

Must always be treated seriously. Careful examination is essential to decide whether it is a simple case which can be dealt with locally or whether it should be referred to hospital. It must be remembered that leprosy patients who have lost sensation get very severe infection with gross swelling of the hands and feet to a degree seldom seen in non-leprous patients, merely because of delay in recognizing their trouble. (Frequently medical staff mistake these infections for severe leprosy, with disastrous consequences.)

Treatment must be on general surgical principles and the golden rules are:

—free drainage

—soaks

—splinting.

NB. Antibiotics are the second, not the first, line of defence.

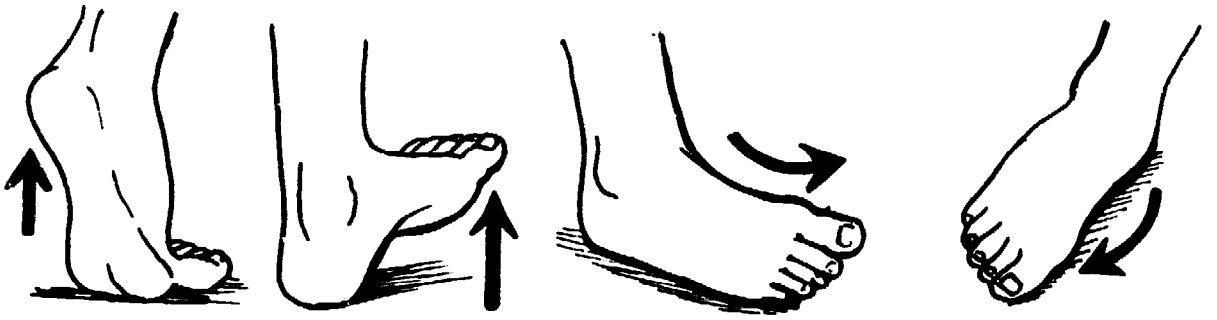
Provision of shoes: Feet that cannot feel need protection against injury. Shoes may protect leprosy patients against injury but can also increase damage if not properly fitting. Shoes too tight or too big will rub the feet and cause ulcers. Shoes which have the straps fixed with nails can cause ulcers of feet that have no feeling.

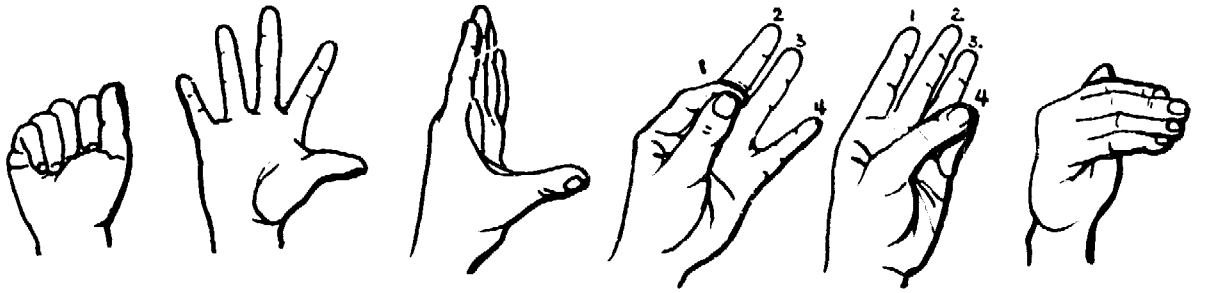
EVERY INSENSITIVE FOOT NEEDS A SHOE WITH MICROCELLULAR CUSHIONING AND A TOUGH UNDERSOLE

Physiotherapy: Joints which are not used will become stiff. Scar tissue tends to retract resulting in contractures. Muscles which are not used will shrink and result in muscle atrophy. All patients with weak or damaged hands and feet, or weak eyelids, should do suitable exercises.

Check each patient to see if he has normal ranges of motion. Exercises are of benefit in the following ways:

1. Exercises can prevent loss of the normal range of motion and prevent stiffness. Stiffness may cause the patient to injure himself and do further damage.
2. Exercises can stop further stiffness.
3. Exercises can improve or help to restore normal ranges of motion, can help to strengthen weak muscles, and can help muscles to function more effectively.





Check normal range of motion in each patient.

There are two types of exercises: passive and active.

Passive exercises are done with the other hand or by another person. They are useful because they can lengthen ligaments and thus loosen stiff joints and can lengthen skin and muscles. Patients should do passively whichever normal motion they cannot

do actively because of stiffness or weakness.

Active exercises are done with muscles which are weak. Only active exercises can strengthen weak muscles and help the patient. Only weak muscles can be strengthened by active exercises. Completely paralysed muscles cannot be strengthened by any type of exercise.

DO PASSIVELY WHAT CANNOT BE DONE ACTIVELY

Exercises should not be done when injuries, infection, or cracked skin are present, or when nerves are painful. If the skin cracks when a patient is exercising, splint the hand or foot as straight as possible. After the skin heals the scar will be long enough to allow as much movement as possible.

If the aim of the exercise is to prevent or stop further stiffness, each movement should be done 5–10 times each day. This is enough provided each part is moved through a full range of movement.

If the aim of the exercise is to strengthen weak muscles and improve the functioning of muscles, the more often the exercises are done the better.

But, of course, they must be done carefully so the skin does not split or crack. Start with very gentle exercise. Gradually increase to several periods of maximum effort each day. Help your patient to set the most convenient time for the exercise each day. This will help him to be regular with his exercises. If patients learn to do their exercises well and regularly they can usually expect good results. Patients may even learn to do some hand exercises while walking about.

Examples of exercises for different conditions are given in Appendix 1.

Health education: All leprosy patients with loss of sensation should be taught how to prevent further damage by avoiding injuries and infection.

Hands—Teach patient to think before acting, to look before acting, hot or cool? sharp or blunt? dangerous or safe?

—Teach about gripping tools too strongly.

—Teach home exercises (physiotherapy).

Feet —Teach every leprosy patient with loss of sensation to wash and inspect his feet daily; to detect minor injuries or ulceration.

—Teach the value of shoes and of not walking too far.

—Teach first aid for small injuries.

—Explain the dangers of closed injury to the feet.

—Teach patients not to stand too long on one foot.

Teach personal hygiene and awareness of infectiousness of nasal discharges.

Prevent blindness

Ask every leprosy patient to close his eyes once during his visit . Do this not only during the first examination, but regularly during the many years he attends for treatment. In doing so you will detect cases of lagophthalmos early and you can refer the patient for treatment in time.

In every patient with a patch of leprosy in the face test the sensation of the cornea. If the cornea is insensitive the patient will not feel foreign bodies in the eye and scars from corneal ulceration will lead to serious loss of sight. Constant awareness of the possibility of eye damage is essential. Refer patients with red eyes immediately.

IN ALL LEPROMATOUS PATIENTS
LOOK ROUTINELY FOR DILATED CILIARY VESSELS
AND CHECK THEIR VISION

Prevent defaulting

There may be many reasons why a patient gives up his treatment.

Examples and suggestions on how to overcome these problems are given below:

No confidence in treatment	Explain to every new patient that leprosy is curable.
Shame	Explain to every new patient that leprosy is a disease like any other.
Thinks he has not got leprosy	Explain to every new patient that he is suffering from leprosy and what can be done to cure him.
Thinks he is cured	Explain to all patients that macules may disappear, and they may feel better, but that treatment must not be stopped until the patient is declared cured by the doctor.
Treatment too long	Explain to all patients why treatment has to be continued and what the dangers are of stopping treatment.
Clinic too far	Supply drugs for 2 or 3 months if patients are living far away.
Irritations over treatment	Order your DDS in time to avoid running out of stock; organize your clinic in such a way that waiting time is as short as possible. Treat leprosy patients yourself. Give proper advice on ulcer care. Give adequate treatment for tender nerves.
Disability	Provide adequate ulcer care. Refer patients with reactions for adequate treatment.
Ignorance	Explain to every new patient for the first 3 or 4 visits that leprosy is curable provided the patients take their drugs regularly for many years until they are declared cured.

One of the reasons patients 'default' or do not turn up for treatment is because of ill-mannered, bad-tempered medical workers. If the patient has missed a treatment the medical worker may speak rudely and abuse him. It is far more effective to explain calmly,

yet again, the absolute necessity of regular treatments. The patient must feel that the medical worker is understanding and helpful. He must not be frightened away from the clinic. Unfortunately, however much the medical worker tries to encourage all patients to attend, there will always be a few who come irregularly. Try to prevent this by:

1. A fixed day for clinics, well organized.
2. Health education to all new patients (see above).
3. Home visits to all patients living nearby.
4. Contacting patients through village chairman if they live far away.
5. Tracing all non-attenders immediately.

Outpatient care

It is the task of every hospital, health centre, and dispensary to take care of all chronically diseased patients in their catchment area. This is achieved most easily by holding a clinic for chronic diseases on a fixed day of each week. Such a clinic can take care of leprosy, tb, and other chronic patients (e.g. epileptics). The patients attending such a clinic should be given priority on that special day. But if, for one reason or another, they come on a day other than the fixed clinic day, do not refuse treatment but advise them to come next time on the clinic day. To refuse treatment to a patient who has walked a long way to come to the health centre (and then tell him to come back tomorrow or the day after) will destroy the doctor-patient relationship. Even better than a weekly clinic is a daily clinic in which the most senior staff member sees all cases referred from OPD and MCH clinics, together with a combined clinic for leprosy and tb cases. This will remove the stigma of attending 'special clinics'.

The work should be divided among all staff. The normal health centre routine can be used—dressing ulcers in the dressing room, dispensing tablets at the dispensary—but the attendance register should be filled by the most senior member of the team, i.e. the person examining the patient. The requirements for leprosy outpatient care are described in Appendix 2. There are many

advantages to outpatient care. If a patient can be treated while he lives at home there will be no problem with readjusting to home life after a long stay in a hospital. Patients prefer outpatient treatment and will therefore report earlier in the course of his disease. Infectivity is nil after a few months of treatment. So outpatient treatment gives 'chemical' isolation. Isolation in hospital is not necessary. Hospital treatment, if indicated, should be as short as possible. There is no need to admit patients in a special leprosy ward. Special leprosy wards, leprosy hospitals, and leprosy clinics contribute to the idea that leprosy is something special, thus making it more difficult for the patient to continue a normal life in the community.

Drug treatment

The first-line drug for the treatment of leprosy is dapsone (DDS or Diamino-Diphenyl-Sulphone). Dapsone is very effective against M. leprae and has almost no side effects. The drug is so cheap that the price is not a significant factor in treatment. Possible side effects are: dermatitis, haemolytic anaemia, and acute psychosis (depression).

Up till 1976 dapsone was started with low doses weekly and increased slowly to a maximum of 300 mg weekly. This was done because it was thought that to start with high doses might precipitate reactions. Recent research has shown that more and more leprosy bacilli have become resistant to DDS. The assumed reason for this is the administration of weekly doses, the practice of slowly increasing dosage, and irregular taking of drugs. Trials have now been done with daily DDS treatment. No more severe reactions have been noticed than with the weekly treatment. It is anticipated that a schedule of daily DDS treatment will soon be advised. For the most recent policy on DDS treatment, enquire about the Ministry guidelines from your DMO. The recommendations to the Ministry are:

patients with positive skin smears: 100 mg DDS daily

patients with negative skin smears: 50 mg DDS daily

children up to 12 years, all cases: 25 mg DDS daily

50 mg tablets are to be ordered by Central Medical Stores.

Treatment must continue until the patient is cured. Leprosy is regarded as cured when the undermentioned signs of inactivity are present for a number of years:

- –absence of new lesions and no change in appearance of existing lesions
- –flattening of raised lesions, absence of redness, and appearance of atrophy in all lesions
- –no nerve tenderness, no change in nerve thickening, no change in extent of area of anaesthesia
- –absence of all signs of reactions
- –negative skin smears.

Leprosy is regarded as completely cured and anti-leprosy treatment may be stopped when all these signs have been present for 3 years in true tuberculoid, 5 years in indeterminate, tuberculoid, and borderline tuberculoid (I, T, and BT), and 10 years in borderline (BT and BB). In BL and lepromatous leprosy it is best to continue treatment for life. Signs of new activity in a patient mean he has relapsed and treatment should be started all over again.

Treatment of complications

Reactions–see p 320 for classification and details.

Type I reversal reactions

- –refer to hospital immediately.

Type II or ENL

- –mild reactions can be treated in the health centre with anti-inflammatory drugs: Aspirin 300 mg tds and chloroquine 300 mg bd for 7 days and 300 mg od thereafter
- –rest is a very important healer in cases of neuritis
- –severe reactions must be treated with corticosteroids and other drugs: patients must therefore be referred to hospital.

DO NOT STOP DAPSONE
This used to be done in former years but is no longer recommended.

Indications for referral are:

- –mild reaction not getting better after 4 weeks' treatment at the health centre
- –mild reaction getting worse after one or two weeks' treatment at the health centre
- –all patients with newly swollen and painful nerves (neuritis)
- –all patients with newly swollen hands, feet, or face (oedema)
- –all patients with new paralysis
- –all patients with fever for more than 3 days, not improving on chloroquine and Aspirin.

Eye complications

Iritis: Refer urgently; give atropine eye ointment or drops if available.

Corneal ulcer: Chloramphenicol eye ointment, refer urgently.

Conjunctivitis: Chloramphenicol eye ointment 4-hourly, refer if no improvement after 2 days.

Lagophthalmos: Exercises: frequent forced closure of eyelids while the hands are holding the skin of the forehead. This exercise should be done 20 times, three times daily.

Patients who cannot close their eyes completely should do the following exercise: place the hands on the cheek and forehead to fix the muscles. Then close the eyes as tightly as possible. Keep

Eye exercise for lagophthalmos.

them closed and count slowly to 10. Do this at least 40 times and twice a day The more often it is done, the better the results.

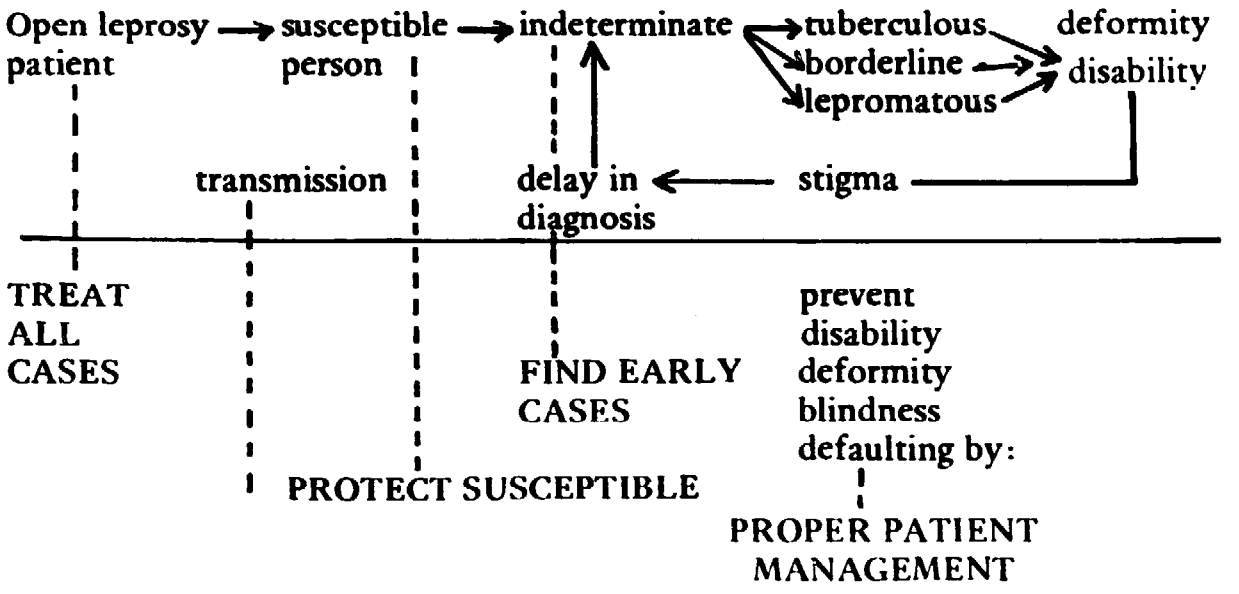
Prevention of drying of the cornea: put drops of saline or water in the eyes frequently and use Vaseline at night.

Refer for surgical correction if insufficient or no improvement rsults from the exercises.

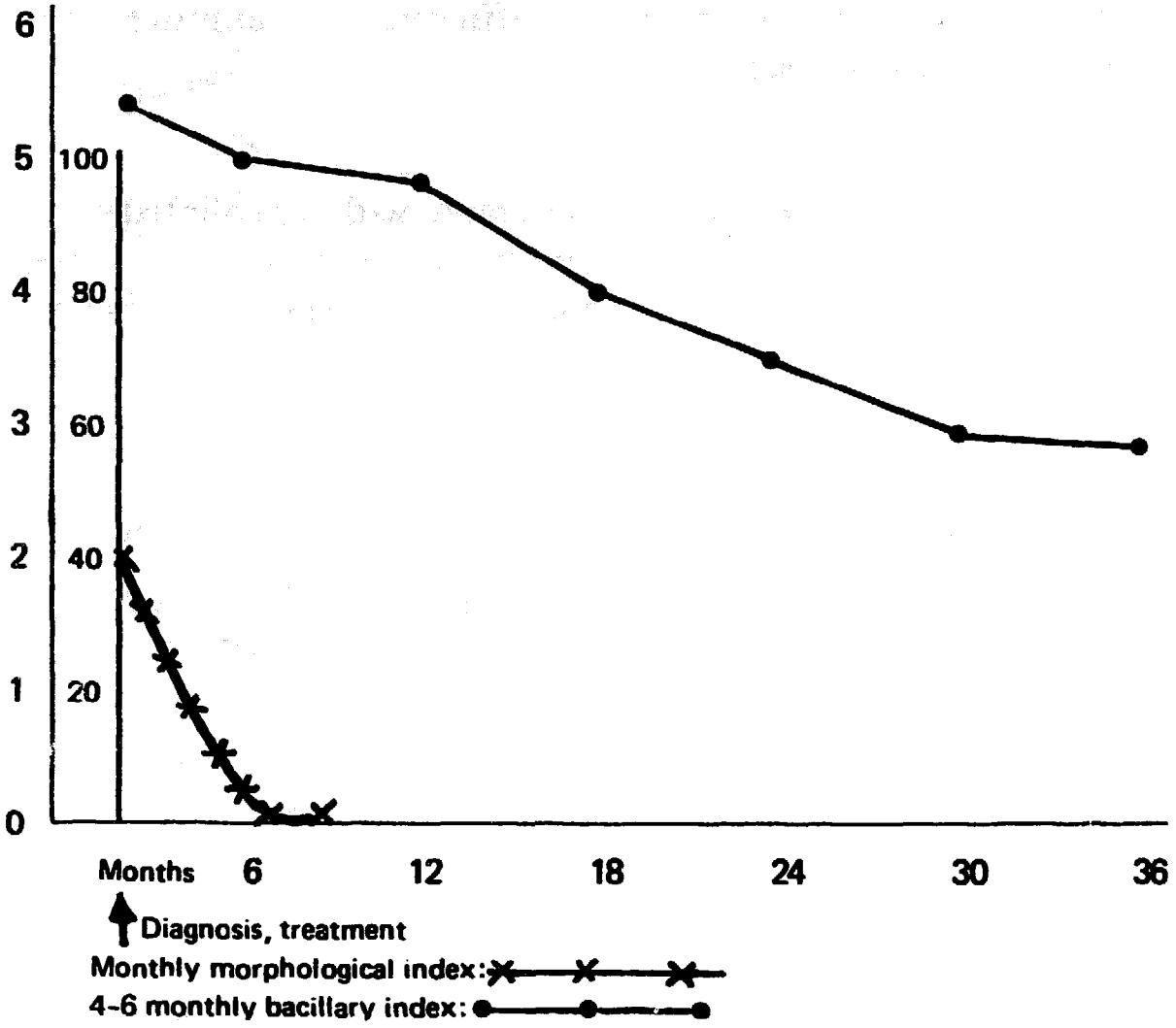
Plantar ulcers

1. In the acute stage rest in bed, preferably with leg splinted on a pillow. In the pre-ulcer stage complete recovery will occur after ten days; once an ulcer has formed recovery will occur after six weeks.
2. Antibiotics may be necessary but only if there are general symptoms.
3. Soaking twice daily and simple dressings are usually sufficient.
4. Osteomyelitis must be treated on general surgical principles, but one should be as conservative as possible with sequestrectomy and amputation.
5. When all this has been done a walking plaster cast can be applied inside which the ulcer will heal spontaneously after six weeks.

6. *Control*



Reduce the number of infective cases. To interrupt transmission it is essential to treat the infective cases. These are the B and L cases. After a few months of treatment the number of viable bacilli is nil.



Number of viable bacteria after start of treatment.

It is essential to continue the treatment. Active case holding is required. This can only be achieved by proper registration and recording, combined with follow-up of non-attenders by home visits. If you have a health auxiliary attached to the health centre or dispensary, it is his duty to look for the non-attenders, to find out (politely) why they did not attend, and to stimulate them to collect their drugs.

If you do not have a health auxiliary, other methods must be devised. If there are village medical workers in your area it is their

duty to see the patients. This should be easy for them as they do not need to travel far since they are living in the villages themselves. When these arrangements are not possible, try to motivate the village chairmen to help in tracing non-attenders. Inform them about patients who have been lost sight of and keep on until answers are obtained from them.

Reduce opportunity for contact:
Leprosy transmission is favoured by overcrowding, poor housing and low socio-economic conditions. The incidence of leprosy will go down when standards of living improve, i.e. better houses and correction of overcrowding.

B and L patients must be taught personal hygiene with regard to their nasal secretions.

Reduce the number of susceptible people:
There is no effective vaccination against leprosy, but *BCG vaccination* is of proven value. It stimulates the production of cell-mediated immunity in those individuals who are able to produce this immunity. These are the people who would get tuberculoid or self-healing indeterminate leprosy when exposed to the M. leprae itself.

BCG has no protective value in individuals who will develop borderline or lepromatous leprosy on exposure.

Drug prophylaxis with DDS is effective but it is impractical to use chemoprophylaxis on a country-wide scale.

Early diagnosis and treatment:
It is impossible to raise socio-economic standards rapidly. Therefore leprosy control is based primarily on the early treatment of as many cases as possible. The aim should, of course, be 100% of the cases. It is generally accepted that control only becomes effective when at least 75% of all infective cases have been found and are taking their drugs at least 75% of the time.

This cannot be achieved by offering treatment only to those who themselves report for treatment. About 25–50% of all cases report spontaneously—on average two years after clinical signs of the disease have been recognized.

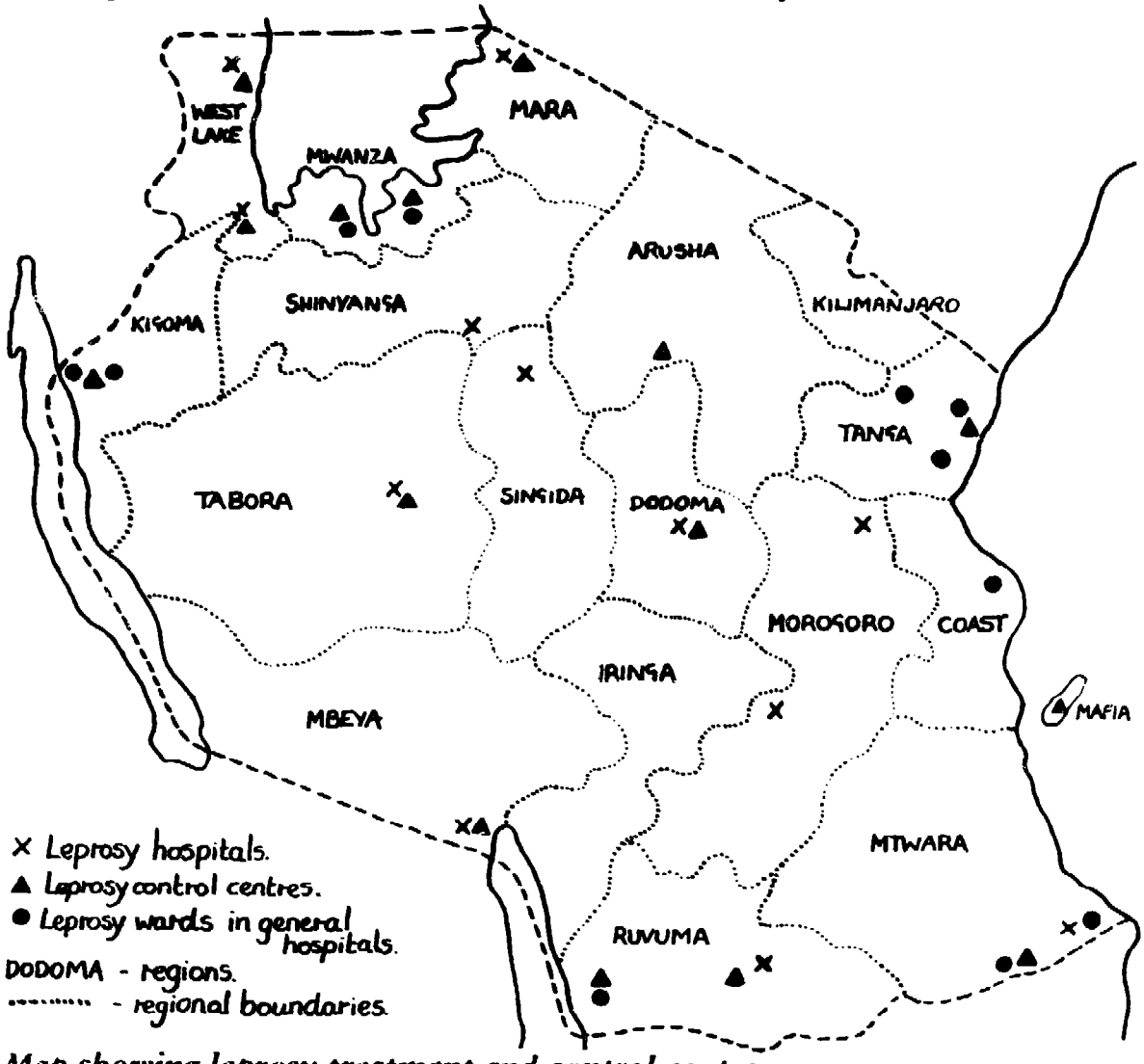
To increase the number who come spontaneously for treatment, extensive health education to the public is necessary. To increase the total number of diagnosed cases active case finding is necessary:

—examine all contacts of known B and L cases

—organize school surveys and village surveys

—be constantly aware of the possibility of leprosy when examining patients.

Only when all health workers diagnose and treat leprosy in all health institutions can the disease be eradicated. Treatment of leprosy should be given as close as possible to the place where the patients are living, that is, in the villages by village medical workers, in dispensaries by RMAs, in health centres by MAs.



Map showing leprosy treatment and control centres.

7. Action

Find out whether the community would accept a control programme. If not, start a careful education programme.

- Get preliminary information about leprosy
  - —observe people in your area for leprosy
  - —review previous reports and statistics about leprosy
  - —find out the attitude of your people towards leprosy.
- Prepare what you need to run a leprosy clinic
  - —inform your DMO and regional leprosy officer
  - —order dapsone, case record cards, attendance register
  - —enquire where you can send your complicated cases.
- Coach your staff
  - —how to deal with leprosy patients
  - —how to spread propaganda.
- Start making propaganda (after consultation and in co-operation with the local leaders)
  - —that leprosy can be cured
  - —that you will treat leprosy on an outpatient basis.
- Register and treat all known leprosy cases
  - —establish a regular leprosy treatment service
  - —provide health education
  - —help patients with late complications
  - —follow-up all defaulters.
- Search for unknown cases
  - —get co-operation from local leaders to find suspicious cases
  - —organize school and village surveys once yearly
  - —encourage all patients to bring their family members for a check-up.
- Organize a contact person (village medical helper, teacher,

village chairman) in every village. They can distribute treatment to the patients, so that no patient needs to walk more than one hour to a treatment centre.

Evaluate your service.

8. *Summary*

Leprosy is a chronic infectious disease of man caused by M. leprae and by the tissue reaction against the leprosy bacillus. It mainly affects the peripheral nerves and skin.

The disease is transmitted by close contact with an infectious case (B or L). Paralysis, anaesthesia and the consequent cycle of injury and infection cause deformity and disability. Reactions and eye complications may hasten this disabling process. Diagnosis, either clinical in T cases (i.e. patches with anaesthesia, nerve enlargement) or clinical + skin smear in B and L cases. Management should concentrate on prevention of disability by regular drug treatment with dapsone. Control is mainly by case finding and case holding. Protection by BCG vaccination is limited.

Chapter 8—Appendix 1

Exercises for hands and feet

Claw hand

Passive exercise: The patient places the back of his claw hand on a flat padded surface like his own thigh. With the side of his other hand he tries to push the four claw fingers straight. Since the patient often does not feel how hard he is pushing, he must start gently and gradually increase his pressure. Otherwise he may tear the skin on the palm side of his contracted fingers.

Active exercise: The patient places the back of his claw hand on a flat padded surface like his own thigh; with his other hand he holds the palm of his clawed hand tight against the surface.

He now actively straightens his four fingers as well as he can, using the muscles of his clawed fingers.

Claw thumb

Passive exercise: The patient places the back of his claw hand on his thigh. He takes hold of his claw thumb between the thumb and index finger of his other hand and pulls the claw thumb out straight towards the end of his hand. He must keep the rest of his thumb and hand down on the surface.

Active exercise: The patient places the palm of his claw hand on his thigh. With his other hand he holds the metacarpal bone (inside the palm) away from his fingers. He then straightens the end joints of his thumb using the muscles of the thumb itself.

Claw foot

Passive exercise: The patient puts the heel of his foot on a chair or box. He grasps the outside (little-toe side) of his foot just behind the toe. He pulls up towards his knee, on the outside of the foot only, so as to turn the sole of his foot outward.

Drop foot

Passive exercise: The patient stands about ½ to 1 metre from a wall, facing the wall. He must keep his feet flat on the floor throughout the whole exercise. He leans against the wall and tries to let his hips come towards the wall. His heels must stay on the floor. He should hold this position for 30 seconds or longer. If he has lost feeling in the soles of his feet, he should keep his shoes on during the exercise.

Chapter 8—Appendix 2

Requirements for leprosy outpatient care

Administrative

1. Patient cards.
2. Leprosy treatment and attendance register (see below).
3. A system for tracing patients who have been lost sight of.

Medical

1. Dapsone tablets (25 and 100 mg); 50 mg tablets are in preparation.
2. Drugs for other skin diseases (e.g. Whitfield's ointment).
3. Cotton wool for testing anaesthesia.
4. Requirements for skin smears.
5. Empty penicillin bottles for patients to take their dapsone.
6. Materials for care of plantar ulcers, empty debe for soaking feet, soap and nailbrush, oil or Vaseline, adhesive plaster.

Treatment and attendance registers to be used in registration of treatment of chronic diseases like leprosy and tb are available from Tanganyika Mission Press, P.O. Box 34, Tabora.

1. The first column is blank so that you can write your own serial numbers and so easily count the total number of patients in your clinic.
2. The registration number must never be changed. If the patient is a previously untreated case, underline this number in red.
3. On the right-hand page enter the dose of dapsone. If you give a supply for several weeks enter each week's dose in the appropriate column and draw a ring around the dose taken at your clinic on the day of attendance. As a general rule you should not give the drugs to people other than the patients themselves. Often relatives or friends will come and ask for the tablets because the patient is sick at home. Be very careful and give only one week's supply. Don't put a ring around this dose in the treatment register, and advise the relatives to bring the patient because sick leprosy patients are in urgent need of examination of the cause of illness and appropriate treatment.
4. After the clinic make a careful list of patients who have not attended for two months and send their names to the Balozi, or your auxiliary.
5. Add up total attendances and total drugs used at the bottom of the page each week.

6. At the end of each year note carefully:
   (a) those who have died
   (b) those who have been discharged
   (c) those who have been transferred
   (d) those who have not attended even once during the year
   (e) write a new list of patients to start on January 1st including all patients except these four groups. Do not change any registration numbers.

Suggestions for care of leprosy patients in a combined chronic diseases clinic:

1. The most senior staff member should conduct the clinic, preferably the same person every day or week.
2. Patients should enter the examination room one by one.
3. Examine all patients for signs of reactions; always check for eye complications; always examine ulnar and radial nerves.
4. Do not write routine treatment on the patient's card but enter dose given in registration book.

Chapter 8—Appendix 3

Indications for referral to hospital

1. Acute eye conditions, i.e. patients with:
   - lagophthalmos —less urgent
   - iridocyclitis —urgent
   - corneal ulcers —urgent.
2. Reactions, i.e. patients with:
   - acute neuritis —urgent
   - sudden paralysis —urgent
   - oedema —urgent
   - ulcerating patches, nodules —urgent.
3. Septic ulcers and osteomyelitis —less urgent.
4. ENL with fever making a patient ill or ENL of more than one month's duration —less urgent.
5. Conditions which require surgical correction, for instance, amputations, tendon transfers, arthrodesis —not urgent, make appointment.
6. All other conditions, not caused by the leprosy itself, which may require admission.

Appendix A

The main communicable diseases of Tanzania and their importance

Mode of transmission	Very important and/or common	Important	Significant but occasional or local only	Others
Contact	Scabies		Trachoma	Ringworm Thrush Conjunctivitis Ectoparasites
Sexually	Gonorrhoea	Syphilis Non-gonococcal urethritis		Chancroid Genital sores Trichomoniasis
Vectors	Malaria	Relapsing fever Schistosomiasis	Trypanosomiasis Plague	Wuchereriasis Onchocerciasis Yellow fever
Faecal contamination (excluding helminths)	Unspecified diarrhoea	Bacillary dysentery Amoebiasis	Cholera Typhoid	Poliomyelitis Viral hepatitis Food poisoning
Intestinal helminths	Hookworm	Ascariasis		Enterobiasis Trichuriasis Taeniasis Strongyloidiasis
Contact with animals		Tetanus Rabies		Brucellosis Anthrax Hydatidosis
Airborne	Measles Acute upper respiratory tract infections Pneumonia	Streptococcal infections Whooping cough Meningitis		Chickenpox/ Smallpox Rubella Mumps Staphylococcal infections
Chronic	Tuberculosis Leprosy			

Appendix B

NOTIFICATION

Notification means that you immediately inform the local health authorities (DMO) that you suspect a patient is suffering from an infectious disease. The authorities can then take measures to have your suspicion confirmed and to prevent the disease from spreading.

Notification must be done immediately and by the most rapid means. When you do not have a telephone, it is best to send a messenger to the DMO's office.

Some diseases spread so quickly that they need international control measures. These diseases will be reported by the authorities to the World Health Organization.

Internationally notifiable diseases are:

CHOLERA
PLAGUE
SMALLPOX
YELLOW FEVER

DO NOT REFER, BUT ASK DMO TO COME

These diseases are so infectious that in case of suspicion you should *not* refer the patient to the hospital to get the diagnosis confirmed, but you should report the case and request your superiors to come to see the patient. Referral would result in infection of others and make spread of the disease easier.

The Tanzania Ministry of Health also wants to be informed immediately about persons suffering from:

MENINGOCOCCAL MENINGITIS
ACUTE POLIOMYELITIS
INFLUENZA (not 'flu')

REFER FOR MANAGEMENT

Patients with meningitis and polio should be treated in a hospital. Those two diseases will not spread so fast, therefore you may refer the patients. In case of notification provide the following minimal information:

Name of patient
Age
Sex
Name of Balozi
Village
Suspected diagnosis
Short history.

Other measures to be taken are discussed under control of each individual disease.

Appendix C

Diseases for which immunization is possible

Disease	Type of immunization	Indication for use/comments
Anthrax	Killed bacteria	Extensive exposure in certain occupations
Cholera	Killed bacteria	International travel (Zambia–Malawi)
Diphtheria	(a) Toxoid (DPT)	Routine for all under-fives
	(b) Antitoxin	Treatment of diphtheria
Hepatitis (infectious)	Immunoglobulin	Expensive, limited protection
Gas gangrene	Antitoxin	Exposure and treatment
Influenza	Live attenuated virus	No priority in Tanzania
Leprosy	BCG	See Tuberculosis
Malaria	Irradiated sporozoites	Experimental only
Measles	Live attenuated virus	Routine for all under-fives
Meningitis (meningococcal type A)	Killed bacteria	Control of outbreaks
Mumps	Live attenuated virus	No priority in Tanzania
Paratyphoid	Killed bacteria (TAB)	See typhoid
Plague	Killed bacteria	Control of outbreaks
Pneumonia (pneumococcal)	Killed bacteria	Adequate treatment more reliable
Polio	Live attenuated virus	Routine for all under-fives and schoolchildren
Rabies	(a) Inactivated virus	Exposure
	(b) Hyperimmune serum	Severe exposure
Rubella	(a) Live attenuated virus	No priority in Tanzania
	(b) Immunoglobulin	Exposure in pregnancy

Smallpox	Live attenuated virus	Routine for all under-fives International travel Control of outbreaks Eradication
Snake bite	Polyvalent antiserum	Signs of intoxication
Tetanus	Toxoid DPT	Routine for all under-fives
	TT	Routine in pregnancy and schoolchildren Booster after exposure Full course when ATS is given
	Serum ATS	Tetanus prevention in previously unvaccinated Treatment of tetanus
Trachoma	Inactivated chlamydiae	None, effectiveness in doubt
Typhoid	Killed bacteria—TAB	Personal protection of individuals
Tuberculosis	Live attenuated bacteria BCG	Routine for all under-fives Booster at primary school
Whooping cough	Killed bacteria (DPT)	Routine for all under-fives
Yellow fever	Live attenuated virus	International travel Control of outbreaks

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