CONTENTS

## PRINCIPLES OF COMMUNICABLE DISEASE CONTROL

**Introduction**

- ✓ In this you will look at the main methods (also known as principles) that are used to control the occurrence and spread of communicable diseases.
- ✓ It is a short but intense section which will give you the foundation you need to prevent and control the diseases you shall cover in subsequent lessons
- ✓ Communicable are diseases spread from one person to another or from an animal to a person.
- ✓ The spread often happens via airborne viruses or bacteria, but also through blood or other bodily fluid.

## Basic definitions related to communicable diseases

**Infection**: Infection is the entry and development or multiplication of an infectious agent in the body of man or animals. An infection does not always cause illness.

**Contamination**: The presence of an infectious agent on a body surface, on or in clothes, beddings, surgical instruments or dressings, or other articles or substances including water and food

**Infestation**: It is the lodgment, development and reproduction of arthropods on the surface of the body or in the clothing, e.g. lice, itch mite. This term could be also used to describe the invasion of the gut by parasitic worms, e.g. Ascariasis

**Contagious disease**: is a disease that is transmitted through contact. Examples include scabies, trachoma, STD and leprosy

**Host**: A person or an animal that affords subsistence or lodgement to an infectious agent under natural conditions.

An animal or an organism,or human being that harbours an infectious agent or disease

**Vector**: An insect or any living carrier that transports an infectious agent from an infected individual or its wastes to a susceptible individual or its food or immediate surroundings

**Reservoir**: Any person, animal, arthropod, plant, soil, or substance, or a combination of these, in which an infectious agent normally lives and multiplies, on which it depends primarily for survival, and where it reproduces itself in such a manner that it can be transmitted to a susceptible host. It is the natural habitat of the infectious agent.

## Incidence and prevalence of infectious diseases

**Incidence of an infectious disease**: number of new cases in a given time period expressed as percent infected per year (cumulative incidence) or number per person time of observation (incidence density).

**Prevalence of an infectious disease**: number of cases at a given time expressed as a percent at a given time. Prevalence is a product of incidence x duration of disease, and is of little interest if an infectious disease is of short duration (i.e., measles), but may be of interest if an infectious disease is of long duration (i.e. chronic hepatitis B).

**Epidemic**: The unusual occurrence in a community of disease, specific health related behavior, or other health related events clearly in excess of expected occurrence

**Endemic**: It refers to the constant presence of a disease or infectious agent within a given geographic area or population group. It is the usual or expected frequency of disease within a population.

**Pandemic and Exotic**

An epidemic usually affecting a large proportion of the population, occuring over a wide geographic area such as a section of a nation, the entire nation, a continent or the world, e.g. Influenza pandemics.

Exotic diseases are those which are imported into a country in which they do not otherwise occur, as for example, rabies in the UK

**Sporadic**

- The word sporadic means “scattered about”. The cases occur irregularly, haphazardly from time to time, and generally infrequently. The cases are few and separated widely in time and place that they show no or little connection with each other, nor a recognizable common source of infection e.g. polio, meningococcal meningitis, tetanus….

- However, a sporadic disease could be the starting point of an epidemic when the conditions are favorable for its spread

**Zoonosis, epizootic and enzootic**

- Zoonosis is an infection that is transmissible under natural conditions from vertebrate animals to man, e.g. rabies, plague, bovine tuberculosis…..
- An epizotic is an outbreak (epidemic) of disease in an animal population, e.g. rift valley fever.
- An Enzotic is an endemic occurring in animals, e.g. bovine TB

**Nosocomial infections**

Nosocomial (hospital acquired) infection is an infection originating in a patient while in a hospital or another health care facility. It has to be a new disorder unrelated to the patient's primary condition. Examples include infection of surgical wounds, hepatitis B and urinary tract infetions

**Opportunistic infection**

This is infection by organisms that take the opportunity provided by a defect in host defense (e.g. immunity) to infect the host and thus cause disease. For example, opportunistic infections are very common in AIDS. Organisms include Herpes simplex, cytomegalovirus

**Eradication and Elimination**

Termination of all transmission of infection by the extermination of the infectious agent through surveillance and containment. Eradication is an absolute process, an "all or none" phenomenon, restricted to termination of infection from the whole world.

The term elimination is sometimes used to describe eradication of a disease from a large geographic region. Disease which are amenable to elimination in the meantime are polio, measles and diphtheria

**Source or Reservoir**

The starting point for the occurrence of a communicable disease is the existence of a reservoir or source of infection.

The source of infection is defined as "the person, animal, object or substance from which an infectious agent passes or is disseminated to the host (immediate source). The reservoir is "any person, animal, arthropod, plant, soil, or substance, or a combination of these, in which an infectious agent normally lives and multiplies, on which it depends primarily for survival, and where it reproduces itself in such a manner that it can be transmitted to a susceptible host. It is the natural habitat of the infectious agent

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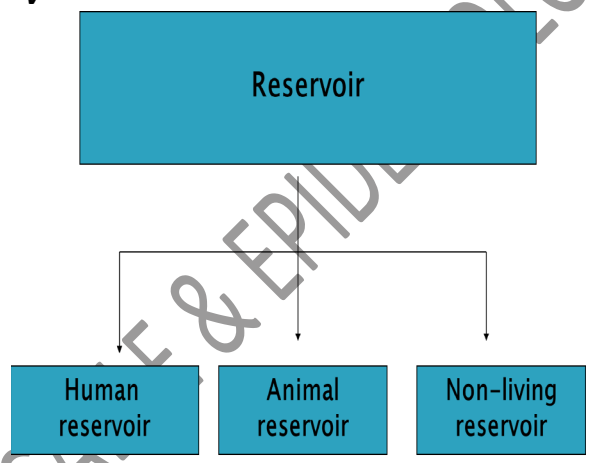
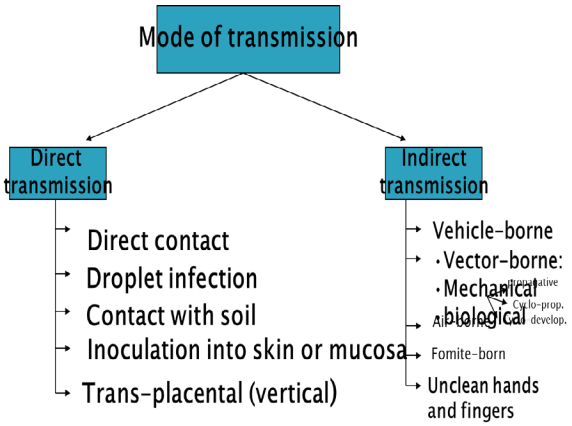



***Types of reservers***

**Cases**

A case is defined as "a person in the population or study group identified as having the particular disease, health disorder, or condition under investigation"

**Carriers: It is "an infected person or animal that harbors a specific infectious agent in the absence of discernible (visible) clinical disease and serves as a potential source of infection to others**




***Modes of transmission***

**Susceptible host**

- Host capable of acquiring a disease
- An infectious agent seeks a susceptible host aiming "successful parasitism".
- Four stages are required for successful parasitism:

1. **Portal of entry**
2. **Site of election inside the body**
3. **Portal of exit**
4. **Survival in external environment**

**Virulence and Case Fatality Rate**

**Virulence**: is the degree of pathogenicity; the disease evoking power of a micro-organism in a given host. Numerically expressed as the ratio of the number of cases of overt infection to the total number infected, as determined by immunoassay. When death is the only criterion of severity, this is the case fatality rate.

**Case fatality rate for infectious diseases**: is the proportion of infected individuals who die of the infection. This is a function of the severity of the infection and is heavily influenced by how many mild cases are not diagnosed.

**Incubation and Latent periods**

**Incubation period**: time from exposure to development of disease. In other words, the time interval between invasion by an infectious agent and the appearance of the first sign or symptom of the disease in question.

**Latent period**: the period between exposure and the onset of infectiousness (this may be shorter or longer than the incubation period).

**Methods of Communicable Disease Control/Principles of communicable disease control**

- ✓ Communicable disease can be controlled and eradicated from the community. When thinking about the control of diseases it is always good to think of all the possible methods.
- ✓ In most cases one or two specific methods will have the greatest effect and should be the focus of your activity, in other cases some methods will be useless against the disease. The aim of control is to tip the balance against the agent.

## The control and eradication of communicable diseases can be done by:

There are three main methods of communicable disease control.They include:

1. Attacking the source of the disease causing organism
2. Interrupting the transmission route
3. Protecting the susceptible host

### Attacking the Source

- ✓ There are various specific measures which can be used to control the spread of an infectious disease. They include:
- ✓ Treating the infected person or animal with the appropriate antibiotics that destroy the disease causing-organism.
- ✓ Treating the carriers and sub-clinical cases after carrying out screening tests among suspected individuals or groups.
- ✓ Treating specific groups of persons who are at high risk of being infected(mass treatment). This is called chemoprophylaxis.
- ✓ Isolating those persons who are infected with highly infectious diseases such as ebola, marburg fever, lassa fever; so as to prevent the spread of the organism to other healthy people.
- ✓ Treating sick animals such as cattle suffering from brucellosis, immunizing animals such as cows from anthrax, and dogs from rabies; killing sick animals such as rats to control plague and dogs to prevent rabies; separating humans from animals.
- ✓ Notifying the local health authorities immediately you suspect a patient is suffering from an infectious disease.
- ✓ Though this does not directly affect the source, it is an essential way of keeping watch on the number of new cases and thereby monitoring the effectiveness of the control programme
- ✓ All of the methods mentioned in the above slides are methods of controlling the reservoir - where an animal is the reservoir.

***In summary you can state that the measures for attacking the source are:***

- ✓ Treating the infected person/s

- ✓ Treating the carrier
- ✓ Mass treatment of persons at risk
- ✓ Isolating the infected person/s
- ✓ Treating the sick animal such as cows
- ✓ Immunizing animals such as dogs and cattle Killing the animal reservoir such as rats Separating humans and animals

**There are five ways of attacking the source of disease micro-organism**

**I. Treatment:**

Refers to the use of drugs that destroy organism. When organism is destroyed, none of them are available to spread to the new host. It is useful in Control of TB,leprosy and STDs.

II. **Isolation:**

Refers to complete separation of a person or individual with a certain disease from others except those providing care. When the sick person is not allowed to come in contact with other people, the organism cannot spread. Is used to control highly infectious diseases such as Ebola, SARS, Measles.

III. **Reservoir control:**

Reservoir is any human , animal ,arthropod, plant,soil or inanimate matter in which the agent normally lives and multiplies.

In those diseases that have their main reservoir in animals, mass treatment, chemoprophylaxis or immunization of animals can be carried out e.g brucellosis.

Other ways of control at reseivor level include **separating humans from animals** or **killing** the affected animals and so **destroying the reservoirs** e.g Rabies

IV. **Chemoprophylaxis :**

Refers to the administration of a medication for the purpose of preventing disease or infection.

Chemoprophylaxis can be carried out in animals that can transfer diseases to human beings

V. **Notification :**

Refers to informing the local health authorities of the presence or suspicion of infectious disease. The local health authorities can be sub county medical officer , sub county nurse, county public health nurse of County health director. It is an essential means of keeping watch on the number of new cases and therefore monitoring effectiveness of the control program.

## Interrupting the Transmission Cycle

A number of methods are used to interrupt the transmission cycle.

Refers to breaking the chain of disease transmission

There are four main methods of interrupting transmission of micro-organisms.

**They include :**

- ✓ Environmental sanitation
- ✓ personal hygiene and behavior change
- ✓ vector control and disinfection and sterilization
- ✓ Personal hygiene
- ✓ Environmental health
- ✓ Water and sanitation
- ✓ Vector control
- ✓ Good and adequate housing
- ✓ Effective food handling and adequate nutrition

***Remember:***

***A clean environment and good personal hygiene are the most important measures in the primary prevention of diseases.***

- ✓ In addition to the measures covered in unit three, add sterilization of medical equipment and the use of sterile surgical equipment. These methods are useful for interrupting the transmission of diseases such as, Human Immunodeficiency Virus (HIV) infection and hepatitis-B infection.
- ✓ *Remember:*
- ✓ *The most effective way of controlling communicable diseases is to use a combination of methods: attacking the source of the infecting organism, interrupting the route of transmission, and protecting the susceptible host.*
- ✓ There are various specific and general measures for protecting the host.
- ✓ Immunisation using vaccines such as the KEPI vaccine
- ✓ Chemoprophylaxis using for example: Proguanil (PaludrineR) to suppress malaria parasites Tetracycline during cholera outbreaks Cotrimoxazole during plague
- ✓ outbreaks surveillance) on the number of new cases of communicable diseases in your area of work and to immediately inform the local health authority when you come across a patient suffering from an infectious disease.
- ✓ One of the main reasons for notification is to help the health authorities take measures to confirm

your suspicion and to control the spread of the disease.

- ✓ Notification of infectious communicable diseases is the responsibility of all health care workers. It is also a legal requirement according to the Public Health Act, Chapter (cap) 242, section eight of the laws of Kenya.
- ✓ *Remember: It is your responsibility to notify your local health authority immediately should you suspect the presence of an infectious disease.*

**Protecting the host through:**

- Immunization
- Chemoprophylaxis
- Personal protection
- Better nutrition

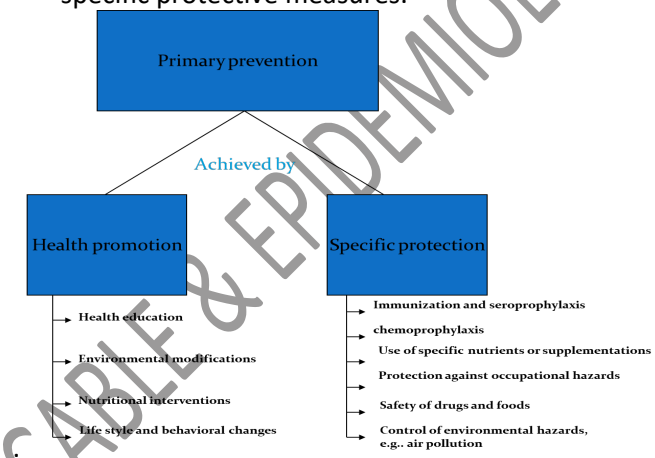
**Levels of prevention**

**Primordial prevention**

- Primordial prevention consists of actions and measures that inhibit the emergence of risk factors in the form of environmental, economic, social, and behavioral conditions and cultural patterns of living etc.
- Primordial prevention (cont.)
- It is the prevention of the emergence or development of risk factors in countries or population groups in which they have not yet appeared
- For example, many adult health problems (e.g.., obesity, hypertension) have their early origins in childhood, because this is the time when lifestyles are formed (for example, smoking, eating patterns, physical exercise).

**Primary prevention**

- Primary prevention can be defined as the action taken prior to the onset of disease, which removes the possibility that the disease will ever occur.
- It signifies intervention in the pre-pathogenesis phase of a disease or health problem.
- Primary prevention may be accomplished by measures of "Health promotion" and "specific protection"
- Primary prevention (cont.)
- It includes the concept of "positive health", a concept that encourages achievement and maintenance of "an acceptable level of health that will enable every individual to lead a socially and economically productive life".
- Primary prevention may be accomplished by measures designed to promote general health and well-being, and quality of life of people or by specific protective measures.




**Secondary Prevention**

- It is defined as " action which halts the progress of a disease at its incipient stage and prevents complications."
- The specific interventions are**: <u>early diagnosis</u>** (e.g.. screening tests, and case finding programs….) and adequate **<u>treatment.</u>**
- Secondary prevention attempts to arrest the disease process, restore health by seeking out unrecognized disease and treating it before irreversible pathological changes take place, and reverse communicability of infectious diseases.
- It thus protects others from in the community from acquiring the infection and thus provide at once secondary prevention for the infected ones and primary prevention for their potential contacts.

***Early diagnosis and treatment***

- WHO Expert Committee in 1973 defined early detection of health disorders as " the detection of disturbances of homoeostatic and compensatory mechanism while biochemical, morphological and functional changes are still reversible."

- The earlier the disease is diagnosed, and treated the better it is for prognosis of the case and in the prevention of the occurrence of other secondary cases.

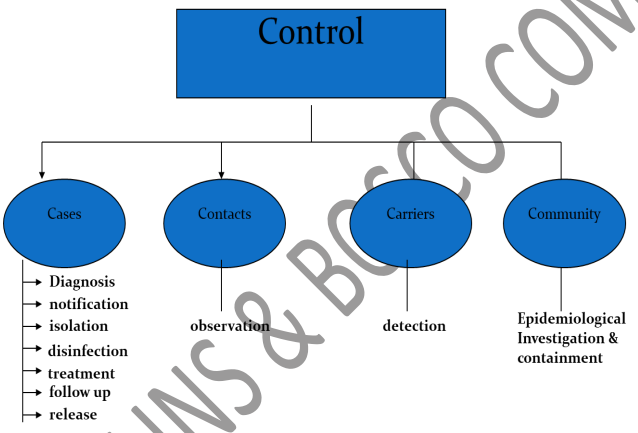
### Tertiary prevention

- It is used when the disease process has advanced beyond its early stages.
- It is defined as "all the measures available to reduce or limit impairments and disabilities, and to promote the patients' adjustment to irremediable conditions."
- Intervention that should be accomplished in the stage of tertiary prevention are **disability limitation,** and **rehabilitation.**
- Control
- Concept of control:

The term disease control describes ongoing operations aimed at reducing:

- The incidence of disease
- The duration of disease and consequently the risk of transmission
- The effects of infection, including both the physical and psychosocial complications
- The financial burden to the community.

Control of infectious diseases (the 4 "C"s




**Application of Communicable Disease Control Measures**

✓ The actual application of the control methods you have just seen can be undertaken by different groups of people and institutions at various levels.

✓ These include individuals and village level, dispensary and health center level and the district and central government (Ministry of Health) level.

***Remember: A successful communicable disease control program is the one that involves members of the community.***

**Control Measures at Individual and Village Level** At this level, each person and indeed every member of the village is responsible for:

✓ Completing the immunization Personal and environmental hygiene

✓ Food hygiene and adequate nutrition Using bed nets and protective wear

✓ Food hygiene and adequate nutrition Using bed nets and protective wear

✓ Abstaining from casual sex, being faithful to one sexual partner or using condoms

✓ Protecting water supply and using clean water

✓ Digging and using pit latrines

✓ Controlling vectors

✓ Healthy habits, for example not smoking, consuming alcohol and abuse of drugs

**Control Measures at Dispensary and Health Centre Level**

✓ The health care workers should support and encourage their clients and community to establish and sustain community based disease control programs. In addition, the health care workers should:

✓ Increase immunization coverage Participate in vector and reservoir control

✓ Emphasize water protection and purification Inspect food, markets and eating places

✓ Encourage sanitation and refuse disposal

✓ Promote health and prevent diseases using Information, Education and Communication (IEC) Notify diseases

**Control Measures at District, Regional and National Level**

✓ At these higher levels, health care workers are responsible for:

✓ Vector control schemes Mass immunization campaigns

✓ Mass treatment and chemoprophylaxis Mass media IEC programmes

✓ Health statistics registration

✓ Research on disease control methods

✓ Emergency, epidemiology and control teams

✓ Manpower training and continuing education for staff

**A)** VECTOR BORNE DISEASES
**B)** DISEASES CAUSED BY FECAL CONTAMINATION
**C)** HELMINTHIC DISEASES
**D)** ZOONOTIC DISEASES
**E)** SELECTED AIRBORNE DISEASES
**F)** EMERGING AND RE-EMERGING DISEASES

**A. VECTOR BORNE DISEASES**
**1.** Malaria
**2.** Bancroftian filariasis
**3.** Onchocerciasis
**4.** Yellow fever
**5.** Schistosomiasis
**6.** Dranculosis
**7.** Leishmaniasis
**8.** Yellow fever
**9.** Relapsing fever
**10.** Tryponosomiasis

B. **DISEASES CAUSED BY FECAL CONTAMINATION**
**1.** Cholera
**2.** Giardiasis
**3.** Amoebiasis
**4.** Bacillary dysentry
**5.** Poliomyelitis
**6.** Enteric fevers
**7.** Food poisoning

C. **HELMINTHIC DISEASES**
**1.** Ascariasis
**2.** Enterobiasis
**3.** Trichuriasis
**4.** Strongyloidiasis
**5.** Taeniasis
**6.** Hydatidosis

D. Hookworm:

**ZOONOTIC DISEASES**
**1.** Rabies
**2.** Tetanus
**3.** Anthrax
**4.** Brucellosis

E. **EMERGING AND RE-EMERGING DISEASES**
**1.** Ebola
**2.** chikungunya
**3.** Zika Virus disease
**4.** SARS
**5.** Bird flue

F. **SELECTED AIRBORNE DISEASES**
**1.** Influenza
**2.** Mumps
**3.** Rubella

## OVERVIEW OF THE PATTERNS TO COMMUNICABLE DISEASES

**Communicable disease**: a disease that can be spread to a person from another person, an animal or object. Ex: common cold, influenza, tuberculosis, etc.

**Non-communicable disease**: a disease that can NOT be spread from person to person. Ex: cancer, heart disease, cirrhosis, etc

**Communicable versus non-communicable diseases**

| **Communicable versus non-communicable diseases** | |
|---|---|
| Sudden onset | Gradual onset |
| Single cause | Multiple causes |
| Short natural history | Long natural history |
| Short treatment schedule | Prolonged treatment |
| Cure is achieved | Care predominates |
| Single discipline | Multidisciplinary |
| Short follow up | Prolonged follow up |
| Back to normalcy | Quality of life after treatment |

## Vector borne diseases

- The organisms which cause vector-borne diseases usually undergo part of their development inside the vectors themselves. The time taken by the disease-causing organism to develop inside the vector is called

the extrinsic incubation period. Although the housefly is an insect that is known to carry bacteria and chlamydia, it is not considered a vector. This is because it is merely a mechanical transmitter of disease; the organisms do not develop inside its body.

- The majority of vectors are insects with the mosquito being the most common. This is because the mosquito is responsible for transmitting more diseases than any other vector. Insect vectors usually acquire disease organisms by sucking blood from infected persons. They then transmit the infection by depositing infected faeces or body fluids in skin cracks or abrasions. Most vectors have quite specific breeding, feeding and attacking behaviour. They therefore only thrive in areas where suitable conditions exist for their survival. As a result, vector-borne diseases tend to be present all the time (endemic) in a given geographical area or population.
- Many of the diseases transmitted by vectors can also become epidemic, especially when there are environmental or other changes leading to increased transmission. Some serious epidemics which have occurred in Africa have been as follows:

**Yellow fever**: Ethiopia, Sudan, Nigeria, Ghana
**Trypanosomiasis**: Uganda
**Kala Azar**: Kenya, Sudan
**Plague**: Uganda, Kenya, Tanzania
**Typhus fever**: Burundi, Rwanda, Ethiopia

When communicable diseases are present in animals all the time, such as the case of yellow fever in monkeys and plague in rats, the disease is said to be enzootic (epidemic in animals).

**Some Diseases and Their Vectors**

| Disease | Causative Organism | Vector |
|---|---|---|
| Malaria | Plasmodium falciparum<br>Plasmodium ovale<br>Plasmodium malariae<br>Plasmodium vivax | Anopheles funestus (female mosquito)<br>Anopheles gambiae (female mosquito) |
| Filariasis (elephantiasis) | Wuchereria bancrofti | Culex pipiens<br>Culex quinquefasciatus<br>Anopheles spp (female mosquito) |
| Yellow fever | Flavivirus | Aedes aegypti (mosquito) |
| Visceral Leishmaniasis (Kala Azar) | Leishmania donovani | Phlebotomus lutzomyia (Sandfly) |
| Trypanasomiasis (Sleeping sickness) | Trypanosoma gambiense<br>Trypanosoma rhodesiense | Glossina palpalis<br>Glossina morsitans (Tsetse fly) |
| Onchocerciasis (River blindness) | Onchocerca volvulus | Simulium damnosum (black fly) |
| Plague | Yersinia pestis | Xenopsylla cheopis (Rat flea) |

## Malaria

Malaria is an acute infection of the blood caused by protozoa of the genus **plasmodium**.
Malaria is directly or indirectly responsible for much ill-health and death, especially of children.
The **vector** responsible for the transmission of malaria is the **anopheles mosquito** (anopheles gambiae and anopheles funestus), which thrive in humid, warm climates where water is available.The parasites develop properly in the mosquito in places where the mean temperature is 16 - 32°C.The cooler the environmental temperature the longer it takes for the parasites to develop in the mosquito.The parasite takes about 35 – 36
days to develop at a mean daily temperature of 16°C, and nine days when the mean daily temperature is 30°C or above.Mosquitoes have an average life span of two to four months.

**There are three Anopheles species in Africa**

1. **Anopheles gambiae :** most important vector in Africa.Feed in temporary water bodies such as pools
2. **Anopheles funetus :** Breed in permanent vegetation such as swamps and rice fields
3. **Anopheles melas :**adapted to slightly salty water.

**The people at risk from malaria include:**

1. Children under 5 years of age
2. Travelers from non-malaria area
3. People with sickle cell disease
4. Women in their first pregnancy {malaria thrives in placenta
5. Splenectomised patients
6. G.Transmission and life cycle of malaria

## Malaria Epidemiology

Malaria is caused by the plasmodium (parasite) that is transmitted to human beings by the bite of the infected female anopheles mosquito. There are four plasmodium species and any of them can cause malaria. They are:

- Plasmodium falciparum
- Plasmodium malariae
- Plasmodium ovale
- Plasmodium vivax

In Kenya 98% of malaria is caused by plasmodium falciparum. The other 2% of the cases are caused by plasmodium malariae and plasmodium ovale. Malaria caused by plasmodium vivax is very rare. Malaria due to plasmodium falciparum is usually the most severe form of malaria and is called malignant malaria. The mortality rate due to malaria is highest in children under five years of age

**In Kenya, malaria occurs in two patterns:**

**Endemic Malaria**

Endemic malaria (also called 'stable malaria') is transmitted all the year round. This type of malaria is found around Lake Victoria and the coastal region of Kenya. Endemic malaria causes severe infection in children under five years of age and in pregnant mothers. The mortality rate is high among infected children. After repeated bites by infected mosquitoes older children and adults develop partial immunity to malaria.

***NB// Endemic malaria is transmitted all the year and severely affects children under five years old and pregnant mothers.***

**Epidemic Malaria**

- Epidemic malaria (also called 'unstable malaria') occurs seasonally and affects people of all ages. Seasonal malaria occurs in Machakos, Embu, Kitui, Tharaka and Marigat in Baringo.
- Another form of epidemic malaria occurs in the highlands and those areas bordering endemic zones. This type of malaria is called highland malaria and is seen seasonally and affects all people severely. Highland malaria epidemics have had high mortality rates. The areas in Kenya which have been affected by highland malaria include Kisii, Nyamira, Kericho, Turkana and Narok.

***Note// Epidemic malaria occurs seasonally and affects people of all ages.***

**Transmission and Life Cycle of Malaria**

***Malaria parasites develop in two cycles:***

- The ***first cycle takes place in the mosquito*** and the other cycle ***in the infected human being***.
- The first cycle which takes place in the mosquito is called the **sexual cycle**, while that which takes place in the human being is called the **asexual cycle**.

**Asexual Cycle**

1. **Sporozoites** in mosquito's salivary gland are injected into host via proboscis of mosquito. They stay in circulation for 30 minutes then rapidly migrate to the liver. A sporozoite is a spore formed after fertilization.

2. **Hepatic {liver} stage***:* {exo-erythrocytic stage}
- Takes 9-14 days
- sporozoite is taken up by the kupffer cells of the liver and then passes through into hepatocytes or liver cells.
- Sporozoite mature into hepatic *Schizont*
- Each schizont multiplies into thousands of other forms called merozoites which are released into general circulation

3. **Hypnozoites**: This stage only occurs in Plasmodium ovale and P.vivax. The hypnozoite mature unpredictably and release new merozoites responsible for recurrent infections upto 18 months after primary infection.

4. **Erythrocytic or blood stage**: Merozoites released from liver invade individual red blood cells and mature to become *Trophozoites* and then develop with RBCs into *blood schizonts.*

The schizonts then rapture the RBC releasing numerous merozoites which invade new RBCs.This part of cycle causes clinical illness and the length of time each RBC cycle gives periodicity of the symptoms.

5. **Gametocytes**: some of the merozoites in red blood cells develop into sexual forms of the parasite, called male and female gametocytes

**In mosquitoes {sexual reproduction}**

6. **Sexual reproduction**:male and female gametocytes develop into gametes and then fuse in stomach to form a zygote. Develop into a mobile form called ookinete which migrate through the wall of stomach to form an oocyst

7. **Oocyst**: This matures and releases sporozoites

8. **Sporozoites**: these migrate into salivary gland ready for delivery with mosquito's next meal.

**Sexual Cycle**

- The sexual cycle of the malaria parasite takes place in the body of the female anopheles mosquito.
- This cycle begins when the feeding mosquito sucks blood containing the male and female gametocytes.
- In the stomach of the mosquito, th`e male gametocytes mate with the female gametocytes.
- The fertilised gametocyte is called the ookinete
- The ookinete stays in the stomach of a mosquito for about 12 - 18 hours after which it penetrates the stomach wall.
- Upon reaching the outer surface of the stomach wall, the ookinete changes into an oocyst
- The oocysts grow rapidly and burst releasing large numbers of sporozoites into the body cavity of the mosquito.
- Many of the sporozoites move to the salivary glands of the mosquito (8) from where they are injected into the body of the next human being when the mosquito feeds.

## Pathophysiology of malaria

- A female Anopheles mosquito carrying malaria-causing parasites feeds on a human and injects the parasites in the form of sporozoites into the bloodstream. The sporozoites travel to the liver and invade liver cells.
- Over 5-16 days*, the sporozoites grow, divide, and produce tens of thousands of haploid forms, called merozoites, per liver cell.
- The merozoites exit the liver cells and re-enter the bloodstream, beginning a cycle of invasion of red blood cells, known as asexual replication. In the red blood cells they develop into mature schizonts, which rupture, releasing newly formed merozoites that then reinvade other red blood cells. This cycle of invasion and cell rupture repeats every 1-3 days* and can result in thousands of parasite-infected red blood cells in the host bloodstream, leading to illness and complications of malaria that can last for months if not treated
- Clinical picture
- Sporozoites and liver stages do not cause clinical symptoms
- Symptoms develop when the invaded RBCs rapture to release new merozoites and vasoactive peptides which activate immune system
- Vasoactive peptides are hormones produce in intestine.

**Clinical Features of Malaria**

The incubation period of malaria is about 10 - 14 days after the infection. The symptoms appear once the invaded erythrocytes rupture to release new merozoites. This stimulates the body's immune system and the signs and symptoms of malaria then appear:

**SYMPTOMS**

- Fever and chills
- General malaise
- Joint pains
- Backache
- Nausea
- Vommiting and diarrhoea
- Headache
- dizziness

**SIGNS**

- High temperatur
- Rigors:sudden feeling of cold accompanied by rise in temperature
- Splenomegaly
- Jaundice

- Convulsions
- anemia

A typical attack of malaria progresses through the following three stages:

**1. The Cold Stage**

This stage starts suddenly and lasts for fifteen minutes to one hour. The patient's body temperature rises and they shiver. During this stage, the infected erythrocytes rupture releasing merozoites in the blood circulation.

**2. The Hot Stage**

The hot stage last for two to six hours. The body temperature is high (40 - 41°C) with severe headache, nausea and vomiting. The skin is hot and dry.

**3. The Sweating Stage**

The fever drops rapidly and the patient sweats profusely. This stage last for two to four hours.
The patient is relieved but exhausted.

- The spleen enlarges with each attack of malaria because the spleen has to clear the damaged red blood cells and produce antibodies
- Anemia results from 2 main factors,hemolysis of parasitized red cells and some bone marrow suppression
- Mild jaundice is usually the result of hemolysis

**Complications of Malaria**

Severe malaria can cause serious complications and is life threatening.

**Brain**

Mental disturbance appearing as acute psychosis, meningitis-like symptoms and coma.

**Diagnosis of Malaria**

Diagnosis is made through:

• **Clinical diagnosis** is based on the patient's symptoms and on physical findings at examination. The first symptoms of malaria (most often fever, chills, sweats, headaches, muscle pains, nausea and vomiting) are often not specific and are also found in other diseases (such as the "flu" and common viral infections)

- Dosage of tablets of artemether lumefantrine a sper guidelines according to age and weight
- Patients presenting with coma, convulsions, respiratory distress, acute renal failure, jaundice,

• **Microscopic diagnosis**: malarial parasites can be identified by examining under the microscope a drop of the patient's blood, spread out as a "blood smear" on a microscope slide

• Laboratory examination of thick and thin peripheral blood films/slides (smears) which demonstrate the parasites (Trophozoites)

**Management of Malaria**

The treatment of malaria depends on whether the disease is complicated malaria or uncomplicated malaria (severe malaria). Uncomplicated malaria is usually treated on an outpatient basis.

- Refers to failure to achieve the desired therapeutic response after initiation of therapy.
- May result from: poor adherence to treatment; unusual pharmacokinetic properties in patient and drug resistance
- Treatment failure should be suspected if the patient deteriorate clinically or if symptoms persist 3-14 days after initiation of drug therapy in accordance with recommended treatment regimen.
- Re-appearance of symptoms 14 days after initiation of treatment should be considered as a new infection and be treated with 1st line treatment
- In all suspected treatment failures, examination of well prepared thin and thick blood smears under light microscopy must be performed.
- Confirmed cases of treatment failure should be treated with second line drugs
- In cases where the cause of treatment failure is established to be non compliance, the same course of first line drugs may be used.

**Treatment of Uncomplicated Malaria**

shock, hypoglycaemia, or acidosis due to malaria should be admitted into the ward for complicated malaria.

**Treatment of Complicated Malaria**

| Age Group | Expected Weight (Kg) | Number of tablets per dose given eight hourly | |
|---|---|---|---|
| | | Quinine Sulphate 200mg | Quinine Bisulphate 300mg |
| < 3months | <5 | ¼ | ¼ |
| 3mo to 11 mo | 5 to 9 | ½ | ½ |
| 12mo to 3yr | 10 to 14 | ¾ | ¾ |
| 4yr to 5yr | 15 to 18 | 1 | 1 |
| 6yr to 7yr | 19 to 25 | 1 ¼ | 1 ¼ |
| 8yr to 12yr | 26 to 37 | 1 ¾ | 1 ¾ |
| 13yr to 15yr | 38 to 49 | 2 ½ | 2 ½ |
| 16 yr and above | 50 and above | 3 | 3 |

Intravenous quinine in dextrose is used in severe complicated Malaria where the patient presents with vomiting and coma.

***Remember: The treatment of malaria keeps changing depending on current research findings.***

Management of severe complicated malaria

**Malaria is said to be complicated if there is any episode of slide confirmed *P.falciparum* with any of the following:**

- Hyperparasitemia > 5%
- Severe anemia<5g/dl
- Hypoglycemia<2.2 mmol/litre
- Cerebral dysfuction ranging from confusion to coma
- Convulsions
- Cocurrent infections-pneumonia,septicemia
- Major organ failure:renal,hepatic,heart failure
- Pulmonary edema

**Severe malaria is a medical emergency**

**Common presentations in children include:**severe anemia,respiratory distress and celebral malaria

- **It can occur in absence of fever and use of parasitological diagnosis is recommended**

***Treatment of choice for severe malaria is paranteral quinine***

**Guidelines on quinine administration**

- **Quinine should be given only as IV INFUSION and NOT INTRAVENOUS {BOLUS} injection**
- **Loading does should be ommited if patient has received quinine in the last 24 hrs or mefloquine in the last 7 days**
- **In hepatic insufficiency,the dose of quinine should be reduced by 25%,**
- **Hypoglycemia is a potential side effect and quinine should be administered in 10% dextrose infusion**

**In absence of quinine**

- In an emergency and in absence of quinine,the following antimalarials may be used:
- Artenusate administered by IM route at 2.4mg/kg stat then 1.2mg/kg at 12 hrs,then 1.2mg/kg daily for 6 days
- Artemether administered by IM route at a loading dose of 3.2 mg/kg stat then 1.6mg/kg daily for five days
- Rectal artenusate administered at a dose of 10mg/kg

**Prevention and Control of Malaria**

**Chemoprophylaxis**

Antimalarial chemoprophylaxis using oral proguanil (PaludrineR) may be given according to the national guidelines for diagnosis, treatment and prevention of malaria for health workers. Individuals who will benefit from chemoprophylaxis include:

- Patients with leukaemia (lowered immunity)
- Patients with sickle cell disease
- Patients with tropical splenomegally
- Non-immune visitors to malaria-endemic areas

**Intermittent Preventive Treatment (IPT)**

IPT is based on the assumption that the pregnant woman is infected with malaria. According to the Ministry of Health (MoH) guidelines, the drugs used for IPT are the ones that contain Sulfadoxine and Pyrimethamine (SP) such as FansidarR, MalaraxinR, FansidinR, MetakelfinR, OrodarR, and FalcidinR. The first single dose of three tablets of SP is given to the pregnant woman between 16 and 24 weeks of gestation; the second and last dose of three tablets of SP is given between 24 and 36 weeks of gestation. (MoH, 2002)

**Vector Control**

Actions to reduce mosquito-breeding areas include:

- Using insecticide-treated bed nets
- Using mosquito screens in houses
- Using chemical mosquito repellents
- Cleaning drainages and water disposal systems
- Clearing bushes and burying or burning rubbish heaps
- Use of larvicides and insecticides

**Health Education**

You should encourage community members to seek early diagnosis and prompt treatment for malaria and to use insecticide treated bed nets every night.

## FILARIASIS (ELEPHANTIASIS)

This is a disfiguring disease caused by a tiny worm (nematode) called ***wuchereria bancrofti***. It is mainly transmitted by mosquitoes; the ***culex quinquefasciatus*** found in heavily contaminated water especially in the urban areas and the culex pipiens and the anopheles mosquito in rural areas. These mosquitoes transmit the worm from person to person in the same way as malaria. The parasitic worm lives in the lymphatic system of the patient causing inflammation of the lymphatic vessels and lymph glands (lymphangitis, lymphadenitis), filarial fever, and eventually elephantiasis of the arms, legs and genitals. The disease is most frequent in the tropical coastal belts and the lake region.

**Disease caused by filarial worms affecting lymphatic system.**

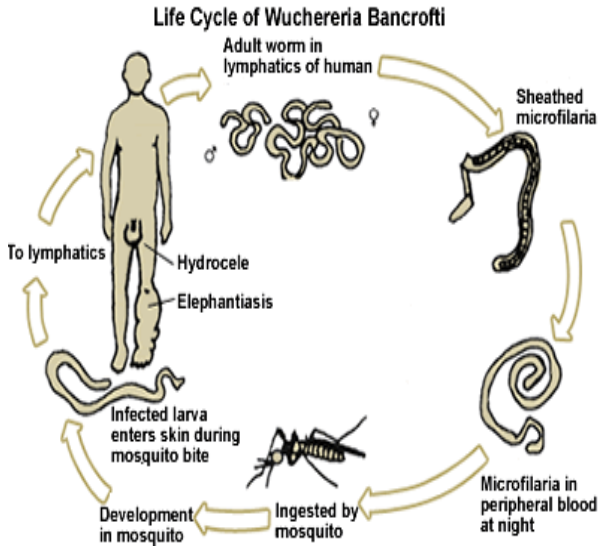
**Also called Lymphatic Filariasis**

**Causal organism:** *Wuchereria bancrofti*

It is one of the three parasitic worms, together with Brugia malayi and B. timori, that infect the *lymphatic* system to cause *lymphatic filariasis*. These *filarial* worms are spread by a variety of mosquito vector species

**Mode of Transmission**

The microfilariae ingested by the feeding mosquito exsheath in the stomach and become first stage larva. They then penetrate the mosquito stomach wall and migrate to the thorax muscles where they moult twice and develop into the infective stage. Mature infective microfilariae
migrate to the mouthparts of the mosquito. The extrinsic incubation period takes 10 -12 days.




**LIFE CYCLE OF FILARIASIS**

**W. bancrofti, is acquired via the bite of mosquitoes. Ø**

1. When mosquitoes bite humans, they deposit third-stage infective larvae into the skin. Ø
2. These larvae travel through the dermis and enter local lymphatic vessels. Over a period of approximately nine months, these larvae undergo a series of molts and develop into mature adult worms, which range from 2 to 5cm in length. Ø These adults reside in the lymphatics, generally several centimeters from lymph nodes.
3. They survive for approximately five years during which time male and females worms mate and produce microfilariae.
4. Female parasites can release more than 10,000 microfilariae per day into the bloodstream. These microfilariae are also known as embryonic or first-stage larvae.
5. Mosquitoes, which bite infected individuals, can take up these circulating microfilariae. Within the mosquito, these embryonic larvae develop into second then third stage larvae over a period of 10 to 14 days. The mosquito is then ready to bite and infect a new human host, thereby completing the life cycle

**Clinical Features**

The presence of mature filarial worms in the lymphatic vessels triggers an inflammatory reaction in the walls of these vessels. When the worms die, more foreign proteins are released causing calcification of the lymphatic walls which eventually leads to obstruction of the flow of lymph fluid. If the obstruction of the lymph flow is extensive, chronic oedema develops in the affected areas of the body. Filariasis progresses through three stages.

**Acute Phase**

≡ Starts few months after infection and is characterized by fever,lymphadenopathy and eosinophilia

≡ Microfilariae cannot be seen in peripheral blood because the worms are not yet mature

≡ The acute phase is due to hypersensitivity reaction

- Fever
- Eosinophilia
- Enlarged lymph nodes
- Inflamed lymph vessels (lymphangitis)

**Sub Acute Phase**

≡ Sets in after about a year

≡ The worms are mature and microfilariae are present in the peripheral blood

≡ Adult worms causes fever,lymphadenitis,funiculitis-inflammation of spermatic cord,Epididymitis-inflammation of epididymis and Hydrocele-collection or accumulation of fluid in scrotal sac.

≡ Microfilariae causes hypereosinophilia,asthma like attacks and fever

- Fever
- Eosinophilia (severe)
- Attacks of dyspnoea (asthma-like)
- Funiculitis (pain and swelling of the spermatic cord/s)

- Epididymitis
- Hydrocele
- Lymphadenitis (tender lymph nodes)

**Chronic Phase**

≡ After many years of repeated attacks, lymph glands and lymph vessels become obstructed.

≡ As a result,lymphoedema develops.It is often seen in in the legs or scrotum {elephantiasis}

≡ Chronic phase is characterize by presence of four main features:Lymphoedema,elephantiasis,chyluria and hydrocele

≡ Lymphedema, lymphoedema, or lymphatic obstruction is a chronic (long-term) condition in which excess fluid (lymph) collects in tissues causing edema (swelling)

≡ Elephantiasis a condition in which a limb or other part of the body becomes grossly enlarged due to obstruction of the lymphatic vessels, typically by the nematode parasites that cause filariasis.

≡ Chyuria refers to discharge of white milky urine

- Lymphoedema
- Elephantiasis
- Chyluria
- Hydrocele

**Diagnosis**

- Fluid aspirated from swollen lymph glands or from hydrocele can be examined under a microscope to show the microfilariae.
- Thick blood slides for microfilariae should be taken between 10:00pm and 2:00am. This is because microfilariae are not present in the peripheral blood during the day.
- Blood slides for microfilariae may be taken 45 minutes after administration of a provocative dose of diethylcarbamazine 100mg.

**DIFFERENTIAL DIAGNOSIS**

- Bacterial lymphagitis
- Relapsing fever
- Malaria
- Septicemia
- TB
- AIDS

**Management**

The drug of choice for filariasis (adult worms and microfilariae) is diethylcarbamazine (DEC, hetrazan, benocide, notezine) 6mg/kg body weight daily in divided doses (150mg) eight hourly for 12 days for an adult. Diethylcarbamazine may be combined with levamisole. This combination kills microfilariae and reduces the parasite worm count in the body more rapidly.

- Drug of choice is Diethylcarbamazine {Hetrazan or Banocide
- Kills microfilariae and some few effects on adult worm
- Dose is 6mg/kg in 3 divided doses
- In adult,the dose is 150mg tds for 12 days
- Ivermectin:Very effective against mf (Microfilariacidal)
- Lowers mf level even in single dose of 200µg – 400µg/Kg body weight
- Albendazole :This antihelmenthic kills adult worms.No action on microfilariae.Dose: 400mg/twice day /2 weeks
- antihistamine or steroids given during the first days of treatment to avoid reactions
- Surgical treatment indicated for hydrocele

**Prevention and Control**

The prevention and control of filiariasis includes:

- Anti-mosquito measures; the same as those used for control and prevention of malaria
- Use of larvicides such as polystyrene powder in the pit latrine
- Reduction of human-mosquito contact including the use of insecticide treated bed nets and screening of houses
- Regular treatment of septic tanks and pit latrines with larvicides
- Reducing exposure to the infection by screening houses and using mosquito bed nets
- Health education on personal protection, screening of houses and usefulness of mosquito treated nets

- Annual mass treatment reduces the level of microfilariae in the blood and thus, diminishes transmission of infection

## Schistosomiasis/ Bilharzia

This disease is commonly known as Bilharzia after Theodor Bilharz who discovered it in Cairo in 1861. The incidence of schistosomiasis is related to water use. Irrigation schemes or water projects for electricity provide the habitat for the snail vectors. Up to 75% of schistosomiasis is transmitted by infected humans while 25% is said to be transmitted by dogs, cows, rats, and baboons. In East Africa, there are two types of schistosomiasis, both of which are named after the causative parasite. They are schistosoma mansoni and schistosoma haematobium.

Schistosomiasis, also known as bilharzia or "snail fever", is a parasitic disease carried by fresh water snails infected with one of the five varieties of the parasite Schistosoma.

Can contract it swimming in lakes, ponds and other bodies of water infested with the parasite's **snail host**

**Causative organisms**

- S. mansoni
- S. haematobium

**Snail hosts**

The different species of Schistosoma have different types of snails serving as their intermediate hosts; these hosts are:

Biomphalaria for S mansoni

Oncomelania for S japonicum

Bulinus for S haematobium and S intercalatum

**Risk factors**

Contact with freshwater sources where infected snails carrying the disease live

Children under age 14

Individuals with labor or domestic chores centered around freshwater areas

Swimming, bathing, fishing and even domestic chores such as laundry and herding livestock can put people at risk of contracting the disease.

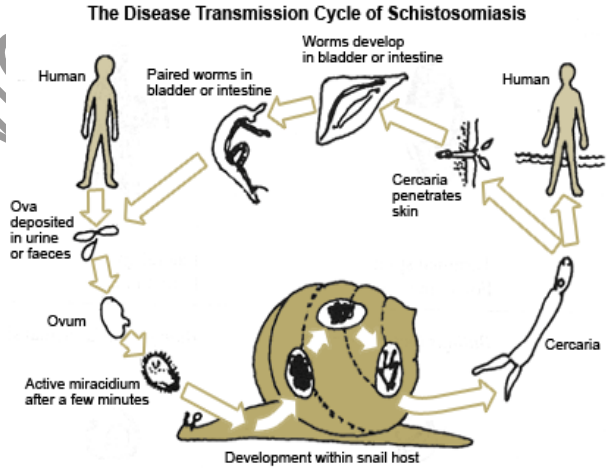
**Mode of Transmission**

S. mansoni is spread in infected stool while S. haematobium is spread in infected urine. When the schistosome eggs in the urine enter a body of water such as a lake, dam, rice paddy or pond, they hatch into free-swimming larvae called miracidia. The intermediate host for S. mansoni is a vector snail of the genus biomphalaria pfeifferi; while for the S. haematobium it is bulinus africanus. The miracidia, after being shed from the ovum, must enter the appropriate snail host within 24 hours or die.

**Transmission Cycle of Schistosomiasis**

Inside the body of the snail host, it takes the miracidia four to seven days to develop and multiply.

The snail sheds them in water where they can only live for 48 hours unless they infect a human. A human being becomes infected when they enter cercariae-infested water, such as when bathing, swimming, laundering, cultivating or fishing.




The cercariae penetrate the skin and enter the bloodstream from where they are carried to the liver or bladder to develop into adult worms. Within four to six weeks, paired adults reach mesenteric and pelvic veins.

**LIFE CYCLE**

(Eggs → larvae → into snail

1. Parasite eggs are released into freshwater (from human urine, feces)
2. Eggs hatch into *miracidia*, free swimming larvae
3. Miracidia find & infect snail host

4. In the snail miracidia develop into many free swimming forms called cercariae within 4-6 weeks of entering snail.THEY ARE INFECTIVE AGENTS OF SCHISTOSOMIASIS
5. Cercarie are shed from the snail 4-7 weeks
6. They can live upto 48 hrs unless they infect human beings.Human become infected when they enter cercarie infected water while bathing,playing,laundering,cultivating or fishing.

7. The cercarie penetrate skin,enter the blood stream,and are carried to the liver or bladder where they develop into adult worm

**DISTINGUISHING BETWEEN S.HAEMATOBIUM AND S.MANSONI**

- Adult worm reside in venous plexus of bladder
- Eggs are excreted with urine
- Vector snail is Bulinus which is found in temporary water bodies such as ponds
- Presents with hematuria in early stages
- Reside in mesenteric veins of the bowel
- Eggs are excreted with feaces
- Vector snail is Biomphalaria found in permanent water bodies such as lakes,rivers dams
- Presents with bloody diarrhoea"mansoni dysentry in early stages
- Disease development

**Schostosomiasis has several stages of development in the body.**

**The development include Invasion,maturation,established infection and late stage**

**1. Invasion**

Cercarie penetrate into the skin.This causes cercarial dermatitis with itching papules and local edema.

The cercarie then enter the circulation and reach the liver via right side of the heart and lungs

**2. Maturation**

**Schistosomes mature in the liver.**
**The stage presents with:**

- Fever
- Eosinophilis
- Abdominal pain
- Transient generalised urticaria.It is also known as Katayana syndrome

➢ **After maturation,the adult worm descend into the portal vein.**

➢ **S.mansoni migrate to the mesentric veins in the intestinal wall and S.haematobium finds its way to the venous plexus of the bladder**

**3. Established infection**

- Is the stage of egg production
- Eggs are produced by female in small veins of the bowel or bladder.
- Some eggs penetrate tissues and other do not.They remain in blood and are carried to the liver and lungs.
- The eggs which do not reach lumen provoke inflammatory reaction and formation of granulomas. A *granuloma* is a small area of inflammation in tissue
- These inflammatory reaction causes bloody diarrhoea and cramps in,S.mansoni and Terminal haematuria and dysuria in S.haematobium

**Late stage**

- Late stage is characterized by fibrosis and calcification which occur when there are many eggs in the tissues.
- Fibrosis refers to the thickening and scarring of connective tissue, usually as a result of injury.

- Calcification is the process in which calcium builds up in body tissue where there normally isn't any calcium. Over time, the buildup can harden and disrupt your body's normal processes.

## LATE STAGE IN THE BLADDER

**Late stage around the bladder results in:**

- Obstruction and dilatation of ureters and hydronephrosis possibly leading to kidney failure
- Pyelonephritis
- Calcification of the bladder which shows on x-ray or ultrasound investigation
- Cancer of the bladder

**Late stage in the liver and lungs**

- Late stage in the liver leads to: portal hypertension,splenomegaly, anemia,oesophegeal varices and massive bleeding
- In the lungs,fibrosis leads to pulmonary hypertension which leads to congestive heart failure

Acute and chronic infection

**ACUTE INFECTION:**

Occur when Cercariae penetrate skin → rash called schistosome or swimmer's itch develop.

**Eggs laid in target organs release antigens which cause Katayama fever characterised by:**

- Fever
- Urticaria
- Malaise
- diarhea

**CHRONIC INFECTION**

- Symptoms of chronic infection caused by eggs that travel to various parts of body
- Eggs remain trapped in host tissues→ granulomatous inflammatory immune response
- Granulomas: macrophages surrounded by lymphocytes
- Fibrosis develops in the late stages

**SYMPTOMS**

- Symptoms for the disease vary depending on the type of worm involved and the location of the parasite inside the body:
- initial itching and rash at infection site ("swimmer's itch")
- The classic sign of urogenital schistosomiasis is haematuria (blood in urine)
- Abdominal pain and bloody diarrhea
- Anemia
- Fever, chills and muscle aches
- Inflammation and scarring of the bladder
- Lymph node enlargement

**DIAGNOSIS**

**Microscopic Detection**

- Take stool or urine sample to detect eggs
- *S. haematobium* eggs are oval and have a spike at the tip
- *S. mansoni* eggs have a spike on the side (spine)
- If they are repeatedly negative,a rectal or bowel biopsy can be done

**MANAGEMENT**

- Praziquantel {biltricide} is the drug of choice for all schistosomal species
- Single dose of 40mg/kg is effective in S.haematobium and S.mansoni
- For patients with heavy infections {over 800 eggs per gramme of stool} ,a total dose of 60mg/kg in two equally divided doses 4-6 hrs apart.
- Drug is best administered after food and in the evening
- Oxamniquine {vansil} is effective only against S.mansoni.
- The drug is used in all stages of infection
- Dose varies with the geographical origin of S.mansoni between 30mg/kg to 60mg/kg.

**Clinical Features**

Schistosomiasis as a disease develops in four stages, each of which is characterised by specific signs and symptoms.

**Effects of Late Stage Schistosomiasis**

| | |
|---|---|
| **Urinary Bladder:** | • Obstruction to and dilation of ureters leading to hydronephrosis which may cause kidney failure<br>• Pyelonephritis<br>• Bladder polyps<br>• Calcification of bladder<br>• Cancer of bladder |
| **Liver:** | • Portal vein fibrosis leading to portal hypertension<br>• Portal hypertension leading to oesophageal varices which may cause massive haematemesis<br>• Caput medusae and ascites |
| | • Hepatomegally |
| **Lungs:** | • Pulmonary fibrosis leading to pulmonary hypertension, causing congestive heart failure |
| **Bowel:** | • Bowel fibrosis and glanulomas<br>• Gastric varices<br>• Haemorrhoids |

**Drugs Used in the Oral Treatment of Schistosomiasis**

| Type of drug | Dose | Contraindications | Side effects | Remarks |
|---|---|---|---|---|
| **Praziquantel** (Biltricide, Distocide, Cysticide, Cesol, Cestox, Coestocide) | S. mansoni: 30mg/kg twice in one day<br>S. haematobium 40mg/kg once (single dose) | None (drug of choice if liver diseased) | Mild giddiness | Expensive but equally effective for both types |
| **Oxaminoquine** (Vansil) | S. mansoni 15mg/kg 12 hourly (twice daily) for 2 days | Epilepsy | Occasional drowsiness, dizziness Or psychosis | Not effective for S. haematobium |
| **Metrophonate** (Bilarcil) | S. haematobium 7.5-10 mg/kg body wt. given in 3 doses at intervals of 14 days; that is a total dose of 22.5 - 30mg/kg. | None | None if correct dose | Not effective for S. mansoni Cheap |

**Diagnosis**

The diagnosis of schistosomiasis is confirmed by finding eggs in stools or urine during a microscopic examination. If this test is found to be negative, a colonic or urinary bladder biopsy can be done. Serological tests are also highly sensitive and yield specific results.

**Management**

The main aim of treatment is to kill the adult worms and to stop their egg-laying activity.

**Prevention and Control**

The prevention of schistosomiasis can be achieved through the following measures:

- Prevention of ova-containing urine and stool from reaching the water by:
  Digging and using pit latrines
  Safe water supply
  Treating the infected persons
- Attacking the intermediate host (the snail) using molluscicides such as copper sulphate which kills snails and their eggs.
- Avoiding contact with infested water by using protective clothing when laundering, cultivating, swimming and wading. Bathing should be done at home (storing water at home for three days will kill the cercariae).
- Conducting mass treatment campaigns for communities at risk using oral praziquantel, especially school-going children.

## LEISHMANIASIS (KALA AZAR)

**A7**This is an infection caused by a parasite of the **leishmania group**. The disease is also known as **Kala Azar**. There are three forms of leishmaniasis which are caused by different parasites.

The vector of leishmaniasis is the **female sandfly** (***phlebotomus***). The four types of sand flies are:

- Phlebotomus martini
- Phlebotomus orientalis
- Phlebotomus longipes
- Phlebotomus pedifer

In Kenya, the main vectors are phlebotomus **martini** which transmit the parasite **leishmania donovani**, responsible for visceral leishmaniasis. The species P. orientalis is common in Sudan while P. longipes and P. pedifer are commonly found in Ethiopian and Kenyan highlands. Together they transmit the parasite leishmania aethiopica which is responsible for cutaneous leishmaniasis.

| Type of Leishmaniasis | Causitive Parasites |
|---|---|
| Visceral | Leishmania donovani<br>Leishmania infantum |
| Cutaneous | Leishmania tropica<br>Leishmania aethiopica<br>Leishmania mexicana |
| Mucocutaneous | Leishmania braziliensis |

**Mode of Transmission**

The zoonotic hosts of leishmaniasis are mainly dogs and rodents, although in some parts of Kenya humans have become the reservoir as well as host. The parasites of leishmaniasis are transmitted when the sandfly bites an infected person and ingests amastigotes. On reaching the sandfly's stomach, the amastigotes change into promastigotes. After four to seven days, they migrate to the foregut where they develop into infective promastigotes. The infective promastigotes are then conveyed in the saliva of the sandfly during feeding. During feeding, the sandfly tears the host's tissue to feed on blood and at the same time deposits infective promastigotes at that site. From here the promastigotes enter the bloodstream and into the macrophages. On entering the macrophages, the parasites escape detection by the body's defences and are spread to various body tissues.

**Visceral Leishmaniasis**

Visceral leishmaniasis is found in many areas of the North Eastern region of Kenya in Machakos, Kitui, Masinga, Tseikuru (Mwingi), Makueni, Kibwezi, and Wajir.

**Clinical Features of Visceral Leishmaniasis**

Visceral leishmaniasis is characterised by fever, splenomegaliy, hepatomegally accompanied by anaemia and weight loss. Visceral leishmaniasis has a rather long incubation period of four to ten months or longer, before definitive signs and symptoms manifest. Most of the patients (96%) are killed by secondary bacterial infections of the lesions.

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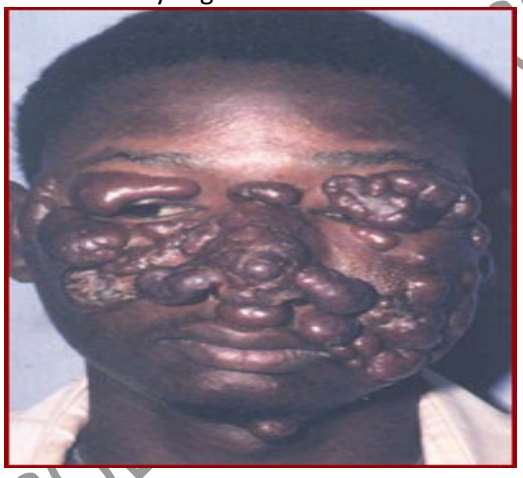
**Cutaneous Leishmaniasis**

Cutaneous leishmaniasis is found in West Pokot, Turkana, Baringo, Laikipia and Kerio valley. It is characterised by single or several painful chronic ulcers in those parts of the body exposed to sandfly bites, such as arms, legs or face. In the lower hotter areas of Kenya such as Baringo, the vector is the P. orientalis, while in the highlands of Kenya, the high altitude sandflies, P. longipes and P. pedifer are the vectors. Phlebotomus longipes bites human beings in their houses at night transmitting the parasite leishmania aethiopica, which is responsible for cutaneous leishmaniasis.

**Clinical Picture of Cutaneous Leishmaniasis**

In about two to eight weeks following a bite from an infected sand fly, a small itchy papule appears at the site of the bite. Over several weeks, the papule grows in size expanding to form a single indolent ulcer or multiple ulcers. The disease may be mistaken for leprosy. There may be enlargement of the local lymph nodes. The lesions begin to heal spontaneously two to twelve months later. Cutaneous leishmaniasis does not spread to other body organs.



**Management of Cutaneous Leishmaniasis**

Small lesions may be treated surgically by curettage or by freezing, using liquid carbon dioxide or by infiltrating them with 1 - 2ml sodium stibogluconate. Large disfiguring or multiple skin lesions are treated in the same way as for visceral leishmaniasis using IV or IM sodium stibogluconate 20mg/kg daily for 20 - 30 days. The drug of choice for visceral leishmaniasis caused by leishmania donovani is IM pentamidine isothianate 3 - 4mg/kg once or twice a week.

**Mucocutaneous Leishmaniasis**

This form of leishmaniasis occurs primarily in the tropics of South America. The disease begins with the same sores noted in localised cutaneous leishmaniasis. Sometimes these primary lesions heal, other times they spread and become larger. Some years after the first lesion is noted (and sometimes several years after that lesion has totally healed), new lesions appear in the mouth and nose, and occasionally in the area between the genitalia and the anus (the perineum). These new lesions are particularly destructive and painful. They erode underlying tissue and cartilage, frequently eating through the septum (the cartilage which separates the two nostrils). If the lesions spread to the roof of the mouth and the larynx (the part of the wind pipe which contains the vocal cords), they may prevent speech. Other symptoms include fever, weight loss, anaemia (low red blood cell count). There is always a large danger of bacteria infecting the already open sores.

Treatment is similar to that of cutaneous leishmaniasis. Prevention or early detection and appropriate treatment are preferred. Corrective surgery can be done where need arises.

**Prevention and Control**

Kala Azar can be prevented through:

- Use of insecticide treated curtains in homes (these have been used with success in Baringo district)
- Destruction of infected dogs and rodents
- Early diagnosis and treatment of the infected persons
- Health education for communities on preventive measures

## ROUNDWORM (ASCARIASIS)

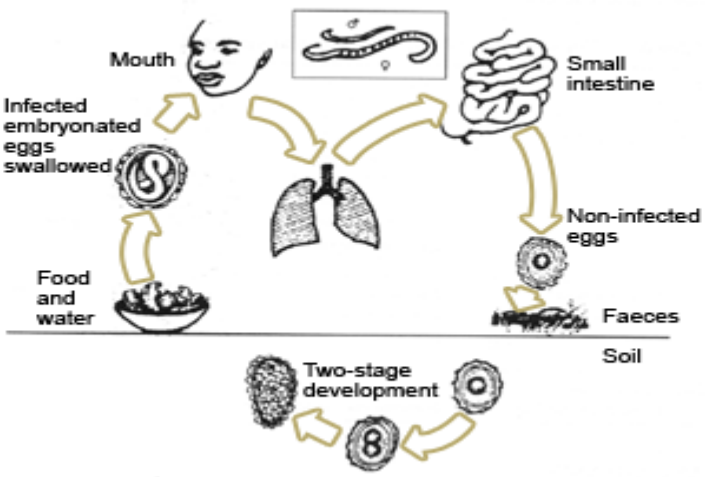
- This disease is caused *by Ascaris lumbricoides,* which infects the small intestine.
- Ascaris is a large intestinal parasite which often infects children because of their habit of putting all kinds of things in their mouth.
- It is one of the commonest and most widespread infections of the small intestine.

- The worms may multiply in large numbers in the intestinal lumen and cause intestinal obstruction at the
  ileocaecal valve.
- The worms also contribute to severe malnutrition and vitamin A deficiency, and may wander out of the intestinal lumen into the peritoneal cavity.

**MODE OF TRANSMISSION**

- Ascariasis is a soil transmitted parasite. Once the eggs are passed out in faeces, they require embryonation in the soil before they can become infective.
- This takes 8-50 days. Embryonated eggs can be carried away from the contaminated place into houses by feet, footwear or in dust by the wind.
- Human beings may injest the eggs as they eat or drink using contaminated hands and utensils, or through eating raw contaminated foods like fruit. Once the eggs are ingested by a human being they hatch into worms
- Once the eggs are ingested by a human being they hatch into worms.
- In order to reach maturity, the larvae need to pass through the lungs and trachea to the pharynx. Once in the pharynx they are swallowed and return to the gastrointestinal tract where they can live for about a year.

**LIFE CYCLE OF ASCARIS LUMBRICOIDES.**




**Clinical Features**

- Is usually asymptomatic and if symptoms are present, they are not characteristic.
- There may be vague abdominal discomfort or occasionally the worm may leave the body in vomitus or stool.
- Also during the stage of larval migration through the lungs there may be temporary symptoms of pneumonitis (cough).
- Infection with a few ascaris is usually asymptomatic and if symptoms are present, they are not characteristic.

**Diagnosis**

Stool microscopy which should show ascaris ova and cyst.

**Management**

- Oral mebendazole 100mg 12 hourly for three days
- **Oral levamisole (3 tabs or 5mg/kg body wt) single dose**
- **Oral piperazine 150mg/kg body wt single dose**
- **Note: For intestinal obstruction, surgical operation is indicated**.

**PREVENTION AND CONTROL**

- Improved environmental sanitation such as proper excreta disposal, clean supply of water
- Discouraging the use of raw (fresh) human faeces for manure (Composting for six months kills the ascaris eggs)
- Washing of fruit and vegetables before eating
- Use of drying racks for utensils so that they do not come into contact with soil and dust
- Washing hands after opening bowels
- Washing hands before handling food

**TRYPANOSOMIASIS (SLEEPING SICKNESS)**

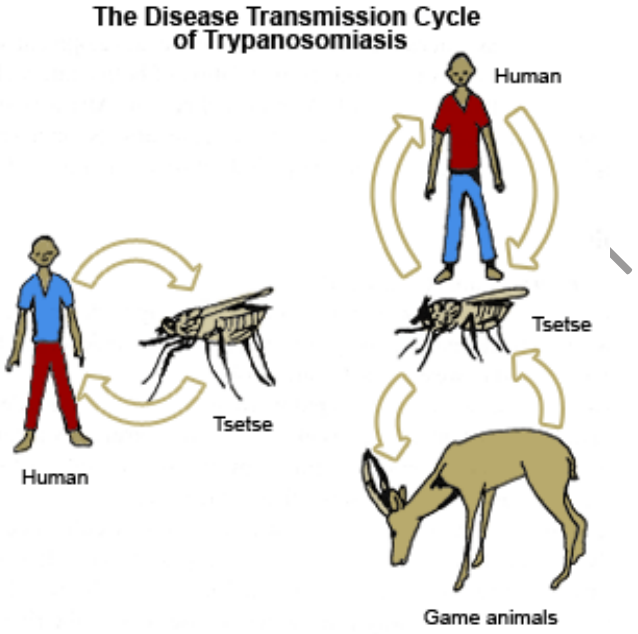
Trypanosomiasis is a tropical disease **caused by protozoa** called ***Trypanosoma brucei gambiense (Tbg)*** and **Trypanosoma brucei rhodesiense (Tbr)**. The important reservoir of Tbr in the wild is the bushbuck. Trypanosomiasis affects both humans and cattle and is invariably fatal over varying periods of time if not

treated. Trypanosoma brucei gambiense causes an acute, rapidly progressive illness with death from cardiac complications within several weeks or months. Reservoirs include antelope and pigs. Tb is found in eastern Africa, now mostly in south-east Uganda.
Trypanasomiasis spreads very rapidly unless the source (the very first case) is identified early, isolated and treated properly. Trypanasomiasis is found in the same areas in Africa where yellow fever is found.

**Mode of Transmission**

- Trypanosomiasis is transmitted by **tsetse flies** which live in areas of wooded vegetation. Tsetse flies are usually not found in flat plains, closely cultivated areas or areas densely inhabited by people.
- There are two important types of tsetse flies known to transmit the disease to humans.
- There is ***glossina palpalis***, a riverine type which breeds along rivers and lakes, and ***glossina morsitans*** the woodland type which lives away from water. ***glossina palpalis is the main vector of Tb gambiense***, while ***glossina morsitans is the main vector for Tb rhodesiense*** and it prefers to bite cattle and game but will also bite humans. Of the two types of tsetse flies, ***glossina palpalis*** (which transmit Tb gambiense parasite) is the main vector in East Africa.

Tsetse flies become infected with sleeping sickness parasites when they take a blood meal from infected persons or animals. After a period of time, during which the trypanosomes undergo development changes, the fly is able to transmit the infection when it bites another susceptible animal or person.




**Clinical Features**

- There is considerable variation in the clinical picture of African trypanosomiasis (AT). Within a few days of a tsetse bite, fever develops due to the invasion of the blood stream by the trypanosomes. The incubation period between the tsetse bite and the onset of fever varies from as short as a few hours following the chancre to several weeks.
- The early stages of trypanosomiasis are characterised by **irregular episodes of fever** with **headaches**, **malaise**, **weight loss**, **muscle** and **joint pains**, **pruritus**, **anaemia**, **skin rash**, and **deep hyperaesthesia (Karandel's sign).**

**The clinical features of trypanosomiasis depend on the infecting parasite as follows:**

- Trypanosoma brucei gambiense (Tbg) infection causes a slow chronic sleeping sickness, resulting in death from the disease in several months or years. Pigs, dogs and antelopes are the reservoirs.
- Trypanosoma brucei rhodesiense (Tbr) infection is acute and rapidly progressive unless prompt treatment is administered. The parasites damage the heart causing cardiac complications and death within several weeks or months. Pigs and antelopes are the reservoirs for Tb rhodesiense.

**Trypanosomiasis presents in the following three stages:**

**1. Primary Stage (chancre stage)**

Within a few days of the tsetse bite, a painful indurated erythematous nodule may appear at the site of the bite.

This chancre may last for one to two weeks and then resolve spontaneously. The chancre occurs in 70% of cases in Europeans but is rare in Africans.

**2. Blood Stage (systemic illness)**

During this stage, the trypanosomes spread to the blood, lymph and lymph nodes. There is fever, which does not follow any typical pattern but recurs at intervals of days or weeks. After the fever resolves, the patient develops anaemia, debilitation and general body weakness. The spleen becomes enlarged as well as the lymph nodes. The cervical lymph nodes especially of the lower back of neck become visibly enlarged in 80% of patients - this is called Winterbottom's sign. The other signs and symptoms of trypanosomiasis include:

- Pruritic rash (beginning six to eight weeks after infection)
- Hepatosplenomegaly
- Poor appetite resulting in weight loss, debility, pitting oedema of face and lower legs
- Impotence and menstrual irregularities
- Heart failure

**3. Cerebral Stage (Sleeping sickness stage)**

This is the terminal stage of trypanosomiasis. During this stage of the disease, the parasites invade the brain leading to mental deterioration and coma. Convulsions and localised signs such as hemiplegia and facial palsy may occur. Patients are very weak, they sleep during the day but are restless at night. As the disease progresses, the patients become severely ill and die if not treated.

**Diagnosis**

- Microscopic examination of the chancre fluid to demonstrate the trypanosomes
- Examination of blood (buffy coat) for trypanosomes
- Wet blood smear for microscopy
- Thick blood smear for microscopy
- Serological test (card agglutination test)
- Lymph node aspiration (microscopy)

**Management with suramin**

| Day 1 | Day 2 | Day 3 | Week 2 | Week 3 | Week 4 |
|---|---|---|---|---|---|
| 0.5ml | 1.0ml | 1.0ml | 2.5ml* | 3.5ml* | 5.0ml* |

***Nb:The drugs used for the treatment of trypanosomes are highly toxic.***

**Common Side Effects of Trypanosomiasis Drugs**

| Drug | Toxicity |
|---|---|
| Suramin | Mild proteinuria, arthralgia, severe dermatitis, diarrhoea, nephritis |
| Pentamidine | Hypoglycaemia, nephritis, diabetes mellitus, injection abscess, collapse if injected intravenously |
| Melarsoprol | Jarisch-herxheimer reaction, arsenical encephalopathy, mortality up to 100% |
| Difluoromethyl ornithine | Diarrhoea, abdominal pain |
| Nitrofurazone | Haemolysis, neuropathy |

***Remember: The drugs used for the treatment of trypanosomes are highly toxic. As such the patient should be monitored carefully and the drugs administered very carefully.***

**Prevention and Control**

The following measures are effective in the prevention and control of sleeping sickness.

- Chemoprophylaxis; IM pentamidine 250mg single dose protects against Tb gambiense infection for six months in those working in endemic bush land areas such as wildlife personnel.
- Bush clearing (which may harm the environment) and establishment of agricultural settlement will in the long run destroy tsetse fly breeding areas.
- Use of baited flytraps which have an efficacy of 95% at reducing the tsetse fly population.

***Notifiable Diseases in Kenya***

- ✓ Plague
- ✓ Cholera
- ✓ Measles
- ✓ Poliomyelitis
- ✓ Diphtheria
- ✓ Tuberculosis
- ✓ Anthrax
- ✓ Trypanasomiasis
- ✓ Typhoid fever
- ✓ Whooping cough
- ✓ Meningococcal
- ✓ meningitisRabies

**YELLOW FEVER**

- Yellow fever is an acute viral disease transmitted to human being by the ***aedes aegypti*** mosquito.
- Yellow fever can spread rapidly, and case fatality rate may reach as high as 30% in non-immune populations. Yellow fever is a disease of forest monkeys (zoonoses) and is transmitted among them by the aedes africanus mosquito.
- Humans may be bitten outside the forest by mosquitoes which have acquired the disease from monkeys feeding on bananas and other agricultural plantations. In urban areas, yellow fever is transmitted by the aedes aegypti.
- Yellow fever is a disease of tropical African countries, especially in the rain forests.
- Yellow fever is an acute viral disease transmitted by infected mosquitoes.
- It is caused by *yellow fever virus*
- The "yellow" in the name refers to the jaundice that affects some patients.
- It is characterised by sudden onset of fever, rigors,headache, jaundice, muscle pain, nausea and vomiting.
- It is a zoonotic disease of forest monkeys
- It is transmitted by a mosquito vector called *Aedes aegypty*

**PATHOGENESIS**

- After transmission of the YF virus from a mosquito the viruses replicate in the lymph nodes. They then reach the liver and infect hepatocytes, wc leads to eosinophilic degradation of these cells.
- When the disease takes a deadly course, there's cardiovascular shock and multi organ failure with strongly increased cytokine levels (cytokine storm) follow.
- Yellow fever begins after an incubation period of 3 – 6 days. Most cases only cause a mild infection with fever, headache, chills, back pain, loss of appetite, nausea, and vomiting. In these cases the infection lasts only three to four days.
- In 15% of cases, sufferers enter a second, toxic phase of the disease with recurring fever accompanied by jaundice due to liver damage, and abdominal pain. Bleeding in the mouth, the eyes, and the gastrointestinal tract will cause vomitus containing blood (i.e. **black vomit**).
- The toxic phase is fatal in approximately 20% of cases. In severe cases the mortality may exceed 50%.

**Mode of Transmission**

- The mosquito becomes infected after feeding on the blood of an infected monkey or person on the ***third day of fever***.

- The incubation period takes 18 days at a daily temperature of 18°C and four days at 37°C. The cycle takes four days and once infected, the mosquito remains infected and infective for its entire life (about two to four months).
- A person may also become infected with yellow fever through handling of blood from an infected individual in the first three days of the disease or handling infected monkeys in the early stages of viraemia.
- Laboratory staff may become infected when working on infected monkeys or infected mosquitoes.

**Yellow fever virus has three transmission cycles: jungle (sylvatic), intermediate (savannah), and urban.**

- The jungle (sylvatic) cycle involves transmission of the virus between non-human primates (e.g., monkeys) and mosquito species found in the forest canopy. The virus is transmitted by mosquitoes from monkeys to humans when humans are visiting or working in the jungle.
- In Africa, an intermediate (savannah) cycle exists that involves transmission of virus from mosquitoes to humans living or working in jungle border areas. In this cycle, the virus can be transmitted from monkey to human or from human to human via mosquitoes.
- The urban cycle involves transmission of the virus between humans and urban mosquitoes, primarily *Aedes aegypti.* The virus is usually brought to the urban setting by a viremic human who was infected in the jungle or savannah

**Clinical Picture**

The onset is sudden with the following signs and symptoms:

- Fever
- Headache
- Backache
- Nausea and vomiting
- A bleeding tendency (epistaxis, bleeding gums, haematemesis, malaena)
- Liver cell necrosis (in severe illness) resulting in jaundice
- Nephritis leading to albuminuria which may proceed to anuria and renal failure
- Once contracted, the yellow fever virus incubates in the body for 3 to 6 days.
- The most common Symptoms are fever, muscle pain with prominent backache, headache, loss of appetite, and nausea or vomiting. In most cases, symptoms disappear after 3 to 4 days.
- A small percentage of patients, however, enter a second, more toxic phase within 24 hours of recovering from initial symptoms
- High fever returns and several body systems are affected,
- usually the liver and the kidneys. In this phase people ar develop and abdominal pain with vomiting.
- Bleeding can occur from the mouth, nose, eyes or stomach. Half of the patients who enter the toxic phase die within 7 - 10 days.

**DIAGNOSIS**

- A presumptive diagnosis of yellow fever is often based on the patient's clinical features, places and dates of travel (if the patient is from a non-endemic country or area), activities, and epidemiologic history of the location where the presumed infection occurred
- Laboratory diagnosis of yellow fever is generally accomplished by testing of serum to detect virus-specific IgM and neutralizing antibodies. Sometimes the virus can be found in blood samples taken early in the illness.

**DIFFERENTIAL DIAGNOSIS**

- Viral hepatitis
- Malaria
- Leptospirosis
- Relapsing fever
- ebola

**Management**

- Yellow fever like most other **viral haemorrhagic diseases** has no specific drug for treatment.
- You only give supportive treatment and ensure that the patient is nursed in strict isolation using ordinary barrier nursing techniques.

- You also ensure that the patient has no further contact with the mosquitoes through the use of insecticide treated bed nets and ensuring that the room is well screened. This is to prevent further spread of the disease from the patient to other healthy people.

**TREATMENT**

- Treatment is directed at symptomatic relief or life-saving interventions. Rest, fluids, and use of analgesics and antipyretics may relieve symptoms of fever and aching
- There is currently no specific anti-viral drug for yellow fever but specific care to treat dehydration, liver and kidney failure, and fever improves outcomes. Associated bacterial infections can be treated with antibiotics.

**Prevention and Control**

- Administering yellow fever vaccine to all travellers coming from or going to yellow fever endemic areas.
- Spraying aircraft coming in from yellow fever endemic areas with insecticides to kill imported mosquitoes, which may be infected.
- Isolating all persons who have been in contact with the infected persons. Such individuals should be quarantined in screened houses for seven days.
- Mass immunisation campaign for the community in areas infested with the aedes aegypti mosquito.
- Spraying of larvicides in all possible mosquito breeding places including water holding plants.
- Immunization against yellow fever in regional hospitals,internation airports and border entry points
- Isolating all contacts in screened quarters to prevent spread of infection
- Disease notification of all suspected cases
- Mass immunisation campaigns to cover all sorrounding areas infected with aedes mosquito
- urging all county health departments and relevant stakeholders to enhance disease surveillance, in particular for yellow fever, at points of entry (PoEs) and within the country
- providing information to travellers on yellow fever vaccination and implementation of inspection of yellow fever certificates at PoEs.

**MANAGEMENT OF YELLOW FEVER OUTBREAK**

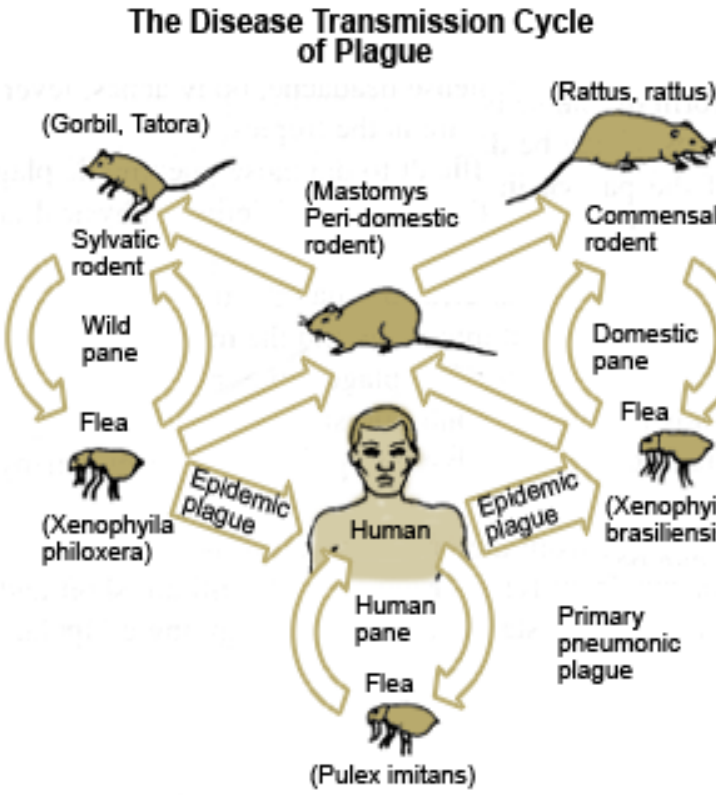
- Notify the health authorities of all suspected cases
- Verify the presence of outbreak by checking medical and laboratory records in health facilities
- Isolating all contacts in screened quarters to prevent spread of infection
- Mass immunisation campaigns to cover all sorrounding areas infected with aedes mosquito
- Carrying out social mobilization activities;
- Activating a national task force to manage the detected imported cases;
- Strengthening the testing capability of the reference laboratory
- providing information to travellers on yellow fever vaccination and implementation of inspection of yellow fever certificates at PoEs.

**PLAGUE**

- This is a highly infectious disease caused by ***bacteria*** called ***yersinia pestis***.
- Plague is a disease of rodents, especially rats and is spread from rat to rat by a rat flea called xenopsylla cheopis.
- Plague is a very rare but serious disease because it can spread very rapidly unless the first case is recognised early and appropriate action taken.
- It is also a serious disease with a high mortality rate (case fatality rate in the absence of treatment can be as high as 60%).

**Mode of Transmission**

Plague occurs when infected wild rats, especially the sewer rat (R. norvegicus) die from the disease and their fleas look for substitute domestic rat (rattus italia) hosts. The domestic rat becomes infected and after it dies the fleas start biting human beings. When the first human is infected, the disease causes bubonic plague. People working in the fields may also be bitten by fleas from the dead infected wild rats and develop bubonic plague.




**Clinical Picture**

Plague has three clinical presentations, bubonic, septicaemic and pneumonic.

**Diagnosis**

The diagnosis of plague can be confirmed by doing a microscopy (staining) of sputum or pus from the bubo to demonstrate the bacilli.

***Remember: Early recognition of plague followed by correct action is a matter of life or death.***

You must start treatment as soon as you confirm the diagnosis from clinical and laboratory findings. The plague bacillus (Yersinia pestis) is sensitive to most common antibiotics except penicillin. Drug treatment with any of the following antibiotics is effective:

- IM streptomycin 30mg/kg two to four times daily for ten days
- Oral or IV tetracycline 10mg/kg six hourly for ten days
- Oral cotrimoxazole two tabs twelve hourly for seven days
- Oral chloramphenicol 500mg six hourly for seven days

***Nb: Plague is an internationally notifiable disease.***

**Management**

**Prevention and Control**

The prevention and control of plague depends on the following measures:

- Early diagnosis and notification so that the patients are not moved or referred to the hospital
- Chemoprophylaxis of all contacts of the patients such as family, visitors and health care workers using tetracycline or cotrimoxazole
- Isolation of the infected and quarantine of the contacts for ten days
- Use of insecticides to kill fleas
- Eradication of rats, for example using rat poison
- Vaccination during epidemics using an anti-plague vaccine
- Health education for communities on preventive measures

## RELAPSING FEVER

- This is an ***acute infectious bacterial disease*** which is characterised by alternating febrile periods.
- It is also known as **Recurrent fever**, **Spirillum**, **Tick fever**, or **Tick Bite fever**. It is transmitted by ticks and lice.

***There are two types of relapsing fever, namely:***

1. Louse-borne relapsing fever
2. Tick-borne relapsing fever

**Mode of Transmission**

The disease is transmitted from person to person by the bite of the head louse, body louse or soft tick.

**Louse-borne**

- The louse-borne relapsing fever is spread by the human head louse, pediculus capitis, and the body louse, pediculus corporis. They transmit spirochaetes of the genus borrelia reccurentis.
- The human louse transmits louse-borne relapsing fever from person to person. When the louse feeds on the blood of an infected person, it takes up the bacteria.
- The bacteria multiply within the body of the louse (but these spirochaete are not found in the saliva or the excreta of the louse).
- The infection is transmitted to another person only when the louse is crushed on the body surface near a bite wound.

- The offspring of an infected louse does not carry the spirochaetes. Epidemics of louse-borne relapsing fever are associated with times of war and famine when refugees are crowded together in unsanitary conditions, which promote infestation with human body lice.
- Body louse (*Pediculus humanus humanus*) is a vector for *Rickettsia prowazekii, Bartonella quintana* and *Borrelia recurrentis* wc cause louse-borne relapsing fever (more severe than the tick-borne variety).
- It is currently prevalent in Ethiopia and Sudan.

**Tick-borne**

- The tick-borne relapsing fever is transmitted by soft ticks (ornithodoros moubata) which live in cracks and crevices of walls and floors.
- They transmit spirochaetes of the genus borrelia duttoni, which cause tick-borne relapsing fever. Children, visitors and pregnant women travelling to endemic areas are more susceptible to the disease. Adults in endemic areas are semi-immune to relapsing fever.
- Tick-borne relapsing fever is transmitted when a tick sucks blood from an infected person. The spirochaetes are taken up and multiply in the tick's body. In seven days, the spirochaetes appear in the tick's salivary glands and the coxal fluid ready to be transmitted to a new host. The organisms can either be injected directly when the tick feeds on the host, or they can infect a new host by penetrating intact mucous membranes (for example in laboratory infections).
- Unlike in louse-borne fever where the offspring does not carry the organism, in tick-borne fever the borrelia duttoni organisms pass into the ovary of the tick, thus automatically infecting the offspring of the ticks (vertical transmission). In this way, a house once inhabited by infected ticks will remain dangerous for up to ten years.
- In an infected pregnant woman, the spirochaete can cross the placenta to the foetus resulting either in abortion, stillbirth, premature delivery, or congenital infection in the newborn.
- Tick-borne relapsing fever is found primarily in Africa, Spain, Saudi Arabia, Asia in and certain areas of Canada and the western United States.
- *Borrelia hermsii, Borrelia parkeri* and *Borrelia duttoni* are transmitted by the soft-bodied African tick *Ornithodoros moubata*, is responsible for the relapsing fever found in central, eastern and southern Africa. Rodents act as reservoir hosts.

**Clinical Features**

The patient presents with **sudden onset of fever** which ranges between 39.5°C - 40.5°C. There is **rapid pulse**, **headache**, **aching joints**, **vomiting** and **infected conjunctiva**. Often there is **potential rash, epistaxis**, and **herpes labialis**. After five to seven days, the temperature drops by crisis. In about 60% of the patients, a less severe relapse of the symptoms occurs five to ten days after the first attack. A second relapse may occur in about 25% of the patients. In untreated cases, there may be up to ten relapses. The fever and clinical symptoms become less severe each time after the relapse. Relapsing fever has a high mortality rate of 40%. Common complications of relapsing fever include meningitis, iritis, optic nerve atrophy (blindness), myocarditis and liver failure bleeding.

**Diagnosis**

You can confirm relapsing fever by doing a microscopic examination of a thick blood smear for the spirochaetes.

**Management**

- Treatment should eradicate the spirochaete from the body without eliciting Jarisch-Herxheimer reaction.
- Some deaths occur after starting treatment as a result of a severe Jarisch-Herxheimer reaction. The antibiotics suddenly kill a large number of spirochaetes which release toxins into the circulation causing the patient to collapse.
- This reaction is characterised by chills, rapid breathing, elevated temperature (40 – 42°C), confusion, delirium, and sometimes convulsions and coma. The patient then develops very severe hypotension, and may go into heart failure. This complication is however not seen in tick-borne infections. Patients must be nursed flat, given adequate fluids and be confined to bed for at least 24 hours.
- The treatment of relapsing fever is **IM procaine penicillin 400,000 units stat**, followed the next day by **oral tetracycline 500mg six hourly for five to seven days**. An alternative to tetracycline is oral doxycyline 200mg once (single dose).

***Remember: Tetracycline should not be given to children and pregnant women because it discolours the teeth***

***permanently and also causes premature calcification of bones.***

**Prevention and Control**

**Louse-borne**

To eradicate lice you should advise the patient to do the following:

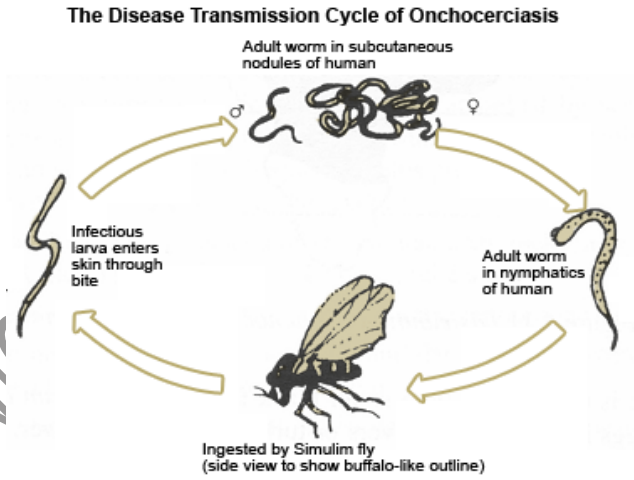
- Improve their personal hygiene
- Use insecticides to kill lice, for example malathion powder
- Boil clothes to kill lice and eggs (delousing)

## ONCHOCERCIASIS

- Onchocerciasis is a chronic disease caused by a ***filarial worm called onchocerca volvulus***.
- It ***lives in the subcutaneous and connective tissue of the infected person***. It manifests mainly as skin nodules on bony surfaces, and **causes eye lesions** which **result in blindness**.
- ***That is why it is also known as river blindness.***
- The vector for ***O. volvulus is the female black fly of the genus simulium***. In western African countries where the disease is more prevalent, the vector is simulium damnosum, while in East Africa the vector is simulium neavei.
- The disease is found in western Uganda, southern Sudan, and eastern Democratic Republic of the Congo (DRC). Blackflies are able to travel up to 80km in a day.
- The simulium fly breeds in fast running well aerated rivers or turbulent areas of a river such as at the waterfalls and rapids.
- The eggs of the simulium fly are able to develop into larvae only in water that is rich in oxygen, such as fast flowing rivers. Larvae are attached to submerged plants, rocks and living crabs.
- The female O. volvulus worm is only about 0.3mm in diameter but can be as long as half a meter (50cm) long. The male is about 0.2mm in diameter and 4 - 13cm long.

**Mode of Transmission**

- River blindness is spread from person to person by the bite of an infected blackfly. Black flies feed during the day both inside and outside houses.
- They usually bite early in the morning or late in the evening.
- The blackfly takes up microfilariae when it sucks the blood of an infected person.
- Once in the stomach, the microfilariae penetrate the stomach wall and travel to the thoracic muscles where they develop further for about seven days.
- They then move to the head of the fly ready to be transmitted to the next susceptible person when the fly feeds.
- When the fly bites again, it injects the larvae of O. volvulus into the skin of the healthy host. The larvae mature in the human subcutaneous tissue into adult worms in about one to three years.




## LIFE CYCLE

- Humans become infected when blackfly bites and deposit *Onchocerca* infective larvae into the skin.
- Once inside the human body, the larvae mature into adults in around 3 months to 1 year.
- The mature males and females then collect in balls ,bound together by fibrous tissue which forms the typical nodule.The nodules are best seen on bony skin surfaces like elbow,shoulder scapula,skull,ribs and iliac crest
- Most adult female worms live in fibrous nodules under the skin and sometimes near muscles and joints
- If a blackfly bites an infected person, onchocerciasis larvae can be ingested by the blackfly after whichThey migrate to the biting parts of the fly where they can be transmitted back to humans when it bites again.

**Clinical Features**

After the adult O. volvulus has lived in the body of an infected person for about one year, it begins to give birth to microfilariae. One adult female worm can produce up to one million microfilariae every year. The microfilariae of O.volvulus have a strong liking for the skin and eyes of the infected host. Adult worms live up to 17 years in nodules in the subcutaneous and connective tissue. Most nodules are found on the bony skin surface such as the elbow, skull, ribs, iliac, crests, and shoulder scapula.

**THE DISEASE HAS FOUR DIFFERENT CLINICAL PRESENTATIONS:**

1. **Severe Itching**

- This is one of the early symptoms and mainly affects the buttocks. The severe itching is often accompanied by skin depigmentation giving rise to a 'leopard skin'.

2. **Skin Nodules**

These are caused by the adult worms which you saw earlier like to live in the skin. They contain adult worms and are painless, rubbery, and firm; ranging in diameter from 3mm - 3cm.

Are caused by adult worms.Are firm and non tender ranging from 3mm to 3 cm in diameter.

3. **dermatitis**

This is caused by a reaction to the presence of microfilariae in the epidermis and manifests as itchy papules and macules. Later, the skin becomes loose, scaly, atrophic and depigmented. Caused by presence of microfilariae in epidermis and manifests as itchy papules or macules.Later the skin become loose,scaly,atrophic and depigmented

4. **blindness**

This is caused by the presence of microfilariae in the cornea and the anterior chamber of the eye. It starts with oedema of conjunctiva, then corneal spots and a pannus begin to develop. Finally cataracts, iritis, sclerosing keratitis, and glaucoma develop leading to blindness. You can differentiate between trachoma and river blindness because in river blindness the pannus start at the lower limbus, while in trachoma it affects the upper limbus.

**Diagnosis**

The diagnosis is made by examining skin snips from the thighs, buttocks and iliac crests under a microscope for microfilariae.

- The most common method of laboratory diagnosis used to identify microfilariae is a biopsy of the skin called a skin snip. Microfilariae emerge when the skin snip is placed in a saline solution.
- Nodules can also be surgically removed to identify adult worms
- A physical examination of the patient can detect localized dermatitis or subcutaneous nodules

***NB: Onchocerca volvulus is not found in the blood therefore it cannot be used for diagnosis***

**MANAGEMENT**

- First line treatment is ivermectin 150µg/Kg orally either yearly or bi-annually.
- Ivermectin kills circulating microfilariae as well as those that are still in adult female worms; this reduces the numbers of microfilariae in the skin and the production of new microfilariae by adult worms so the disease does not progress
- Contra-indicated in pregnancy and breastfeeding women.
- Six weeks course of doxycycline
- Diethylcarbamazine {Hetrazan or Banocide
- Kills microfilariae though it has no effect on adult worm
- Surgical excision when nodules are located in the heade
- MAIN GOAL OF THERAPY: to prevent deelopment of irreversible lesions and alleviate symptoms

**TREATMENT**

Onchocerciasis is not a fatal disease. If the patient has no serious complaints and is likely to be re-infected, there is no urgency for treatment, since the traditional drugs used have been known to cause severe reactions. However, the following groups of patients do need treatment:

- Patient with eye lesions
- Patients with severe skin lesions
- Patients with heavy infections

***Two types of treatment are used in the management of this disease.***

**i)** The first one is to kill the microfilariae. Give the patient oral Ivermectine (mectizan) 150 microgram/kg single dose repeated once every six to twelve months.

**ii)** The second type of treatment is aimed at killing or removing the adult worms by surgical resection of the nodules.

**Prevention and Control**

The following measures have been found to be useful in preventing onchocerciasis:

- Addition of insecticide to the water of rivers known to be breeding places of the simulium fly
- Wearing of long clothing which covers most of the body
- Moving the whole community away from sites near where black flies breed
- Treating infected people with microfilaricides
- Mass treatment of communities using ivermectine

**SUMMARY**

- Onchocerciasis, commonly known as "river blindness", is caused by the parasitic worm *Onchocerca volvulus*.
- It is transmitted to humans through exposure to repeated bites of infected blackflies of the genus *Simulium*.
- Symptoms include severe itching, disfiguring skin conditions and visual impairment, including permanent blindness
- Community-directed treatment with ivermectin is the core strategy to eliminate onchocerciasis in Africa. In the Americas the strategy is biannual large-scale treatment with ivermectin

## LEPROSY (HANSEN'S DISEASE)

- Leprosy is one of the oldest diseases of human beings.
- It is caused by a **bacteria** belonging to the same family as the **mycobacterium** that causes tuberculosis, known as ***mycobacterium leprae*.**
- Leprosy is a major public health and socio-economic problem because it is a disabling and deforming disease. Leprosy is not a killer disease in that it runs a chronic course and does not significantly reduce the life expectancy of the infected individual.
- In some communities patients suffering from leprosy are discriminated against or stigmatised due to ignorance and unfounded traditional beliefs.
- This causes a lot of distress and misery to those infected and their families. In Kenya, leprosy has almost been eradicated except for a few endemic areas in the Coast, Eastern, Nyanza and Western Province.

**Mode of Transmission**

- Leprosy has a long incubation period and runs a chronic course if it is not adequately treated at an early stage.
- The *mycobacterium leprae* bacillus multiplies very slowly (dividing only once every 14 - 30 days). That is why the incubation period is long, about five to eight years. Just like tuberculosis, the leprosy bacillus is transmitted by droplets, by sneezing, coughing, spitting and unhygienic nose cleaning habits.
- The organism is also suspected to enter the body through broken skin such as small wounds. Leprosy is common among family members of the infected.

**There are certain factors that increase the incidence of leprosy in the community:**

- Presence of many untreated cases
- Overcrowding in living houses
- Presence of susceptible new comers in a leprosy endemic area
- Hiding patient with leprosy and starting treatment late

**Classification (Types) of Leprosy**

Broadly speaking, there are two forms of leprosy:

**i)** Tuberculoid form

**ii)** Lepromatous form.

***Pauci-Bacillary Leprosy (PBL), also called*** *tuberculoid leprosy* ***is characterised by:***

- Absence or presence of very few of bacilli in the skin smears or skin biopsy (skin smear is negative)
- Skin patches 1 - 5cm
- Reaction type I
- Nerve involvement/damages affects one or more peripheral nerves
- Disability and deformities are common as a result of irreversible nerve damage and most are disfiguring

***Multi-Bacillary Leprosy (MBL), also called*** *lepromatous leprosy****, is characterised by:***

- Presence of numerous bacilli in most tissues of the body, except brain and spinal cord
- Skin patches six or more cm
- Skin smears positive (numerous bacilli present)
- Reaction both type I and type II

- Nerve damage comes late
- Disability and deformities usually develop at a later stage of the disease

**Nerve Involvement in Leprosy**

- The main cause of disability in leprosy is the destruction of the nerves.
- Damage to the sensory nerve fibres causes anaesthesia, while damage to the motor nerve fibres causes paralysis.
- Impaired circulation, loss of sweating and skin atrophy is caused by damage to autonomic nerve fibres.
- Leprosy patients may get burned or injured on their limbs and fail to notice because of anaesthesia. The patient may walk on an injured foot without realising it.
- In the eye, the cornea may become anaesthetic so that foreign bodies may enter unnoticed leading to corneal damage.
- Anaesthetic eyelids may lose the blinking reflex or fail to close the eye (lagophthalmos) leading to dryness, iritis, adhesions, glaucoma and blindness.

**Clinical Features**

After infection, the mycobacterium leprae bacilli multiply in macrophages of the skin and the schwann cells of the peripheral nerve fibres. The bacillus has a preference for the relatively cool places in the body such as the face and the limbs.

***The early signs of leprosy are as follows:***

- Hypopigmented patches on the skin with loss of sensation to pain, touch and temperature
- Loss of sweating or loss of hair over the affected part
- Burning sensations in the skin
- Weakness of eyelids, hands or feet
- Thickening of cutaneous nerves especially the ulnar, median and lateral popliteal nerves
- Nodules in the skin especially of the nose, face and ears
- Painless wounds (ulcers) and burns on the hands and feet

**Reaction Types**

Reactions are sudden unexpected changes which occur in all types of patients with leprosy. These reactions are caused by a change in the balance between the immunity of a patient and the bacilli. There are two main types of reactions, type I or reversal reaction and type II or erythema nodusum leprosum.

**Type I Reaction (Reversal Action)**

Type I reaction (reversal action) is common in Pauci-Bacillary Leprosy (PBL). It occurs after a sudden increase in immunity results in a rapidly increased response of the body to the leprosy bacilli. This reaction causes sudden inflammation in places where the leprosy bacilli are present. It causes nerve damage, inflamed and raised red skin lesions and oedema of hands, face or feet.

**Type II Reaction (Erythema Nodosum Leprosum)**

This appears six months or more after treatment and is caused by a reaction between dead leprosy bacilli and circulating antibodies. Nerve damage is not common in this reaction. Eyes, joints and testes become inflamed, nerve become tender and ulcerating tender nodules appear on the skin. Thus, reaction is usually of sudden onset and tends to recur.

Generally, reactions in leprosy are provoked by a number of factors. These include:

- Malaria, malnutrition, anaemia
- Severe emotional or physical stress
- Menstruation, pregnancy, abortion, puberty and childbirth
- Using drugs containing iodine
- BCG vaccination
- Osteomyelitis
- Septic wounds

***Remember:***
***Drugs for leprosy do not cause reactions and therefore should not be stopped.***

**Late Deformities of Leprosy**

The following are the late deformities of leprosy:

- Paralytic deformities including claw hand, claw fingers, wrist drop, food drop, claw toes, lagophthalmia, corneal ulcers, and facial paralysis
- Depression of the nasal bridge
- Wrinkling of facial skin
- Disfigured ears
- Stiffness of finger joints
- Shortening and loss of fingers and toes

**Diagnosis**

The diagnosis of leprosy can be made using the following:

- Clinical signs: presence of pigmented anaesthetic patches on skin and thickened nerves
- Bacteriological examination: skin slit and skin crap, nasal smears for leprosy bacilli

- Chemical tests: histamine test, lepromin test

**Management**

The aim of leprosy treatment is to prevent nerve damage, deformity, blindness and defaulting. The National Leprosy and Tuberculosis Programme (NLTP) in Kenya uses the WHO recommended multiple drug therapy for the treatment of the two classes of leprosy.

**Pauci-Bacillary (Tuberculoid) Leprosy (PBL)**

This type of leprosy is treated for six months as shown in the table below.

Six months treatment for pauci-bacillary leprosy for all ages*.

| | 0-5 years | 6-14 years | Over 14 years |
|---|---|---|---|
| Rifampicin every four weeks supervised | 150mg | 300mg | 600mg |
| Dapsone daily | 25mg | 50mg | 100mg |

*Adapted from the Kenya National Leprosy and Tuberculosis Programme (NLTP)

**Multi-Bacillary Leprosy (MBL)**

Multi-bacillary or lepromatous leprosy is also treated for six months as shown in the following table.

Six months treatment for Multi-Bacillary Leprosy for all ages*

| | 0-5 years | 6-14 years | Over 14 years |
|---|---|---|---|
| Dapsone daily | 25mg | 50mg | 100mg |
| Clofazimine (lamprene) four weekly supervised | 100mg | 200mg | 300mg |
| Clofazimine (lamprene) unsupervised | 50mg on alternate days | 50mg daily | 50mg daily |
| Rifampicin every 4 weeks supervised | 150mg | 300mg | 600mg |

*Adapted from the Kenya National Leprosy and Tuberculosis Programme (NLTP)

Having looked at drug therapy you will now find out what else can be done to prevent blindness and deformity.

**Wound Prevention in Leprosy**

Wounds are caused and made worse by the loss of sensation to pain, pressure or burning. Therefore to prevent further damage you should advise the patient to do the following:

- Wear protective footwear
- Wear heatproof gloves when working and handling hot objects
- Inspect the feet and legs regularly for swelling, cracks, bruises, injuries, dryness - a small mirror can be used to inspect the soles of feet
- Soak feet for 20 minutes twice daily in salty water, then rub oil on the skin to keep it moist and prevent cracks
- Remove grit from inside the shoes

**Eye Care**

For the patients who are suffering from lagophthalmos, you should advise them as follows:

- Wear sun glasses
- Check the eye daily in front of a mirror for inflammation and foreign bodies
- Cover the eyes with pads at night
- Avoid rubbing the insensitive eyes

**Exercises**

It is common knowledge that joints which are not used become stiff, while muscles atrophy and become weak. Also scar tissue tends to retract resulting in contractures. That is why all patients with weak or damaged hands should do suitable exercises. For paralysed muscles, passive exercises help to loosen the stiff joints and lengthen the skin. The exercises should be done for five to ten minutes daily on a regular basis.

**Prevention and Control**

The cornerstone of leprosy control is to reduce the number of infective cases and interrupt transmission. These can be achieved through the following preventive measures:

- Treatment of all infective cases until cured
- Searching for unknown cases, registering and treating them
- Administration of BCG vaccine which gives some immunity against leprosy

## HELMINTHIC DISEASES

Helminthic diseases are still a very common problem in Kenya, despite the fact that it is known how to prevent and treat them. They are common in low income areas

such as slum settlements due to lack of proper facilities for human waste disposal as well as poor attitudes.

***The other factors which promote the spread of some helminths are:***

- Moist warm soil in the case of hookworms
- Cattle keeping areas in the case of tapeworms
- Lack of latrines in the case of roundworms
- Unwashed hands in the case of threadworm

Helminthic diseases can be categorised into two groups:

**i)** Nematodes

**ii)** Flatworms

## NEMATODES (CYLINDRICAL-SHAPED WORMS)

This group is made up of cylinder-shaped worms and includes

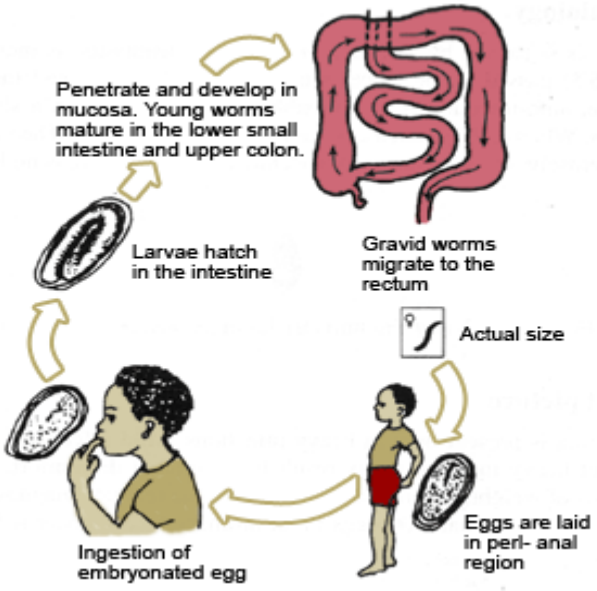
**a.** Threadworms/Pinworm

**b.** Whipworms,

**c.** Roundworms

## THREADWORM/PINWORM

The threadworm is caused by **enterobius vermicularis**. It has a worldwide distribution and mainly affects school aged children, especially in boarding schools. The children reinfect themselves when they scratch their anus and then transfer the eggs on their fingers to the mouth.




**Mode of Transmission**

Infection with enterobius vermicularis is maintained by direct transfer of infective eggs from the anus to the mouth (auto infection) or indirect contact through clothing, bedding, food and other articles.

After ingestion, the eggs hatch in the stomach and small intestine. The worms mature in the lower small intestine and upper colon and then they migrate to the rectum where they discharge eggs on the perianal skin, especially during the night. This causes itching and consequently scratching.

The graphic illustrates the life cycle of the pinworm.

**Clinical Features**

**List four clinical features of pinworm infestation.**

Your list should include the following signs and symptoms of pinworm infestation:

- Mainly pruritus ani leading to intense scratching of the perianal region
- Disturbed sleep
- Restlessness
- Loss of appetite and weight loss

**Diagnosis**

Diagnosis is mainly made by a laboratory examination of stool microscopy for ova and cyst.

**Management**

You should treat the whole family with mebendazole 100mg given as a single dose. During treatment you should impress on the patient the importance of avoiding auto-infection.

**Prevention and Control**

The prevention and control of this disease lies in improved personal hygiene and proper disposal of faeces. You should give health education on the importance of bathing and hand washing, keeping nails short, and how to prevent reinfection.

## WHIPWORM

This infestation is called **trichuriasis** because it is caused by an intestinal worm called **Trichuris trichiura**. The worm infects the large intestine and infestation is usually asymptomatic.

**Mode of Transmission**

The transmission of trichuriasis is indirect, as the eggs passed in the faeces require embryonation in soil. Therefore unlike the threadworm, auto-infection is not possible.

When the embryonated eggs are ingested, they hatch and eventually the mature worms attach themselves to

the mucosa of caecum and colon. They are mainly transmitted through food that is contaminated by soil or dirty fingers.

**Clinical Features**

Often, mild infections are asymptomatic, but heavy infections may result in abdominal discomfort, bloody diarrhoea, loss of weight and prolapse of rectum.

**Diagnosis**

Diagnosis is made by examining a stool sample microscopically for ova and cyst.

**Management**

You can eliminate this infection by giving oral mebendazole 100mg 12 hourly for three days.

**Prevention and Control**

Just like the threadworm, the prevention of trichuriasis can be achieved through good personal hygiene and proper disposal of faeces.

## ROUNDWORM (ASCARIASIS)

This disease is caused by **Ascaris lumbricoides**, which infects the small intestine.

Ascaris is a large intestinal parasite which often infects children because of their habit of putting all kinds of things in their mouth.

It is one of the commonest and most widespread infections of the small intestine. The worms may multiply in large numbers in the intestinal lumen and cause intestinal obstruction at the ileocaecal valve.

The worms also contribute to severe malnutrition and vitamin A deficiency, and may wander out of the intestinal lumen into the peritoneal cavity.

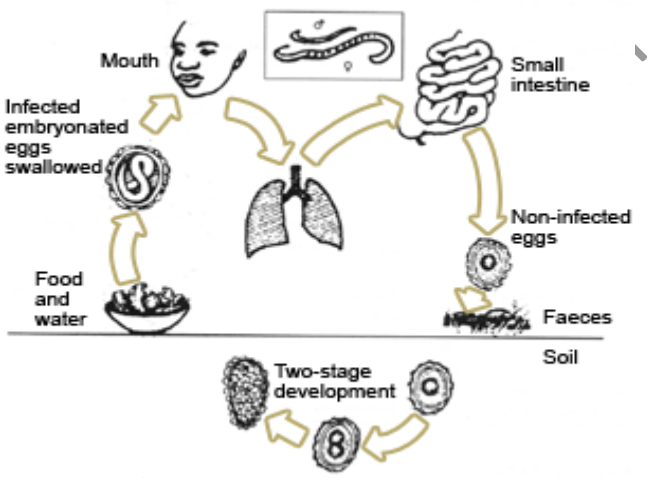
**Mode of Transmission**

Ascariasis is a soil transmitted parasite. Once the eggs are passed out in faeces, they require embryonation in the soil before they can become infective. This takes 8-50 days. Embryonated eggs can be carried away from the contaminated place into houses by feet, footwear or in dust by the wind. Human beings may injest the eggs as they eat or drink using contaminated hands and utensils, orthrough eating raw contaminated foods like fruit. Once the eggs are ingested by a human being they hatch into worms.

In order to reach maturity, the larvae need to pass through the lungs and trachea to the pharynx.

Once in the pharynx they are swallowed and return to the gastrointestinal tract where they can live for about a year.

The graphic illustrates the life cycle of Ascaris lumbricoides.




**Clinical Features**

Infection with a few ascaris is usually asymptomatic and if symptoms are present, they are not characteristic.

There may be vague abdominal discomfort or occasionally the worm may leave the body in vomitus or stool. Also during the stage of larval migration through the lungs there may be temporary symptoms of pneumonitis (cough).

**Diagnosis**

Diagnosis is by stool microscopy which should show ascaris ova and cyst.

**Management**

Any one of the following drug treatments is useful in the management of ascariasis.

- Oral mebendazole 100mg 12 hourly for three days
- Oral levamisole (3 tabs or 5mg/kg body wt) single dose
- Oral piperazine 150mg/kg body wt single dose

Note: For intestinal obstruction, surgical operation is indicated.

**Prevention and Control**

The prevention and control of ascariasis involves the following measures:

- Improved environmental sanitation such as proper excreta disposal, clean supply of water
- Discouraging the use of raw (fresh) human faeces for manure (Composting for six months kills the ascaris eggs)
- Washing of fruit and vegetables before eating

- Use of drying racks for utensils so that they do not come into contact with soil and dust
- Washing hands after opening bowels
- Washing hands before handling food

## HOOKWORM (ANCYLOSTOMIASIS)

This is an infection of the small intestine by a blood-sucking worm called Ancylostoma duodenale or necator americanus.

In East Africa, necator americanus is the cause of the disease.

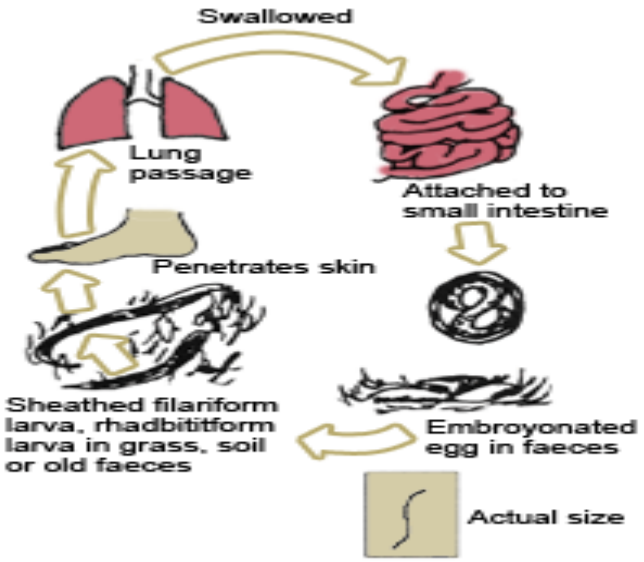
The worm causes severe iron deficiency anaemia and protein loss.

Each adult necator americanus worm causes a daily loss of 0.03ml of blood from the patient. In many infected individuals the disease is asymptomatic because the hookworm load is light.

**Mode of Transmission**

Hookworm eggs are embryonated by the time they are passed out with faeces. Indeed, when the faeces stand for a long time before examination the free larvae can be found.

When an infected person passes faeces in the soil, the larvae bury themselves in the moist damp soil. The larvae are called rhabditiform and only become infective after five days, when they change into the sheathed filariform stage.




As soon as the filariform larvae come into contact with 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.

Once in the lungs they penetrate the alveoli and are carried to the larynx and pharynx, from here they are swallowed into the stomach.

When they reach the stomach they attach themselves to the wall of the abdomen with hook-like teeth and start to suck blood from the patient's body.

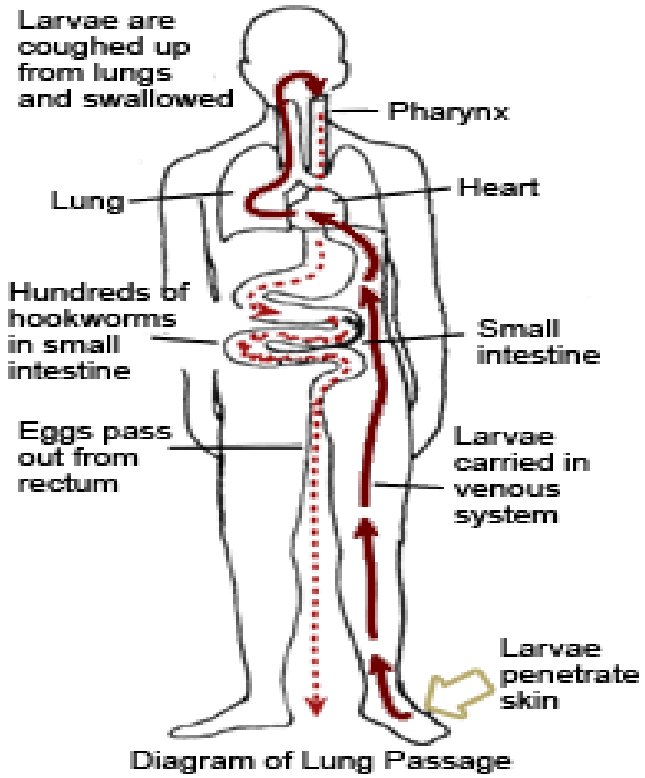


Diagram of Lung Passage

**Clinical Features**

**How would you diagnose a hookworm infection?**

In most of the cases, hookworm infestation tends to be asymptomatic. However the following signs and symptoms are indicative of hookworm infestation:

- Itching of the skin at the site of entry (local irritation)
- Anaemia (due to haemorrhage), pallor
- Weakness, puffy face, malnutrition
- Flatulence, constipation
- Pain in abdomen
- Some little blood in stool

**Diagnosis**

Diagnosis of hookworm infestation is made by stool microscopy which should show ova and cysts and in some cases occult blood. More than 100 eggs in an ordinary faecal smear indicate heavy infection.

**Management**

The following drugs are commonly used in the treatment of hookworm infections:

- Levamisole 25mg/kg body weight as a single dose

- Mebendazole 100mg bd. for three days
- Albendazole 400mg stat

## FLATWORMS

This group is made up of flat or segmented worms, their intermediate hosts are mainly animals, such as cattle, pigs and dogs.

You will look at two worm diseases under this group, namely

i. Tapeworms
ii. hydatidosis

## TAPEWORM (TAENIASIS)

There are various types of tapeworms, but in human beings the infestations are commonly caused by:

**a.** Taenia saginata or beef tapeworm (commonest infection)
**b.** Taenia solium or pork tapeworm

You will now consider each type in turn.

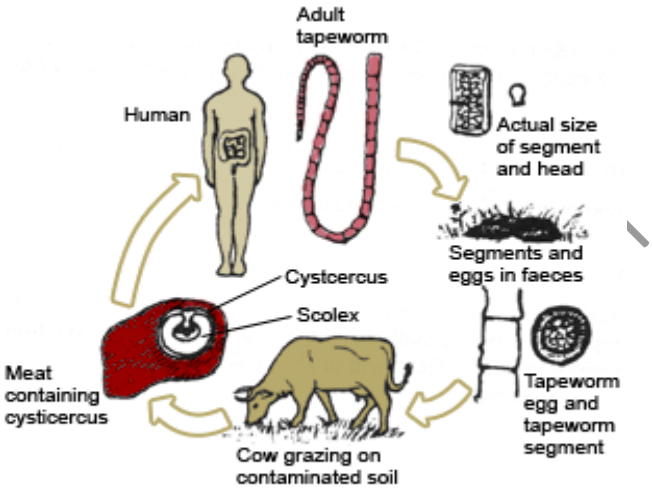
## TAENIA SAGINATA OR BEEF TAPEWORM

Infection with the beef tapeworm is common in areas where beef is eaten raw or lightly cooked.

**Mode of Transmission**

The eggs of adult tapeworms living in the small intestines of human beings are passed in the stools. They are then ingested by cows as they feed on contaminated grass. Once in the gastrointestinal tract of the cow, the embryos hatch and penetrate the bowel wall and are carried via the bloodstream to striated muscles. Here the larvae grow and form infective cysts called cysticerci.

When human beings ingest cow meat containing these cysts, the cysts are dissolved by the gastric acid in the stomach to release embryos.




**Clinical Features**

Most tapeworm infections caused by taenia saginata do not cause any signs or symptoms.

However, some people may complain of loss of weight, abdominal discomfort and itching around the anus (pruritis ani).

**Diagnosis**

Diagnosis of tapeworm infestation can be made by the presence in the stool of segments or eggs.

The eggs are not laid singly and appear only accidentally in the stools.

**Management**

Drug treatment with oral niclosamide is effective. The dose is 1gm chewed and swallowed with water followed one hour later with 1gm (a total of 2gm).

## TAENIA SOLIUM (PORK TAPEWORM)

This disease occurs when a person ingests pork infected with the taenia solium larvae. Whereas in the beef tapeworm the embryo attaches itself to the wall of the small bowel and grows into an adult worm, the pork tapeworm behaves differently. The embryo penetrates the intestinal wall of the human as it does the pig, and it is carried to organs like striated muscle or the brain. This can cause serious problems such as epilepsy and death.

**Clinical Features**

Taenia solium is a dangerous worm and the signs and symptoms depend on the organ it has invaded as follows:

- In the brain it causes epilepsy
- In the skeletal muscles it causes myositis (severe pain), which may make movement temporarily impossible
- In the laryngeal muscles it causes difficulty in speaking
- In the myocardium it causes (myocarditis), heart failure or cardiac arrest
- In the eyeball it can cause unilateral or bilateral blindness

**Diagnosis**

Diagnosis of taenia solium infections can be made by doing the following tests:

- Biopsy examination of the infected tissue
- X-ray examination to locate the calcified cysticercus
- Stool microscopy for ova and cyst

**Management**

The management involves both the surgical removal of calcified cysticercus where possible as well as drug treatment with niclosamide 2gm.

The dose is 1gm chewed and washed down with water followed one hour later by 1gm.

**What measures would you recommend for the prevention and control of taeniasis?**

The prevention and control of taeniasis can be achieved through the following simple measures:

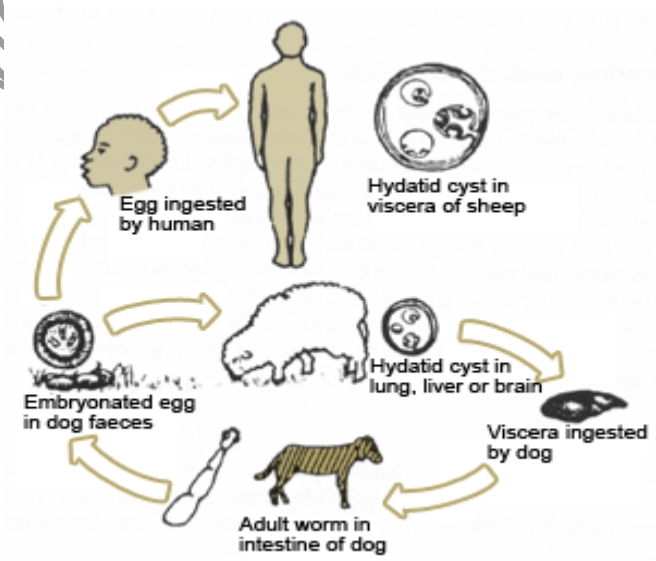
- Proper disposal of human faeces in toilets instead of in the field and within reach of cattle and pigs
- Ensuring that beef, pork and fish are thoroughly cooked
- Eating only meats that have been inspected
- Burying in deep pits or incinerating the carcases of heavily infected cattle and pigs
- Washing hands thoroughly after handling carcases and raw meat
- Early diagnosis and treatment of infected persons

## HYDATIDOSIS (HYDATID DISEASE)

- The hydatidosis disease is actually a disease of dogs (zoonotic).
- Human beings become infected only by accident. Nevertheless, the disease is a serious problem among the Turkana community of northern Kenya.It is also known as ***echinococcosis or hydatid disease.***

**Mode of Transmission**

- Hydatidosis is caused by the cysts of the dog tapeworm known as Echinococcus granulosis.
- Dogs and other carnivores such as jackals and lions are the hosts of the dog tapeworm.
- The eggs are passed in the faeces of an infected dog and ingested by domesticated animals such as sheep, goats, cattle, camels, donkeys, and wild antelopes
- The eggs hatch in the animal's intestine and penetrate through the intestinal wall to the portal circulation.
- They are then carried to the liver and lungs where they form many cysts.
- When a dog eats the diseased animal it becomes infected with these cysts, which then proceed to develop into mature worms.
- Human beings become infected when they accidentally ingest eggs from dog faeces.
- The larvae migrate from the intestine to the liver or lungs causing cysts.
- The larvae can also cause cysts in other tissue in the body.




**Clinical Features**

In the liver, the cyst grows slowly over time thereby enlarging the liver. The abdomen may also become grossly distended.

**Diagnosis**

This is done through a chest x-ray or an abdominal ultrasound investigation. A serological test can also be done to assist in making the diagnosis.

**Management**

The treatment of hydatid disease can either be medical or surgical.

The medical treatment is as follows:

- Oral albendazole 20mg/kg in divided doses twice daily for 30 days (The cure rate with this treatment is 20%). The treatment can arrest the growth of the cyst and reduce its size
- PAIR (Puncture, Aspiration, Instillation of 95% alcohol and Re-aspiration). This is the treatment for the liver or spleen. The ultrasound machine is used to guide the PAIR procedure. This treatment is very effective and has a high cure rate

The surgical treatment is known as endocystectomy. It is the surgical removal of the cysts contents, especially those cysts that are easily accessible like abdominal cysts.

**Prevention and Control**

The prevention and control of the hydatid disease can be achieved by eradicating stray dogs and deworming them. Deworming should be done every six weeks with praziquantel.

You should also provide health education on the dangers of close contact with dogs (licking), especially among children. Also, infected meat should not be fed to dogs.

## DISEASES FROM CONTACT WITH ANIMALS OR ANIMAL PRODUCTS (ZOONIC DISEASES)

**Diseases from Contact with Animals**

Diseases that are transmitted between infected vertebrate animals (animals with a backbone) and humans are called zoonotic.

In some of these diseases, humans are usually the last in the transmission cycle or the final host as in the case of hydatidosis, unless of course the person's body is eaten by a predator.

Similarly in other diseases like rabies and brucellosis, the disease transmission ends with mankind, though possibilities of further transmission can occur if for example, a rabid patient bites another person, or a patient with brucellosis accidentally transmits it to another person.

Zoonoses are transmitted between animals and humans through the following means:

**Vectors**

These include:

- The rat flea which transmits plague among rats and other rodents
- The tsetse fly which transmits trypanosomiasis among game animals and nagana in cattle
- Mosquitoes which transmits yellow fever among monkeys

**Ingestion of Contaminated Material**

Ingestion of meat or dairy products from sick animals, leading to diseases such as:

- Anthrax (meat from cattle and game animals)
- Brucellosis (milk from infected cattle)
- Taeniasis (milk and meat from infected cattle and pigs)

**Animal Bites**

Bites, resulting in diseases such as:

- Rabies (from rabid domestic and wild dogs or foxes)

**Direct Contact with Infected Animal**

Close contact resulting in diseases such as:

- Hydatidosis (close contact with infected domestic dogs or other carnivores)
- Cutaneous anthrax (contact with infected cattle or their products)

In this section you will cover anthrax, rabies and brucellosis, looking at their mode of transmission, clinical picture, diagnosis, management and prevention.

## ANTHRAX

- Anthrax is an acute bacterial disease of herbivores (plant eating animals). However, it occasionally also infects human beings especially those who process hides, skins and wool or work in slaughterhouses.
- Anthrax is caused by a **rod shaped bacteria (bacilli) called bacillus anthracis**.
- The disease can occur in large numbers among cattle (epizootic), especially during drought and flooding when they are moved from one place to another. In humans, this infection takes various forms depending on the route of entry.
- There is anthrax of the skin which affects people who handle cattle, anthrax of the lungs which occurs in people working with infected wool; and anthrax of the bowels which affects families who eat the meat of dead animals.
- The type of disease caused depends on the route of entry of the bacillus or its spores. In animals, anthrax causes a fever which is followed by septicaemia and death. Vultures, which feed on the dead animal can spread the spores.

**Mode of Transmission**

- The bacillus anthracis forms spores when exposed to the air.
- The spores can survive for years in the soil even under harsh weather conditions.
- The spores enter the animals orally (through the mouth or ingestion).
- The body of a sick or dead animal contains millions of anthrax bacilli. These bacilli are shed through animal urine, droppings, saliva milk and blood.
- If any of these body fluids are touched or the meat of an infected animal eaten, a person becomes infected with anthrax.

**Clinical Features**

The clinical features depend on the route of entry of the anthrax bacillus.

Skin or cutaneous anthrax presents with a malignant pustule with a black necrotic centre. The wound is usually painless and has swollen edges. Skin anthrax has low mortality.

Respiratory tract anthrax on the other hand has a high mortality rate and presents with severe respiratory distress and shock.

Digestive tract anthrax is characterised by fever, sepsis, watery diarrhoea and vomiting.

**Diagnosis**

The diagnosis of anthrax is made by taking a specimen (fluid from vesicles, sputum or stool) for a culture to confirm gram-positive rods.

**Management**

Bacillus anthracis responds to penicillin and most other antibiotics.

Patients with anthrax of the respiratory tract need respiratory support and oxygen therapy in a high dependence care unit.

Those with anthrax of the digestive tract may need fluid replacement due to diarrhoea and vomiting.

**Prevention and Control**

Although the main responsibility for the prevention and control of anthrax falls on the veterinary department, you as a health worker also have a role to play.

You should ensure that all meat offered for sale is inspected and educate the community on proper disposal of all infected animals. The carcasses must be burnt or buried two meters deep in the ground in calcium oxide powder (quick lime).

Other measures include annual vaccination of cows at risk, proper disinfection of hides and skins, and vaccination of members of the community who are at risk of getting anthrax.

**Infectious Agent** Bacterium *Bacillus anthracis (B. anthracis),* an aerobic, Gram-positive,
encapsulated, spore forming, nonmotile rod.

**Clinical**

**Clinical**

**Presentation**

Depending on the route of transmission of infection, there are four clinical
syndromes:

**Cutaneous Anthrax**: Characterized by initial itching of exposed skin, an
initial vesicle at the site of inoculation develops into a painless black
eschar; fever, malaise and headache may be present.

**Inhalational Anthrax**: The most lethal form of disease. Initial
presentation includes, sweats, malaise, mild cough, dyspnea, nausea or
vomiting, and this is followed by acute onset of respiratory distress and
shock; there is also radiological evidence of mediastinal widening and
pleural effusion present. Fatality rate is extremely high.

**Anthrax meningitis**: Begins with hypotension, quickly followed by
delirium or coma; refractory seizures, cranial nerve palsies, and
myoclonus have been reported.

**Intestinal Anthrax:** Presents with acute vomiting, abdominal distension,
gastrointestinal bleeding, and peritonitis. Symptoms of oropharyngeal
Anthrax include fever, neck swelling due to lymphadenopathy, throat
pain, oral ulcers and sepsis.

## RABIES

- Rabies is a serious viral disease of canines which is incidentally transmitted to humans by the bite of a rabid animal.
- It is caused by a virus known as lassa virus type I. The disease is of public health importance because it has a case fatality rate of 100%. If a patient is not treated immediately after the bite, once the clinical signs appear it is too late.
- Rabies is found all over the world and in canines. It occurs all the time and in great numbers (enzootic and epizootic). In human beings, rabies is a zoonotic disease, and humans usually do not transmit it any further.

- The main reservoirs of lassa virus type I are felines, hyenas, and mongoose.

**Mode of Transmission**

- The rabies virus is transmitted to humans through the saliva of an infected animal such as a dog or cat.
- This happens when humans get bitten by a rabid animal or when its saliva comes into contact with the mucous membranes or open wound of a person.
- The main reservoirs of the disease are wild animals such as mongooses, jackals and hyenas. These wild animals infect domestic animals including cattle, donkeys and horses, which in turn infect mankind.
- In North and South America, rabid bats have been known to infect humans. All warm blooded animals are susceptible to rabies.

**Clinical Features**

The incubation period of rabies ranges from two weeks to a year, with an average of two to three months. The length of the incubation period is influenced by the following factors:

- The size of the bite - the deeper the bite the shorter the incubation period
- Distance of the wound from the brain - the nearer the wound is to the brain the shorter the incubation period
- Type of wound - if the wound is big with extensive tissue damage the shorter the incubation period

**Write down three symptoms of rabies infection.**

- The earliest symptoms usually consist of increasingly severe pain in the bite wound, depression, irritability, nausea, sore throat, headache and loss of appetite.

***Later, two clinical presentations emerge:***

1. **Furious rabies** whereby the infected person develops convulsions, intense fear of death and irrational excitement, which alternates with periods of alertness and calmness. The patient is also unable to tolerate noise, bright light and cold drought (aerophobia - fear of cold air). There is increased reflexes, muscle spasms, excessive sweating, dilatation of pupils, excessive salivation and lacrimation. The patient develops intense hydrophobia (fear of water) because of the intense pain experienced when swallowing water due to spasms of the pharyngeal muscles. This stage is also known as the 'furious' rabies stage and it lasts for two to three days and sometimes for five to six days. Death usually occurs due to cardiac or respiratory failure during a convulsion.

2. The next stage is the **paralytic rabies** stage which is characterised by paralysis of muscles causing paraplegia, quadriplegia and coma. Patients who reach this stage do not survive for more than a week.

**PATHOPHYSIOLOGY**

- Following inoculation, it begins to replicate in the skin or muscle tissue before it works its way into the peripheral nerves.
- It then spreads to the CNS in the endoneurium of the Schwann cells.
- Terminally, there is widespread CNS involvement and the patient presents with paralysis then to death .

**Diagnosis**

Diagnosis of rabies is made if a person is bitten by a dog with abnormal behaviour and without any provocation. In addition the presence of negli bodies in the brain of a suspected animal should confirm the disease.

**Management**

There is no cure for rabies once the disease has started. It is however possible to prevent it from reaching that stage by doing the following:

**Post Bite Prophylaxis**

Immediately someone is bitten you should give first aid treatment of the bite with the aim of removing as much virus as possible. This involves immediate flushing of the wounds and scratches preferably with running water and washing the surrounding skin with a lot of soap and water. Puncture wounds should be irrigated with a sterile catheter using methylated spirit and povidone. Iodine is also virucidal and may be used to clean the wound.

Bite wounds should not be sutured immediately to prevent more traumas from the suturing needle, which will increase the areas for viral entry into the body tissue. Suturing may be done 24 to 48 hours after the bite using very few sutures under the cover of anti-rabies serum locally.

**ANTI-RABIES VACCINE**

- This is a very safe and effective treatment following a rabid animal bite. The vaccine HDCV (Human

Diploid cells tissue Culture Vaccine) is administered in six doses IM as follows:

- 1ml immediately after exposure (day 0), 1ml on day 3, 1ml on day 7, 1ml on day 14, 1ml on day 28, 1ml on day 90.

**Other Drugs**

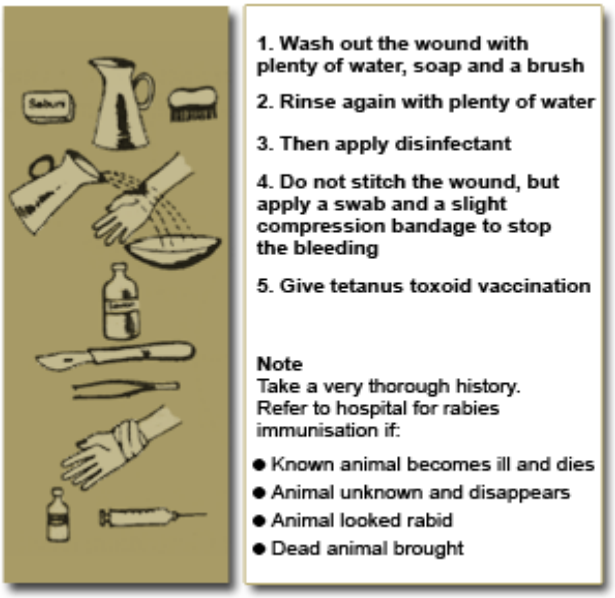
In order to prevent wound infection and tetanus you should give the patient broad spectrum antibiotics.

***Note: The animal, which inflicted the bite, should be quarantine and observed for ten days from the day of the bite.***

***If it shows signs of rabies it should be killed anditshead removed and sent under refrigeration for rabies examination.***

**Prevention and Control**

- Vaccinate dogs and cats against rabies as required by law. All dogs and cats more than four months of age must be vaccinated against rabies.
- Keep dogs and cats under control. Animal control laws prohibit allowing animals to roam unsupervised
- Immediately wash the wound thoroughly, cleaning and flushing with plenty of soap and water for several minutes.
- Immediately report all animal bites to your animal control agency, police department of health department for follow-up.
- Identification and continual observation of the animal (if wild or stray) to aid identification of signs and symptoms of rabies
- Rabies is a notifiable disease. It is very important to give immediate first aid to a person who has been bitten by a suspect animal.
- In addition, you should educate the community members on the importance of immunising their domestic dogs and cats every three years and eliminating all stray dogs and cats.




## BRUCELLOSIS

Brucellosis is a zoonotic disease or disease of animals. It is caused by a bacteria called brucella melitensis in goats, sheep and camels, brucella abortus in cattle and brucella suis in pigs. All these bacteria however can be transmitted to mankind causing brucellosis.

**Distribution**

Brucellosis has a worldwide distribution, predominantly in rural areas among pastoral communities.

It is also an occupational health hazard of farmers, veterinarians, abattoir workers and butchers.

**Transmission**

Brucellosis is transmitted through ingestion of unpasteurised milk or milk products such as cheese.

It can also be transmitted by contact with blood, urine, tissues, through splashing of amniotic fluid or milk on the conjunctiva and blood transfusion.

**Clinical Presentation**

The incubation period takes about two to four weeks. Initially the signs and symptoms are non-specific and include the following:

- Headaches
- Fever
- Weakness
- Anorexia
- Rigors
- Night sweats

- Constipation

Patients may also complain of pain in the large joints like the hips and knees although any other joint may be affected. Hepatomegally, splenomegally and lymphadenopathy may also be present. If untreated, the disease can continue for many months and the patients may become depressed.

**Diagnosis**

A serological diagnosis of brucellosis can be made by doing an agglutination test in dilutions. A level of 1:160 or above is associated with the infection.

Blood cultures rarely give positive results but a bone marrow aspirate culture gives better yields of up to 90%.

Full haemogram - normochromic, normocytic anaemia, neutropenia and lymphocytosis is common.

**Treatment**

The treatment of brucellosis is doxycycline 200mg daily for 14 - 21 days and cotrimoxazole tabs 2 bd. for 14 - 21 days.

**Prevention**

You should educate the community and especially farmers on the importance of boiling or pasteurising milk.

Animal handlers and those at special risk should be advised to take extra precautions.

## CHILDHOOD IMMUNIZABLE DISEASES

### TETANUS:

***Definition:***

An infection of a neonate by anaerobic organism called clostridium tetani.

A fatal infection common in the developing countries

- The infection is acquired by exposure to the spores of the bacterium to the umbilicus stamp which are universally present in the soil/faeces
- The disease is caused by the action of potent neurotoxins *(tetanospasmin*) produced during the growth of bacterium in dead tissues e.g. umbilical stump.
- **Organism** - Clostridium tetani

**Characteristics**

- gram positive
- spore forming
- rod shaped
- anaerobic

**PREDISPOSING FACTORS:**

- Delivery in dirty environment
- Local cutting of the cord using contaminated instrument
- Local treatment of the cord to hasten healing- use of cow dung
- Lack of antenatal immunization

**PATHOPHYSIOLOGY:**

- When the organism enters the body,
- it remains at the site of required medium
- It liberates the toxins which have the affinity of the nerves
- The toxins cause muscles spasms and rigidity.

**CLINICAL FEATURES**

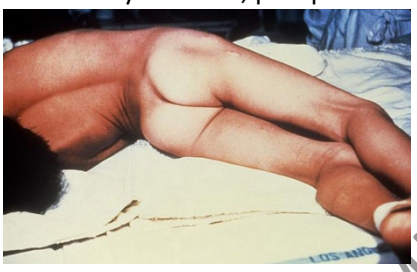
- Inability to suck due to lockjaw (trismus)

The generalised muscle spasms or "convulsions" are often precipitated by stimulation such as handling or loud noises

- convulsions
- Irritability
- Opisthotonus position(back curled/back may arch backwards)
- Cyanosis – Spasms of the diaphragm
- Sardonic us smile- spasms of face muscles(risus sardonicus)
- Respiratory failure and death in untreated infants, due to spasm of the respiratory muscles

**DIAGNOSIS:**

- History – labour, puerperium



***Opisthotonos***

**INCUBATION PERIOD:**

4 – 14 days – average 7 days

- The shorter the incubation period the more severe the symptoms.

**TREATMENT:**

**Antibiotics –** Penicillin 50,000 iu/kg 6 hourly

**Anticonvulsant –** Diazepam 0.5 mg/kg 8 hourly

**Antitetanus serum(ATS)-** 15,000iu after a test dose

**Principles of Treatment**

**Neutralization of unbound toxin** with Human tetanus immunoglobulin.It is antitoxin given 10,000IU IM or IV.A test dose is given while keeping adrenaline at hand because allergic reactions are common and severe

**Prevention of further toxin production** by eliminating the source of toxin through:

- Wound debridement
- Antibiotics (Metronidazole) and penicillins eradicate vegetative cells the source of toxns

**Control of muscle spasms through:**

IV diazepam 10-40mg start

maintain sedation by giving crushed tablets every 3 hours through the Nasogastric tube.

avoid unnecessary stimuli.Nurse the patient in a quiet dark room to minimise provoked spams

Protecting the airway

**SUPPORTIVE CARE.**

- **Observations**- vital signs 4 hourly, spasms – site, duration, frequency
- Airway keep clear to ease breathing- suction and position
- **Nutrition** – EBM through nasogastric tube
- **Hygiene** – daily bed bath, change of cot clothes
- Quiet environment to prevent stimulation of spasms
- Daily cord dressing if the entry point
- **Bonding** – Mother to hold the baby after sedation

**PREVENTION:**

- Antenatal vaccination to stimulate passive immunity- TT
- Individual birth plan- skilled attendant at health facility
- Counsel on dangers of local traditional ways of treating the cord
- Good cord care

**COMPLICATIONS:**

- Aspiration pneumonia
- Mental retardation
- Contractures.

**DIPTHERIA**

- It is acute infectious disease caused by Bacteria – **corynebacteria diptheriae**
- Agent ; **corynebacteria diptheriae**
  - Gram positive, non motile

**Three types;**

1. **Gravis**
2. **Mitis**
3. **intermedius**

  - They are sensitive to penicillin and readily killed by heat and chemicals
  - They survive for short time in dust and formites
- Its characteristically confined to the resp tract

**SOURCE OF INFECTION**

  - Nasopharyngeal secretions, discharge from the skin lesions, contaminated formites and infected dust
  - May be cases or carriers. Carriers are most common and dangerous source
  - Immunization does not prevent carrier state

**MODE OF TRANSMISSION**

  - Respiratory route
  - Skin through cut wounts, ulcers or umbilical cord
  - Raw milk has served as a vehicle

**COMMUNICABILITY**

  - From 14 to 28 days from onset

**HOST FACTORS**

  - Affects children 1 to5 yrs
  - Immunity may be attained by repeated sub clinical attacks of low virulence
  - Infants born of immune mothers have passive protection which is lost before the 6$^{th}$ month

**INCUBATION PERIOD**

2 to 6 days

**PATHOPHYSIOLOGY**

- Starts as acute inflammation of the pharynx. They remain in tonsils and in upper resp mucous membrane. The bacilli secretes an exotocin that is more toxin than cobra venom
- Exotocin irritates tissues which give fibrinous exudates that coagulates into a tough, leathery grayish white pseudomembrane
- The membrane along with swelling due to inflammation may occlude air passages esp if larynx is invaded
- Death may result from mechanical obstruction unless tracheotomy is done
- The toxin may cause myocarditis and sudden heart failure, paralysis, nephritis with albuminuria
- It may also affect mucous membrane of the conjuctiva or vagina

**CLINICAL FEATURES**

- Grayish white or yellowish membrane over tonsils, pharynx or larynx
- Marked congestion, edema or local tissue destruction
- Enlargement of lymph nodes
- Signs of toxaemia

**MANAGEMENT**

- Isolation by medical aseptic technique. Disifect any material that is contaminated
- Strict bed rest for pharyngeal and tonsillardiptheria. Do not allow them sit up even for meals, use bed baths and be fed by nurses

- During acute phase use soft food or fluid esp fruit juices. Milk is not usually easily swallowed. Spare the patient of feeding herself
- Warm throat irrigation esp before meals ease swallowing. They should not gargle
- Encourage fluid intake and keep record either orally or IV
- Nurse the patient in a warm humid room to allow easy breathing.

Control measures

**Cases and carriers**

– Early detection by cultural methods
– Isolation in the hospital for atleast 14 days or till proved free of infection

**Treatment**

– Of cases With diphtheria antitoxin IM or IV plus penicillin and erythromycin
– Of carriers with 10 day oral erythromycin

**Active immunization with diphtheria toxoid in pentavalent**

## PERTUSIS (WHOOPING COUGH)

- Is an acute infectious respiratory disease, extremely dangerous esp during infancy, which is characterised by an inflammation of the mucous membranes of the lungs, pharynx, trachea and bronchi

**Causes**

Bacteria – gram-negative aerobic bacillus called bordetella pertusis

**Source of infection**

- Case of pertusis (it only infect human) in nasopharyngeal and bronchial secretions
- Objects contaminated by the secretions

**Mode of transmission;**

- Through direct contact with soiled fomites
- Through **airborne droplets** spread from the infected person

**Period of infectivity**

– It is most infective during catarrhal stage
– A week after exposure to about 3 weeks after paroxysmal stage

**Host characteristics**

– Is primarily the disease of infancy and preschool children esp < 5yrs
– Infants below 6mo have the highest mortality
– It is more in females than males
– Recovery from whooping cough or immunization boosts immunity hence second attacks may only occur in immunocompromised persons but are usually mild

**The incubation period is 7 to 14 days**

**Pathophysiology**

- Child gets infected by inhaling droplets
- The bacteria liberates toxins that irritates surface cells and cause marked lymphocytosis
- Later, epithelial necrosis and macrophage infiltration
- There is also increased secretions. In severe cases exudates may be found in alveoli
- This leads to congestion of resp tract esp bronchi and bronchioles. Other parts involved are nasopharynx, larynx and trachea
- Obstruction of the small bronchioles by mucus plugs results in atelectasis and diminished oxygenation

**Clinical Manifestations of Whooping Cough**

Occur in three stages

1. **Catarrhal stage (1st 14 days) is characterized by;**

– Acute illness, with a slow onset of cough and fever resembling common cold
– The cough is severe at night and terminates in vomiting. It gradually assumes a paroxysmal character
– Leukocyte count in blood is high with relative lymphocytosis
– Culture of coughed material has ***B. pertusis***

2. **Paroxysmal stage: last 3 or more weeks**

– Fever and catarrhal symptoms improves
– The cough increases and occur in paroxysmal bouts i.e a short deep crowing or whooping inspiration, is followed by a quick bouts of cough which continue until it appears as if there is no more air left in the chest
- The mouth appears open with a protruding tongue
- The child becomes cyanosed and the eyes start watering
- The child has a picture of suffocation; protruded eye balls, congested face, engorged neck veins and sweating

- There is copious secretions from the nose and mouth (strings of sticky mucus hanging down the sides of the mouth)
- The child may vomit suddenly, pass urine or stool, bleed from the nose, bite the tongue or get an attack of convulsion
- Sub conjuctival hemorrhage and rupture of the membrane tympani may occur
- Paroxysmals are common at night
- When the attack is over, the appears tired and may sleep

**3. Convalescent stage;**

– The patient improves gradually, symptoms may disappear within 1 to 3 weeks
– Most death occur in children under one year

**Complications**

– Bronchopneumonia
– Massive lung collapse
– Pneomothorax, surgical emphysema and spasm of glottis
– Prolapse rectum
– Convulsions
– Right cardiac failure
– Inguinal hernia
– Sub conjuctival hemmorrhages
– Detatchment of retina

**DIAGNOSIS**

Culture of postnasal swabs

**Control measures**

- Early diagnosis of cases and contacts; for isolation and treatment, disinfection of nasal phayngeal discharges
- Antibiotics; eryrthromycin is the DOC. Others are ampicillin, tetracycline
- Keep away infants and young children from cases
- Prophylaxis; active immunization with pentavalent vaccine or pertusis vaccine or DPT
- Health education; avoid contact with infected person for atleast 4 wks after onset. Single cough produce lots of bacteria and any time a child spend with an infected person can lead to transmission

**Medical Management**

- Erythromycin 40mg/kg daily or Septrin 30mg/kg BD for two weeks. This can reduce the duration of infection if administered within the first week of the illness.
- Mild sedatives to keep the child quiet should be given, for example, chloral hydrate or Phenobarbitone (heavy sedation must be avoided).
- Phenegram in a dose of 5mg in the morning and 10mg in the evening may reduce whooping and vomiting and ensure a good nights sleep.

**Nursing Management**

- **Keep the child in warm and humid** env free of draughts, plenty of fresh air free from dust, wind and smoke, and sunshine (out doors)
- **Keep the patient quiet**, avoid un due excitement or anything that provokes crying or fright
- **Isolation**; isolate patient, teach child how to use paper handkerchief while coughing, cleans all article used by patients immediately, remove contaminated linen so as not to soil clean area
- **Diet**; give an extra meal; avoid to hot or too cold (avoid ice drinks) meals, give small frequent meals but slowly. If the child vomits, wait for approx 15 minutes before continuing. After feeding burp the child and place her in the left lateral position
- **Fever**; it is usually high when bronchopneumonia is present. Maintain adequate fluid intake
- To prevent hernia, may use tight abdominal binder, which may also control vomiting
- **Oxygen therapy** is given continuously or when the child is cyanosed.
- **Maintain a fluid balance chart and observe the child's vital signs such as the temperature, pulse and respiration. Record any findings every two to four hours until the condition improves.**
- Give health education to the parents. Continue to maintain the child's personal hygiene.

**Prevention**

- The primary form of prevention is immunisation with pentavalent vaccine.
- Good nutrition, especially breast feeding is also necessary.
- Children should be kept in well ventilated houses.
- Prevent contact between small babies and children who have pertussis whenever possible.
- Children who have household contacts should receive a course of erythromycin.

## POLIOMYELITIS

- In Greek, polio means '**gray'** and mulleas means '**marrow'**
- Is an acute highly infectious viral disease of the anterior horn cells of the **spinal cord** and sometimes of the lower part of the brain (medulla and cortex)characterized by sudden onset of fever.
- Is a complication of a viral infection which is **usually confined to the pharynx and gastro-intestinal tract, but the virus can gain entry into the nervous system and settles in the motor cells of the anterior horn of the spinal cord or in the medulla oblangata**
  - Some factors, which contribute to the invasion of the nervous system include muscular exhaustion, tooth extraction, tonsillectomy, injections and damaged nerve endings. The invasion finally leads to paralysis
- It occurs sporadically or is epidemiologically
- It is common in Africa and Asia
- It was classified as a public health problem in this country in 1950.
- It is characterised by varying degrees of paralysis.
- **The poliovirus** is of **three types** and **child has to be immunized against all these viruses to develop full immunity**

**Type one** is called **Brunhilde**; is commonly associated with paralytic illness (85%) and major epidemic.
**Type two** is called **Lansing** usually causes sporadic cases
**Type three** is known as **Leon**.

**Causative organism**; poliovirus

- It is the smallest human pathogen
- Is an RNA virus that mainly replicates in GIT
- It is resistant to freezing and drying, **can remain in feces at 4 degrees for several months**
- Can survive for many years at -20 or -70 degrees
- Temp of 50 degrees destroys virus rapidly but milk and ice cream protects the virus to up to 60 degrees but destroyed by **pasteurization at 62 degrees**
- In absence of organic matter that protect the virus, **free chlorine at 0.3 to 0.5 mg/L inactivates it**
- Its also **inactivated by formalin** and **U/V** rays
- Other commonly disinfectant used are potassium **permanganate and hypochlorides**

- **It has an incubation** period of 7 to 14 days but can extend between 3 to 35 days.

**Transmission**

- Mainly Feco-oral route then spread to lymphatic system;
- Other minor route is droplets; coughing and sneezing
- Routes of entry are **The Five F's: Fingers, Flies, Faeces, Fluids and Food**

**Host factors**

- Is common btn 6mo to 3 yrs
- Risk factors include trauma, fatigue, IM injections and operative procedures such as tonsillectomy
- Paralytic type is suspected to be determined by genetics
- Child is more susceptible from 6mo to 3 yrs

**Reservoir**

- Human is the only reservoir for both clinical and sub clinical cases
- Virus is present in the throat, feces, oropharyngeal secretions

**Communicability**

- Patients are most infectious at acute stage; a week before and a week after onset of symptoms
- In feces the virus is excreted for 2 to 3 weeks

**Pathophysiology**

- Poliovirus enters GIT by ingestion and then spread to various organs.

***Replication occurs in 3 phases***

**Phase 1; primary replication**

- Occurs in the oropharynx and intestinal mucosa and also subjacent lymphoid tissue

**Phase 2**

- Virus spread via draining lymphatic into regional lymph nodes and undergoes further replication and amplification.
- It then enters blood stream and results into in transient viraemia which manifest as mild and febrile illness

**Phase 3**

- Virus disseminate into various extraneural tissues. Extensive replication takes place producing persistent viraemia. From blood the virus enters the CNS
- The virus attacks the anterior horn cells of the spinal cord where the motor pathways are located

and may cause motor paralysis. The posterior horn is not affected
- It can also attack medullar and basal structures of the brain including the cranial nerves causing bulbar poliomyelitis

**Signs and symptoms**

**1. Prodromal stage; onset of disease**
- Resp infection; coryza, sore throat or cough
- GIT; vomiting diarrhoea or constipation
- Constitutional; fever, headache, drowsness, restlessness, irritability and sweating
- Temperature falls to normal within 34 to 48 hrs and rises again in pre paralytic stage

**2. Pre paralytic stage; start of neural phase**
- Fever that **rises to 39 degrees** with associated pain and stiffness in the back
- Moderate headache, nausea and sometimes vomiting
- Pains provoked by movement of the back, neck, limbs and abdomen
- Hyperesthesia
- Nuchal and spinal rigidity confirmed by:

***Active tests***
- Tripod sign; when the child is made to sit up unassisted, the knees flex upwards and the child places the hands on the bed behind due to spinal rigidity
- Kiss the knee test; when the child is sitting, he can only kiss the knees by flexing them

***Passive tests***
- Positive Kernig's and Brudzinski signs
- Nuchal rigidity
- Head drop sign; the head fall backwards when the shoulders are elevated
- Muscle fasciculation; flickering movements of the muscle
- Micturation disturbances; retention
- Reflexes are usually active
- CSF; clear, pressure increased, **protein normal** at first but rises in the 2nd week, sugar normal, lymphocytes may appear later
- Patient is usually alert, rapid pulse with excessive persipiration

**3. Paralytic stage**
- Develops 2nd to 5th days after onset of the signs of meningeal irritation and 1-5 days after onset of illness
- Appear when fever is still present
- It is **asymmetrical**
- Distribution of paralysis
  - Any part of the spinal cord; **either upper or lower limbs are paralysed** but <u>without sensory loss</u>
  - Trunk; abdominal muscles, back muscles, intercostal or diaphragm
  - Resp disturbances; resp centre may be affected, intercostal muscles and diaphragm are also affected

**Convalescence**
- Paralysis begin to diminish from 2 to 3 weeks. The affecetd muscles become flaccid, while contraction may produce severe deformities

## CLINICAL TYPES OF POLIOMYELITIS

**Abortive poliomyelitis**
- Characterised by influenza like symptoms pus one or more of the malaise, anorexia, nausea, vomiting, headache, sorethroat, constipation, localized abdominal pain and fever more than 103F

**Non-Paralytic Type**
- Symptoms like abortive but headache, nausea, vomiting are more intense. There is also stiffness of the posterior muscles of the neck, trunk and limbs
- **Non paralysis but can** change to a paralytic type as a result of any kind of stress, by IM injection, walking long distances and cold weather

**Paralytic Type**
- **Spinal form**
- Paralysis of flaccid type usually asymmetric and scattered in distribution, with legs affected most
- **Bulbar form**
- Involves facial, palatal and pharyngeal muscle; change in voice, difficult in swallowing, nasal regurgitation and choking
- Resp paralysis is common and causes death.

**Diagnostic Investigations**
- A lumbar puncture should be performed to exclude the possibility of meningitis.

- Cerebral Spinal Fluid is usually clear in colour. Both lymphocytes and polymorphs may be present in the CSF. High WBC, Normal sugar
- Isolation of the virus from nasopharyngeal swabs in the first 5days of illness, and from stools and rectal stools up to 5weeks afteronset
- Serology for the risen antibodies

**Treatment**

**No special treatment is available.**

- Symptomatic management is encouraged
- Mild analgesics and sedatives for pain and sleep induction
- Laxatives for constipation
- Sulfonamides to prevent resp and oral complications
- Immunization with live vaccine (OPV) eradicates wild polio by displacing it from intestines thereby interrupting transmission
- Passive immunization with human normal immunoglobulin

**Management of Poliomyelitis**

**Acute stage**

- The patient is strictly confined to bed : activities in the first two weeks of the infection risk possible increased paralysis
- The patient is to be nursed in isolation
- Pain is controlled through the administration of analgesics, for example, paracetamol, valium or phenobarbitone
- Regular respiratory suction and postural drainage should be performed
- N.G tube feeding should be high calorie and include substantial amounts of protein
- Change the patient's position every four hours to prevent bedsores
- Surgical procedures should also be avoided
- No injections are to be administered during this acute stage as they may precipitate paralysis
- Immobilise the affected limbs during the acute stage of the illness, using splints to prevent flexion deformities and promote rest.

**After the acute stage has passed, (6wks from onset)**

- Begin gradual and gentle exercise of the affected limbs. Recovery may take 6mo but may extent to 2yrs
- Ensure proper disposal of faeces and urine to prevent spread of infection.
- Urinary catheterisation must be passed but principles of asepsis must be observed strictly.
- Maintain an intravenous infusion and fluid balance chart.
- Oxygen therapy may be used when necessary. A tracheotomy and use of a mechanical respirator may be used should the patient's condition deteriorate.
- Hydrotherapy (in swimming pool), the limbs are bit lighter in water and the patient gets psychological uplift

**Discharge**

- After being discharged, the child should return to the clinic at regular intervals to ensure flexion deformities do not occur.
- A plaster of Paris or back slab should be applied to the limbs if these deformities actually occur.
- Special shoes and callipers may help severely affected children.
- Prevention and control
- Protection of the susceptible by immunization is most effective method. It breaks the chain of transmission. Immunize all the children in infancy before 6months

– Report the case to the authorities
– Isolate the patient for 1 wk for the onset of the disease or as long as there is fever
– Ensure proper disposal of urine and feces of the patient
– All sources of water must be protected, swimming baths must be chlorinated
– Milk must be pasteurized and fruits should be washed with weak potassium permanganate solution before using
– Avoid overcrowding of children in schools, cinemas, playgrounds etc
– Children must avoid excessive physical straining during epidemic
– Search for sick persons, investigate contacts and source of infection
– Antifly measures should be adopted
– Active immunization **with OPV** during outbreak is important

– All suspected carriers must gargle with potassium permanganate solution and take four small spoons of sulfadiazine in a day
– Educate the community about the disease and its consequences

## METHODS OF IMMUNIZATION

**Active immunization**

• Done using inactivated polio vaccine(IPV) and OPV

**1. IPV (Salk type)**

– Is a liquid **killed** vaccine (by formalin), contains all three types of polioviruses, given as IM
– It only stimulate the production of only systemic (humoral) antibodies **but not intestinal or local immunity** so the wild polio can still multiply in the intestines hence become a source of infection
– It is **not recommended during epidemics** because immunity is not developed with one dose. Besides its injection may provoke paralysis
– It does not provide life immunity, booster doses are required once in 5 yrs
– However, **its safe for immunocompromised** persons and stable

**2. Oral polio vaccine (sabin vaccine)**

– It's **a live liquid** vaccine that contain all three types of polioviruses
– It's **the drug of choice** for immunization against polio
– It is given as oral two drops per dose
– Polio **zero given at birth results in development of gut immunity only** not systemic immunity, subsequent three doses helps develops both local gut and systemic immunity
– It stimulate production of Ig A in the gut and Ig G in the circulation
– It can help develop **herd immunity** and can **help eradicate poliomyelitis** i.e

• When immunized child drinks contaminated water, the virus is inactivated by Ig A, which when it enters the mouth of a susceptible child it induces immunity indirectly
• If all children are immunized simultaneously and no single child is left, the virus replaces the wild polio virus

– It is the **vaccine of choice in epidemics**
– Its adm is simple and easy
– However, since it's a live vaccine, its multiplication in the intestines may result in a resistant strain that **may cause vaccine associated paralytic poliomyelitis**
– Do not the vaccine in acute febrile illness, diarrheal disease, steroid therapy etc

**WHY IT IS POSSIBLE TO ERADICATE POLIOMYELITIS**

• Human beings are the only reservoir of infection
• There is no chronic carrier state
• Half life of wild polio virus in feces is short (hardly 48hrs)
• The available vaccine is a live vaccine, stabilized, highly potent, cheap and easily adm and safe
• An OPV can develop herd immunity indirectly
• OPV replaces wild polio viruses from nature by inducing gut and systemic immunity
• Correct and complete dosage schedule confers life long immunity

**Poliomyelitis resembles GBS, transverse myelitis and traumatic neuritis (differentials)**

However

• **In Gullain-Barre' syndrome**
– There is sensory deficit
– Has a chronic onset
– Paralysis is symmetrical
– Fever, headache, nausea, vomiting are absent
– CSF; high protein and low cell count
• **In transverse myelitis**
– Absence of fever
– Symmetrical paralysis lower limbs usually paraplegia ass. with loss of rectal and bladder sphincters
– Marked sensory loss
– CSF is normal
– Common in children above 4yrs
• **In traumatic neuritis**
– There is history of IM injection
– Paralysis of limb is accompanied by pain
– Knee jerk is present, ankle jerk is absent
– Child has foot drop

## MEASLES

- This is an acute highly infectious disease characterized by fever and catarrhal symptoms of upper respiratory(nasal and respiratory membrane) then followed by a rash and Koplik spot
  - Is an endemic disease but epidemics can occur
  - Any body who has not had the disease or not been immunized

**Caused by an RNA paramyxovirus called measles virus**

- The viruses cannot survive outside human body

**Communicability**

- It is highly infectious during prodromal period and at the time of eruption
- Secondary attack rate is 80% among susceptible HH contacts
- From 4 days before rash to 4 days after rash

**Host characteristics**

- Affects infancy or children btn 6mo to 3 yrs in developing countries and > 5yrs in developed countries
- Both sexes affected equally, no age is immune
- One attack confers a life long immunity
- Its usually severe in malnourished and in young children
- Children with vitamin A deficiency places a child at high risk

**Source of infection**

- Case of measles, no carriers involved
- Secretions from the nose, throat and resp tract during prodromal period and early stages of the rash

**Transmission**

- By droplet or direct contact with secretions from the nose and throat of infected persons from 4 days before onset of rash to 5days thereafter
- Port of entry is resp tract

**Incubation period,**

- Period of seven to 14 days to appearance of rash but usually 10 to 12 days is sufficient

**Pathophysiology**

- It causes hyperplasia of the lymphoid tissue of the tonsils, adenoids, spleen, appendix and lymphoid tissue during prodromal phase
  - Its characterized by the presence of a multinuclear giant cell observed in lymphoid cells and inflamed pharyngeal and bronchial mucosa
- There is and slight edema and hyperemia with perivascular infiltration of lymphocytes in the skin where the rash appears
- It also involves mucous membrane of the eyes, nasopharynx, bronchi and the lungs
- These changes disappear in 10 days
- The commences in the superficial vessels of the cranium where it causes serious exudation, proliferation of endothelial cells and then vascularization and necrosis of endothelial cells and finally desquamation
- The skin pills off in small flakes
- There is also peribronchial inflammatory reaction. Secondary infection may occur in lungs
- In the brain and spinal cord, edema congestion and petechial hemorrhages occur (encephalomyelopathy)

**Clinical Manifestations**

**Prodromal phase: begin 10 days after infection and may last three to seven days**

- Charcterized by fever, coryza with sneezing and nasal discharge
- Cough, redness of the eyes, lacrimation and pften photophobia
- There may be vomiting or diarrhoea
- Koplik spots appear on buccal mucosa , opposite the first and the second upper molars, 24 – 48 hours before the main rash. They are small, bluish white spots on a red base

**Eruptive phase:**

- The maculopapular or macular rash starts behind the ears and spread rapidly over few hours on the forehead, face and neck. It spreads downwards to the body
- It takes 2 to 3 days to reach the feet, at which point it starts to fade.
- The rash always bocomes confluent and blotchy
- Fever is high and lasts for 3 to 5 days

**Post measles phase**

- The child will have lost weight and will remain weak for few days.

- There may be growth retardation, diarrhoea, pyogenic infections, candidiasis and reactivation of pulmonary TB
- **Complications**
- Otitis media
- BronchoPneumonia - This is usually a viral pneumonitis
- Reactivation of Pulmonary TB
- Kerato - conjunctivitis
- Post measles Encephalitis is a serious complication often fatal or with residual brain damage
- Gastroenteritis
- Oral thrush and/or oral herpes
- Cervical adenitis

**Nursing Care Management**

- Uncomplicated cases can be nursed at home but complicated cases, infants and malnourished children should be treated in a hospital with isolation facilities
- There is no specific treatment available other than supportive which includes:
- **Isolation in a private room**
- **Nutrition, During febrile stage**; penty of nourishing liquids and soft bland diet e.g soup, milk, juices, ice cream **Convalescence;** general diet
- **Care of the eyes;** Avoid direct light(may use dark glasses or eye shade ) In case of discharge, apply boric ointment or liquid petroleum
- **Care of the ears;** Report signs of mastoiditis, un explained mastoid swelling, redness or tenderness, sudden rise in temperature, ear discharge
- If there is discharge; cut the hair above the ear so that it will not be in contact with the discharge, wipe the discharge with sterile cotton
- Never plug a discharging ear, ear wicks may be used to absorb discharge
- **Care of the nose**, use cotton swabs soaked in liquid petroleum or saline solution or hydrogen peroxide to clean the nose
- **Care of the mouth and throat**, Good oral hygiene with mouth washes or gargles before and after meals
- Brush teeth at least twice daily

Apply glycerin or lemon miture on the lips and tongue

- Encourage bowel elimination. May use enema such as milk of magnesia
- Antipyretics to control temperature
- **If there is itching**; sodium bicarbonate or magnesium sulphate may be used
- Vitamin A 200,000 units orally daily for two days

**Convalescence**

- Uncomplicated measles recovers quickly
- The patient should be protected from cold, draughts and un due fatique for several weeks to prevent developing complications

**Prevention**

- Active immunisation with attenuated live virus vaccine esp after 9 months, 0.5ml SC, it must be kept cold at 4 to 8 degrees
- Immunity develops 11 to 12 days after vaccination
- The infection can be aborted if vaccine is given within 12 hours of exposure

## RUBELLA

**Infectious Agent** Rubella virus.

**Clinical Presentation**

- Rubella is most often a mild viral illness associated with rash in approximately half of infections. Asymptomatic infections occur.
- Generally, a prodrome occurs 1-5 days before the onset of rash which may be characterized by low-grade fever, headache, mild coryza, malaise and conjunctivitis. Children usually have few or no symptoms. Lymphadenopathy, involving occipital, post auricular and posterior cervical nodes, may start 5-10 days prior to the rash onset and is the most characteristic sign. Transient arthralgia and, less frequently, arthritis may occur in up to one-third of women and usually begins at the same time as the rash or shortly afterwards.
- The rash is diffuse, punctate (red pinpoint), and maculopapular. It is often itchy and begins on the face.
- The usual duration is 3-5 days. Clinically, Rubella is indistinguishable from febrile rash illnesses caused by measles, parvovirus B19, human herpes virus 6 (HHV6), Coxsackie virus, ECHO virus, adenovirus and dengue virus, and laboratory confirmation is required for diagnosis unless there is an epidemiological link to a confirmed case.
- Rare complications include chronic arthritis and encephalitis in adults, and thrombocytopenia in children.
- Rubella is important because of its ability to produce anomalies in the developing fetus if the

infection is acquired in the first trimester of pregnancy (refer to Section 7.9.1 Congenital Rubella protocol).

**Diagnostics** The clinical diagnosis of Rubella is often difficult as Rubella has many symptoms in common with other rash illnesses.

- IgM antibodies develop about 5 days post rash onset. IgM antibodies can persist for 6 weeks after the rash onset. If the IgM is negative and the sample has been taken on or before the fifth day post rash onset, another specimen should be taken as soon as possible (more than 5 days post rash onset).
- Seroconversion or a significant rise in rubella IgG between acute an convalescent sera collected at least 10 days apart will assist with the diagnosis of infection.
- Isolation of rubella virus from the nose, throat, cerebral spinal fluid, urine or blood can be used to confirm the diagnosis. Ideal specimens are respiratory (nasopharyngeal or throat swabs). Specimens should be obtained as soon as possible after onset (less than 4-5 days post onset).
- Follow your regional laboratory procedures for testing.
- **Treatment** Supportive treatment as indicated. No specific treatment is available for
- Rubella.

## CHICKENPOX (VARICELLA)

This is a **mild** viral infection, which is **extremely** contagious.

Its characterized by Fever, malaise and a typical pruritic skin lesions.

**The causative organism is;** Varicella Zoster Virus (VZV) a member of Herpesvirus group

**Source of virus**; oropharynx, skin, mucous membrane and blood

**Transmission.**

- Airborne droplet infection, from the respiratory tract
- Direct or indirect contact, the wind can transmit the virus particles from the skin of the infected person over a distance of meters to another person.
- Dry scabs and nut infections.

- Once infected, the disease leaves immunity against chicken pox but the virus remains within the body and may reappear later in adult life as the herpes zoster when the person's immunity is weakened, for example in AIDS, diabetes, leukaemia and old age.

**Incubation Period**

- It has an incubation period of 14 to 16 days but may vary from 7 to 21 days.

**Infectivity period**

- The period of infectivity is about 1 to 2 before the rash and 4 to5 days after the appearance of the rash.

**Host characteristics**

- Common in under 10 children
- One attack gives durable immmunity, second attack is rare
- Overcrowding favors transmission
- Reservoir; human beings

**Pathophysiology**

- Virus gain entry thro mucosa of upper resp tract
- Causes viraemia and circulation through blood and then becomes localized to the skin
- The virus then produces swelling of the epithelium cells and accumulation of fluid producing vesicle**Clinical Manifestation**
- Begins with mild headache, backache, moderate fever and malaise in the first 24hrs then followed by a rash and itching itching
- Later, lesion appear in **oropharynx which ruptures to form an ulcer**, that causes painful swallowing
- **Maculopaular** Cutaneous rash appear over 1 to 5 days, **first appearing in the back, chest, or on the forehead or face, becomes numerous on the trunk and face and relatively sparse over the extremities**
- On the extremities , the flexor muscles appear more affected than in extensor (distribution to palms and soles is seldom)
- The skin lesions develop as small, deep pink, slightly raised, ovoid papules, which within few hours become fragile, thin walled translucent, umbilicated, glistening bleb like **vesicle**s containing clear fluid surrounded by small red areolar **in the super layer** of the skin
- The **vesicles later change to crusts and finally slough out in 7 to 14 days**

***SUMMARY MACULOPAPULAR FOR FEW HRS, VESICULAR FOR 3 TO 4 DAYS AND LEAVES A GRANULAR SCAB***

***Note***: Once infected, the disease leaves immunity against chicken pox but the virus remains within the body and may reappear later in adult life as the herpes zoster when the person's immunity is weakened

**Nursing Care**

- Isolate the patient in a warm well ventilated room.
- Confine the child to bed until the pyrexia settles down.
- Monitor the vital signs at regular intervals. Temperature is usually mild
- The child requires plenty of fluids and a nourishing diet.

**Personal hygiene**

- Daily warm cleansing bath using a soft towel
- Good oral hygiene twice daily; use antiseptic mouth washes or gargles in case of lesions
- The nose should cleaned by cotton soaked antiseptic solution then dried with sterile cotton
- Clean the eyes with moist sterile cotton
- Clean the ear in case of purulent otitis media
- The fingernails should be kept short and the child has to be restrained from scratching.
- Soothing lotions such as Calamine, should be applied to the skin to soothe itching, lesions in the vagina can be relieved by douching with sodium bicarbonate
- Encourage elimination
- Bowel elimination at least once a day
- Catheterization may be necessary if lesions appear in or near urinary meatus
- Antibiotics are given prophylactically..

**Convalescence** ; it is usually rapid and uneventful

**Terminal disinfection**

- When the patient can no longer tolerate the disease,
- He should be given cleansing bath and shampoo,
- He should be dressed in clean clothes and placed in clean area
- All equipment that he has come incontac with should be cleaned up or burnt
- The matresses/ pillows should be thoroughly aired on sunshine for atleast 6hrs
- Bed, chairs, table and walls should be washed

**Complications**

- Secondary infections of skin lesions.
- Pneumonia or encephalitis may also occur but are rare.
- Other possible complications may be thrombocytopenia, arthritis and nephritis.
- Otitis media, myocarditis

**Prevention and Control**

**Prevention**

- Active immunization with live attenuated varicella vaccination in pts with good immunity
- Protect high risk group who can not be immunized by immuinizing the house holds or close contacts
- Passive immunization with varizella zoster immunoglobulin within 72 hrs of exposure 0.4 to 1.2 ml/kg body weight

**Control of patients, contacts and immediate environment**

- Isolation of the patient from school till vesicles dry
- Report to the local authority
- Disinfection of oronasal discharge and soiled articles
- Protection of contacts with varizella zoster immunoglobulin within 72 hrs of exposure up to 5 days of exposure
- Specicif treatment with acyclovir (DOC), others are vidarabine
- Terminal disinfection of the room

**In case of an epidemic**

- Isolate those affected
- Immunize the contacts
- If not eligible for immunization such as tendency to be pregnant, give varicella zoster IG

**Differentiate btn chicken pox and small pox??**

## MUMPS (INFECTIVE OR EPIDEMIC PAROTITIS)

- Is acute febrile contagious, generalized infectious disease characterized by non suppurative swelling and tenderness of salivary glands majorly the parotid glands and less commonly sublingual and submaxillary
- Caused by a virus called **Myxovirus parotitis**, which is a member of family **paramyxoviridae**
- Mumps does not kill but patient may die because of complications

– Its largely endemic

**Host characteristics**

– Occurs in children btn 5 to 15 yrs,
– it is more severe in adults than children
– One attack of clinical or sub clinical induce life long immunity
– Most infants below 6 months are immune coz of maternal antibodies
– Most infections in children under 2 yrs are subclinical

**Source of infection**

– Human are the only reservoir i.e those suffering from mumps or those with subclinical mumps

**Methods of spread**

– It can spread by droplets or contact with the salivary secretions of the infected person, airborne, formites
– The virus can be found in blood, urine, human milk, CSF, saliva

**Incubation Period**

– On average between 14 and 21 days of infectivity after the onset of the parotid glands swelling

**Communicability**

– Is 4 to 6 days after onset before parotitis and a week or more (9 days) after the onset of parotitis

• Pathophysiology
• The infection causes desquamation of epithelium in the ducts of parotid gland, interstitial edema and Lymphocytic infiltration within the lumina
• The gland enlarges, displacing the ear lobe outwards and upwards
• The enlargement is painful and tender but begins to subside in 7 to 10 days where temperature subsides
• Unilateral parotitis is the most common form
• It may affects other organs esp in adults e.g testes, pancrease, CNS, ovaries, prostate, breast, joints, eyes, ears

**Clinical Manifestations**

**The salivary glands namely the parotid, sublingual and submaxillary glands may be infected by painful swelling, this may be one side or both sides**

• Swelling of parotid gland is the first indication; the patients complains of pain and stiffness on opening the mouth before the swelling
• There may be also sore throat, fever, ear ache and painful chewing before the onset
• Fever subsides after 48hrs of onset
• Complains of headache and malaise may be present
• The tongue is furred and mouth dry due to diminished saliva
• Moderate lymphocytosis is noted on blood examination
• The tenderness may last two to three days then gradually subside

**Nursing Care**

• Isolate during period of communicability
• Maintain bed rest in a warm room until swelling subsides esp in acute phase
– An adult should remain in bed until swelling has subsided for atleast 3 days because ambulation increases the incidence of orchitis in males
• Give analgesics and antipyretics as required to control pain and temperature
• Encourage fluids and soft bland foods that do not require chewing
• Avoid foods which contain acid because they may increase pain
• Apply heat or cold compress to neck whichever is more comfortable
• Observe the child's vital signs of temperature, pulse and respiration and record them every four hours
• Cleanliness; daily cleansing bath and brushing because the secretions are usually dirty
• Encourage bowel movement at least daily

**Prevention and control**

**Prevention**

– Health education to encourage mumps immunization
– Active immunity of a live attenuated vaccine is available for those who are not already infected. The mumps virus vaccine is best given before puberty (btn 12 to 18 mo) as single dose

**Control of patient, contact and immediate environment**

– Report to local authority
– Isolation measures for 9 days from onset of parotitis, exclusion from school, work
– Concurrent disinfection of articles soiled with nose and throat secretions
– Immunization of contacts
– Investigation of contacts and source of infection

**Complications**

- The child may develop sensory neural hearing loss
- Inflammations of genital organs i.e ovaries - oophoritis and testes - orchitis, in both cases this may result in sterility in adulthood
- Meningoencephalitis (inflammation of the meninges and brain)
- Pancreatitis, which is inflammation of the pancreas
- Spontaneous abortion if it occurs in first trimester

**Orchitis**

- Is a common complication in adults developing a week after onset
- It may be accompanied by a rise in temp
- It may cause testicular atrophy and cancer
- It is usually unilateral
- In all males past puberty with mumps, the scrotum must be supported by suspensory from the start. An adhesive tape bridge across the thighs and under the scrotum is used

**Oophoritis**

- Its onset is characterized by nausea and vomiting, fever, headache and low abdominal pain

## TUBERCULOSIS

- Pulmonary Tuberculosis is an infectious disease of the parenchyma of the lungs caused by ***Mycobacterium tuberclosis***
- ***Agent*** *; Mycobacterium tuberclosis*
  - *Is a slender, straight or slightly curved bacillus*
  - *They are acid fast, non sporing, non capsulated non motile that stain blue with Ziehl Neelsen stain*

***Source of infection***

- Human; The pulmonary tuberculosis which affects the lungs accounts for 90%
- Bovine type acquired through the drinking of infected animal milk. Accounts for 10%
- *Infective material is sputum of patient suffering from TB bacilli, pus, pleura and peritoneal fluid*

***Host factors***

- *More in male than females, common in males who are >40 yrs*
- Children under the age of two years are more susceptible than older ones
- *There is no inherited immunity for TB but is acquired through natural infection and BCG vaccine. This immunity can break in case of severe infection*
- *There is increased tendency to develop disease during puberty and adolescent due to increased contacts*
- *Poor nutrition increases risk*
- *Infectious diseases such as HIV, measles and pertusis may reactivate TB*

**Environmental factors; common in**

- Overecrowded, insanitary and sub standard houses
- Poverty/ low income families and low level of education and ignorance
- Large families, malnutrition and occupation (doctors, nurses)

**Pathophysiology**

- Source of infection in children is usually a member of house hold or a frequent family visitor
- The child inhales micro droplets after someone coughs or sneezes
- The droplets passes into bronchial tree and implants in a bronchiole or alveolus, and the tubercle starts to multiply
- Epithelial cells surrounds and encapsulate the multiplying bacilli in attempt to wall off the organism forming the typical tubercle
- During inflammation, some bacilli leave the focal area and are carried to the regional lymph nodes, causing fever. At this stage tuberculin is positive
- Extension of the primary lesion at the original site causes progressive tissue destruction as it spreads within lungs, discharges material from foci to other areas of the lungs (bronchi or pleura) or produces pneumonia
- Erosion of blood vessels may causes wide spread of tubercle bacillus to near and distant sites (miliary TB). Organisms deposited in the upper lung zones, bones, kidneys and brain may grow but those in bone marrow, liver and spleen are inhibited
- Extra pulmonary TB may be manisted as superior lymphaadenitis, meningitis, osteoarthritis and may appear in the middle ear and mastoid and on the skin
- Children with good immunity may remain aymptomatic and lesions usually heal. TB infection is manifestd only by positive skin tes
- Active disease is manifested by positive skin test, postive chest x ray, positive sputim culture and signs of disease

**Clinical Features**

- General malaise
- Prolonged fever
- Anorexia
- Weight loss and local signs of infection
- Prolonged cough, aching pain, tightness in the chest and rarely hemoptysis
- Examination of the affected lung; affected side does not expand as well as the other, diminished breath sounds, crackles are present, dullness on percusion
- Fever in infants persist, the child develops pallor, anemia, weakness and weight loss

***Consider TB in any child with:***

**A history of:**

– Unexplained weight loss or failure to grow normally
– Unexplained fever, especially when it continues for longer than 2 weeks
– Chronic cough (i.e. cough for > 14 days, with or without a wheeze)
– Exposure to an adult with probable or defi nite infectious pulmonary TB.

**On examination:**

– Fl uid on one side of the chest (reduced air entry, stony dullness to percussion)
– Enlarged non-tender lymph nodes or a lymph node abscess, especially in the neck
– Signs of meningitis, especially when these develop over several days and the spinal fl uid contains mostly lymphocytes and elevated protein
– Abdominal swelling, with or without palpable lumps
– Progressive swelling or deformity in the bone or a joint, including the spine

**Diagnosis**

**History and physical signs**

- Try to obtain specimens for **microscopic examination** of acid-fast bacilli (**Ziehl-Neelsen stain**) and for **culture of tubercle bacilli**.

– Possible specimens include three consecutive early-morning, fasting gastric aspirates, CSF (if clinically indicated) and pleural fl uid and ascites fl uid (if present). A positive result confi rms TB, but a negative result does not exclude the disease

- **Tuberculin test/Mantoux test**; 2 to 3 weeks after primary infection, given intradermally. The test is usually positive in children with pulmonary TB (reactions of > 10 mm suggest TB; < 10 mm in a child previously vaccinated with BCG is equivocal).

– The purifi ed protein derivative test may be negative in children with TB who have HIV/AIDS, miliary disease, severe malnutrition or recent measles

- **Obtain a chest X-ray**. A diagnosis of TB is supported when a chest X-ray shows a miliary pattern of infi ltrates or a persistent area of infi ltrate or consolidation, often with pleural effusion, or a primary complexIn some patients, pleural effusion may be present also to include lymphatic nodes enlargement
- **Xpert MTB/RIF should** be used as the initial diagnostic test in children suspected of having multidrug-resistant TB (MDR-TB) or HIV-associated TB.
- **Routine HIV testing should be offered** to all children suspected of TB.

**Treatment**

- **Isoniazid (H): 10 mg/kg** (range, 10–15 mg/kg); maximum dose, 300 mg/day
- **Rifampicin (R): 15 mg/kg** (range, 10–20 mg/kg); maximum dose, 600 mg/kg per day
- **Pyrazinamide (Z): 35 mg/kg** (range, 30–40 mg/kg)
- **Ethambutol (E): 20 mg/kg** (range, 15–25 mg/kg).
- Follow national guidelines or WHO for regimen

**WHO**

- ***Four-drug regimen:*** HRZE for 2 months, followed by a two-drug (HR) regimen for 4 months OR
- *Three-drug regimen:* HRZ for 2 months, followed by a two-drug (HR) regimen for 4 months
- In cases of suspected or confirmed tuberculous meningitis, spinal TB with neurological signs or osteo-articular TB, treat for 12 months with a four drug regimen (HRZE) for 2 months, followed by a two-drug (HR) regimen for 10 months;
- **Precautions:** Streptomycin should not be used as part of fi rst-line treatment regimens for children with pulmonary TB or tuberculous peripheral lymphadenitis but reserved for treatment of multidrug-resistant TB

TB treatment

**Monitoring**

- Confirm that the medication is being taken as instructed, by direct observation of each dose.
- Monitor the child's weight gain daily and temperature twice a day in order to check for resolution of fever

**Public health issues**

- Notify the case to the relevant authority
- Children < 5 years of age who are household or close contacts of people with TB and who, after an appropriate clinical evaluation, are found not to have active TB should be given 6 months of isoniazid preventive therapy (10 mg/ kg/day, range 7–15 mg/kg, maximum dose 300 mg/day)

**Nursing Management**

- If admitted, the mother should be encouraged to stay and help with the child's care.
- Nursing care should aim for infection control, bed rest and high protein diet with oral fluid intake.
- The patient's personal body hygiene should be maintained at all times.
- Usually the child will be prescribed daily anti-tuberculous drugs, which you should administer continuously for a minimum of six months with at least two drugs. The drugs regimen can be selected from the following:
- The dose will be determined according to the child's age and weight. The drugs have to be administered daily in combination to prevent bacterial drug resistance to one particular drug.
- The drugs are very toxic and so the child should be observed very closely

**Preventive Measures**

- All children have received their BCG vaccination.
- Health education to members of the community so that the parents may bring their children to the clinic when infection is suspected and also avoid conditions that favour the disease
- Maintain good health and nutrition
- Pasteurization of milk and routine testing of milk
- Isolation of suspected and confirmed cases

**THE AIM OF TB TREATMENT**

## MAIN AIMS OF TB TREATMENT:

1. Cure patients, prevent suffering and death from tb
2. Prevent long-term complications or sequelae of tb
3. Prevent relapse of the tb disease
4. Prevent transmission of the tb infection
5. Prevent the development of drug resistant tb

## BASIC PRINCIPLES OF TB TREATMENT:

1. Never use single drugs
2. Always use drugs in combinations –using fixed dose combinations (fdcs)
3. Drug dosage is based on weight
4. Drug intake should be directly observed
5. Ensure the entire treatment is taken as recommended

**Before initiating treatment**

***Classify patients according to the following classifications;***

1. HIV status
2. Anatomical site
3. History of previous treatment
4. Drug resistance pattern
5. The regimen, duration of treatment, follow up investigations and adjunct medications and investigations is based on the above

## CLASSIFICATION BASED ON ANATOMICAL SITES

**Pulmonary TB(PTB)**- Any bacteriologically confirmed or clinically diagnosed case of TB involving the lung parenchyma or the tracheobronchial tree. This exclude pleural effusion

**Extra pulmonary TB (EPTB**)- ny bacteriologically confirmed or clinically diagnosed case of TB involving organs other than the lung parenchyma, e.g. pleura, lymph nodes, abdomen, genitourinary tract, skin, joints and bones, meninges.

**Classification based on history of previous TB treatment (patient registration group)**

**New patients-** Patient who has never been treated for TB or has taken anti-TB drugs for less than 1 month

**Previously treated patients**- Patient who has received 1 month or more of anti-TB drugs in the past. They are further classified by the outcome of their most recent course of treatment as follows:

a. **Relapse patients;** previously treated for TB, declared cured or treatment completed at the end

of their most recent course of treatment, and are now diagnosed with a recurrent episode of TB (either a true relapse or a new episode of TB caused by reinfection)

b. **Treatment after failure patients;** previously treated for TB and whose treatment failed at the end of their most recent course of treatment
c. **Treatment after loss to follow-up patients;** previously treated for TB, and declared lost to follow-up at the end of their most recent course of treatment. (These were previously known as return after default patients.)

***Patients with unknown previous TB treatment history do not fit into any of the categories listed above***

## CLASSIFICATION BASED ON HIV STATUS

**HIV-positive TB patient**- Any case of TB who has a positive result from HIV testing conducted at the time of TB diagnosis or other documented evidence of enrolment in HIV care, such as enrolment in the pre-ART register or in the ART register once ART has been started

**HIV-negative TB patient-** Any case of TB who has a negative result from HIV testing conducted at the time of TB diagnosis. Any HIV-negative TB patient subsequently found to be HIV-positive should be reclassified accordingly

**HIV status unknown TB patient-** Any case of TB who has no result of HIV testing and no other documented evidence of enrolment in HIV care. If the patient's HIV status is subsequently determined, he or she should be reclassified accordingly

## CLASSIFICATION BASED ON DRUG RESISTANCE, BASED ON DRUG SUSCEPTIBILITY TESTING

**Monoresistance**- Resistance to one first-line anti-TB drug only

**Multidrug resistance-** Resistance to at least both isoniazid and rifampicin

**Extensive drug resistance-** Resistance to any fluoroquinolone and to at least one of three second-line injectable drugs (capreomycin, kanamycin and amikacin), in addition to multidrug resistance

**Rifampicin resistance-** Resistance to rifampicin detected using phenotypic or genotypic methods, with or without resistance to other anti-TB drugs. It includes any resistance to rifampicin, whether monoresistance, multidrug resistance, polydrug resistance or extensive drug resistance

## FIRST LINE ANTI TB DRUGS

- These are drugs that are prescribed for the treatment of drug sensitive TB
- 1st line treatment is given to all drug sensitive new patients, relapse, previous failures, and return after default depending on DST results
- The first line drugs include;

1. Rifampicin
2. Isoniazid
3. Pyrazinamide
4. Ethambutol

## PROPERTIES OF INDIVIDUAL ANTI TB DRUGS

| Drug | Mechanism of action | Target bacilli | Media | Compartment it works in |
|---|---|---|---|---|
| Isoniazid(H) | Bactericidal after 24 hours. High potency: kills >90% bacilli in first few days of treatment. | Rapid and intermediate growing bacilli | Alkaline and acid media. | Intracellular and extracellular |
| Rifampicin (R) | Bactericidal within 1 hour. High potency. Most effective sterilizing agent. | All populations including dormant bacilli. | Alkaline and acid media. | Intracellular and extracellular |
| Pyrazinamide (Z) | Bactericidal with a low potency. Achieves its sterilizing action within 2-3 months. | Slow growing bacilli | Acid medium. | Intracellular bacilli only ( macrophages) |
| Ethambutol (E) | Bacteriostatic. Low potency. Minimises the emergence of drug resistance. | All bacterial populations. | Alkaline and acid media. | Intracellular and extracellular |

**Advantages of using FDCs:**

1. Reduced risk of resistance developing to the drugs in the event of missed doses
2. Reduction of pill burden
3. Fewer medication errors

4. Fewer prescription errors

**Disadvantages of FDCs include:**

1. Reduced bioavailability of some drugs
2. Flexibility in obtaining an optimal dose of some agents

**Anti TB regimen for both adult and children**

| | Intensive phase | Continuation phase |
|---|---|---|
| All forms of TB except TB Meningitis and osteo-articular TB | 2 RHZE | 4 RH |
| TB Meningitis and osteo-articular TB | 2 RHZE | 10 RH |

Directly Observed Therapy (DOT) should be provided using a treatment supporter who is acceptable and accountable to the patient and to the health system

**Adult dosage of anti-TB drugs according to body weight**

| Drug | Recommended dose in mg/kg | Range in mg/kg | Maximum dose |
|---|---|---|---|
| Isoniazid | 5 | 5-10 | 300mg |
| Rifampicin | 10 | 10-15 | 600mg |
| Pyrazinamide | 35 | 30-40 | 1.5g |
| Ethambutol | 20 | 15-25 | 1.6g |

**FDC treatment dosage for adults**

| FDC Dosages | Formulation | 30-39kg | 40-54 kg | Over 55 kg |
|---|---|---|---|---|
| Rifampicin 150 mg + Isoniazid 75 mg + Pyrazinamide 400 mg + Ethambutol 275 mg | 4-FDC tablet RHZE | 2 | 3 | 4 |
| Rifampicin 150 mg + Isoniazid 75mg | 2-FDC tablet RH | 2 | 3 | 4 |

**Dosage of pyridoxine**

| Weight (kg) | Dose of pyridoxine (available in both 25mg and 50mg tablets) |
|---|---|
| 1-13.9 kg | 12.5mg |
| 14-25 kg | 25mg |
| >25 kg | 50mg |

**POINTS TO NOTE**

- Monthly monitoring of weight should be done and recorded in the patients tb record card and doses adjusted accordingly
- No trial of therapy should be done to minimize emergence of drug resistance
- Children above 30kg should be treated as adults
- All patients taking anti-tbs should also receive daily pyridoxine to reduce the risk of developing peripheral neuropathy
- However, lack of pyridoxine should hinder tb therapy

**ADDITIONAL MANAGEMENT DECISIONS**

**HOSPITALIZATION**

1. Severe forms of ptb and eptb severe malnutrition for nutritional rehabilitation
2. Signs of severe pneumonia (i.e. Chest in-drawing)
3. Other co-morbidities e.g. Severe anemia
4. Social or logistic reasons to ensure adherence
5. Severe adverse reactions such as hepatotoxicity

**steroid therapy should be offered in the following cases;**

1. Tb meningitis
2. Ptb with respiratory distress
3. Ptb with airway obstruction by hilar lymph nodes
4. Miliary tb
5. Pericardial effusion

| *Dosage of prednisone for adults and children* | | | |
|---|---|---|---|
| Dosage | Week 1-4 | Week5-6 | Week7 |
| Adult and Children>30kg | 1mg/kg (max 60mg) | 0.5mg/kg | 0.25mg/kg |
| Children < 30kg | 1-2mg/kg (max 60mg) | 0.5-1mg/kg | 0.25-0.5 mg/kg |

**TREATMENT OUTCOME DEFINITIONS**

- The new treatment outcome definitions make a clear distinction between two types of patients:

**A.** Patients treated for drug-susceptible tb

**B.** Patients treated for drug-resistant tb using second-line treatment

- Defined as combination chemotherapy for drug-resistant tuberculosis which includes drugs other than those in group 1

- The two groups are mutually exclusive
- Patients found to have drug-resistant tb and placed on secondline treatment are removed from the drug-susceptible tb outcome cohort
- Management of the standard tb register and of the second-line tb treatment register needs to be coordinated to ensure proper accounting of the outcomes of treatment

## TREATMENT OUTCOMES FOR DRUG SENSITIVETB PATIENTS

**CURED-** A Pulmonary Tb Patient With Bacteriologically Confirmed Tb At The Beginning Of Treatment Who Was Smear Or Culture Negative In The Last Month Of Treatment And On At Least One Previous Occasion

**TREATMENT COMPLETED-** A Tb Patient Who Completed Treatment Without Evidence Of Failure But With No Record To Show That Sputum Smear Or Culture Results In The Last Month Of Treatment And On At Least One Previous Occasion Were Negative, Either Because Tests Were Not Done Or Because Results Are Unavailable

**TREATMENT SUCCESS-** the sum of cured and treatment completed. This is calculated based on bacteriologically confirmed cases

**TREATMENT FAILED-** A Tb Patient Whose Sputum Smear Or Culture Is Positive At Month 5 Or Later During Treatment

**DIED-** A TB PATIENT WHO DIES FOR ANY REASON BEFORE STARTING OR DURING THE COURSE OF TREATMENT

**LOST TO FOLLOW-UP-** A Tb Patient Who Did Not Start Treatment Or Whose Treatment Was Interrupted For 2 Consecutive Months Or More

**NOT EVALUATED-** a tb patient for whom no treatment outcome is assigned. This includes cases "transferred out" to another treatment unit as well as cases for whom the treatment outcome is unknown to the reporting unit

## COMPLICATIONS OF PTB

**SPONTANEOUS PNEUMOTHORAX**

- Presence of air in the pleural cavity resulting to impairment of oxygenation and ventilation
- Medical emergency
- Results from rupture of a tb cavity adjacent to the pleura
- May be associated with formation of pus in the pleural space (empyema) leading to a pyopneumothorax

**Presentation:**

- Acute shortness of breath
- Chest pain

**Management:**

- The patient should be admitted

**BRONCHIECTASIS**

- Chronic Lung Disease Often Secondary To An Infectious Process That Results In The Abnormal And Permanent Distortion Of The Conducting Brochi Or Airways

**Presentation**

- Cough
- Copious amounts of sputum which is mainly greenish, blood stained and foul smelling
- Hemoptysis

**Management:**

- Chest physiotherapy which involves postural drainage to improve drainage of respiratory secretions
- Infective exacerbations will require antibiotics.
- Broad spectrum antibiotics like amoxycillin-clavulanate, metronidazole or clindamycin for anaerobic infection.
- Antipseudomonal antibiotic like ciprofloxacin, 3rd generation cephalosporin (ceftazidime) should be used when colonization with *pseudomonas* is suspected.

- If hemoptysis is severe and life threatening, patients should be admitted to hospital for more specialized treatment

**FIBROSIS OF THE LUNGS**

- Sequelae of extensive tuberculous disease
- In severe terminal cases, long term oxygen therapy may be required
- Patients should be referred for review and specialised care by a physician

**LUNG ABSCESS**

- Seen in patients with extensive damage to the lungs after tuberculosis
- Antibiotic treatment is given aided by the results of a pus culture-sensitivity test
- Surgical intervention may also be necessary

**ASPERGILLOMA**

- Result from colonization of tuberculous cavities or bronchiectatic lesions with the fungus *aspergilus*

**Presentation**

- Recurrent or persistent haemoptysis in patient previously treated for tb.
- Malaise
- Fever
- Chest x-ray shows a cavity with an air crescent (halo) around it
- High levels of specific immunoglobulin g against aspergillus in blood (confirmatory test)
- The only effective treatment is surgical removal of the aspergilloma

**HEPATITIS**

- A systemic infection affecting the liver predominantly.
- Viral **hepatitis** is caused by one of five viral agents:- **a-e**.
- **Except for hepatitis b, all other are rna viruses.**
- All types of viral **hepatitis** produce **clinically similar illnesses.**

- This is an acute viral disease which mainly affects the liver, causing hepatocellular inflammation , followed by jaundice. The disease is found in all the countries of the world.

- **Viral hepatitis** is liver inflammation due to a viral infection. It may present in acute or chronic forms. The most common causes of viral hepatitis are the five unrelated hepatotropic viruses

***There are five types of viruses which cause hepatitis. These are:***

1. Hepatitis A Virus (HAV)- incubation 1-4 weeks
2. Hepatitis B Virus (HBV
3. Hepatitis C Virus (HCV)
4. Hepatitis D Virus (HDV)
5. Hepatitis E Virus (HEV) - incubation 3-12 weeks

| TYPE | A | B | C | D | E |
|---|---|---|---|---|---|
| Source | fecal | Blood and blood derived body fluids | Blood and blood derived body fluids | Blood and blood derived body fluids | fecal |
| Inc. period | 15-50 | 60-90 | 15-160 | 30-180 | 14-60 |
| Mode of transmission | Feco-oral | Percutanious/ Permucosal | Percutaniou s/ Permucosal | Percutanious/ Permucosal | Feco-oral |
| Chronic infection | NO | YES | YES | YES | NO |
| Prevention | Pre/Post-expo. imm | Pre/Post-expo. imm | Blood Screening | Pre/Post-exp. imm | Safe water |

- Hepatitis A virus causes infectious hepatitis and is the most infectious of these viruses, while hepatitis B virus causes serum hepatitis (also called epidemic hepatitis).
- The hepatitis B virus causes chronic active infection of the liver (hepatitis) which may be followed some ten years later by liver cirrhosis (in 10 - 20% of the patients).
- In some of the patients who develop cirrhosis, the disease progresses to liver cancer (hepatocellular carcinoma).
- Hepatitis B infection occurs in about 1 - 3% of the human beings, but the incidence may be higher in patients undergoing kidney dialysis and in cancer wards (due to repeated blood transfusions), and among children because of close personal contact.

- It is difficult to tell the difference between infectious hepatitis and serum hepatitis on clinical observations alone.

**Mode of Transmission**

- Hepatitis A and E infections are transmitted through faecal-oral route and are both called infectious hepatitis.
- Hepatitis B, C and D are transmitted through blood products and close personal contacts and are called serum hepatitis. Serum hepatitis may cause chronic liver infection and liver cirrhosis.
- The human being is the only known reservoir and host of viral hepatitis.
- Hepatitis B infection transmission

***Takes place through two main routes:***

**Percutaneous route** - this is through injections and transfusion of blood and blood products.

**Non-percutaneous routes** - these include close personal contact for example, kissing and sexual intercourse, from mother to foetus through placenta or to baby during delivery.

**Clinical Features**

- All types of hepatitis infections are characterized by a similar clinical picture which manifest in two phases.

**Pre-icteric Phase (no jaundice)**

- Fever of sudden onset
- Malaise
- Loss of appetite
- Nausea
- Abdominal discomfort

**Icteric Phase**

- Appearance of jaundice
- Enlarged tender liver (hepatomegaly)
- Extreme tiredness and myalgia
- Feelings of deep sadness (depression)
- Pale stools
- Dark urine (contains bilirubin)

**Diagnosis**

- The urine of a person suffering from viral hepatitis is dark and contains bilirubin, while the stool is usually pale.
- In the blood, both direct and indirect bilirubin levels are raised. For hepatitis B, diagnosis is made by detecting various immunological markers in the blood. The most important is the hepatitis B surface antigen (HBsAg) which is present when the virus is in the blood in the acute stage and in the chronic carrier state.

**Management**

No specific treatment is available for both hepatitis A and B.

The patient should be given symptomatic RX together with diet and bed rest at home to prevent the spread of the disease

If admission is indicated - ensure that the patient is isolated and extra precautions taken during handling and disposal of excreta. Alcohol ↑ the risk of cirrhosis, advise patient to avoid alcohol for at least six months.

**Prevention and Control**

- Improve environmental sanitation to prevent the transmission of hepatitis A.
- Isolating patients suffering viral hepatitis
- Administration of hepatitis vaccine
- Screening blood for hepatitis B surface antigen before giving it for transfusion and excluding all donors with a history of jaundice
- Effective sterilisation and high level disinfection of all instruments, needles and syringes

In addition to the hepatitis viruses, other viruses that can also cause hepatitis include:

Herpes simplex

- Cytomegalovirus
- Epstein-Barr virus
- Yellow fever

## HEPATITIS A

- *It* is caused by hepatitis A virus (HAV), a picornavirus transmitted by the fecal-oral route often associated with ingestion of contaminated food.
- It causes an acute form of hepatitis and does not have a chronic stage. The patient's immune system makes antibodies against HAV that confer immunity

against future infection. People with hepatitis A are advised to rest, stay hydrated and avoid alcohol.

- The time between the infection and the start of the illness averages 28 days (ranging from 15 to 50 days), and most recover fully within 2 months, although approximately 15% of sufferers may experience continuous or relapsing symptoms from six months to a year following initial diagnosis.
- Is present in the faeces of infected persons and is most often transmitted through consumption of contaminated water or food.
- Certain sex practices can also spread HAV. Infections are in many cases mild, with most people making a full recovery and remaining immune from further HAV infections.
- However, HAV infections can also be severe and life threatening. Most people in areas of the world with poor sanitation have been infected with this virus. Safe and effective vaccines are available to prevent HAV.

**HEPATITIS A VIRUS TRANSMISSION**

• Close personal contact
—(household contact, sex contact, child day care centers)
• Contaminated food, water
—(infected food handlers, raw shellfish)
• Blood exposure (rare)

**LABORATORY DIAGNOSIS**

• **Acute infection** is diagnosed by the detection of HAV-IgM in serum by EIA.
• **Past Infection** i.e. immunity is determined by the detection of HAV-IgG by EIA (test Anti/HAV/total Ab).

**HEPATITIS A VACCINATION STRATEGIES**

**EPIDEMIOLOGIC CONSIDERATIONS**

• Many cases occur in community-wide outbreaks
—no risk factor identified for most cases
—highest attack rates in 5-14 year olds
—children serve as reservoir of infection

**Persons at increased risk of infection**

—travelers
—homosexual men
—injecting drug users

**HEPATITIS A PREVENTION - IMMUNE GLOBULIN**

• Pre-exposure
—travelers to intermediate and high HAV-endemic regions
• Post-exposure (within 14 days)
Routine
—household and other intimate contacts

**Selected situations**

—institutions (e.g., day care centers)
—common source exposure (e.g., food prepared by infected food handler)

**Hepatitis B**

- *It* is caused by hepatitis B virus, a hepadnavirus that can cause both acute and chronic hepatitis. Chronic hepatitis develops in the 15% of adults who are unable to eliminate the virus after an initial infection. Identified methods of transmission include blood, tattoos, sexually, or via mother to child by breast feeding. However, in about half of cases the source of infection cannot be determined.
- Patients with chronic hepatitis B have antibodies against hepatitis B, but these antibodies are not enough to clear the infection of the affected liver cells.
- The continued production of virus combined with antibodies is a likely cause of the immune complex disease seen in these patients.
- A vaccine is available that will prevent infection from hepatitis B for life.
- Is transmitted through exposure to infective blood, semen, and other body fluids.
- HBV can be transmitted from infected mothers to infants at the time of birth or from family member to infant in early childhood.
- Transmission may also occur through transfusions of HBV-contaminated blood and blood products, contaminated injections during medical procedures, and through injection drug use.
- HBV also poses a risk to healthcare workers who sustain accidental needle stick injuries while caring for infected-HBV patients. Safe and effective vaccines are available to prevent HBV.

## HEPATITIS B - CLINICAL FEATURES.

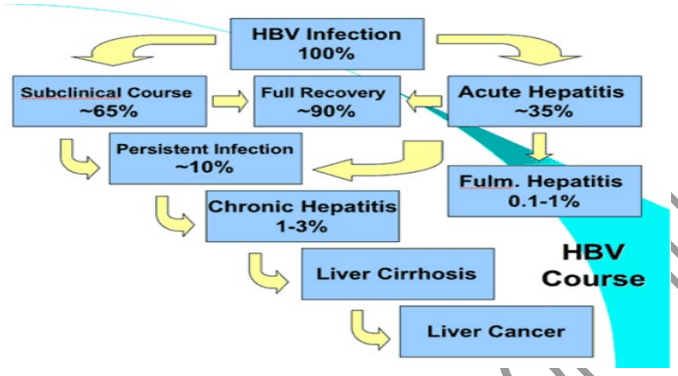
- Incubation period: Range 45-180 days
- Clinical illness (**jaundice**): <5 yrs : <10%
  5 yrs : 30%-50%
- Acute case-fatality rate: 0.5%-1%
- Chronic infection: <5 yrs: 30%-90%
  > 5 yrs: 2%-10%
- Premature mortality from (15%)

chronic liver disease: %-25%

### SPECTRUM OF CHRONIC HEPATITIS B DISEASES

1) Chronic Persistent Hepatitis – asymptomatic.
2) Chronic Active Hepatitis - symptomatic exacerbations of hepatitis.
3) Cirrhosis of Liver.
4) Hepatocellular Carcinoma.

**HBV infection – evolution (adults)**




**Concentration of Hepatitis B Virus in Various Body Fluids**

| High | Moderate | Detectable |
|---|---|---|
| •Blood<br>•Serum<br>•Wound exudates | •Semen<br>•Vaginal fluid<br>•Saliva | •Urine<br>•Feces<br>•Sweat<br>•Tears<br>•Breast milk |

### HEPATITIS B VIRUS

### MODES OF TRANSMISSION

**Sexual** - sex workers and homosexuals are particular at risk.

**Parenteral** - IVDA, Health Workers are at increased risk.

**Perinatal**-Mothers who are ***HBeAg positive*** are much more likely to transmit to their offspring than those who are not.

Perinatal transmission is the main means of transmission in high prevalence populations.

### DIAGNOSIS.

- **HBsAg** - used as a general marker of infection.
- **HBsAb** - used to document recovery and/or immunity to HBV infection.
- **anti-HBc/IgM** - marker of acute infection.
- **anti-HBc/IgG** - past or chronic infection.
- **HBeAg** - indicates active replication of virus and therefore infectiveness.
- **HBV-DNA** - indicates active replication of virus, more accurate than HBeAg especially in cases of escape mutants. Used mainly for monitoring response to therapy.

### TREATMENT

- Interferon - for HBeAg positive carriers with chronic active hepatitis. Response rate is 30 to 40%.
- Lamivudine - a NRTI

### PREVENTION.

- **Vaccination** of those at **increased risk of HBV infection**
  - **Health care workers.**
  - **Neonates.**
- **HBIG** may be used to protect persons who are exposed to hepatitis B.
- It is efficacious within ***48 hours of the incident***.
- It may also be given to ***neonates*** whose mothers are HBsAg and HBeAg positive.
- Other measures - screening of blood donors, blood and body fluid precautions.

## HEPATITIS C

- *It* is caused by hepatitis C virus (HCV), an RNA virus that is a member of the Flaviviridae family.
- HCV can be transmitted through contact with blood and can also cross the placenta. Hepatitis C usually leads to chronic hepatitis, culminating in cirrhosis in some people. It usually remains asymptomatic for decades. Patients with hepatitis C are susceptible to severe hepatitis if they contract either hepatitis A or B, so all persons with hepatitis C
- should be immunized against hepatitis A and hepatitis B if they are not already immune, and avoid alcohol. HCV viral levels can be reduced to undetectable levels by a combination of interferon and the antiviral drug ribavirin.

- Is mostly transmitted through exposure to infective blood.
- This may happen through transfusions of HCV-contaminated blood and blood products, contaminated injections during medical procedures, and through injection drug use.
- Sexual transmission is also possible, but is much less common. There is no vaccine for HCV.

**Risk Factors Associated with Transmission of HCV**

- Transfusion or transplant from infected donor
- Injecting drug use
- Haemodialysis (years on treatment)
- Accidental injuries with needles/sharps
- Sexual/household exposure to anti-HCV-positive contact
- Multiple sex partners
- Birth to HCV-infected mother

**Laboratory Diagnosis**

- **Serologic diagnosis**
- **HCV antibody** - generally used to diagnose hepatitis C infection.
- For acute infection: anti-HCV/IgM type.

**Treatment**

- **Interferon** - may be considered for patients with chronic active hepatitis. The response rate is around 50% but 50% of responders will relapse upon withdrawal of treatment.
- **Ribavirin** - there is less experience with ribavirin than interferon. However, recent studies suggest that a combination of interferon and ribavirin is more effective than interferon alone.

**Prevention of Hepatitis**

- Screening of blood, organ, tissue donors
- High-risk behavior modification
- Blood and body fluid precautions

## HEPATITIS D

- Also known as the **Delta Virus.**
- **Small RNA virus**
- **Defective virus (need the presence of HBsAg)**
- It is a small virus that requires concomitant infection with hepatitis B to survive. HDV cannot survive on its own because it requires a protein that the hepatitis B virus makes to enable it to infect liver cells.
- The ways in which hepatitis D is spread are by shared needles among drug abusers, contaminated blood, and by sexual contact.
- Infections occur only in those who are infected with HBV. The dual infection of HDV and HBV can result in a more serious disease and worse outcome. Hepatitis B vaccines provide protection from HDV infection.
- Hepatitis E virus (HEV) is mostly transmitted through consumption of contaminated water or food. HEV is a common cause of hepatitis outbreaks in developing parts of the world and is increasingly recognized as an important cause of disease in developed countries

**Hepatitis D - Clinical Features**

- **Coinfection** (both viruses are transmitted)
- severe acute disease;
- low risk of chronic infection.
- **Superinfection** (HDV on HBsAg (+): healthy carrier, acute of chronic infection)
- usually develop chronic HDV infection (75%);
- high risk of severe chronic liver disease;
- may present as an acute hepatitis.

**Hepatitis D Virus Modes of Transmission**

- Percutanous exposures-injecting drug use
- Permucosal exposures-sexual contact

**Hepatitis D - Prevention**

HBV-HDV Co-infection

- Pre- or post exposure prophylaxis to prevent HBV infection.

HBV-HDV Superinfection

- Education to reduce risk behaviors among persons with chronic HBV infection

## HEPATITIS E

- Incubation period: Average 40 days
- The *Hepatitis E virus* (HEV) produces symptoms similar to hepatitis A, although it can take a fulminant course in some patients, particularly

pregnant women; it is more prevalent in the Indian subcontinent.

- HEV has a fecal-oral transmission route
- Transmitted via the faecal-oral route
- It is water-borne
- Symptomatic infection more common in young adults aged 15-40 years
- High fatality rate in pregnant women (5-25%)

**Hepatitis E - Epidemiologic Features**

- Most outbreaks associated with faecally contaminated drinking water.
- Minimal person-to-person transmission.

**Clinical picture**

- Incubation period is 3-12 weeks
- Typical signs and symptoms include: jaundice, loss of appetite, an enlarged, tender liver, abdominal pain, nausea, and vomiting.

**Diagnosis**

- Blood tests to detect elevated antibody levels of specific antibodies to hepatitis E.
- Suspect hepatitis E infection in outbreaks of water-borne hepatitis if the disease is more severe in pregnant women, and hepatitis A has been ruled out.

**Management**

- Prevention is key
- Admission in hospital for supportive therapy
- Hospitalisation is required for fulminant hepatitis should be considered especially for pregnant women

**Prevention and control**

- Safe water supply
- Good personal hygiene
- Improved sanitation
- Avoid drinking water (and beverages with ice) of unknown purity, uncooked shellfish, and uncooked fruit/vegetables not peeled or prepared by traveler.
- Vaccine?

**MODE OF TRANSMISSION**

- Hepatitis A and E infections are transmitted through faecal-oral route and are both called infectious hepatitis.
- Hepatitis B, C and D are transmitted through blood products and close personal contacts and are called serum hepatitis. Serum hepatitis may cause chronic liver infection and liver cirrhosis.
- The human being is the only known reservoir and host of viral hepatitis.
- The disease is transmitted from the infected person to the susceptible host through faeces, contaminated food and objects, blood, blood serum and other body fluids
- In the case of hepatitis B infection, transmission takes place through two main routes:
- Percutaneous route - this is through injections and transfusion of blood and blood products.
- Non-percutaneous routes - these include close personal contact for example, kissing and sexual intercourse, from mother to foetus through placenta or to baby during delivery.

**Clinical Features**

- All types of hepatitis infections are characterised by a similar clinical picture.
- The incubation period is one to four weeks in the case of hepatitis A and 12 weeks or longer in the case of hepatitis B.
- Hepatitis infections manifest in two phases.

**a. Pre-icteric Phase (no jaundice)**

- Fever of sudden onset
- Malaise
- Loss of appetite
- Nausea
- Abdominal discomfort

**b. Icteric Phase**

- Appearance of jaundice
- Enlarged tender liver (hepatomegaly)
- Extreme tiredness and myalgia
- Feelings of deep sadness (depression)
- Pale stools

- Dark urine (contains bilirubin)

**People At Risk of Hepatitis**

Reservoirs of hepatitis B virus include :

- ❖ Sexually promiscuous individuals,
- ❖ Spouses of acutely infected persons
- ❖ Health care workers exposed to blood
- ❖ Family members of chronically infected persons
- ❖ Anyone who requires repeated blood transfusions

**Diagnosis**

- The urine of a person suffering from viral hepatitis is dark and contains bilirubin, while the stool is usually pale.
- In the blood, both direct and indirect bilirubin levels are raised.
- In the case of hepatitis B, diagnosis is made by detecting various immunological markers in the blood.
- The most important is the hepatitis B surface antigen (HBsAg) which is present when the virus is in the blood in the acute stage and in the chronic carrier state.

**MANAGEMENT**

- No specific treatment is available for both hepatitis A and B.
- The patient should be given symptomatic treatment together with diet and bed rest at home to prevent the spread of the disease.
- If admission is indicated for one reason or the other, you should ensure that the patient is isolated and extra precautions taken during handling and disposal of excreta.
- Since alcohol increases the risk of cirrhosis, you should advise the patient to avoid alcohol for at least six months

**PREVENTION AND CONTROL**

- Improvement of environmental sanitation will prevent the transmission of hepatitis
- Isolating patients suffering viral hepatitis
- Administration of hepatitis vaccine
- Screening blood for hepatitis B surface antigen before giving it for transfusion and excluding all donors with a history of jaundice
- Effective sterilisation and high level disinfection of all instruments, needles and syringes

**SPECIFIC PREVENTIVE MEASURES FOR HEPATITIS B**

- Vaccination
- Wash your hands thoroughly after any potential exposure
- Practice safe sex with all partners
- Avoid direct contact with blood and bodily fluids
- Clean up blood spills with a fresh diluted bleach solution
- Cover all cuts carefully
- Avoid sharing sharp items such as razors, nail clippers, toothbrushes, and earrings or body rings
- Discard sanitary napkins and tampons into plastic bags
- Avoid illegal street drugs (injecting, inhaling, snorting, popping pills)
- Do not donate blood or body organs
- Make sure new, sterile needles are used for ear or body piercing, tattoos, and acupuncture

## CHOLERA

- An acute diarrhoeal infection caused by ingestion of food or water contaminated with the bacterium *Vibrio cholerae.*
- Researchers have estimated that there are 1.4 to 4.3 million cases, and 28 000 to 142 000 deaths worldwide due to cholera every year.
- Up to 80% of cases can be successfully treated with oral rehydration salts.
- Has a short incubation period of **two** hours to **five** days hence a potentially explosive pattern of outbreaks.
- Provision of safe water and sanitation is critical in reducing the impact of cholera and other waterborne diseases.
- Oral cholera vaccines are considered an additional means to control cholera

**CAUSES**

- Vibrio cholerae which Has 2 strains; O1 and O139
- Evidence of other strains in Africa and Asia shown to be more virulent and high case fatality

- Main reservoirs of *V. cholerae* are people and aquatic sources such as brackish water and estuaries, often associated with algal blooms

**CLINICAL FEATURES**

- About 75% of people infected with *V. cholerae* are asymptomatic, though the bacteria are present in their faeces for 7–14 days after infection and are shed back into the environment, potentially infecting other people.
- Of the remaining 25%, 80% have mild or moderate symptoms, while around 20% develop acute watery diarrhoea with severe dehydration
- The ISS have a poor prognosis upon infection
- Incubation period is about 2-3 days

***In severe cholera, the disease develops in three stages:-***

**Stage 1:**

- Lasts for 3-12 hours
- Profuse watery diarrhea pours from the patient
- Soon fecal matter disappears and clear fluid with mucus flakes (rice water) appears.
- Vomiting follows diarrhea
- Abdominal cramps starts due to salts loss

**Stage 2:**

- There is collapse from dehydration
- The body becomes cold, dry and inelastic.
- Rapid and feeble pulse
- Low (unrecordable) blood pressures
- Anuria
- Death may occur from shock

**Stage 3:**

- Is stage of recovery
- Diarrhea decreases, patient is able to drink and dehydration improves rapidly

**PATHOPHYSIOLOGY**

- Most bacteria, when consumed, do not survive the acidic conditions of the human stomach. The few surviving bacteria conserve their energy and stored nutrients during the passage through the stomach by shutting down much protein production. When the surviving bacteria exit the stomach and reach the small intestine, they need to propel themselves through the thick mucus
- The small intestine to get to the intestinal walls, where they can thrive. *V. cholerae* bacteria start up production of the hollow cylindrical protein flagellin to make flagella, the cork-screw helical fibers they rotate to propel themselves through the mucus of the small intestine.
- Once the cholera bacteria reach the intestinal wall, They stop producing the protein flagellin. On reaching the intestinal wall, *V.*
- *Cholerae* start producing the toxic proteins that give the infected person a watery diarrhea. This carries the multiplying new generations of *V. cholerae* bacteria out into the drinking water of the next host if proper sanitation measures are not in place.

**Risk factors and disease burden**

- Inadequate environmental management
- Peri-urban slums and IDP/refugee camps
- Poor water and sanitation practices
- Inadequate or lack of good personal hygiene measures
- Lack of adequate appropriate domestic practices

**Treatment**

- Cholera is an easily treatable disease.
- Up to 80% of people can be treated successfully through prompt administration of oral rehydration salts
- Very severely dehydrated patients require administration of I.V fluids.
- Appropriate antibiotics to diminish the duration of diarrhoea, reduce the volume of rehydration fluids needed, and shorten the duration of *V. cholerae* excretion.
- Mass administration of antibiotics is not recommended, as it has no effect on the spread of cholera and contributes to increasing antimicrobial resistance.

**Prevention and control**

- A multidisciplinary approach based on prevention, preparedness and response
- An efficient surveillance system
- Practice other preventive and control measures
- ➢ All these will help mitigate cholera outbreaks, control cholera in endemic areas and reduce deaths.
- Currently there are 2 WHO pre-qualified oral cholera vaccines (OCVs) (Dukoral® and Shanchol®) .Dukoral® is administered to adults and children aged >6 years in 2 doses; and to children aged >2 years and <6 years in 3 dose. Shanchol's immunization schedule is 2 doses given at an interval of 2 weeks for those aged >1 year

## BACILLARY DYSENTERY/SHIGELLOSIS

- This refers to a type of diarrheoa restricted to shigella infection and mainly as a result of Enterobacteriaceae family (non-motile gram –ve bacilli)
- Three species are associated with bacillary dysentery: *Shigella sonnei, Shigella flexneri* and *Shigella dysenteriae*
- *Shigella boydii* has also been reported to cause dysentery
- Mainly fecal oral using the 4F's with the infected mainly being asymptomatic for up to 3 months
- Dependent on sanitary conditions, quality or availability of water, population of the flies, nutrition status, overcrowding (IDP, refugees, prisons etc) and seasonality

**Clinical picture and epidemiology**

- Humans are the only reservoir for epidemics
- Reproduce in food, dirty dishes & hands
- Incubation is 1-4 days with the symptoms being mild or severe even leading to death
- Presents with sudden bloody stool, fever, vomiting, abdominal cramps(colicky pain), diarrhea with tenesmus and rapid pulse
- Severe cases; convulsions, dehydration (oliguria, muscle cramps) and rectal prolapse

**DIAGNOSIS**

- Dark red stool with lots of mucous
- Leukocytosis and erythrocytosis on microscopy
- Culture and sensitivity of the stool for shigella

**Management**

- Prevent and Rx dehydration
- Rehydration
- Appropriate antibiotics vis; cotrimoxazole 960 BD*5/7, amoxyl 500mg QID*5/7, ciprofloxacin 500mg OD *5/7
- Use of PPE while handling such patients

**Prevention and control**

- Currently, there is no vaccine to prevent shigellosis.
- Frequent & careful hand washing with soap
- Correct disposal of infected waste e.g. diapers
- Disinfecting (using Lysol or bleaching agents) areas suspected to be contaminated
- Isolate infected children from the non-infected ones
- Basic food safety precautions & disinfection of drinking water prevents shigellosis from food and water.
- People with shigellosis should not prepare food or drinks for others
- Practice hand washing and disinfection in the common swimming areas and day care centers

**Possible complications**

- Dehydration and electrolyte disturbance
- Bacteraemia
- Seizures may occur in young children and are common where there is fever
- Haemolytic uraemic syndrome
- Reactive arthritis (or Reiter's syndrome when arthritis is combined with uveitis and urethritis) can occur. This is most often associated with *S. flexneri* infection.
- Toxic megacolon is occasionally a complication of *S. dysenteriae.*

## AMOEBAISIS

- It is an infection caused by a pathogenic amoeba, *Entamobea histolytica.*
- Infection with amoeba is in most cases asymptomatic. Under certain circumstances, the amoeba may invade the bowel wall causing amoebic dysentery.

**Epidemiology**

- It is acquired when the cysts are ingested. The emerging trophozites takes up residence in the ascending large bowel where it can live as a commensal or begin penetrating the intestinal wall causing small mucosal ulcerations which result in dysentery.
- The parasite penetrates through the sub mucosa and into the muscularis layer of the colon.
- If the muscularis layer becomes extensively involved, scarring can result (amoeboma).
- Invasion of bloodstream by the trophozite may led to infection of the liver, especially the right lobe. After a variable incubation period, a liver abscess may form.
- The only infective form of E. histolytica is the cyst. If trophozites are ingested they are destroyed in the stomach.
- An asymptomatic cyst-passer forms the main reservoir for spread of amoebiasis.

- Cysts are passed from person to person by the faecal-oral route. Amoebiasis can occur in families or spread through institutions but usually doesn't occur in epidemics like Bacillary dysentry.

**Persons at greatest risk for amoebiasis include:**

- people who have traveled to tropical locations with poor sanitation
- immigrants from tropical countries with poor sanitary conditions
- people who live in institutions with poor sanitary conditions, such as prisons
- Food handlers may transmit the cysts while preparing or handling food.
- Transmission is also possible during anal sex or colonic irrigation.
- people with compromised immune systems and other health conditions

**Clinical picture**

- When amoeba penetrate the intestinal wall, they multiply in the sub-mucosa causing bottle shaped ulcers.
- From the ulcers the amoeba may be transported to the liver.
- Amoeboma- this is where by the scar in the colon heals while the parasite is still in the tissue. This results in an abscess and keloids.
- Amoebic dysentery is insidious and associated with abdominal discomfort. There may be mildly loose stool or frank diarrhoea with or without blood and mucous
- Distended colon
- Chronic amoebiasis may resemble duodenal ulcer, gall bladder disease or carcinoma of the colon.

**Other extra-intestinal manifestation**

- Amoebiasis of the skin may occur where amoeba come into contact with the skin, that is:
  - Around the anus
  - Around incision wounds
- An amoebic skin ulcer is irregular and painful. The ulcer enlarges continuously because of necrosis of the edges.
- When a liver abscess breaks through into the chest cavity, a lung abscess or empyema may occur.

**Diagnosis**

- Stool is checked for cysts.
- colonoscopy

**Management**

- asymptomatic infection should be treated: paromomycin, iodoquinol, and diloxanide furoate are best suited for such therapy.
- Metronidazole 25mg/kg/day for 3 days in children-tabs should be taken slowly to reduce vomiting.
- Tinidazole is an effective alternative.
- Unlike pyogenic liver abscess, uncomplicated amebic liver abscess generally responds to medical therapy alone

**INDICATIONS FOR DRAINAGE OF AMEBIC LIVER ABSCESS INCLUDE THE FOLLOWING:**

- Presence of a left-lobe abscess more than 10 cm in diameter
- Impending rupture and abscess that does not respond to medical therapy within 3-5 days

## ENTERIC FEVERS

1. Typhoid fever
2. Paratyphoid A and B fevers

## TYPHOID FEVER

- infectious disease characterized by high continuous fever, malaise and involvement of lymphoid tissue and spleen.

**Etiology**

- This is an infectious bacterial disease caused by *Salmonella typhi.*

**Epidemiology**

- Endemic in many regions of East Africa, although epidemic outbreaks have occurred when a source of water or food used by many people has been contaminated
- Human beings are the only known reservoir and host.

**Mode of transmission**

- Mainly spread by the faecal-oral route through contaminated food, water and milk.
- Flies are also important in the transmission

**Incubation Period**

- The incubation period of typhoid fever is 7 – 21 days

**Clinical Features**

***The disease has a gradual onset which progresses through the following four stages.***

***First Stage***

- patient has severe headache, malaise, loss of appetite, body pains and aches and a tendency to nose-bleed
- The body temperature rises day by day or in steps to 39.5ºC or higher.
- Most patients cough because they develop bronchitis and may also complain of constipation.

**Second Stage**

- temperature continues to rise, but the pulse rate is slower than would be expected for that temperature.
- There is swelling of lymphoid tissue in the intestines as well as Peyer's patches necrosis and ulcers, which cause the abdomen to become distended and tender
- The high temperature and toxaemia causes mental confusion and disorientation in the patient.
- Half the patients may develop greenish watery ('pea-soup') diarrhoea and bronchopneumonia.

**Third Stage**

- Body temperature decreases step by step and the patient improves slowly
- If there is no improvement, the Peyer's patches in the intestines perforate and toxaemia increases.
- The patient becomes delirious and incontinent of urine and stool, muscles twitch and coma may precede death

**Fourth Stage:**

- For the patients who do not suffer the serious complications of the third stage, the fourth stage is a period of convalescence.
- The temperature drops back to normal and the patient recovers gradually.

**Diagnosis**

- best way to diagnose typhoid fever is through a blood culture.
- Stool and urine cultures can also be made although they are only positive after the first week
- Other tests:
- Widal test during the first and second week
- WBC count which indicates low levels (leucopenia) with raised lymphocyte count
- Stool to check for presence of occult blood which is found in 100% of the cases

**Management**

- Fluid replacement due to diarrhoea
- Oral norfloxacin 400mg 12 hourly for 10 - 14 days
- Oral ciprofloxacin 500mg bd. for 14 days
- Oral corticosteroids to prevent Jarisch-Herxheimer's reaction

**Complications**

- Intestinal haemorrhage
- Chronic carrier state for salmonella typhi
- Intestinal perforation leading to peritonitis and sepsis

**Prevention and Control**

- Identification of the carriers especially those who work as food handlers and treat them promptly
- Administration of typhoid vaccine
- Safe water supply
- Improvement in food hygiene
- Treating healthy carriers
- Patient should be isolated in fly-proof room
- Contaminated articles should be disposed by incineration
- Stools and urine should be disposed of in a pit latrine or septic tank
- Surgical treatment for perforated bowels

## PARATYPHOID FEVER

- It is caused by bacteria known as salmonella paratyphi types A, B and C. The disease runs a milder course than typhoid fever and also has enlargement of the spleen, bloodstained diarrhoea and swelling of the Peyer's patches
- Paratyphoid fever may present like typhoid fever, but in most cases it presents as gastroenteritis or transient diarrhoea

***Treatment***

- The treatment of paratyphoid fever is as follows:-
- Intravenous fluid if diarrhoea is severe
- Oral rehydration if diarrhoea is mild
- Oral contrimoxazole two tablets bd. for five to seven days

***Prevention and Control***

- The prevention and control measures are similar to those that were covered under typhoid fever.

## BACILLARY DYSENTERY (SHIGELLOSIS)

- Bacillary dysentery, also known as ***shigellosis***, is an acute bacterial disease of the intestines.

**Aetiology**

- It is caused by a ***non-motile gram-negative bacilli*** of the genus ***shigella spp***.
- The organisms responsible for outbreaks are:

**i)** *Shigella sonnei*
**ii)** *Shigella dysenteriae*
**iii)** *Shigella flexneri*
**iv)** *Shigella boydii*

However, the first three organisms are the most common causes of outbreaks.

**Epidemiology**

- It is common especially in areas where the standards of hygiene are low, particularly, where there is scarcity of safe water, improper human excreta disposal, large population of flies and child malnutrition. Humans are the only known reservoir

**Mode of Transmission**

- Faecal-oral route
- Directly through flies or contaminated hands
- Indirect transmission may also occur through dishes which are poorly washed.
- The shigella multiply in food which when ingested causes dysentery.

**Incubation period**

- a short incubation period of one to four days.

**Clinical Features**

- Is sudden with fever, headache, diarrhoea with streaks of blood, and colicky abdominal pains
- After a few motions (usually in a few hours) diarrhoea stops and is followed by severe colicky abdominal pain known as dysenteric syndrome, and painful contractions of the sphincter ani which produce an irresistible urge to defecate
- Patient they pass small amounts of purulent mucus and blood.
- Vomiting may also occur
- Toxins produced by the shigella on the wall of the colon may be absorbed into the blood stream resulting in toxaemia -high fever and rapid pulse
- Dehydration is also common and dangerous as it may cause muscular cramps, oliguria and shock.
- In infants, rectal prolapse may occur as well as convulsions.

**Diagnosis**

- The following laboratory examinations are undertaken:
  - ❖ Stool examination which shows the presence of blood and mucus
  - ❖ Stool microscopy which shows presence of large numbers of white blood cells and erythrocytes
  - ❖ Stool culture for shigella spp.

**Management**

- Mild bacillary dysentery is self-limiting and all it requires is prevention or treatment of dehydration
- Severe infection? combine rehydration with antibiotics as follows
- Antibiotics: oral ciprofloxacin 500mg q12h for five to seven days
- Analgesics for colic
- Rehydration due to diarrhoea and fluid loss

**Prevention and Control**

- Depends on stopping the faecal-oral transmission through the following ways
- Safe water supply
- Improvement in personal hygiene
- Digging and use of pit latrines
- Practising food hygiene
- Giving health education that emphasises environmental hygiene and breastfeeding
- Inspection of public eating places, markets, boarding schools and camps

## AMOEBIC DYSENTERY

- An infection of the intestines, especially the colon and caecum, caused by the amoeba-like protozoa *Entamoeba histolytica*.
- *E. histolytica* is usually a commensal of the intestines and can live there without causing disease. It is a *gastrointestinal* infection that may or may not be *symptomatic* and can remain *latent* in an infected person for several years.
- It can however invade the colon wall causing colitis, acute dysentery, or long term chronic diarrhoea.

**Epidemiology**

***Distribution***

- Warmer areas (tropics) because the parasitic cysts surviving longer in warm moist conditions and also because of poorer hygiene.
- It is therefore endemic in regions of the world with limited modern *sanitation* systems
- In East Africa liver amoebiasis is especially rampant in Arusha and around Mount Kilimanjaro.
- Can occur in families or spread through institutions but usually does not occur in epidemics unlike bacillary dysentery.

- According to WHO (2008), it is estimated to cause 70,000 deaths per year worldwide.

**Risk Factors**

- Malnutrition
- Old age
- Pregnancy
- Immunosuppresion
- Use of corticosteroid therapy to suppress the immune system

**Causative Agent**

- Entamoeba histolytica.
- Definitive host is the human being.
- Habitat is the large intestine of the host especially the caecum and colon where it can exist in two forms:
- As a cyst
- As a trophozoite
- Trophozoite form is the pathogenic form (active form), which feeds on bacteria and tissues
- Trophozoites dominates in liquid stools but rapidly die once discharged out of the body as it cannot withstand the outside environment
- Some trophozoites in the colonic lumen become cysts and are excreted with stool.

❖ cystic form predominates in formed stools and is resistant to the external environment

- They are non-pathogenic but infective. They do not invade the cells but once swallowed they are capable of hatching out (encysting) after passing the stomach and the small intestines.
- The daughter cells become the trophozoites.
- The cyst can live outside the body for 10 days even more and can resist extreme temperatures such as on heating and refrigeration. They are however killed by boiling (temperature above 100$^{o}$C).

**pathogenesis**

- Once the cysts are ingested, the emerging trophozoites take up residence in the intestinal mucosa.
- The organisms multiply in the mucosa (causing the formation of bottle-shaped ulcers each 1-2cm in diameter). Too many such ulcers may cover the large intestine. Some of the ulcers may become perforated leading to severe peritonitis with shock. In the small intestines, the *entamoeba histolytica* may pass through the mucous membrane and enter the liver. After a variable incubation period a liver abscess may form.

**Transmission**

- *fecal-oral route*
- can also be transmitted indirectly through contact with dirty hands or objects (fomites) as well as by anal-oral *contact*.
- Infections can occur from drinking water but it' really not a water-borne disease.

**Incubation Period**

- about 2 to 4 weeks.
- It can however be as long as 11 weeks.

**Clinical Picture**

- Many people remain asymptomatic (the cyst-passers).
- With tissue invasion, symptoms can range from mild *diarrhea* to *dysentery* with *blood* and *mucus* in the stool (foul smelling diarrhoea)

**Other symptoms**

- intermittent diarrhea with constipation (pea-soap diarrhea)
- flatulence and cramping abdominal pain.
- Tenderness over the liver or ascending colon may occur and stools may contain mucus and blood.
- Severe amoebiasis infections occur in two major forms

❖ Invasion of the intestinal lining causes *amoebic dysentery* or *amoebic colitis*

❖ If the parasite reaches the bloodstream it can spread through the body, most frequently ending up in the liver where it causes *amoebic liver abscesses*

## EXTRA-INTESTINAL AMOEBIC DISEASE

- The most common site for extra-intestinal amoebiasis is the liver where it forms a liver abscess. Other secondary sites include lungs and skin leading to:
- Amoebic infection of the skin
- Amoebic balanitis
- Amoebic lung abscess
- Amoebic brain abscess

**Investigations**

❖ Stool for Cyst (Microscopy)-The *E. histolytica* cyst is quadrinucleated. Asymptomatic human infections are usually diagnosed by finding cysts shed in the stool. Since cysts are not shed constantly, a minimum of three stools should be examined. In symptomatic infections, the motile form (the trophozoite) can often be seen in fresh feces.

❖ *Serological* **Tests -***Serological* tests exist and most individuals (whether with symptoms or not) will test positive for the presence of *antibodies*.

- levels of antibody are much higher in individuals with liver *abscesses*.
- Serological tests are useful in detecting infection by *E. histolytica* if the organism goes extra-intestinal and in excluding the organism from the diagnosis of other disorders.
- Note that positive serological result may not necessarily indicate an active infection.why?
- A negative serological result however can be equally important in excluding suspected tissue invasion by *E. histolytica*.

❖ **Sigmoidoscopy-** To visualize the GI mucosa for ulcerations but this is not specific.

**Case Management**

- two different types of amoebicides (tissue and luminal) are needed to treat the infection, one for each location.

➢ Asymptomatic cyst carriers (luminal amoebiasis)

- Treat only if patient is a food handler.
- Use entamizole/dilazole (metronidazole-diloxanide furoate)
- Adults: 500mg TDS x $^{10}/_{7}$
- Children: 30 – 50 mg/kg TDS x $^{10}/_{7}$

❑ Dilazole/dirazole is a combination of metronidazole and *diloxanide furoate* hence effective for both luminal and tissue amoebiasis.

## TISSUE AMOEBIASIS (AMOEBIC DYSENTERY)

- *Metronidazole*, or a related drug such as *tinidazole*
- *Metronidazole* (Flagyl) 400 - 800mg po TDS for 5 days (children: 30 – 50 mg/kg/day in 3 divided doses.
- *Tinidazole* (fasigyn, tinycos) 2g OD po for 3 days (children: 50mg/kg per dose

**Luminal Amoebiasis**

- *paromomycin* (Humatin) and *diloxanide furoate* (also known as furamide). These are however not essential in Kenya.
- Paromomycin: 500mg po TDS x $^{10}/_{7}$
- Furamide: 500mg po TDS x $^{10}/_{7}$
- Entamizole/Dilazole: 500mg po TDS x $^{10}/_{7}$
- Children: 30 – 50 mg/kg po TDS x $^{10}/_{7}$

**Prevention and Control**

- Health Education
- Food Handlers- They should be medically examined between 6 months and 1 year so that cyst-passers, if any, are treated and stopped from handling food
- Food Hygiene- must be properly and hygienically handled to avoid contamination for instance by fecal matter that may contain the cysts.
- Environmental Hygiene and Sanitation
- Water Supplies-Public water supplies should be protected from any contamination by fecal matter

## EBOLA VIRUS DISEASE

- Ebola, previously known as Ebola hemorrhagic fever, is as deadly disease caused by infection with one of the Ebola virus species.
- The illness is characterized by abrupt onset of headache,myalgia,sorethroat,rash and hemorrhage
- Ebola can cause disease in humans and nonhuman primates (monkeys, gorillas, and chimpanzees).
- Ebola was first discovered in 1976 near the Ebola River in what is now the Democratic Republic of the Congo. Since then, outbreaks have appeared sporadically in Africa

**SUBTYPES/SPECIES OF EBOLA VIRUS**

- There are five identified Ebola virus species, four of which are known to cause disease in humans: They include:

**a.** *Zaire ebolavirus*
**b.** *Sudan ebolavirus*
**c.** *Taï Forest ebolavirus*, formerly *Côte d'Ivoire ebolavirus*)
**d.** *Bundibugyo ebolavirus*
**e.** *Reston ebolavirus*, has caused disease in nonhuman primates, but not in humans.

**Transmission of the Ebola Virus**

- Natural reservoir is suspected to be fruit bats
- Aperson is infectious WHEN they are symptomatic.
- Transmission is through the broken skin or mucous membranes due to:

– Close contact with blood, secretions, organs or other bodily fluids of infected animals

Direct physial contact with blood, saliva, stool, urine, sweat and other body fluids of an infected person and soiled linen of a patient

Contact with objects, such as needles, contaminated with infected secretions
Direct contact with a deceased infected person during burial ceremonies

**Signs and Symptoms**

- Incubation period is 2-21 days before onset of signs & symptoms
- EVD is a severe acute viral illness often characterized by sudden onset of fever and accompanied by fatigue, muscle pain, headache and sore throat.
- General: (0-3 days)
- Fever, headache, sore throat, chills, weakness, tiredness
- Gastrointestinal symptoms: (3-10 days)
- Vomiting, diarrhoea, abdominal pain,
- Severe symptoms (7-12 days)
- Severe diarrhoea and vomiting, bleeding

***Other signs of Ebola Virus Disease***

- Redness in the whites of the eyes
- Rash on the trunk
- Bleeding in 45% of cases (historically)
- Mild: nose bleed, bruising
- Severe: gastrointestinal bleeding, shock

***Ebola and infectivity***

- People infected with Ebola can only spread the virus to others once they have developed symptoms
- People with no or very mild symptoms (low-grade fever), level of virus is low and unlikely to pose a risk to others
- Once a person is unwell, all body fluids are infectious, with blood, vomit and diarrhoea being the most infectious
- Semen can remain infectious for up to three months after recovery

***Differential diagnosis***

- Malaria
- Typhoid fever
- Cholera
- Other viral hemorrhagic fevers (e.g., Lassa)
- Brucellosis

**Diagnosis**

- Confirmation is by: §
- Antibody tests (ELISA) :detects and measures antibodies in blood
- Antigen tests : Detect and measures antigens in the blood
- Serum neutralization test
- Reverse transcriptase polymerase chain reaction assay {RT-PCR}:It is a technique used in genetic studies that allows the detection mRNA
- Virus isolation by cell culture

**Management of patients**

- There are no approved treatments available for EVD
- Fluids to correct hypovolemia and electrolytes,
- Blood transfusion incase of hemorrhage
- maintenance of oxygenation through administration of o2,
- Pain control by administration of pain killers,
- Nutritional support
- Treating secondary bacterial infections and pre-existing comorbidities

**Preventive measures of Ebola**

- Isolate patients with Ebola from other patients
- Wear protective clothing such as masks,gloves.gowns and googles.
- Complete sterilization of equipments
- Practice careful hygiene. For example, wash hands with soap and water or an alcohol-based hand sanitizer and avoid contact with blood and body fluids (such as urine, feces, saliva, sweat, urine, vomit, breast milk, semen, and vaginal fluids).
- Do not handle items that may have come in contact with an infected person's blood or body fluids (such as clothes, bedding, needles, and medical equipment).
- Notify health officials if you have had direct contact with the blood or body fluids, such as but not limited to, feces, saliva, urine, vomit, and semen of a person who is sick with Ebola
- cont
- Avoid funeral or burial rituals that require handling the body of someone who has died from Ebola.

- Avoid contact with bats and nonhuman primates or blood, fluids, and raw meat prepared from these animals
- Avoid contact with semen from a man who has had Ebola until you know Ebola is gone from his semen.

## SEVERE ACUTE RESPIRATORY ILLNESSES

- Severe acute respiratory syndrome
- A severe respiratory viral infection
- Caused by coronavirus (SARS-CoV) a stable strain of virus that continues to be effective in faeces and urine at room temp for at least 1-2 days
- It loses infectivity after exposure to various disinfectants and fixatives
- The virus can be destroyed by heat above 56˚C

**Epidemiology**

- First recognized in 2003, originated in china
- Spread from person to person through:
- ✓ Droplets and fomites
- ✓ Mechanical vectors such as aerosolised sewerage
- Health workers are at greatest risk
- Transmission occurs during the second week of illness
- The majority of the affected group is the adults
- Incubation period is 2-10 days

**Clinical features**

- Sudden onset of fever, malaise, myalgia, followed by cough that may quickly progress to respiratory distress

**Diagnosis**

- Suspected if a patient presents with a history of fever and one of the following: cough, difficulty breathing, shortness of breath and gives a history of close contact with a case or suspect within 10 days prior to onset of symptoms, travel to affected areas, or residing in those areas i.e. transmission areas.
- Immunohistochemistry.
- Virus isolation
- PCR of blood or tissue specimen

**Management**

- Barrier nursing
- Wearing of PPE when dealing with the patient
- Strict isolation and putting a surgical mask on them
- Pulmonary support with oxygen and mechanical ventilation for respiratory failure
- Prevention and control
- Investigation should be conducted within24-48hrss to identify the source of infection incase of a suspected case.
- Barrier nursing of hospitalised patients
- Quarantine for contacts and follow up for 10 days until SARS has been ruled out
- Proper handling of all specimen by the lab workers
- Public health awareness and education
- Notification to the relevant authority

**SEVERE ACUTE RESPIRATORY SYNDROME (SARS)**

- **Infectious Agent** Severe Acute Respiratory Syndrome (SARS) is caused by a coronavirus similar
- on electron microscopy to animal coronaviruses. Coronaviruses are large,
- enveloped RNA viruses.

**Clinical Presentation**

- SARS illness generally presents with malaise, myalgia and fever, quickly
- followed by respiratory symptoms including cough and shortness of breath.
- Diarrhea may occur.
- Symptoms may worsen for several days coinciding with viraemia at 10 days
- after onset. Nearly all confirmed infected adult cases developed pneumonia or
- acute respiratory distress syndrome.
- **Diagnostics** Consult your local laboratory.
- **Treatment** Consult with physician.

**Pathogen**

- **Occurrence Worldwide:** First recognized in February 2003, the disease is thought to have
- originated in the Guangdong province of China, with emergence into human
- populations sometime in November 2002. By July 2003, major outbreaks had
- occurred at 6 sites, with disease occurrence reported in more than 20 additional
- sites throughout the world, following major airline routes. Most cases occurred
- in hospitals and among families and close contacts of hospital workers. There
- have been no cases of SARS identified since the 2003 outbreaks.
- **Canada:** The first case of SARS was detected in Toronto, which was the
- epicenter of the outbreak in Canada. In all, there were 251 cases of SARS
- diagnosed in Canada, mostly in the Toronto area; 43 patients died.

- **Nunavut:** No reported cases.
- **Reservoir** Unknown.
- **Transmission** SARS is transmitted from person-to-person by close contact (i.e. within 1 or 2
- meters); caring for, living with, or direct contact with infectious respiratory
- secretions or body fluids of a suspected, or confirmed case of SARS.
- The virus is thought to be transmitted most readily through respiratory droplets
- and possibly fomites (a surface or object contaminated with infectious droplets).
- **Incubation Period**
- 3–10 days.
- **Communicability** Not yet completely understood. Initial studies suggest that transmission does
- not occur before onset of clinical signs and symptoms, and that maximum
- period of communicability is less than 21 days.
- During the 2003 outbreak, health workers were at great risk of disease
- acquisition, especially when exposed to aerosol-generating procedures such as
- intubations. In 2003, health care workers served as an entry point of the
- disease into the community in North America.
- **Susceptibility**
- **and Resistance**
- Unknown but susceptibility is assumed to be universal. At present race and sex
- do not appear to alter susceptibility. Because of the small number of cases reported among children, it has not been possible to assess the influence of age. The clinical course appears to be much milder and shorter among cases

less than 12 years of age.

## MENINGITIS

This is a common complication of mumps. It presents with fever, headache, vomiting, neck rigidity and spinal rigidity. The condition resolves spontaneously

**Meningococcal Meningitis (Epidemic Meningitis)**

- ✓ This is an acute and dangerous bacterial disease, which occurs sporadically and in epidemics.
- ✓ The causative bacterium is the Neisseria meningitides, also known as meningococcal

**There are two types of meningitis.**

- ✓ The first type known as **meningococcal meningitis** is spread by droplets from one person to another and may cause epidemics in crowded institutions such as army barracks, boarding schools, prisons and camps.
- ✓ The second type is caused by a variety of other organisms usually occurring as a complication of some other disease in the body, or by direct extension from neighboring structures such as the middle ear (otitis media).
- ✓ This type of meningitis occurs one case at a time, that is, it is sporadic.

**Mode of Transmission**

- ✓ About 20 - 25% of people may be healthy carriers of the meningococcal and the other organisms which cause meningitis, such as, *haemophilus influenzae* type B and *streptococcus pneumonia* (pneumococci).
- ✓ Transmission of the *neisseria meningitides* occurs by direct contact and by droplets from nasal and throat discharges of infected persons.

**Clinical Features**

- ✓ When a susceptible host is infected the organism causes blood poisoning (septicaemia ) and pyogenic meningitis.
- ✓ The onset is sudden with the following signs and symptoms:
- ✓ Severe headache and neck
- ✓ Rigidity Fever and rigors
- ✓ Pain in the back and limbs Irritability and confusion
- ✓ Drowsiness and coma
- ✓ Positive Kernig's and Brudzinki's signs On lumbar puncture
- ✓ Cerebral Spinal Fluid (CSF) is under pressure and contains high levels of White Blood Cells (WBCs)
- ✓ Has raised protein and lowered glucose
- ✓ Petechial haemorrhages
- ✓ Circulatory collapse (Waterhouse- Friderichson syndrome)

**Diagnosis**

- ✓ The following tests are useful to confirm a diagnosis:
- ✓ Lumbar puncture

- ✓ Positive Kernig's sign
- ✓ Positive Brudzinki's 1 and 2 signs
- ✓ Blood culture for neisseria meningitides

**Management**

- ✓ The patient must be admitted in the hospital and antibiotic therapy started immediately. Either one of the following drugs can be given:
- ✓ IM crystalline penicillin (benzyl penicillin) six mega units stat followed by three mega units every six hours
- ✓ IM chloramphenicol 500mg every six hours

**Prevention and Control**

- ✓ The prevention of meningitis follows the same principles that were covered in the other airborne diseases. They include:
- ✓ Improvement in housing:
- ✓ adequate space
- ✓ good ventilation
- ✓ Proper disposal of respiratory secretions
- ✓ Health education to avoid overcrowding in poorly ventilated houses
- ✓ Proper disposal of respiratory secretions
- ✓ Health education to avoid overcrowding in poorly ventilated houses
- ✓ Isolation of the suspected patients
- ✓ Notifying the District Medical Officer of Health
- ✓ Immunization during epidemics using meningitis A and C vaccine
- ✓ Chemoprophylaxis (single dose of oral floxacillin 500mg or rifampicin) for all household and other
- ✓ Contacts of the patient including the health care workers
- ✓ Use of gowns, gloves and masks while caring for these patients

## CORONAVIRUS 2019 (COVID-19)

**Coronavirus 2019 (COVID-19)** is a disease caused by a new strain of coronavirus called *severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2)* that can cause symptoms from common cold to more severe disease such as pneumonia and eventually it may lead to death especially those in vulnerable groups such as the elderly, the very young, and people with an underlying chronic health condition.

- ✓ Limited information is available to characterize the spectrum of clinical illness associated with COVID-19.
- ✓ The CDC clinical criteria for a COVID-19 patient under investigation (PUI) have been developed based on what is known about MERS-CoV and SARS-CoV and are subject to change as additional information becomes available.
- ✓ Early on, many of the patients in the outbreak in Wuhan, China reportedly had some link to a large seafood and animal market, suggesting animal-to-person spread.
- ✓ However, a growing number of patients reportedly have not had exposure to animal markets, indicating person-to-person spread is occurring

**Pathophysiology**

- ✓ Coronaviruses are common in many different species of animals, including bats, camels, cats, and cattle
- ✓ COVID-19 is a betacoronavirus, like MERS and SARS, all of which have their origins in bats.
- ✓ The sequences from US patients are similar to the one that China initially posted, suggesting a likely single, recent emergence of this virus from an animal reservoir.
- ✓ When person-to-person spread has occurred with MERS and SARS, it is thought to have happened mainly via respiratory droplets produced when an infected person sneezes, similar to how influenza and other respiratory pathogens spread
- ✓ Most coronaviruses infect animals, but not people; in the future, one or more of these other coronaviruses could potentially evolve and spread to humans, as has happened in the past.
- ✓ Many of the patients have direct or indirect contact with the Wuhan Huanan Seafood Wholesale Market that is believed to be the original place of the outbreak of COVID-19.
- ✓ However, the transmission of COVID-19 from fish to humans is unlikely.
- ✓ The COVID-19 and fish coronaviruses such as Beluga Whale CoV/SW1 belong to different genera and apparently have different host ranges.
- ✓ As the Wuhan market seafood market also sells other animals, the natural host of COVID-19 awaits to be identified.
- ✓ Due to the possibility of transmission from animal to human, CoVs in livestock and other animals

including bats and wild animals sold on the market should be constantly monitored.

- ✓ In addition, more and more evidence indicates the new virus COVID-19 is spread via the route of human-to-human transmission because there are infections of people who did not visit Wuhan but had close contact with family members who had visited Wuhan and got infected.

**Causes**

Coronaviruses are named for the crown-like spikes on their surface.

There are four main sub-groupings of coronaviruses, known as alpha, beta, gamma, and delta.

Human coronaviruses were first identified in the mid-1960s.

The seven coronaviruses that can infect people are 229E (alpha coronavirus), NL63 (alpha coronavirus, OC43 (beta coronavirus), and HKU1 (beta coronavirus).

Other human coronaviruses are MERS-CoV, SARS-CoV, and COVID-19

**Statistics and Incidences**

An outbreak of pneumonia of unknown etiology in Wuhan City was initially reported to WHO on December 31, 2019

Chinese health authorities have confirmed more than 40 infections with a novel coronavirus as the cause of the outbreak.

Reportedly, most patients had epidemiological links to a large seafood and animal market; the market was closed on January 1, 2020.

Globally, there are 597,250 confirmed cases and 27,365 deaths confirmed as of March 28, 2020

**Clinical Manifestations**

- ✓ For confirmed COVID-19 infections, reported illnesses have ranged from people being mildly sick to people being severely ill and dying; these symptoms may appear in as few as 2 days or as long as 14 after exposure based on what has been seen previously as the incubation period of MERS viruses.
- ✓ Fever
- ✓ Dry cough
- ✓ Shortness of breath
- ✓ Sore throat
- ✓ Runny nose
- ✓ Diarrhea
- ✓ Fatigue/tiredness
- ✓ Difficulty of breathing (in severe cases)

**Assessment and Diagnostic Findings**

- ✓ To increase the likelihood of detecting infection, CDC recommends collection of three specimen types: *lower respiratory, upper respiratory, and serum specimens* for testing

**Medical Management**

- ✓ The best way to prevent infection is to avoid being exposed to this coronavirus.
- ✓ **Hand hygiene.** Wash hands often with soap and water for at least 20 seconds; if water and soap are not available, use an alcohol-based hand sanitizer.
- ✓ **Keep hands off your face.** Avoid touching the eyes, nose, and mouth with unwashed hands.
- ✓ **Maintain social distancing.** Avoid close contact with people at least 3 feet (1 meter) who are sick, and stay at home when you are sick.
- ✓ **Proper cough and sneeze etiquette.** Cover your cough or sneeze with a tissue, then throw the tissue in the trash.
- ✓ **Supportive care.** People infected with COVID-19 should receive supportive care to help relieve symptoms.
- ✓ **Severe cases.** For severe cases, treatment should include care to support vital organ functions.

**For Healthcare Workers**

- ✓ Healthcare workers are the very people who will be working day-and-night to treat and assist coronavirus patients are among the most exposed population for becoming infected.
- ✓ The protection of vulnerable members is one of the priorities for the response to COVID19 outbreaks.
- ✓ Occupational health services in healthcare facilities play a vital role in helping, supporting, and ensuring that workplaces are safe and healthy and addressing health problems when they arise.
- ✓ WHO emphasizes the rights and responsibilities of health workers, including explicit criteria required to preserve occupational safety and health.

**Health worker rights include that employers and managers in health facilities:**

- ✓ Assume overall responsibility to ensure that all necessary preventive and protective measures are taken to minimize occupational safety and health risks.
- ✓ Provide information, instruction, and training on occupational safety and health, including;
- Refresher training on infection prevention and control (IPC)
- Use, putting on, taking off and disposal of personal protective equipment (PPE).

- ✓ Provide adequate IPC and PPE supplies (masks, gloves, goggles, gowns, hand sanitizer, soap and water, cleaning supplies) in sufficient quantity to healthcare or other staff caring for suspected or confirmed COVID-19 patients, such that workers do not incur expenses for occupational safety and health requirements.
- ✓ Familiarize personnel with technical updates on COVID-19 and provide appropriate tools to assess, triage, test and treat patients and to share infection prevention and control information with patients and the public.
- ✓ As needed, provide appropriate security measures for personal safety.
- ✓ Provide a blame-free environment for workers to report on incidents, such as exposures to blood or bodily fluids from the respiratory system or to cases of violence, and to adopt measures for immediate followup, including support to victims.
- ✓ Advise workers on self-assessment, symptom reporting and staying home when ill.
- ✓ Maintain appropriate working hours with breaks.
- ✓ Consult with health workers on occupational safety and health aspects of their work and notify the labor inspectorate of cases of occupational diseases.
- ✓ Not be required to return to a work situation where there is continuing or serious danger to life or health, until the employer has taken any necessary remedial action.
- ✓ Allow workers to exercise the right to remove themselves from a work situation that they have reasonable justification to believe presents an imminent and serious danger to their life or health. When a health worker exercises this right, they shall be protected from any undue consequences.
- ✓ Honor the right to compensation, rehabilitation, and curative services if infected with COVID-19 following exposure in the workplace. This would be considered occupational exposure and resulting illness would be considered an occupational disease.
- ✓ Provide access to mental health and counseling resources.
- ✓ Enable co-operation between management and workers and/or their representatives

**Nursing Management**

- ✓ Nursing management for patients with COVID-19 infection include the following:
- ✓ Assessment of a patient suspected of COVID-19 should include:
- ✓ **Travel history.** Health care providers should obtain a detailed travel history for patients being evaluated with fever and acute respiratory illness.
- ✓ **Physical examination.** Patients who have fever, cough, and shortness of breath and who has traveled to Wuhan, China recently must be placed under isolation immediately

**Nursing Diagnosis**

- ✓ Based on the assessment data, the major nursing diagnosis for a patient with COVID-19 are:
- ✓ **Infection** related to failure to avoid pathogen secondary to exposure to COVID-19.
- ✓ **Deficient knowledge** related to unfamiliarity with disease transmission information.
- ✓ **Hyperthermia** related to increase in metabolic rate.
- ✓ **Impaired breathing pattern** related to shortness of breath.
- ✓ **Anxiety** related to unknown etiology of the disease.
- ✓ **Nursing Care Planning and Goals**
- ✓ The following are the major nursing care planning goals for COVID-19:
- ✓ Prevent the spread of infection.
- ✓ Learn more about the disease and its management.
- ✓ Improve body temperature levels.
- ✓ Restore breathing pattern back to normal.
- ✓ Reduce anxiety

**Nursing Interventions**

- ✓ Listed below are the nursing interventions for a patient diagnosed with COVID-19:
- ✓ **Monitor vital signs.** Monitor the patient's temperature; the infection usually begins with a high temperature; monitor the respiratory rate of the patient as shortness of breath is another common symptom.
- ✓ **Monitor O2 saturation.** Monitor the patient's O2 saturation because respiratory compromise results in hypoxia
- ✓ **Maintain respiratory isolation.** Keep tissues at the patient's bedside; dispose secretions properly; intsruct the patient to cover mouth when coughing or sneezing; use masks, and advise those entering the room to wear masks as well; place respiratory stickers on chart, linens, and so on.
- ✓ **Enforce strict hand hygiene.** Teach the patient and folks to wash hands after coughing to reduce or prevent the transmission of the virus.

- ✓ **Manage hyperthermia.** Use appropriate therapy for elevated temperature to maintain normothermia and reduce metabolic needs.
- ✓ **Educate the patient and folks.** Provide information on disease transmission, diagnostic testing, disease process, complications, and protection from the virus.

**Evaluation**

- ✓ Nursing goals are met as evidenced by:
- ✓ Patient was able to prevent the spread of infection.
- ✓ Patient was able to learn more about the disease and its management.
- ✓ Patient was able to improve body temperature levels.
- ✓ Patient was able to restore breathing pattern back to normal.
- ✓ Patient was able to reduce anxiety.

**Documentation Guidelines**

- ✓ Documentation guidelines for a patient with COVID-19 include the following:
- ✓ Individual findings, including factors affecting, interactions, nature of social exchanges, specifics of individual behavior
- ✓ Cultural and religious beliefs, and expectations.
- ✓ Plan of care.
- ✓ Teaching plan.
- ✓ Responses to interventions, teaching, and actions performed.
- ✓ Attainment or progress toward the desired outcome

# EPIDEMIOLOGY

## CONCEPTS IN EPIDEMIOLOGY

of **Epidemiology**: it is the study of occurrence, distribution and determinants of health and illness (or health related statuses) in a population and the application of this study in the control health problems.
Prevalence: refers specifically to all current cases (old and new) existing at a given time in a given population. It can either be point prevalence and period prevalence.
**Point prevalence**: it is the total number of cases (old and new) of a disease known to have existed at some point in time (a specific point)
**Period prevalence**: it is the total number of cases of a disease known to have existed during a specified period e.g., Jan 2018- Dec 2018.
**Incidence**: the number of new cases occurring in a defined population during a specified period of time
**Epidemic**: refers to the occurrence of a disease in a community or region in excess of normal expectancy.
**Endemic**: the constant occurrence of a disease or infectious agent in a given geographical area or population group.
**Pandemic**: an epidemic that usually affects a large proportion of the population occurring over a wide geographical area. It goes beyond boundaries.
**Host**: a person or an animal that gives subsistence or harbors an infectious agent under natural conditions.

**The types of hosts include:**

1. Obligate
2. Definitive/primary
3. Intermediate
4. Transport hosts.

**Susceptible host**: a person or animal lacking sufficient resistance to a particular pathogenic agent to prevent disease if or when exposed
**Agent**: these are the organisms which are usually responsible for the occurrence of infections and infectious diseases. They include ***viruses***, ***bacteria***, ***fungi***, ***helminthes***, ***protozoa*** and ***anthropoids***.
**Source**: this is the site from which a pathogen is transmitted to a susceptible host either directly or indirectly through an intermediary object.
**Reservoir**: the environment in which a pathogen lives and multiplies. Reservoirs include man, animals and non-living things.
**Environment**: refers to extrinsic factors that affect the agent and the opportunity for exposure.
Environmental factors include

**a) Physical Factors**

Geology and climate

**b) Biologic Factors**

Insects that transmit the agent, and

**c) Socioeconomic Factor**s

Crowding, sanitation, and the availability of health services.

**Infection**: the successful entrance and the replication of organisms in host tissue and may result in causing disease.
**Disease**: a stage of dysfunction which is subjectively or objectively apparent.
**Carrier**: an individual with no overt disease who habours the infectious micro organisms in the sub-clinical stage for a long time.
**Risk factor**: a disease factor that may cause a person to acquire an infection or disease.

## SCOPE OF EPIDEMIOLOGY

Epidemiology is divided into

1. Descriptive epidemiology.
2. Analytical epidemiology.

### Descriptive epidemiology

- Descriptive epidemiology ***uses existing data*** to compare how mortality or morbidity may vary among certain groups.
- It describes the distribution of a disease in terms of **place**, **person** and **time** i.e. where, who and when.
- Descriptive epidemiology is used in formulation of hypothesis.

**The essential characteristics of disease that we look for are as follows:**

1. **Person**: who is getting the disease? age, gender, ethnic group, genetic predisposition, diet, physical activity, smoking, concurrent disease, risk taking behaviour, education, SES, occupation etc
2. **Place (geographic):** where are the rates of the disease highest or lowest? Presence of agents or vectors, climate, geology, population density, economic development, nutritional practices, medical practices.

3. **Time**: when does the disease occur? Is the frequency of the disease now different from the corresponding frequency in the past? Calendar time, seasonality, time since an event, physiological cycles, age/time since birth, temporal trends.

## ANALYTICAL EPIDEMIOLOGY

- It is the branch of epidemiology that identifies ***aetiology*** or cause of disease, ***risk factors*** for disease ***causation*** and therefore the ***determinants*** of disease with the aim of developing preventive measures.
- It is more focused on the determinants of disease.
- It uses a comparison group to establish an association between risk factors and illness in the two groups.
- It analyses the problem by testing the causes and determinants.
- It is ***used in hypothesis testing***.

**The following studies are useful in analytical epidemiology:**

**A. Retrospective**

- Looking back at previous morbidity or mortality of a disease.

**Case-control**: compares the odds of past exposure to a suspected risk factor between cases.
Cases refer to diseased individuals while controls refer to non-diseased people.

**B. Prospective studies**

**a)** ***Longitudinal***: study of a population over a usually long period of time.

**b)** ***Cohort***: a group of health people are identified and followed for a specified period of time. Exposed and unexposed participants are compared in relation to the diseases incidence.

**c)** ***Cross-sectional studies***: a sample of (or total) population is examined at a given point in time. Takes a snapshot of a cohort.

**d)** ***Experimental***: establishes cause and effect relationships through control of variables

**i.** Clinical trials……assignment

**ii.** Field trials/community trials: they test or address preventive intervention. Subjects are allocated to receive the preventive measure or placebo. e.g vaccines to prevent polio…etc

## OBJECTIVES OF EPIDEMIOLOGY

**Epidemiology seeks to answer the following questions:**

1. What is the problem? Health related phenomena.
2. How big is the problem? How common is the health related phenomena.
3. Who has the problem? Sex, age, ethnic group etc
4. Where is the problem? Geographical place with the highest and lowest cases of the health related phenomena,
5. What causes the problem? Analytical epidemiology.

## USES OF EPIDEMIOLOGY

1. Detect changes of the patterns of diseases/ determine geographic patterns.
2. Discovery of new diseases.
3. To determine, describe and report on the natural course of disease, disability, injury and death.
4. To aid in planning and development of health services and programs.
5. To provide administrative and planning data.
6. To study the cause (or etiology) of diseases or conditions, disorders, disabilities etc.
7. To determine the primary agent responsible or ascertain causative factors.
8. To determine the characteristics of the agent or causative factors.
9. To determine mode of transmission.
10. To determine contributing factors.
11. To provide a basis for developing disease control and prevention measures for groups at risk-developing measures to prevent and control disease.

## DISEASE OCCURENCE

- Disease does not occur in a vacuum but occurs as a result of interaction of some factors.
- The ***epidemiologic triad*** or triangle has been proposed to explain disease causation.
- The triad consists of an ***external agent***, a ***susceptible host***, and an ***environment t***hat brings the host and agent together.
- These are commonly referred to as disease determinants.

After exposure to the infectious agent, whether an individual will be infected or not or will develop the disease or not depends on the following factors:

### Agent factors

- ***Infectivity/invasiveness***: ability of an agent to cause an infection to a susceptible host
- ***Pathogenicity***: ability of an agent to cause clinical disease to an infected host
- ***Virulence***: ability of an agent to cause severe or fatal conditions to an infected host.
- ***Potency***: how strong the exposure is i.e how large a "dose" of germs is.
- Agents susceptibility to drugs. Some have become resistant to available treatment e.g. TB
- Ability to adapt to change.
- Mode of transmission. Has the agent found the correct route of transmission.

### Host factors

- Age, sex, anatomic structure
- Genetic make up
- SES, poverty
- Nutritional status
- Previous exposure
- Immunization status
- Education status
- Lifestyle and behavioural factors
- Psychological make up
- Occupation

### Environmental factors

- Shelter/housing
- Overcrowding
- Altitude
- Humidity
- Sanitation
- Food supply
- Water supply
- Climate change
- Occupational factors
- Essential services

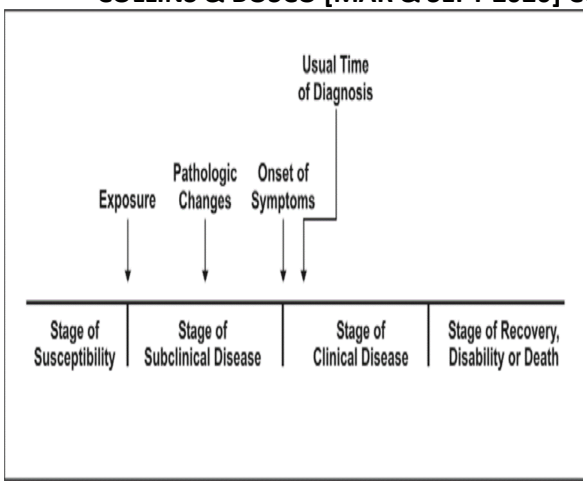
### DISEASE OCCURRENCE

1. Epidemics occur when the host, agent and environmental factors are not in balance. This can be as a result of:
2. Presence of a new agent. The recent introduction of the agent into a setting where it has not been before.
3. Change in exiting agent (infectivity, pathogenicity, virulence and increase in amount).
4. A change in the susceptibility of the host response to the agent.
5. Change in number of susceptible hosts in the population.
6. Environmental changes that affect transmission of the agent and growth of the agent which results in an enhanced mode of transmission so that more susceptible persons are exposed, and/or
7. Introduction of agent through new portals of entry.

### NATURAL HISTORY OF DISEASE

- Natural history of disease refers to the **progression of a disease process in an individual over time, in the absence of treatment.**
- Most, diseases have a characteristic natural history, although the time frame and specific manifestations of disease may vary from individual to individual and are influenced by preventive and therapeutic measures.
- The process begins with the ***appropriate exposure to infectious agents sufficient for the disease process to begin in a susceptible host.***
- After the disease process has been triggered, ***pathological changes then occur without the individual being aware of them.*** This stage is called the **subclinical disease**. It is the stage of disease unaccompanied by signs or symptoms and may only be detected by lab investigations.
- It extends from the time of exposure to onset of disease symptoms.
- It is usually called the ***incubation period for infectious diseases***, and ***latency period for chronic diseases.***
- The onset of symptoms marks the transition from subclinical to clinical disease.
- Clinical disease is disease accompanied by signs and symptoms. Most diagnoses are made during the stage of clinical disease.
- In some people, however, the disease process may never progress to clinically apparent illness.
- Such persons who are infectious but have subclinical disease are called **carriers**.
- ***Ultimately, the disease process ends either in recovery, disability or death.***

NATURAL HISTORY OF DISEASE




## SPECTRUM OF DISEASE

- The disease process may result in illness that ranges from mild to severe or fatal.
- This range is called the spectrum of disease.

## LEVELS OF DISEASE PREVENTION

- Disease prevention is defined as the ***postponement or removal of certain illnesses and conditions by use of interventions that have proved to be efficient.***
- The aim is to restore human health or to make the quality of life better.
- Prevention is always done in three levels; **primary**, **secondary** and **tertiary**.
- However, some epidemiologists usually talk about four levels where we have the **primordial level** right before the primary level.
- The line between these levels of prevention may not be as clear as implicated in theory.

## PRIMORDIAL PREVENTION

- At this level we try to do a **lot of *health promotion*** especially in campaigns to sensitize people to change behaviors or lifestyles that are considered dangerous.
- The outstanding effect here is that we will no longer need other preventive interventions.
- A good example may be that of promoting the use of no cholesterol food stuffs, food stuffs with added salt to prevent hypertension, another one may be the global campaign to eliminate smallpox so that we do not need the immunization anymore.

## PRIMARY PREVENTION

- Focuses on the ***prevention of disease before the biological onset***, thus inhibits the development of disease.
- For example getting measles immunization will prevent the clinical illness before it gets started.
- Other types of primary prevention are focused on the behavioral aspects of individuals.
- These would include quitting smoking, dental care by regularly brushing of teeth and maintaining a regular routine of exercise.

## SECONDARY PREVENTION

- At this level we prevent the clinical illness through the early detection.
- The individual has not started experiencing the symptoms associated with the disease.
- Early detection is done through screening and from there we can manage or even reverse the outcome of the condition.
- Examples of interventions done for secondary prevention are routine cultures for sexually transmitted micro-organisms in the individuals who don't show the symptoms, screening for hypertension, screening for breast cancer or colon cancer.
- Screening becomes useful especially when the disease in question is treatable.

## TERTIARY PREVENTION

- ***Focuses on stopping the disease from progressing further and inhibiting the development of new complications***.
- Mostly the physicians and other specialists who manage chronic conditions are involved.
- Because of this, it creates a thin line between the definition of treatment and tertiary prevention.
- We can have lowering of blood cholesterol after a heart attack to prevent the occurrence of further attacks and other complications like stroke and angina pectoris.
- Another example would be examinations for retinal disease brought about by diabetes and then putting steps to prevent its progression.

## MEASURING HEALTH AND DISEASE

- Common frequency measures are **ratios**, **proportions**, and **rates**.
- **Ratio** is the expression of a relationship between a numerator and a denominator where the two are distinct and separate quantities. The numerator is not included in the denominator.
- **Proportion** is the comparison of a part to the whole. The numerator is included in the denominator e.g. what fraction of patients tested positive for HIV.
- A proportion may be expressed as a decimal, a fraction, or a percentage.
- **Rate** is a measure of the frequency with which an event occurs in a defined population over a specified period of time.
- Usually, rate means how fast something is happening or going.
- Rate is always reported per some unit of time.
- For example, when we say 70 new cases of breast cancer per 1,000 women per year, the measure conveys a sense of the speed with which disease occurs in a population, and seems to imply that this pattern has occurred and will continue to occur for the foreseeable future. This rate is an *incidence rate*.
- Other epidemiologists use the term rate more loosely, referring to proportions with case counts in the numerator and size of population in the denominator as rates.
- For example, an attack rate is the proportion of the population that develops illness during an outbreak. For example, 20 of 130 persons developed diarrhea after attending a picnic. An alternative and more accurate phrase for attack rate is incidence proportion.
- A prevalence rate is the proportion of the population that has a health condition at a point in time. For example, 70 influenza case-patients in March 2005 reported in County X.
- A case-fatality rate is the proportion of persons with the disease who die from it. For example, one death due to meningitis reported among County X's population.
- All of these measures are proportions, and none is expressed per units of time.
- Therefore, these measures are not considered "true" rates by some, although use of the terminology is widespread.
- Ideally, a rate is a proportion in which change over time is considered, but in practice, often used interchangeably with proportion, without reference to time.
- Rates tell us how fast the disease is occurring in the population. Proportions tell us the fraction of the population affected.
- Morbidity has been defined as any departure from a state of physiological or psychological well-being. In practice, morbidity encompasses disease, injury, and disability. Measures of morbidity include incidence and prevalence.
- Mortality rate is a measure of the frequency of occurrence of death in a defined population during a specified interval. Morbidity and mortality measures are often the same mathematically; it's just a matter of what you choose to measure, illness or death.

**Assignment: what are the numerators and the denominators of the measures below?**

Incidence rate
Prevalence rate
Case fatality rate
Cause specific mortality rate
Death rate
Infant mortality rate
Neonatal mortality rate
Maternal mortality rate
Population at risk

***EXERCISES***

***In the United States in 2003, a total of 2,419,921 deaths occurred. The estimated population was 290,809,777. Calculate the crude mortality rate in 2003.***

***In the United States in 2003, a total of 108,256 deaths were attributed to accidents (unintentional injuries). The estimated population was 290,809,777 calculate the cause-specific mortality rate for accidents.***

***In a survey of 1,150 women who gave birth in Maine in 2000, a total of 468 reported taking a multivitamin at least 4 times a week during the month before becoming pregnant. Calculate the prevalence of frequent multivitamin use in this group.***

***In 2003, 44,232 new cases of acquired immunodeficiency syndrome (AIDS) were reported in the United States. The estimated mid-year population of the U.S. in 2003 was approximately 290,809,777. Calculate the incidence rate of AIDS in 2003.***

## INTEGRATED DISEASE SURVEILLANCE AND RESPONSE (IDSR)

- Surveillance in the context of public health means: *the **continued watchfulness over the distribution and trends of incidence [of a disease] through the systematic collection, consolidation, and evaluation of morbidity and mortality reports and other relevant data.***
- Disease Surveillance is both applicable to communicable and non-communicable diseases and injuries.
- The objectives of public health surveillance is ***to use data to monitor health problems*** to facilitate their prevention or control.
- For example, identifying geographic areas or populations with higher rates of disease can be helpful in planning control programs and targeting interventions, and monitoring the temporal trend of the rate of disease after implementation of control efforts.
- Surveillance is keeping over watch to get information for action or its ongoing systematic collection, analysis and interpretation of health related data essential to the planning, implementation and evaluation of public health practice, closely integrated with the timely dissemination of these data to those responsible for prevention and control.
- Kenya has a functional disease surveillance system essential for defining health problems which done by the IDSR under the guidance of the WHO/MOH Kenya.
- Clinicians are the first hand level health workers to come in to contact with the patient after the community. Thus they are actively involved in surveillance for they have the role.

***Note: surveillance only provides and interprets data to facilitate the prevention and control of disease.***

### Generally, the steps followed in disease surveillance are as follows:

1. **Identify, define, and measure the health problem of interest**:

What is the health importance of the problem? Ability to prevent, control or treat the problem? Capacity of health system to implement control measures for the health problem. Make a case definition criteria and define the scope of surveillance.

2. **Collect and compile data about the problem (and if possible, factors that influence it)**.

***Sources of data that can be used are:***

i. Morbidity and mortality reports from heath facilities and community health workers.
ii. Lab reports on isolation and identification of infectious agents.
iii. Reported deaths and from central death registers, health workers, community leaders etc. especially in long standing relief programs or post emergency phases.
iv. Reports on water supply, sanitation, vector control, shelter, food distribution etc. from health related services.
v. Rumours or reports of disease outbreaks from community leaders, school teachers, volunteers and field supervisors.

3. **Analyze and interpret these data.**
4. **Provide these data and their interpretation to those responsible for controlling the health problem**:

- They could start on outbreak investigation and uncovering of outbreaks.
- The team doing outbreak investigation can be composed of: Disease surveillance coordinator, Clinician, Medical laboratory technologist, Veterinarian, Microbiologist, Entomologist, Public health officer, District/ Local administration nominee, Computer specialist, Other personnel as required (health workers)

5. **Deciding Whether to Investigate a Possible Outbreak**

- Severity of illness
- Number of cases
- Source / mode of transmission
- Availability of preventive & control measures
- Availability of staff & resources
- Public, political and legal concerns
- Public health program considerations
- Potential to affect others if the control measures are not taken.

6. **Monitor and periodically evaluate the usefulness and quality of surveillance to improve it for future use.**

## The following tools are used in IDSR:

**Line list**: is like a nominal roll of the cases being reported to the various health care establishments (like dispensaries, general practitioners or admitted to the hospitals)

**Epidemiological case sheet/Case interview form**: Detailed information from the case relevant to the disease under study. The information includes:

- Name, Age, Sex, Occupation, Social class
- Time of onset of disease, Signs & Symptoms
- Personal contact at home, work, school
- Travel history, attendance at large gatherings
- History of previous exposure/injections
- Special events such as parties attended, foods eaten, and exposure to common vehicles such as water, food and milk.

**Kenya has identified 18 communicable disease and conditions in IDSR**

A. **Top cause of high morbidity and mortality in Kenya** e.g malaria, pneumonia, diarrheal disease, tuberculosis, HIV/AIDS
B. **Epidemic potential** e.g cholera, measles, yellow fever, meningococcal meningitis
C. **Surveillance required intervention** e.g polio, yellow fever, cholera
D. **National intervention programme for prevention and control, eradication or elimination of the disease** e.g polio, NNT

- Easily to be identified using simple case definitions

## The role of a clinician in IDSR includes:

1. Detect cases of priority disease by use of the standard case definition
2. Report the cases to the next level
3. Collect or send specimen to the laboratory including KEMRI in Nairobi
4. Institute appropriate management of cases using recommended management guidelines
5. Take appropriate preventive measures

## CATEGORY OF IDSR PRIORITY DISEASE (18) IN KENYA

**Epidemic prone disease**
cholera, dysentery, plague, yellow fever, typhoid fever, meningococcal meningitis, measles, viral hemorrhagic fever

**Disease earmarked for eradication/ elimination**
leprosy, poliomyelitis, dracunculiosis, neonatal tetanus

**Diseases of public health importance**
malaria, new HIV cases, TB, childhood pneumonia, childhood diarrhea, STI

**Other emerging infections**
like pandemic influenza A, H1NI, HPAI

**Kenya through the IDSR has also categorized the disease according to the duration of reporting as follows:**

- **Disease reported within 24 hours-** cholera, plague, yellow fever, typhoid fever, meningococcal meningitis, measles, viral hemorrhagic fever, poliomyelitis, guinea worm, neonatal tetanus (reported in MOH 502 surveillance form).
- **Disease reported weekly-** cholera, dysentery, plague, yellow fever, typhoid fever, meningococcal meningitis, measles, viral hemorrhagic fever, NNT, AFP and other suspected outbreaks
- **Diseases reported monthly-** at the end of the month all the 18 priority diseases are reported in MOH 504 surveillance form
- **Other disease of public health importance other than the 18 priority diseases that need reporting**;- new HIV cases, diarrhea with some/severe dehydration, severe pneumonia, unexplained SARD, TB and STI

## Some uses of public health surveillance

- Detect sudden changes in disease occurrence and distribution
- Monitor trends and patterns
- Portray the natural history of a disease
- Generate hypothesis, stimulate research
- Monitor changes in infection agents
- Detect changes in health practices
- Evaluate control measures
- Facilitate planning

### Surveillance link to action

- Outbreak investigation
- Disease control through vaccination, prophylaxis, elimination of cause, interruption of transmission
- Development, targeting of programmes through education and risk reduction
- Development of policies and regulation

### Types of surveillance in Kenya

1. Human surveillance
2. IDSR for the 18 priority diseases
3. Sentinel surveillance
4. Population based surveillance
5. Animal surveillance
6. Active surveillance- targeted and live bird market surveillance
7. Passive surveillance – reports from the county to the epidemic unit

#### Monitoring surveillance indicators

- Timeliness
- Completeness
- Complete report
- Surveillance performance index

### Challenges and constraints in surveillance

- Surveillance linkage to action is weak at lower levels due to lack of capacity for data analysis
- Weak reporting and communication system from health facilities to County/ Central surveillance unit
- Weak laboratory capacity and network
- Lack of quarantine and adequate isolation facilities
- Insufficient early warning systems for epidemics preparedness and response
- Little or non- involvement of the communities in surveillance
- Limited financial resources to strengthen surveillance and to support epidemic response

### Ways of supporting and improving surveillance

- Capacity building of the clinicians and the laboratory personnel including the surveillance team
- Having an active surveillance team in every sub county level
- Improvement of data and information through innovative approaches in ICT e.g phone
- Ensuring adequate supplies, logistics and commodities
- e.g for case management and laboratory reagents
- Development of more equipped and established laboratory
- Having a kit ready for epidemic rapid response
- Having a team that can do finance mobilization and soliciting of resources for response
- Provision of infection prevention and control measures
- Provision of support in implementation of international health regulation

### Summary

The government of Kenya does surveillance mostly over infectious agents as categorized below:

**FOODBORNE** – botulism, food intoxication, norovirus, salmonellosis, staphylococcal disease
**WATER BORNE** – amoebiasis, cholera, dracunculiasis, Giardiasis, Lgionellosis, Shigellosis, E. Coli
**VACCINE PREVENTABLE** – chicken pox, diphtheria, measles, mumps, influenza, pertussis
**STI**- genital herpes, chlamydial infections, gonorrhea, HPV, HIV/AIDS, syphilis, trichomoniasis
**PERSON TO PERSON** – aseptic meningitis, group A, TB, Viral hepatitis, leprosy, herpes simplex
**ARTHROPOD BORNE**–malaria, dengue fever, lyme disease, plague, viral encephalitis, westnile virus
**ZOONOTIC** – anthrax, brucellosis, rabies, tularemia, Q-fever, psittacosis, leptospirosis
**FUNGAL** – aspergillosis, blastomycosis, candidiasis, cryptococcosis, histoplasmosis, tenia pedis

## CLASSIFICATION OF EPIDEMIOLOGICAL STUDY METHODS /DESIGNS

Classification of Epi study methods
Are strategies used to find out the causative factors of diseases and include:

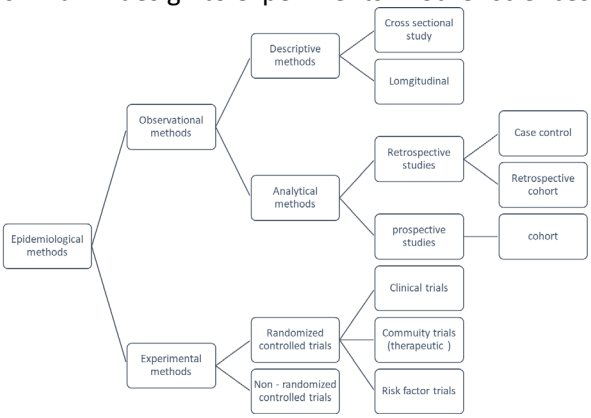
- Observational methods
- Experimental methods

1. **Observational methods**

- Observational studies allow nature to take its course.
- The investigator measures but does not intervene.

2. **Experimental methods**

- Active involvement to change disease determinants.
- Such as an exposure or a behaviour – or the progress of a disease through treatment.
- Are similar in design to experiments in other sciences.




Types of Epidemiologic study designs

| Type of study | Alternative name | Unit of study |
|---|---|---|
| ***Observational studies*** | | |
| Descriptive studies | | |
| Analytical studies | | |
| Ecological | Correlational | Populations |
| Cross-sectional | Prevalence | Individuals |
| Case-control | Case-reference | Individuals |
| Cohort | Follow-up | Individuals |
| ***Experimental studies*** | ***Intervention studies*** | |
| Randomized controlled trials | Clinical trials | Individuals |
| Cluster randomized controlled trials | | Groups |
| Field trials | | |
| Community trials | Community intervention studies | Healthy people Communities |

## OBSERVATIONAL STUDY METHODS

Observations are made as the studies allow nature to take its own course.
Studies do not involve any intervention or experiment.
The investigator measures but does not intervene.
Observational study methods
**Selected Units of study**: individuals, groups
**Study Populations**: Cross-sectional, longitudinal
**Data collection timing**: prospectively, retrospectively, combination
**Data collection types**: primary, secondary data

Observational study methods include :

i. **Descriptive study methods**
ii. **Analytical study methods**

**Descriptive**
Used to formulate a certain hypothesis: small / large scale.
**Examples**: case-studies; cross-sectional studies

**Analytical**
Used to test hypotheses: small / large scale.
Examples: case-control, cross-sectional, cohort.

## Descriptive study designs

i) Descriptive epidemiology

- Is the **study** of the **frequency and distribution** of disease or health status within the population by person, place and time.
- A simple description of the health status of a community.
- It is often the **first step in an epidemiological investigation**.
- ***Describe***: who, when, where & how many
- Is limited to a description of the **occurrence** of a disease in a **population**
- It involves the determination of **incidence, prevalence and mortality** of diseases in populations according to characteristics such as age, sex, and geographical area.
  - In these studies, **data on causes and effects** in an individual are often **unknown.**
  - It studies **factors responsible for distribution** of health and disease in human population such as age, sex, social status, income, occupation, housing, social customs, habits.
  - It is the 1st phase of an investigation in which data are obtained from such sources as **vital records and vital statistics, census information**, **survey and disease reports**.
  - The data collected are then **presented as percentages or in the form of rates.**

***PURPOSE****: to provide statistical overview of the community health problem and give a clue about the aetiological factors involved*

**Uses of descriptive studies**

- Determination of distribution of disease according to person, place and time

- Delineation of syndrome as a disease entity
- Establishment of natural history of disease
- Evaluate the trends in health and make comparisons
- Health planning
- Identify problems to be studied by analytical methods = hypothesis

### Types of descriptive studies

**i)** Case series
**ii)** Case reports
**iii)** Prevalence / cross- sectional studies /surveys
**iv)** Ecological studies

### CASE REPORT

- Unit of study is a single person with a disease
- Limitation : based on experience of a single person
- Provides first clues in the identification of a disease or adverse effects of exposure

### Case-series: Clinical case series

**Clinical case-series**: is a **coherent and consecutive set of cases of a disease** (or similar problem) derived from either the practice of health care professionals or a defined health care setting, e.g. a hospital.
**Unit of study**: group of persons with a similar disease
A case-series is, **effectively, a register of cases**.
**Analyse cases together to learn about the disease**.
**Clinical case-series are of value in epidemiology for:**

**a)** Studying symptoms and signs
**b)** Creating case definitions
**c)** Clinical education, audit and research

- ***Case series***: Population based
- When a **clinical case-series** is complete for a defined geographical area for which the population is known, it is known as **a population based case-series** consisting of a **population register of cases**.
- Epidemiologically the most important case-series are **registers of serious diseases or deaths (usually NCDs),** and of **health service utilisation, e.g. hospital admissions.**
- Usually compiled for administrative and legal reasons.
- Case series: Natural history and spectrum
- By delving into the past circumstances of these patients, including examination of past medical records, and by continuing to observe them to death (and necropsy as appropriate), health professionals can build up a **picture of the natural history of a disease.**
- Population case-series is a systematic extension of this series but which includes additional cases, e.g. **those dying without being seen by the clinicians.**
- Add breadth to the **understanding of the spectrum and natural history of disease.**

***Case series***: Requirements for interpretation
To make sense of case-series data the key requirements are:

1) The diagnosis (**case definition)** or, for mortality, the cause of death
2) The date when the disease or death occurred (**time)**
3) The place where the person lived, worked etc **(place)**
4) The characteristics of the person **(person)**
5) The opportunity to collect additional data from medical records (possibly by electronic data linkage) or the person directly
6) The size and characteristics of the **population at risk**

### PREVALENCE / CROSS- SECTIONAL STUDY

Measures **point prevalence** of disease or a health related event
Information about exposure and outcome are obtained simultaneously in a well –defined population.
In this study design information about the status of an individual with respect to presence/absence of exposure and diseased is assessed at a point in time.
**USES**

- Determination of prevalence of risk factors
- Determination of frequency of prevalent cases
- Determination of health status and health needs
- Formulation / generation of hypothesis rather than test

**Advantages**

- Quick and easy to perform;
- allows study of several diseases / exposures;
- useful for estimation of the population burden, health planning and priority setting of health problems

**Disadvantages**

- Temporal ambiguity (cannot determine whether the exposure preceded outcome);
- Not suitable for rare conditions;
- Liable to survivor bias

**Effect measure**:

- **Odds Ratio**

## ECOLOGICAL STUDIES

These are studies where **exposure data relating to a place** (e.g. hardness of water, which could be collected on individuals) are **correlated with health data collected on individuals** but summarized by place (e.g. Coronary Heart Disease (CHD) rates).

- Crude way of exploring relationship between environment or occupation and disease
- Unit of study is populations or groups of people rather than individuals
- Generates hypothesis

**Advantages**

- Simple to conduct

**Disadvantages**

- Individual link between exposure and effect cannot be made (ecologic fallacy)

**Ecological fallacy example**

Imagine a study of the **rate of coronary heart disease** in the capital **cities of the world relating the rate to average income**.

Within the cities studied, **coronary heart disease is higher in the richer cities than in the poorer ones.**

We might predict from such a finding that **being rich increases your risk of heart disease.**

In the **industrialised world** the opposite is the case - within cities such as London, Washington and Stockholm, **poor people have higher CHD rates than rich ones.**

The **ecological fallacy is** usually interpreted as a **major weakness of ecological analyses.**

Ecological analyses, however, **informs us about forces which act on whole populations.**

**Steps in Descriptive study methods**

1) Define the population to be studied
2) Define the disease under study
3) Describe the disease in relation to when, where and who
4) Measurement of disease
5) Comparing with known indices.
6) Formulation of aetiological hypothesis

### Steps in descriptive studies

### STEP 1: DEFINE THE POPULATION

Done in terms of numbers, composition (age, sex, occupation and cultural characteristics). The defined population can be:

- Whole population
- Sample population
- A selected group / workers
  - Hospital patients
  - School children
  - Elderly
  - Females /Males
- Communities

**Criteria for selecting a defined population**

***Large enough*** – so that the conclusion drawn from the study is meaningful

***Stable community*** – without migration of people into or out of the community

***No migrant or visitor*** – should be included in the defined population

***Access to medical services*** – health facilities should be nearby to provide care to those in need

### Step 2: Define the disease

- The disease under study should be precise and valid.
- An operational definition helps to identify and measure the disease in the defined population.
- An operational definition of a disease is a particular criteria by which the disease is measured.( case definition)
- It can be classified as **suspected, probable or confirmed case definition.**

### Step 3: DESCRIBE the disease in relation to when, where and who

Once the population and the disease has been defined, describe the disease in terms of time, place and person.

**A. *Time distribution***

Describe the time pattern by answering these questions

- Whether the disease is occurring **season wise, monthly, weekly , yearly**
- Whether there is **periodic rise or fall in the occurrence** of disease
- Whether the disease **follows a consistent time trend**

**On the basis of time trend or fluctuation in disease occurrence, disease occurs as:**

1) Short term fluctuation
2) Periodic fluctuation
3) Long term fluctuation

### Short – term fluctuation (Irregular /Epidemic)

- Epidemics can be classified as :

a) **Common source epidemic**: occur due to exposure from one common source. This exposure can be for a brief period or prolonged period leading to two types of common source epidemics:

❖ **Single exposure epidemics**
❖ **Repeated exposure: This** repeated exposure is for prolonged periods which can be **continuous or intermittent.**

b) **Propagated epidemics**

### Periodic fluctuations (Seasonal trends)

The disease occurs either in season, months, days, weeks. These fluctuations are periodic and occur according to:

**Seasonal trend**: communicable diseases occur in particular seasons. The seasonal variations occur due to the following factors :

- Temperature
- Humidity
- Overcrowding
- Rainfall

**Cyclic trend**: disease spreads in cycles over days, weeks, months. E.g.

- Influenza pandemic occur at an intervals of 7 – 10 years, due to antigenic variations
- Automobile accidents occur more frequently over the weekends / holidays

### Long term fluctuations (Secular trends)

Is changes in the occurrence of disease over a long period of time which can be several years or decades. There can either be an increase or decrease in the occurrence of disease.

For example – an increase in non – communicable diseases and a decrease in childhood immunizable diseases like diphtheria, pertussis

**B. *Place distribution***

Helps to compare variations in disease pattern within and between countries.

Variations in disease distribution can be classified as :

- International variation
- National variation
- Rural – urban variation
- Local distribution

Describe a health event by place to **gain insight into the geographical extent of the problem.**

Analyzing data by place- ***get an idea of where the agent that causes a disease normally lives and multiplies, what may carry or transmit it, and how it spreads***.

If the occurrence of a disease is associated with a place, we can

– *Infer that factors that increase the risk of the disease are present either in the persons living there (**host factors**) or in the environment, or both*.

**C. *Person distribution***

Analysis of data by "person" has several person categories.

- **Inherent characteristics** of people (e.g. age, race, sex),
- **Their acquired characteristics** (immune or marital status),
- **Their activities** (occupation, leisure activities, use of medications/tobacco/drugs), or
- The **conditions under which they live** (socioeconomic status, access to medical care).

### Step 4: Measurement of disease

Rates and ratios are used to measure **death, disease and disability patterns**.

Research designs are used to obtain estimates of the magnitude of health and disease in populations.

The research designs are :

**Cross – sectional study designs :** there is a single examination of a cross section of the population at one point in time (snap shot). The results are then generalized to the whole population

**Longitudinal study designs:** the observations are repeated in the same population over a prolonged period of time by means of follow – up examinations

These categories determine **who is at greatest risk** of experiencing undesirable health condition

Age is *important since* ***almost every health-related event or state varies with age***.

- Factors that vary with age are : **susceptibility, opportunity for exposure, incubation period of the disease, and Physiologic response (which affects, among other things, disease development).**

**Sex.** males have higher rates of illness and death than females do for a wide range of diseases.

This sex-related difference is because of ***genetic, hormonal, anatomic, or other inherent differences between the sexes***.

These inherent differences affect their susceptibility or physiologic responses.

For example, ***premenopausal women have a lower risk of heart disease than men of the same age. This difference is attributed to higher estrogen levels in women.***

1. Step 5: Comparing with known indices.
   To arrive at clues to causative factors of disease, comparison is made between different populations and sub groups of the same population

### Step 6: Formulation of etiological hypothesis

Hypothesis can be formulated related to the disease etiology.

A hypothesis is a supposition which is arrived from observations or reflections.

**Criteria for formulating hypothesis should include:**

- Population
- Specific cause
- Expected outcome
- The amount of cause to effect
- Time period between the exposures to cause the effect

### ANALYTICAL STUDY DESIGNS

ANALYTIC EPIDEMIOLOGY

- It is the second phase of an investigation.
- Is concerned with the searching for the underlying causes.
- Answers two other major questions: ***how***? And ***why***? of a health related event
- Analyses the determinants of disease or health related event or reasons for low or high frequency in specific groups.

### PRINCIPAL USES OF ANALYTIC EPIDEMIOLOGY

- Community diagnosis
- Investigation of epidemics
- Determination of disease etiology
- Evaluation of community intervention and programs

**Purpose**: to uncover the source and mode of spread of disease.

Used to determine the multiple factors that caused the disease as agent, host and environment.

It focuses on the determinants for the relatively high or low frequency of disease in specific groups.

Hypothesis are tested by analytical methods.

***Methods of analytical study used for testing hypothesis include***

i. **Retrospective method - case control**
ii. **Prospective method – cohort studies**

### Retrospective method (case control studies)

- It is first approach to **testing causal hypothesis,** especially for rare disease
- Compares **cases and controls** with the regard to the **presence** of some elements in their past experiences.
- A group of people who are diagnosed to be having a particular health problem **(cases)** are compared with a group of people who are free from that particular problem **(control)** from a chosen population
- Both are questioned or their records are reviewed about the presence or absence of a suspected cause / risk factor in the past
- It is **retrospective** coz the investigator **looks backward at the history of the cause**. E.g. the **cause of lung cancer is smoking**.
- The investigator then **looks back at the history of smoking in both the groups.**
- Cases must be representative of all those with disease and clearly defined
- Controls must be representative of all those without the disease and come from same community or sources as the cases

**Has Three features-**

1. Before initiation of the study, the diseases must have occurred due to exposure to the infectious agent **(Both exposure and outcome (disease) have occurred).**
2. The study proceeds backward (effect to cause)
3. Has one control group which it uses to support or refuse a inference.

***Uses of case control***

- To test risk factors
- Preferred if disease is rare
- Preferred if several factors are associated with disease of interest

**Advantages of case control**

- More economical
- Smaller sample size required
- Suitable for rare diseases
- Suitable for diseases associated with multiple exposure

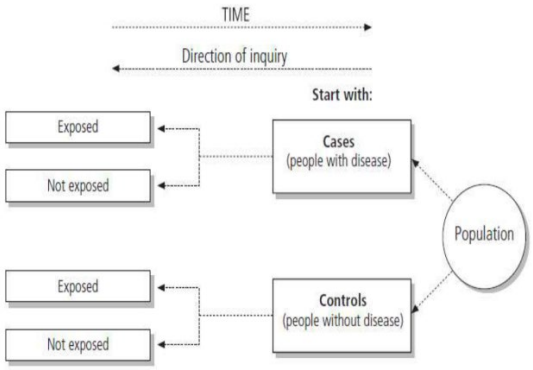
**Disadvantages of case control studies**

**1.** Not suitable for rare exposures;
**2.** liable to selection bias and recall bias;
**3.** not suitable for calculation of frequency measures.

**Effect measure**:

❖ Odds Ratio

**Design of a case control study**




Basic steps in Case-control study

**1.** Selection of cases and controls.
**2.** Matching.
**3.** Measurement of exposure.
**4.** Analysis and interpretation.

**1.** Selection of cases and controls

**Cases**

❖ Case definition – (Diagnostic criteria and Eligibility criteria.)
❖ Source of Cases – (Hospital or General population)

**Controls**

❖ Free from the disease under study.
❖ Similar to the cases in all other aspects.
❖ Source-
❖ Hospital, Relative, Neighbourhood, General population

**2. Matching**

- Is process of selecting controls in a manner that they are similar to cases in all variables.
- Matching is essential for comparability and for elimination of confounding bias.
- A Confounding factor is a factor which is associated with both exposure and disease and unequally distributed in study and control groups. **For example:**

**1.** Alcohol in esophageal cancer, smoking is confounding factor.
**2.** Age for steroid contraceptive as a causative factor in Breast cancer.
**3.** Measurement of Exposure

Information of exposure of risk factor should be obtain in same manner for both cases and controls.
Information obtain by-

a) Questionnaire.
b) Interviews.
c) Hospital records.
d) Employment records.

**Examples of case control studies**

- **Thalidomide Tragedy- A classic example of Case-control study**
- A classic example of a case-control study was the discovery of the relationship between **thalidomide and limb defects** in babies born in the Federal Republic of Germany in 1959 and 1960.
- The study, **done in 1961**, compared affected children with normal children.
- Of **46 mothers** whose babies had **malformations**, **41 had been given thalidomide** between the fourth and ninth weeks of pregnancy, whereas **none of the 300 control mothers**, whose children were normal, had taken the drug during pregnancy.
- Accurate timing of the drug intake was crucial for determining relevant exposure.

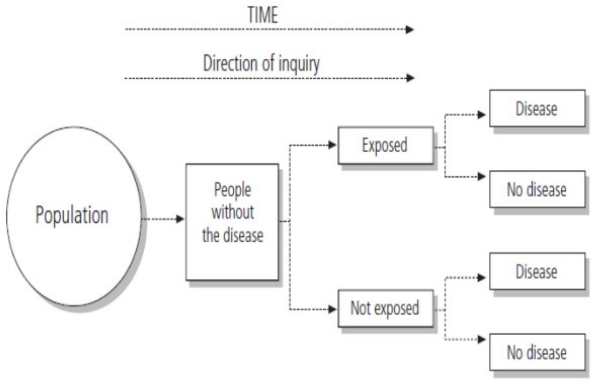
## Prospective methods

- A forward survey in which host, agent and environmental factors are studied.
- The study begins with a disease or condition and watches it over a period of time to see what develops.
- Tests hypothesis on causation of diseases.
- The investigator selects a group of people for study and gathers information about those who do and those who do not have the characteristics in question such that the cases belong to the experimental group while the non-cases belong to the control group.
- It helps make estimation of the risk of developing a particular condition in the presence of certain characteristics

## COHORT STUDIES

- **Cohort is group of people with similar characteristics.**
- Also called **follow-up** or **incidence studies.**
- Groups of subjects are chosen on the basis of having been exposed to a factor or not
- Begin with a group of people who are **free of disease.**
- Whole cohort is **followed up** to see the effect of exposure.
- Groups are followed up to identify those who develop the disease or outcome

## Study design of a cohort study




## Types of cohort studies

- On the basis of occurrence of disease in relation to time cohort studies can be divided into:
- **Prospective cohort studies (Current)–** starts from the present and continues into the future
- **Retrospective cohort studies (Historical)** – the outcome (disease) has occurred before the start of the investigation

**A combination of both** – a group is identified from past records and is assessed up to the present and followed up into the future for assessment of outcome.

**Steps in Cohort studies**

1. Selection of study subjects.
2. Obtaining data on exposure.
3. Selection of comparison group.
4. Follow-up.
5. Analysis.

## Selection of study subjects.

General population or
Special group (Doctors, Teachers, Lawyers).
Cohort should be selected from the group with special exposure under study.

## Obtaining data on exposure.

- Members of the cohort- questionnaire, interview.
- Review of records.
- Medical Examination or tests.
- Environmental surveys.

**Categorized according to exposure –**

a. Whether **exposed or not exposed** to special causal factor.
b. **Degree of exposure**
c. Selection of comparison group.

**A. Internal comparison.**

Subjects are categorized in group according to degree of exposure & mortality and morbidity compared.

**B. External comparison.**

When degree of exposure not known.
Control group with similar in other variable.

## Comparison with general population.

Comparison with the general population as exposed group

**Follow-up.**

Regular follow-up of all participants.
Measurement of variable depends upon outcome.
Procedure-
1. Periodical medical examination.
2. Review of hospital records.
3. Routine surveillance and death records.
4. Mailed questionnaire and phone calls.

**Analysis.**

Data are analyzed in terms of –

***i. Incidence rates.***

• Among exposed and non-exposed

***ii. Estimation of risk.***

• Relative Risk.
• Attributable Risk.

**Relative risk (Risk ratio) (RR)**

Is the ratio of the incidence of disease among exposed and incidence among non-exposed.

**Attributable Risk (Risk difference) (AR)**

AR is the difference in incidence rates of disease among exposed and non- exposed group.

Outline 8 differences between a case control study and a cohort study

| Case control study | Cohort study |
|---|---|
| 1. From ***effect to cause.*** | 1. From ***cause to effect.*** |
| 2. Starts with disease. | 2. Starts with people exposed to risk factors. |
| 3. Tests whether the suspected factor associated more with diseased. | 3. Tests whether disease occur more in those who exposed to risk factor. |
| 4. First approach to ***testing the hypothesis.*** | 4. Reserved for ***precisely formulated hypothesis.*** |
| 5. fewer no of subjects. | 5. Large no of subjects. |
| 6. Suitable for ***rare disease.*** | 6. Inappropriate when exposure is rare. |
| 7. Only estimates ***Odds ratio.*** | 7. YeildS ***IR, RR, AR.*** |
| 8. Relative inexpensive. | 8. Expensive. |

**EXPERIMENTAL EPIDEMIOLOGY**

The investigator intervenes and changes on variable and then observes what happens in the other.
It allows for control of variables, random allocation of subjects and elimination of bias on part of the experiment.
Are used to **confirm aetiological hypothesis** and to **evaluate or assess the effectiveness of the therapeutic preventive measures** before applying them to the community.
**Interventional or experimental study** involves attempting to change a variable in subjects under study. This could **mean the elimination of a dietary factor thought to cause allergy, or testing a new treatment on a selected group of patients**.
The effects of an intervention are measured by comparing the outcome in the experimental group with that in a control group.

**Objectives of Experimental Studies**

1. To provide **'scientific proof'** for etiology of disease and risk factor which may allow **modification of occurrence** of disease.
2. To provide a **method of measurement** for effectiveness and efficiency of **therapeutic / preventive measure for disease**.
3. To provide **method to measurement** for the efficiency **health services** for prevention, control and treatment of disease.

**Key characteristics of experimental studies**

1. Randomization
2. Manipulation
3. Control

**Basic principles in experimental epidemiology**
**Basic principles in experimental epidemiology include:**

- **Random allocation** of subjects to the appropriate subgroups
- **Medical, ethical and moral issues** are observed while conducting experiments
- Ability to **generalize**
- **Double blindness** – neither the investigator nor the subjects receives the treatment and who receives the placebo

**Types of Experimental Studies**

1. Randomized Control Trials.
2. Field Trials & Community Trials.

**Types of experimental studies in epidemiology**

In epidemiology, there are two types of experimental studies:

**a) Prophylactic or clinical trials** (RCT)

Designed to prevent disease or conditions in which the efficacy of preventive of therapeutic agent or procedure is tested on individual subjects
e.g. **administration of BCG vaccine as prophylaxis for TB**

**b) Therapeutic or community trials** –

- ❖ Designed to treat established disease processes in which a group of individuals as a whole is used to determine the efficacy of a drug or procedure
- ❖ e.g. the **evaluation of fluorides in preventing dental carries**

Though similar to cohort in approach, experimental studies are carried out under the direct control of the investigator.

**The study requires the following:**
**Intervention or action** – is application or withdrawal of suspected case or changing variable in the causative chain in experimental group
**Observation** – observe changes occurring in the variable in the experimental group
**Comparison** – compare the findings of experimental group with the control group

- Randomized Control Trials (RCT)
- RCT is a **planned experiment** designed to asses the **efficacy of an intervention** in human beings by comparing the **effect of intervention** in a **study group** to **a control group**.
- The allocation of subjects to study or control is determined purely by chance **(randomization)**.
- For **new programme** or **new therapy** RCT is **best method of evaluation.**

– **Randomized controlled trial (RCT**) – involves the process of random allocation of the subjects to be studied.
– Randomization is done to eliminate bias and allow for comparison.
– The investigator has no control over the grouping of participants under study or control group.
– It is done after the participants have given consent and have qualified the criteria of the clinical trial

## Basic Steps in RCT

1. Drawing-up a protocol.
2. Selecting reference and experimental population.
3. Randomization.
4. Manipulation or Intervention.
5. Follow-up.
6. Assessment of outcome.

## Field trials

- Field trials, in contrast to clinical trials, involve people who are healthy but presumed to be at risk.
- **Data collection takes place "in the field**," usually among non-institutionalized people in the general population.
- Since the subjects are disease-free and the purpose is to prevent diseases.
- Community Trials /therapeutic trials
- In this form of experiment, the treatment groups are **communities** rather than individuals.
- It is designed to treat established disease process in which a group of individuals as a whole is used to determine the efficacy of a drug or procedure
- This is particularly appropriate for diseases that are influenced by **social conditions**, and for which **prevention efforts** target **group behaviour.**

**Examples of community trials**

1. Evaluation of fluorides in preventing dental carries
2. IDD and Iron deficiency Anaemia.
3. Fortification of food.
4. Use of Efavirenz in pregnancy

- **Non – randomized controlled trial** – used when due to some reasons (ethical, moral, administrative) , it is not possible to conduct RCT in human beings.
- There is no randomization and these trials are referred to as non – **experimental studies**