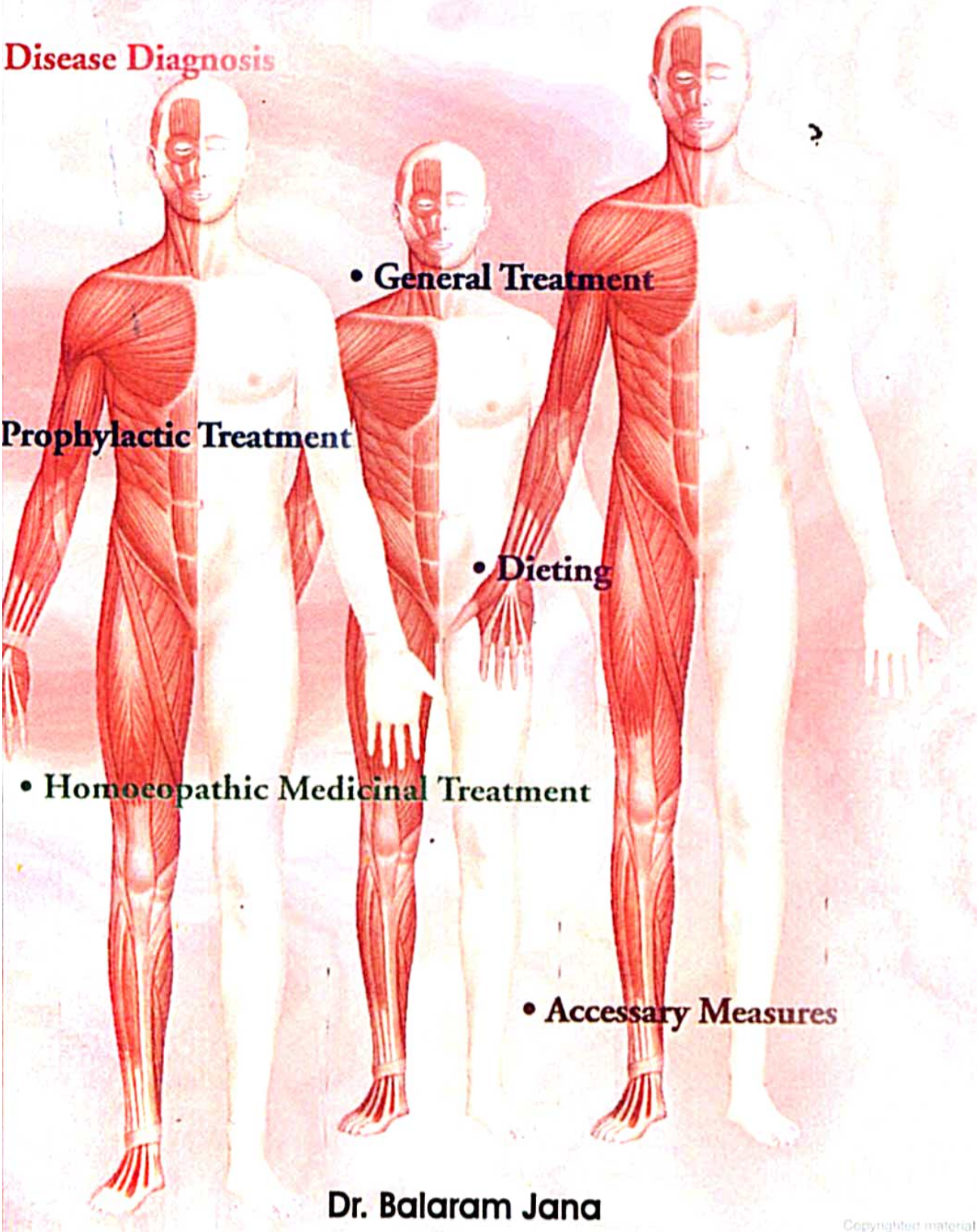
ESSENTIALS OF

PRACTICE OF MEDICINE



Dr. Balaram Jana

ESSENTIALS
OF
PRACTICE OF MEDICINE

BY

Dr. BALARAM JANA

M.Sc., D.M.S., COSIP (Cal.)
P.G.R. (Bombay), DOAST (Delhi)

Research Fellow—University College of Science, Calcutta.
Vice Principal—B.H. Medical College & Hospital
Examiner—Council of Homoeopathic Medicine, West Bengal.
Author—Preventive and Social Medicine, Human Pathology, Gynaecology & Obstetrics

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CARDIO-VASCULAR DISEASES

1. ENTERIC FEVER

• **What is it meant ?**

Enteric fever includes Typhoid and Para-typhoid fevers caused by *Salmonella Typhi* and *Paratyphi A.B.C.*

Enteric Fever is also a generalised septicemic infection with wide spread involvement of all the body tissues. It is not a mere intestinal lesion. So the term Enteric Fever is a misnomer, as the disease is generalised. It was named Enteric fever with the idea that it was primarily a disease of intestine, evidenced by—

(a) portal entry and exit of infecting bacilli being intestinal tract, and

(b) severe intestinal lesions like haemorrhage and perforation.

2. TYPHOID FEVER

• What is Typhoid fever? What are its clinical features ? How can we diagnosed a case of Typhoid fever in 1st week and 2nd week of illness in laboratory ? What are its complications? Describe its general management and medicinal treatment. What is meant by Typhoid state? Where it is found ? What are the cardinal signs of Typhoid Perforation ?

[**Nomenclature of Typhoid** — The word **TYPHUS** means **CLOUD.** Since the disease causes clouding of consciousness, it is called Typhoid.]

Def—Typhoid fever is an acute infectious disease caused by salmonella typhi, a flagellated Gram-negative rod; characterised clinically by intense frontal headache, prolonged high continued temperature having a step-ladder like rise during the first week and ending by lysis by the end of the third week, relative bradycardia during first week, stupor, typhoid rash, gurgling in the right iliac fossa and a tender enlarged spleen.

Infection occurs from human sources only, man being the sole reservoir, through contaminated food, water or through carriers via the alimentary tract.

Typhoid is a cosmopolitan disease, the incidence is maximum between the ages 5–40 years being slightly more common amongst the males, but no age or sex is immune. Immunity after one attack is only partial. Second attacks are not rare.

2.1. CLINICAL FEATURES:

A. THE FIRST WEEK (Stage of invasion or advance)

1. Onset:- The onset is gradual with malaise, anorexia, frontal headache, lethargy, rarely with epistaxis.

2. Symptoms:- For the first few days the patient may be fit to carry on his usual vocations; before the symptoms become severe between the 3rd and 7th days.

On 3rd and 7th days-There are intense frontal headache, constipation, scanty high coloured urine (which may contain trace of albumin), and slight cough due to bronchitis.

3. Signs

i) On examination the patient looks toxic, the face becomes anxious, the *tongue* is coated in the centre with margins.

ii) The abdomen may be distended and *caecal gurgling* is often present.

iii) The *pulse is dicortic* and *relatively slow*, the usual increase by 10 per degree rise of fever being not present (relative bradycardia).

iv) The *temperature* tends to assume *step ladder fashion* i.e., the evening rise is higher 1 °C than the next morning's fall.

v) *Leucopenia and Neutropenia* are *present* in the blood.

vi) *Blood culture* show B.typhosus in 95% cases.

vii) *Urine and stool* cultures are also positive i.e. show B. typhosus.

B. THE SECOND WEEK (stage of fastigium)

1. Symptoms:

i) The fever continues high i.e. the temperatur becomes continous and constant with a range of 103 °F to 105 ° F.

ii) The patient becomes dull, weak, stuporous and apathetic.

iii) The headache disappears because of the dulling of consciousness.

iv) The bowels become loose, 5-6 yellow or green '*pea-soup*' stools may pass without colic or tenesmus, blood being absent.

v) Cough aggravates due to secondary bronchitis and bronchopneumonia.

vi) Delirium may occur—Low, muttering delirium.

2. Signs

i) On examination, the patient looks ill and toxic.

ii) The ***temperature*** remains continuous or remittent by the end of this week 103° F to 105°F.

iii) The ***pulse*** is no more dicortic and becomes accelerated due to myocarditis.

iv) ***Sordes*** appear on the ***teeth,*** the ***tongue*** and ***lips*** become dry and cracked.

v) The ***abdomen*** becomes doughy tumid, tympanitic and tender; the liver and spleen are palpable and soft.

vi) Bronchitis and bronchopneumonia develop.

vii) The ***rose-spots*** (roseola), varing from 6-30 in number usually appear between the 7th-10th day or earlier. These are due to bacterial embolism. These are found in 10-20% of all cases and are ***Pinkish circular maculo papules*** measuring 2-4mm. in diameter.These appear in successive crops on the flank, abdomen and back and last 3.4 days after which these fade away leaving a brownish tint. This fades under pressure. ***Miscroscopically*** aggregation of the mononuclear cells around dilated capillaries of the skin is seen.

viii) ***Blood Examination***—(In the 2nd week) there are leucopenia (4,000/-c.mm), neutropenia (50%) relative lymphocytosis (38%) and monocytosis (12%).

ix) ***Blood culture*** show B.typhosus in 70% cases.

x) ***Widal test*** positive after 9–11 days.

xi) ***Urine and stool*** culture may show B.typhosus.

C. THE THIRD WEEK (stage of defervescence).

Symptoms

i) General symptoms remain more or less the same as in the second week.

ii) Loss of flesh and weakness more marked.

iii) In favourable cases, the temperature starts to fall by lysis on or about the 15th day, the toxaemia diminishes, the appetite returns, the abdominal symptoms subside and convalescence may continue since after.

iv) In *unfavourable cases* the temperature continues high and there may be hyperpyrexia. The patient becomes profoundly weak, toxic, and extremely apathetic and may pass into the **typhoid**

state—lying on the back in a semi-stuporous condition with low muttering delirium.

v) The patient is utterly helpless, lying with half closed eyes, coma vigil (stupor with delirium semiconsciousness with wakefulness), carphology (involuntary picking at the bed-clothes), subsultus tendinum (twisting movement of the muscles and tendons) and convalsions may be present.

2. Signs

i) The ***pulse*** becomes feeble, the ***tongue*** tremulous and dry, the gums ulcerated or bleeding, the ***lip*** dry.

ii) The abdomen is tympanitic; the liver and spleen recede.

iii) Serious pulmonary and abdominal complications may make appearance.

iv) The bowels are traditionally loose.

v) Urinary incontinence may be present.

vi) There is leucopenia with neutropenia. if secondary bacterial complications occur then polymorphonuclear leucocytosis seen.

vii) The widal becomes positive in higher titres.

viii) Blood cultures is positive in 45%.

x) Urine & stool cultures are also positive.

D.FOURTH WEEK (stage of convalescence)

i) Temperature is at first subnormal and remains in a very unstable state.

ii) General condition improves.

iii) Appetite is good and the weight rapidly increases.

iv) The abdominal reflex reappears and the spleen is no longer palpable.

v) Heart sounds feeble and the pulses remains fast.

vi) Slight peeling of the skin, loos of hair and ridging or furrowing of the nails are noticeable.

•• 2.3 How can we diagnose a case of Typhoid fever in its lst week and 2nd week of illness in laboratory?

Typhoid fever refers to a febrile illness caused by ***Salmonella typhi and paratyphi A,B and C.(Sal.paratyphi C is not common in India).***

The laboratory diagnosis of typhoid fever is made by—

a) ***Isolation of the causative agent from clinical specimens,*** and

b) ***demonstrations of antibodies in patient serum.***

The following investigations are usually performed.

1. **Blood Culture**—This is positive in 90% cases in lst week, 75% cases in 2nd week,. 60% of cases in 3rd week and 25% of cases thereafter.
2. **Stool culture**—This is positive through out the course of heart disease.
3. **Urine culture**—This is less valuable than stool culture and blood culture, as the organisms are irregularly shed in urine. Only 25% of cases, it is positive in the 3rd and 4th week of illness.
4. **Widal agglutination test**—This becomes positive from 2nd week onwards and the titre steady rises upto 3rd or 4th week after which it declines.

•A. IN 1ST WEEK OF ILLNESS

The diagnosis is based mainly on the isolation of organisms ***by blood and clot cultures.***

(a) ***Blood culture:***

About 5ml. of blood is collected aseptically by venepuncture and inoculated into 50ml. of 1% glucose citrate broth. After inoculation at 37 °C for 48 hours, subscultures are

made on MacConkey agar and blood agar. These are incubated at 37°C for 18–24 hours. Non-lactose fermenting colonies from MacConkey's agar are tested for motility and biochemical reactions.Once organisms biochemically resemble Salmonella, its confirmation and species identification is performed by slide agglutination tests, according to **Kauffmann and white scheme** using **'O'** and **'H'** antisera.

(b) ***Clot culture:***

This is an alternative to blood culture. About 5ml. of patient's blood is collected and allowed to clot. The serum is separated out and used for the widal test. The clot is lysed by the enzyme streptokinase (100 units per ml.) and used for culture in a blood culture bottle as before. Clot culture is prefered because both culture and widal test can be performed from the same specimen. Clot culture is also said to yield a higher positivity than blood culture.

• B. IN 2ND WEEK ON-WARDS

The diagnosis of typhoid fever from 2nd week onwards depends on the isolation of organisms from stool, urine and sometimes blood and on demonistration of antibodies in patient's serum.

(a) ***Stool culture :*** This is probably more informative than blood culture as treatment which chloram-phenical does not eliminate the organisms from stool as rapidly as it does from the blood. Organisms are isolated from using enrichment media (Selenite F broth and tetrationate broth) and selective media (DCA, SSA, XLD, Wilson-Blair).

(b) ***Urine culture :*** Clean voided urine samples are centrifused and the deposite inoculated into selective and enrich media as for stool culture.

(c) ***Blood culture :*** Same as 1st week.

(d) ***Widal Test*** or ***Dilution Agglutination Test—***

When Blood culture method fails, unequivocal evidence of infection is obtained from the ***widal reaction*** in about 90% of cases.

Vi-antibodies do not appear with any regularity early in the disease and are therefore of more importance in detecting persistence of infection or a carrier state in a healthy individual than in diagnosis. Antibodies begin to appear in patients serum in between the seventh and tenth days of illness. In the uninoculated and in those who have not previously suffered from typhoid, 'O' agglutinins appears first. Most typhoid patients show a four fold rise of titre in 'O' agglutinins with 4 to 5 days. In a virgin case a titre of 1:200'O' agglutitinis is highly suggestive of typhoid fever.

A four fold rise of titre is diagnostic 'H' agglutinins appear more slowly and tend to persists for longer than 'O; agglutinins both after the disease and inoculation. A low 'O' and high 'H' titre suggests an anamnestic reaction. A level of antibodies in the serum bears constant relationship to the severity of illness.

2.4 What are the complication of typhoid fever?

Common Complications

1. **Alimentary system**—Three most serious complications which occur in the 3rd week of typhoid fever are:-

(a) ***Intestinal haemorrhage.***

(b) ***Intestinal performation.***

(c) ***Paralitic ileus.***

(d) ***Others***—Ulceration of mouth, Duguet's ulcer on the pillars of the fauces and pharynx, parotitis, tymphanities, diarrhoea, cholecystitis and cholangitis, hepatitis, portal pyaemia and jaundice.

2. **Respiratory system:**
Bronchitis, bronchopneumonia, lobar and hypostatic pneumonia, empyema, pulmonary abscess and embolism.

3. **Blood and circulation;**
iMyo. endo-.pericarditis, collapse, arteries, femoral venous thrombosis and haemolytic anaemia.

4. **Renal**—febrile albuminuria, nephritis, retention urine.
5. **Lecomotor system**—periostitis, osteomyelities, typhoid spine, arthritis.
6. **Nervous system**—Meningitis, meningism, encaphalitis, paralysis, post typhoid confusional state.
7. **Miscellaneous**—Alopecia, otitis, deafness, vulvovaginitis, orchitis, mastitis.

2.5 TREATMENT AND GENERAL MANAGEMENT.

1. Prophylactic.

(i) T.A.B.C. injection to those who are not affected.

(ii) Complete isolation of patient.

(iii) Disposal of all excreta after being carefully sterilised.

(iv) Feeding vessels, linen etc. soaked in 1 in 20 carbolic lotion for 2 hours and then boiled.

(v) Avoidance of contact.

(vi) Hands should be washed well with soap and water after attending the patient and afterwards ringed in 1 in 2000 per chloride of Hg. lotion.

(vii) Water and milk should be boiled.

(viii) Ice-cream etc. forbidden.

(ix) Control of house fly.

2. Curative:

i) Free ventilation in the room.

(ii) Complete bed rest until the temperature has been

normal for two weeks. The patient should lie flat and turned frequently from side to side to prevent hypostatic congestion and bed-sores. Air-rings are to be given under the pelvis and pressure points e.g. elbows, heels etc. Back treated daily with spirit and powder. Body sponged daily with tapid water and powder. Smoothness of bed clothes is essential.

(iii) Bed pan is to be used.

(iv) ***Oral hygiene*** is to be maintained.

v) ***Hydrotherapy***—water inside and water outside is of great value.

vi) ***Fluid intake***—atleast 5 pints in 24 hrs.

vii) ***Diet***-2000 to 3000 calories in 24 hrs; 6 to 8 ounces every 2 to 4 hrs. and at night 4 to 6 hrs.

Composition

(a) ***Milk and milk products***—Milk alone, diluted, flavoured with tea, coffee, ovaltine, chocolate etc, modified milk e.g., skimmed milk, horlicks, whey, ghole or lassi etc.

(b) ***Protein***—half boiled eggs, eggs, boiled fish etc.

(c) ***Cereals***—khai with milk, corn flour, well boiled rice.

(d) ***Vegetables Samashed*** boiled potatoes (Skin peeled off)

(e) ***Soups***—Chicken, dal etc.

(f) ***Fruits***—orange, sweet lime, grape juices.

(g) ***Drinks***—dextrose lemonade.

• B. MEDICINAL TREATMENT (LEADING REMEDIES).

1. ***Baptisia.*** Especially useful in the early stage. Face is dark,red with a besotted expression, stupid, dull, confusion of ideas, *goes to sleep while answering questions.* Complaints of dull aches and pains all over the body. Pulse is full and soft. The tongue is brown blackish and dry. *Breath*

is offensive. Diarrhoea of dark fluid exceeding fotid.

2. ***Arsenic-***

Useful for the 3nd stage. There is high fever, patient is *very irritable, anxious, restless* and *fears death.* The face is pale and shrunken, cadaveric, lips are dry, cracked and dark in colour. The pulse is small, Extreme prostation of strength and emaciation. The abdomen is very much swollen and tender and diarrhoea. Haemorrhage from the bowels. *Worse 1-2 a.m.* and *p.m. thirst for cold sips.*

3. ***Arnica-***

Says she is "So well" when desperately ill.
Can be roused, answers correctly, then goes back into stupor.
"I am not sick: I did not send for you; go away!"
Foul breath—Stool, Haemorrhagic tendency.
"Bed feels so-hard"
"So sore" can only lie on one part a little time, restless ness from this cause;
Involuntary and unnoticed stools and Urine.

2.5 WHAT IS TYPHOID STATE? WHERE IT IS FOUND?

It is the condition of semi-consciousness or coma attended by elevation of temperature and muttering delirium due to toxaemia.

It is found in typhoid fever; typhus fever, Acute labour pneumonia, Acute pulmonary T.B., Infective endocarditis, meningitis, and encephalitis lethergica.

- **What are the Cardinal Signs of Typhoid Perforation ?**

1. Sudden abdominal pain with rising pulse rate.
2. Patient may sometimes be too ill due to toxaemia, and specially may complain much, as already there may be sufficient abdominal discomfort due to distension.
3. Presence of fever and toxaemia as already mention in pre-page.
4. History of febrile illness of more than 9-10 days.

5. Sudden fall of B. P. and temperature to subnormal degree with abdominal symptoms and occurance of rigidity in the already distended abdomen.
6. Leucocytosis with predominent polymorphs.
7. Plain X-ray of the patient showing gas under diaphram in semi-inclind or sitting position or tilting the-ray table.

Treatment:

Perforation is followed by peritonitis, hence treatment is surgical. But it is now rarely seen.

3. DIPHTHERIA

• What is diphtheria? Describe the signs and symptoms, of Fauceal diphtheria. What are the differences between Acute follicular tonsilitis and fauceal diphtheria. What are itsComplications Sequelae and Treatment?

Def.—Diphtheria is an acute specific infectious disease caused by Corynebacterium diphtheria and characterised by local formation of a false membrane at the site of infection, rise of the temperature, dysphagia, profuse salivation, toxaemia, paralysis and albuminuria. It is of 4 types - (i) Fauceal (ii) Laryngeal (iii) Nasal (iv) Anal, vulval, aural (rare).

3.1 CLINICAL FEATURES OF FAUCEAL DIPHTHERIA.

A. Signs and symptoms

(1) The onset is incidious,

(2) Lasitude, headache, short cough, malaise, sore throat and fever sledom exceeding 101 °F (83.3 °C) are present.

(3) The *typical membrane* may begin on one or both tonsils and often spreads to the pillars of the fauces, uvula and palate.

(4) The *membrane is elevated, pearl-grey in colour,* firm in consistency small or large, adherent, being difficult to swab off; on *forced removal leaves a raw bleeding surface* which fills up again.

(5) It appears like a confluent sheet and has well-defined edge.

(6) Slightly tender enlargement of the cervical lymphnodes may be present.

(7) *In severe faucial diphtheria*—features are restlessness; pallor, gross cervical periadinitis ("Bull neck" diphtheria); recurrent vomiting, heavy albuminuria, rapid pulse and respiration; petechial haemorrhages in the skin;

Characteristic foetor oris; translucent filmy ill-defined membrane in the throat.

(8) Circulatory and wide spread paralysis may occur between 5th—21st day or later.

B. Laboratory Findings:

(1) ***Blood***—A moderate leucocytosis of about 15,000 cu. mm is present in the acute stage.

(2) ***Blood sugar***—Blood Sugar level may be diabetic type in the toxaemic phase.

(3) ***Urine***—Traces of albumin may be present, rarely RBC's.

(4) ***Throat swab examination***—Gram positive bacilli in chinesd letter fashion showing metachromatic granules is strongly supportive of clinical diagnosis. Culture in Loeffler's medium, blood agar or in tellunine medium is confirmatory, if KLB is grown.

(5) ***C.S.F.*** —Albumin-cytogical dissociation in CSF may be found in post-diphtheric neurits.

(6) ***E.C.G.***—Involvement of the myocardium is a late complication, and when myocarditis is present the ECG shows prolonged PR interval, T wave inversion,

arrhythmias and various degrees of heart block.

3.2 TREATMENT

The diagnosis is made on clinical grounds and treatment should not wait for laboratory confirmation.

Allopathically. *Diphtheria antitoxin* should be administered preferably intravenously unless it is a mild case. The risk of anaphyulaxis should be weighed against possible fatal outcome, if treatment is denied. The patient must be under close observation preferably in a hospital. When hospital facilities are not available, do not allow the patient to go home. Better treat him and observed him at least for one hour at the chamber.

• **Homoepathically following medicines are used successfully.**

1. **Lachesis**—Exudate begins on *left side and spreads to the right.* Throat is very sensitive to touch on pressure. Hawking and spiriting of the mucus in the throat. Pain extends to the ear from swallowing. Prostration is severe, heart's action is feeble, whole body aches and the patient *sleeps into an aggravation.*
2. **Lycopodium**—Exudate begins on the Rt.side. Aggravation of all symptoms in the evening typicallty from 4 to 8 p.m. The patient on awaking from sleep is cross.
3. **Belladonna**—To be given only at the commencement when there is dry sorethroat with cerebral symptoms. Difficulty in diglutition. Throat is swollen.
4. **Apis mel**—May be used with success in the first stage, it controls further progress. Great prostration from the begining; thirstlessness, urine scanty and high coloured; burning when urinating. Oedematious swelling of tonsils, throat and uvula. Diptheritic patches over palate, difficulty in swallowing; senses of suffocation. Fever; sweats every now and then. It serves inflammation of the throat with several stupor, prostration in diphtheria.

*Apis is found to be of great value and a frequently indicated remedy. It is almost a specific in true diphtheria; always indicated when the *throat is very much* swollen and oedematous, with severe stinging pains on *attempting to swallow.*

5. **Arsenic**—This is a potent remedy for malignant form of diphtheria in all its stages; oedematous swelling of the throat, throat seems to become narrow unable to swallow; diphtheritic path looks dry, foul breath fetid odour from mouth, unquenchable thirst, burning, great debility restlessness. Fever, burning hot skin, rapid beating of pulse, sometimes delirium.

6. **Mercurius**—Painful swelling of the glands of the throat with profuse salivation. Foul breath, Inflammation of the throat, fauces, tonsils and uvula. Swallowing painful; chocked feeling. Diphtheritic patches.

[According to many *Merc. bin. Iod* is more potent than *Merc. sol., or Merc. viv; Merc. cyanates* may also be sometimes restored to with success. **Allen** says, *Merc. cyn* is a valuable remedy for some forms of diphtheric characterised by the mercuric symptoms and aggravations with extreme prostration; the disease invades the nostrils, there is threatening collapse with very small rapid or intermittent pulse; it follows Apis well].

3.3 WHAT ARE THE DIFFERENCES BETWEEN DIPHTHERIA AND ACUTE FOLLICULAR TONSILITIS?

Diphtheria	Acute Follicular Tonsilitis
1. Local Signs.	
(a) Ashy—grey patch or patches on tonsils, uvula and soft palate (latter situation is path-	(a) Swelling and redness chiefly confined to one or both tonsils. In the

ognomic).	follicular form, tonsils covered with sticky mucus with numerous small, seperate yellow spots of secretion on one or both, which are easily removable. *Nothing on soft palate.*
(b) Patches longer than the follicular tonsilitis; they consist of membrane surrounded by red areolae; difficult to remove leaving raw surfaces.	
(c) Characteristic smell. Corynebactrium diphtheria (K.L.B.) bacillus in membrane. Sometimes a mucopurulent acrid nasal discharge.	
(d) Comparative absence of pain.	
2. General Symptoms	
(a) Onset insidious. Early and marked enlargement of cervical glands.	(a) Onset moderately sudden with moderate fever.
(b) Temperature usually low during whole course.	(b) Temperature may be very high, but local symptoms are usually more troublesome than general symptoms.
(c) Paralytic sequelae	

N.B.1. **Laryngeal diphtheria** — The characteristic features are husky cough, hoarseness of voice and increasing inspiratory dysponia. Restlessness and cyanosis develop. Rest of the symptoms are like the faucila diphtheria.

2. **Nasal diphtheria** — Nasal air entry is obstracted. Nasal discharge is watery or mucoid and later becomes blood stained.

3. **Atypical varieties** — Characteristically on there sites, suchas, conjunctiva, vagina, vulva, and cutaneous wounds, a membrane is formed with marked signs and symptoms of toxaemia.

3.4 WHAT ARE THE COMPLICATIONS OF DIPHTHERIA?

Complications:

1. Bronchitis, bronchopneumonia and asphyxia.
2. Heart failure, peripheral failure, endocarditis, myocarditis, pericarditis,
3. Albuminumia, aneuria and nephritis.
4. Vomiting.
5. Otitis media, mastoiditis.
6. Septicaemia.
7. Bull neck.
8. Embolism.

3.5. WHAT ARE ITS SEQUALAE?

1. Circulatory or cardiac failure.
2. Various paralysis e.g. Palatal; Occular; pharyngeal; paralysis of limbs, trunk, neck etc.

4. WHOOPING COUGH

• What is Whooping Cough? What are its clinical features, complications and treatment. How will you diagnose the case?

Def.- Whooping cough is a highly infectious disease of upper respiratory tract and is characterised by acute episodes of cough followed by inspiratory whoop and vomiting.

It is caused by ***Bordetella pertusis or Haemophilus pertusis,*** a small slender gram negative rod which almost exclusively invades children under the age of five.

This is a contagious disease, spread by infection, with an incubation period of one to two weeks. Outbreaks may

occur in schools. No quarantine period is usually necessary.

4.1. CLINICAL FEATURES

The incubation period is usually 7—10 days.

1. Catarrhal Stage

The child presents with symptoms of acute nasopnaryngitis, conjunctivitis, slight fever and non-productive cough. Catarrhal stage persists for about a week and is the most infective stage.

2. Paroxysmal Stage

This is the second stage of the illness which is characterised by severe paroxysm of Whoop. Often there is expectoration of tenacious mucus and vomiting. At the end of each episode the child gets exhausted and even may go to sleep. These may be as much as 40 bouts of cough in a day. The temperature remains normal at this stage. Moderate conjunctivits, swelling of face and ulceration of fraenum linguae are often noted.

3. Convalescent Stage

In untreated cases the 'whoop' persists for about a month after which the cough gradually disappears and the child returns to normalcy unless some complications supervane.

4.2 DIAGNOSIS

1. Diagnosis During the early stage is clinical; culture of the organisms from pernasal swabs or cough, plate containing the Bordet-Gengoue medium is positive in 80% in first two weeks.
2. Blood picture-Total white cells 15,000-25,000/c.mm or more, Lymphocytes more than 50%. The WBC count may go up to 80,000/c.mm and lymphocytes to 80%.
3. Urine-may show an excess of uric acid.
4. X-ray findings-may show pneumonitis but significanthilar adenopathy may sometimes be present.

4.3 COMPLICATIONS

1. The commonest complication is upper respiratory tract infection. Severe broncho-pneumonia, segmental collapse, lung abscess and even bronchictasis may follow.
2. In severe cases encephalitis, convulsions and cerebraL haemorrhagehave been reported.
3. In a minority of cases otitis media and bleeding episodes from the ear, eyes, nose and gums may occur.The prolonged cough may augment incidence of hernias including prolapse of the rectum.

4.4 TREATMENT

A.Prevention-

1. For active immunization, *triple antigen* (pertussis, tetanus, diphtheria) is usually employed. Three doses of 1 ml, at intervals of at least one month are given preferably beginning when the baby is 4—6 months old with booster doses in older siblings; this is *contra indicated* in fevers, aural and cutaneous sepsis, in premature and frail infants. Pertusis vaccine beyond to age 1 year is sometimes following by neurological involvement like peripheral nuritis or encephalopathy.

B. Management—

Isolation from other children is useful to prevent spread, specially in the early stage. Various cough syrups available in the market, may rarely prevent the paroxysms. But proper Homoeopathic Medicine should be choice and used for this treatment.

C. Homoeopathic Medicinal treatment—

1. *Drosera* (6,30)-Violent coughs which follow each other in quick succession; the patient is scarcely able to breathe. Wakes early in the morning and continues coughing until a quantity of tenacious mucus

is vomited. Coughs at regular intervals. Dry spasmodic cough; bleeding from the nose when coughing; cough worse lying down after midnight. Laughing, singing, drinking, etc. excite cough. It is specific for whooping cough.

2. *Cuprum met* (6,30)-Long lasting, dry, suffocative cough and spasmodic attacks; worseat 3 A.M. Unable to speak; breathless; blue face; stiff; three attacks one after the other. Cough has a gurgling sound; cough better by drinking water.

3. *Coralium* (6.30)-Dry spasmodic and suffocative cough, coughs in rapid succession at regular intervals patient becomes purple in face. Worse open air; changing from a warm to cold room excites cough.

4. *Sticta* 6. Hoarse, incessant, unprofitable cough of spasmodic type which is often barking and at night and morning. It cures whooping cough left by measles and influenza.

5.RHEUMATISM.

• What is Rheumatism? Give signs and symptoms and complications of Rheumatic fever. Give some drugs with indications. What are the differences between Rheumatic fever and Acute Gout?

The term *Rheumatism* is loosely applied to all conditions causing pain and stiffness of the muscles and joints due to unknown etiology. It is of 2 types-

1. *Acute Rheumatism.*
2. *Chronic Rheumatism.*

Acute Rheumatism is an acute fabrile condition affecting the joints, heart, muscles, skin and nervous system. It is also called Rheumatic fever. Chronic Rheumatism consists of the following conditions :

(1) *Rheumatoid Arthritis.*
(2) *Osteo-arthritis.*
(3) *Ankylosing Spondylitis.*
(4) *Gout.*

· RHEUMATIC FEVER

5.1 WHAT IS RHEUMATIC FEVER?

Rheumatic fever is a one type of collagen disease of children between the age of 5 to 14 years and adult up to age 18 years by the infection of *beta-haemolytic Streptococci of Lancefield's group* and is characterised by fever, fleeting joint pains, carditis, chorea, erythema marginatum and rheumatic nodules.

It is sometimes called juvenile rheumatism. Carditis is the most dreadful manifestation, so it is well said that "It licks the joints and bites the heart".

5.2 WHAT ARE IT'S MAIN SIGNS AND SYMPTOMS?

A. Major Manifestations

(a) *Polyarthritis*—usually migratory and mild in children.

(b) *Carditis*—75% incidence of carditis are found in an initial attack of rheumatic fever in an Indian series. Murmurs', pericardial rub, tachycardia, galloprhythm and heart failure may be present.

(c) *Chorea*—often in girls.

(d) *Subcutaneous nodules*—(3 to 10 mm. infrequent in Indian) occur every bony prominences and signify rheumatic activity.

(e) *Erythema marginatum*—recurrent migratory pink rash seen mainly on trunk, sometimes in extremities, never on face (rare in India).

Other cutaneous manifestations, e.g. petechae, urticarial rashes and erythema nodosum may occur, but none are specific of the disease.

B. Minor Manifestations

a) History of previous attack—fever with joint involvement.

b) Fever.

c) Arthralgia.

d) Prolonged P-R interval.

e) Increased E.S.R. or presence of C-reactive protein.

**The diagnosis of the disease requires the presence of *two major manifestations or one major and two minor manifestations.* So these criteria serve well to minimise both over-diagonsis and under diagnosis.

C. Other Signs and Symnptoms (in details).

1. **Fever**—This is perhaps the most common manifestation, though minor. In severe cases the fever may rise to 104 °F (40 °C) or higher and may persist for several weeks before subsiding. It may be accompanied by toxicity, profuse sweating, epistaxis and purpura.
2. **Arthritis**—It is usually accompanied with fever. The large joints of extremities viz. knee, ankle, elbow, wrists are commonly affected but no joint may be spread. One may find spine, hands, feet, sternoclavicular or termporo-mandibular joint affections rarely. Joint effusion may occur but not persistent, pain and swelling may affect one joint and subside in a day or two to shift to another joint. This migratory nature is characteristics but number are invariable, as several large joints may be involved simultaneously.
3. **Carditis**—During the course of acute rheumatic fever the heart may be relatively unaffected or the child may be develop endo-carditis, myo-carditis, pericarditis or pancarditis. Recovery may be complete or the patient may develop valvular lesions. The most constantly affected value is the mitral, followed by the aortic and then the tricuspid.

• During the attack there may be tachycardia out of proportion to the pyrexia which persists when the patient is at rest or is asleep. As long as the sleeping pulse rate remains fast, the disease is considered active. In many cases there is severe endocarditis.

The heart sounds become *muffled and a pansystolic murmur* is heard on the precordium which is transmitted to the left axilla; it is an evidence of mitral incompetence. Sometimes mid-distolic murmurs develop (Careycoombs murmur) which is low-pitched, flowing and tends to vary from day to day and may disappear. A high-pitched early diastolic-murmur at the base of the heart signifies *aortic incompetence.*

Gallop rhytm is frequent owing to exaggeration of 3rd heart sound. Aortic stenosis rarely occurs in children due to rheumatic lesion but may occur in adults. Rheumatic lesion of *Pulmonary values* does not occur. Mitral stenosis probably takes two years to develop after an initial attack of rheumatic endocardits. Pericarditis may occur with high fever, precordial pain, vomiting, dyspoynoea and presence of a pericardial rub which disappears when there is effusion.

4. **Radiological Finding & E.C.G.—**

X-ray shows pear-shaped heart and sometimes pericardial effusions.
E.C.G. reveals depresses ST segmentis in standard leads with subsequent inversion of T waves.

In acute rheumatic fever ECG maybe normal, in mild cases an amount of heart block exists. P-R interval is prolonged (above 0.20 sec), but this itself is not diagnostic or rheumatic carditis. Prolongation of P-R interval occurs in one-third of patients with post-Streptococcal infection regardless of other featured of carditis.

In healthy children the P-R interval is seldom more than 0.06 second, but in myocarditis it is prolonged to 0.24 second without dropped beats occurring. There is a 2:1, 3:1 or 4:1 heart blocks, but complete block is rare: The Wenckebach phenomena consists of an increasing P—R interval with successive heart beats until a beat is dropped.

The subsequent beat or two are normal and the process then repeats itself.

5. Laboratory findings

There are no specific laboratory findings. The most reliable evidence is either an increased or rising Streptococal antibody titre which occurs in an early stage of the disease. The antistreptolysin O'titre is most widely used.

In general, single titre of at least 250 Todd units in adults and least 330 units in children over 5 years is significant. Many patients continue to harbour small numbers of *Group A Streptococci* in the throat, at the onset of acute rheumatic fever. The EST&C-reactive protein are raised along with moderate leucocytosis with anaemia (normocytic normochromic).

• DIFFERENTIAL DIAGNOSIS

5.3. WHAT ARE DIFFERENTIAL DIAGNOSIS OF RHEUMATIC FEVER?

Rheumatic fever is generally distinguished from other varieties of *arthritis, limb pains* (the so called growing pain), and *acute leukaemia.*

1. **Limb Pains**—The pain is mainly in the evening when the child is tired, sometimes waking the child at night. It has nothing to do with growth, as growth is a painless process. The growing pain poses a problem, if the child has got a systolic murmur. But this pain is in the limbs and not in the joints, there is no carditis, no tachycardia and sleeping pulse rate is normal

2. **Pyogenic arthritis**—Acute onset with high fever, intense muscle spasm and severe joints pain which is not migratory and the child passes sleepless nights.

3. **Rheumatoid arthritis**—The diseases affects the hands as well as large joints, carditis seldom occurs. It is a relapsing illness with high fever, adenopathy,

splenic enlargement and no dramatic response to salicylates.

4. **Acute leukaemia**—Limb pains from leukaemia rather than in the joints. There may be other features like severe anaemia with haemorrhages and abnormal cells in blood examination.

5.4 COMPLICATIONS OF RHEUMATIC FEVER.

1. (a) Cardiac—Endocarditis involving the mitral and less frequently aortic valves, so that mitral or aortic stenosis is produced.

   (b) Myo-carditis leading on to congestive heart failure.

   (c) Peri-carditis leading on to adhesions.

2. Pulmonary-Bronchopneumonia, pleurisy.

3. Nervous-Hyperpyrexia, delerium.

4. Cutaneous-Purpura and erythemia.

5.5. TREATMENTS

1. **General**—The patient must be on bed rest for 6 to 8 weeks during which the rheumatic process usually subsides.The patient should not be up till the fever and joint pain go away, and sleeping pulse rate, blood count and ESR become normal.The affected joints should be kept in a position of optimum comfort, wrapped in cotton wool and protected from pressure of heavy bed cloths.

2. **Diet**—Should be nutritious with adequate protein and Vit. C, fruit juice, barely water milk, etc. gradually add bread, butter and semi-solids. Convalescence should be prolonged even up to 5 months, depending on the condition of the heart or in case of relapse of rheumatic process.

• MEDICINAL TREATMENT

1. **Aconite**—This is the main, first and the most favourable remedy of pain in muscles or joints of cutting or

twisting type. High fever, restlessness, loss of appetite, high coloured urine, affected parts are red and inflammed. The patients is irritable, anxious and restless; tosses about and knows he is going to die. Great thirst.The joint is red, swollen and very sensitive and there is shooting and tearing pain in the joints aggravated at night.

2. ***Bryonia***—Bryonia is a good remedy for the joint pains and backache, and is frequently used in cases of rheumatic fever because it has affinity for serious membranes especially of joints.

Joint is swollen, red and hot. One or more joints may be involved. Sharp, stiching or cutting type of pains worse by least motion, touch or pressure. Better by lying on painful side. Copious sour smelling sweat. Fever with dryness of oral cavity and alimentary canal. Lips and tongue dry. Tongue coated thick white. Great thirst for large quantities of cold water at longer intervals.

Backaches, lumbago, sorness of the lumbar muscles, stiching pains worse by slightest motion but better by firm pressure and lying on back.

3. ***Rhus Tox***—It is more useful remedy for rheumatism of extremities, though useful in intercostal rheumatism. Pains are worse during rest, first motion, cold wet rainy weather, at night and getting wet. Better by change of position, motion, movement of affected part, warmth.

4. ***Ranunculus***—It is more useful remedy for intercostal rheumatism, though can be used for rheumatism of extremities.

Pains are worse by motion, touch, wet, stormy weather, atmospheric changes and in the evening.

5. ***Belladonna***—High fever, tendency to cerebral irritation. Dry hot skin, thirst and throbbing carotids. The joint is swollen, red and shiny with tearing, shooting pains. The pains come and go quickly.

6. ***Arnica***—Bruised feelings as is lying on something hard. The joint is hard, swollen and red. With soreness and bruised feeling with great fear being touched. There is numbness of the joint. Pain and stiffness following hurt or fall.

5.6 WHAT ARE THE DIFFERENCES BETWEEN RHEUMATIC FEVER AND ACUTE GOUT?

• **Differences Between Rheumatic Fever and Acute Gout**

Items	Rheumatic Fever	Acute Gout
1. Age.	Common upto 20 years of age.	Common in middle. age and over.
2. Sex.	Either sex	Males preponderate.
3. Joints	Large joints are usually affected e.g. knee, ankle, shoulder etc.	Small joints are affected e.g.great toes.
4. Shifting	Shifting polyarthritis; joint affection wandering from joint to joint.	No shifting. Localised joint affection never wandering from join to joint.
5. Swelling.	Swelling is usually hot but pale	Swelling red, tense and pitting on pressure.
6. Pain.	Pain on pressure or movement of the joints	Pain persists even at rest.
7. Tophi.	No tophi in ears.	Tophi in ears ++.
8. Nodule.	Rheumatic nodules. ++	No nodules.
9. Skin rashes	Sudamina and various rashes present.	No Skin rash.
10. Temperature	High continuous type of temperature with blanket tongue and sour sweat.	Temperature slight and transient.
11. Onset	Onset may be sudden or insidious, but no definite nocturnal aggravation.	Sudden onset in middle of night.
12. Complications.	Rheumatic carditis and other heart affections common.	No heart affection.

- **Homoeopathic medicine used in Rheumatism/Gout.**

1. ***Aconite*** 3x, 30-Acute rheumatism with fever, restlessness and fear of death; worse at night and by warmth; better in open air. Unbearable shooting pains; bruised feeling, Heaviness of limbs. Rheumatic inflammation of joints.
2. ***Actea racemosa*** 30, 200-Rheumatic pain in small joints, wrist, fingers, toes, tearing pains, worse from touch and motion. Rheumatism in females with uterine trouble.
3. ***Colchicum*** 30,200-In acute cases affecting mainly joints, and in persons of vigorous constitution; shifting pain in all the joints; the parts affected are red, hot and swollen & are sensitive to touch & motion, worse at night; feverish. In sudden attack of gout it has a specific action, and in chronic cases it is beneficial.
4. ***Dulcamara*** 6.-Rheumatism due to damp ground, aggravated by every cold change, somewhat relieved by moving about, rheumatism alternates with diarrhoea, after acute skin erruption.

B. DISEASES DUE TO VIRUS

DISCUSSION FOR LEARNING.

1. INFLUENZA (La.Grippe.)
2. MEASLES (Morbilli, Rubeola).
3. SMALL POX (Variola).
4. CHICKEN POX (Varicella).
5. DENGU (Break bone fever, Dandy fever).
6. MUMPS (Parotitis).
7. ENCEPHALITIS (Japanese B.Encehphalitis.)
8. RABIES (Hydrophobia).

6. INFLUENZA:

• What is Influenza? What are its aetiology, clinical features and complications? How can we treat it by homoeopathic medicine?

What are the differences between Influenza and Dengu?

Def. *Influenza is an acute infectious disease caused by* influenza viruses *of type a,B,C or D and is generally characterised by sudden rise of high temperature, pain all over the bodyu, headache, coated tongue, and inducing and living behind it great vital depression.*

6.1 AETIOLOGY:

1. ***Primarily*** :- A,B,C & D type of Influenza viruses. B,C & D sporadic but A viruses are causes of epidemic, which occur at the interval of 2 to 5 years and lasts for 2 to 3 months. Influenza virus, is of 80-100 millimicrons and is filter passing.
2. ***Secondary causes*** are—H. Influenza, N.catarrharalis, Strepto, viridans, Strepto, haemolyticus etc.
   • Incubation period is 1—2 days.
   One attack gives a short staying immunity to the particular type of the virus only.
   No age, sex or race is exempt.
   Transmission is by droplet inhalation.

6.2 CLINICAL FEATURES:

1. The paroxysm starts with a sudden sharp rise of temperature, usually with chill or rigors and reaching maximum-on the same day or the day after. Associated with this is severe headache, disabling pain all over the body, marked prostration, obvious cantarrh of the upper respiratory passages. There is dryness of the throat and dry cough.
2. The temperature is slightly fluctuating and last for 4—6 days and drops almost by crisis.
3. Naso-pharyngeal exudations are nearly absent and prostration is marked.

4. Mild conjunctivitis may be present.
5. After the recovery from the fever, there is marked weakness and tiredness occurs very rapidly.
6. During the fever the face is flushed, the conjuctiva suffused.
7. The pulse is slow or slightly rapid.
8. Moderate leucopenia with neutropenia are usually at the early stage, complications cause leucocytosis.

6.3 COMPLICATIONS

1. *Respiratory* : This is due to associated secondary organisms like Streptococcus haemolyticus, Staphyllococcus, H. influenza and Pneumococcus. There is laryngitis, tracheitis, brochitis, alveolitis and pleurisy,. Ultimately bronchopneumonia may occur. Sinusitis, otitis media, or asthma may occur.
2. *Gastro-intestinal* : complications may be severe tympanitis, melaena, jaundice.
3. *Nervous*-dilirium, coma, meningitis, enchephalitis, myeliis.
4. *Cardiovascular*-produces myocarditis. this also causes palpitation, with soft pulse.

6.4 TREATMENT

A General managements

The three main measures to be secured in a pronounced attack of influenza are—*rest, warmth & nourishment.* Easily assimilated diet must be given frequently, till the acute symptoms are over. The sick room must be well ventilated but the patient must be protected from draught; and after the attack is over, if it has been at all severe case; must be taken to avoid any tax on the strength and exposure till all traces have passed away.

B. Homoeopathic Medicines are used symptomatically

1. *Baptisia*—The drug which comes nearest to being a

specific is Baptisia. The heaviness, bestotted appearance of the eye, headache, foul tongue, sore throat, soreness all over, and general uneasiness with or without fever. This medicine given in any potency will quickly cure a large, proportion of cases.

2. *Eupatorium perf.*—When the pains in the bones are well marked this is preferable than *Baptisia.*
Deep seated bone pains especially in the back, wrists and ankles; eye balls sore on turning; nausea or vomiting and debility.

3. *Gelsemium*—Severe bodily pain with great general prostration, the patient wanting to lie perfectly still, and trembling from weakness with least exertion; fever with no thirst, and th3e tongue tremblemes when protruding it.

4. *Bryonia*—Severe pain in the whole of the body, wanting to lie quiet, and worse on the least motion, mouth and throat dry with great thirst; headache and severe constipation.

5. *Nux Vomica*—After drinking, immediately shivering and chilliness. chilliness on the least movement. On the slightest exposure to the open air, shivering and chilliness for an hour, dreads to go into open air. He can not get worm. Attack, as of fever shivering and drawing in the limbs.
"serious ailments from catching cold are often removed by it"-Hahnemann.

6.6 HOW WILL YOU DIFFERENTIATE INFLUENZA AND DENGU?

- **Differences Between Influenza and Dengu**

	INFLUENZA	DENGU
1.	Def.—It is an acute infectious disease characterised clinically by fever dispro portionate aches and pains all over the body and symptoms of upper respiratory tract infection caused by Influenza viruses.	It is an acute viral infection with diphasic febrile episode, severe headache, myalogia and morbilliform rash caused by Dengu virus.
2.	*Spreads*—From man to man by droplet infection.	Transmitted from man to man by female mosquito Aedes aegypti.
3.	*Types*—Many types.	Only one type.
4.	*Clinical features*-Temperature sudden with headache, with pain in the eyes. No saddle back temperature.	Temperature sudden with rigor and prostration; anorexia and vomiting; pain as if bone are broken so, it is also known as break bone fever. Temperature saddle back like i.e. (Temperature is raised which comes down by crisis on the 3rd day but again goes up on 4th or 5th day given the typical saddle shaped Temp. curve).
5.	Leucopenia-with relative lymphocytosis.	With 50% polymorphs.

7. MEASLES

- Mention the clinical symptoms of measles from it's prodome to it's termination. Mention some of the

common drugs used in measles epidemic wth their differentiation. How will you try to prevent it's spread by drug or otherwise?

- Name three diseases which childrens are very often prone to infection?
- What do you know about measles? What prophylaxis and preventive measures would you adopt if epidemic breaks in your locality?

Def. Measles is an extremely contagious viral disease common in children, caused by droplet infection of medium sized RNA para-mixovirus, characterised by coryza, exanthemata of the skin and upper respiratory catarrh.

7.1 AETIOLOGY

Virus affects man and monkey. The disease is spread by direct contact, through droplet infection or sneezing or coughing.It is very iufectious during the early catarrhal stage, before the appearance of rash.

Age between 8th month to 5 years.

Previous attack gives immunity.

Epidemics during winter and spring.

Incubation period 10-14 days.

School going children are easily infected, specially in cities, Children in rural areas escape infection till their adult life, when they come in contact become infecte.

7.2 CLINICAL FEATURES

Measles passes through its course by *stages*; it has a period of *incubation*, lasting from ten to fourteen days, its *precursory fever*; its *eruptive stage*, and its decline. But the main clinical features can be described into two stages-

1. Pre-eruptive or catarrhal stage.

The onset is sudden, like common cold with nasal catarrah, sneezing, watering and infection of conjunctivae. Hoarseness of voice is due to laryngitis, photophobia swelling of eyelids.

The pre-eruptive stage is characterized by the presence of white spots (Koplik's spots), comparable to grains of salt or a pin head surrounded by a zone of inflammation in the cheek against the molar teeth.

2. The eruptive or the exanthematous stage.

The koplik's spots disappears with appearances of the rashes.

• A. *Temperature* : The temperature generally rises to 37.8 °C (100 °F) on the first day, but falls to normal or below normal on the 2nd to 3rd day; to be elevated to 39 ° C (102 ° F) or higher on the 4th day when the rash appears, after 6-7 days it gradually reaches normal. The rashes and the fever, may however last for a few days more.

• B. *Prodromal rashes* : The prodromal rashes are erythematous or utricarial and may be found before/on the first day of fever.

The *characteristic rashes*: appear on the 4th day on the forehead, temple and behind the ears and spread to the other parts; they are maculo-papules (2-6 mm. in diameters), erythematous, crescentic, may be confluent and form crecentic blotches. The rashes begin to diappear in 2-5 days in the order of their appearance. Staining or branny desquamation may continue for 1-2 weeks.

C. *Laboratory finding*: *Leucopenia*-if complication occurs then there is leucocytosis.

7.3 WHAT ARE IT'S COMPLICATIONS?

1. *Bronchopneumonia*—is a common and the most dangerous complication of measles in the children. Pleurisy and empyema may follow and even death may insure.
2. *Enteritis*—has grave sequelae in under-nourished children. It occurs in late erruptive phase.
3. *Otitis media*—is common and if untreated, may produce mening it is and deafness.
4. *Keratitis, blepheritis, panophthalmitis*—rarely.

5. *Encephalitis*—rare and calls for immediate lumber puncture.

6. *Tremor hyper-pyrexia and delirium*—may occur in some cases labelled 'toxic measles'. convulsion may also occur during appearance of rashes.

7.4 TREATMENT

A. *General*—Treatment is mainly symptomatic.
The children should be kept in well ventilated room with good sanitary conditions.
Quality nursing care is necessary.
Put a lot of bath powder to avoid irritation.
Linen should be kept clean.
Protection of eyes against injury is done by installing Lotio.Boric 2 to 3 times daily in both eyes.

B. *Prophylaxis*—Segregation for 2 weeks.
Disinfection of used articles.
Quarantine of the contacts for 3 weeks.
A life long immunity occurs after immunisation with attenuated measles vaccine (Allopathically).

*Morbillinum is prophylactic for contacts. But it may be prevented or modified by giving children who have not had Measles, a does of *Pulsatilla*, every morning, and one of Aconite every evening, for a week or the days during its prevalance.

C. Medicinal Treatment: (Leading-remedies)

1. *Aconite Nap*-Catarrh and high fever: before rash clinches diagnosis.
   Readness conjunctivae : dry, barking cough.
   Itching, burning skin : rash rough and miliary,
   Restless, anxious, tossing : frightend. (M.L.tyler).

2. *Pulsatilla*-Cough worse towards evening or during the night with rattle of mucus in the air passages or thick yellowish or whitish expectoration, thick greenish or yellowish discharge from the nose; epistaxis; Catarrhal derangement of the stomach, and diarrhoea. Puls, may follow Acon.

3. *Euphrasia*—Cases with great catarrhal intensity.
"A wonderful medicine in measles. When symptoms agree will make a violent attack of measles turn into a very simple form...."
"Streaming, burning tears, photophobiua, running from nose; intense throbbing headache, dry cough and rash". (Kent)
Copious acid lachrymation, with streaming bland discharge from nose. (Tyler)

4. *Gelsemium*—some give it instead of puls.
Chills and cheats chase one another.
Sneezing and sore throat:excoriating nasal discharge.
severe, heavy headache:occipital pain.
thirstlessness is the rule with Gels.
Drowsy and stupid, Lids heavy: Eyes inflamed.
Face dark red, swollen bestotted look.

5. Bryonia alb.
Rash tardy to appear.
Hard, dry cough with bearing pain.
Little or no expectoration.
Or, rash disappears and child drowsy:pale, twitching face, chewing motion of jaws.
Any motion causes child to scream with pain.
Mild delirium, "weants to go home", when at home.
Or, instead of rash, bronchitis or pneumonia with Bry. symptoms.

6. Sulphur.
"Measles with a purplish appearance Sulph, to modify the case when the skin is dusky and the rash does not come out:, Convalescence slow, and the patient is weak and prostrate.

8. SMALL POX AND CHICKEN POX.

• Differentiate a case of Small pox from that of a Chicken Pox?

What are the complications and sequalae of Small pox and how will you avoid them?

Give an account of small pox, What are the differences

between Small pox and Chicken pox? Mention 4 homceopathic drugs for treatment.

8.1. WHAT ARE THE DIFFERENCES BETWEEN SMALL-POX AND CHICKENPOX?

- **Difference Between Smallpox and Chickenpox**

Small Pox	**Chicken Pox**
It is an acute infectious disease caused by variola virus, and clinically characterised by a sudden onset of fever, headache, backache, vomiting and sometimes convulsions, specially in children.	It is an highly contagious viral disease caused by varicella virus, and characterised byvesicular rash.
Epidemic and severe	**Sporadic and Mild.**
1. This is a virus disease. *caused by variola virus.*	1. *Caysed* by *varicalla zoster virus,*
2. *Spread* is direct by inhalation of droplets or by the fomites.	2. *Spread* by droplets, driedscales or through ruptured skin lesions.
3. *Incubation period* 12 days but extreme limit is 7-16 days.	3. About 14 days, ranging between 12 to 21 days
4. *Prodromal feature*—Malaise frontal headache, backache, sore throat, cough hoarseness of voice. Usually associated with 2-4 days of fever before the onset of rash.Before the appearance of smallpox purpuric rash, urticarial rash etc. found.	4. No prodromal features. If any features appear will remain for a short time. Rash usually appears simultaneously with fever.
5. *Apperance of rash*—1st day appearance of typical rash. Small pox rash on hte 3rd day on the forehead and wrist.	Very first day on the Trunk and palate.

6. *Distribution of rash.*	
(a) Most of the rashes are found peripheri to centre.	(a) From the centre to peripheri.
(b) Axilla is free from rash.	(b) Axilla is infected.
(c) More dense on the face and limbs.	(c) More dense on the trunk than on the face and limbs.
(d) Plams and soles frequently involved.	(d) Seldom affected.
(e) Rash predominent on extensor surfaces and bony prominences.	(e) Rash mostly on flexor surfaces.
7. *Characteristic of rashes.* Only one type of rash is found at the sametimes, i.e. when macule, all are macule, when papule all are macule, when papule all are papule.	Polymorphic *f* different types of rash are found in the body at the sametime. *Macule*—rash in the skin but not above the level of skin. Papule—above the level of skin. Vasicule—above the level but contain liquid. Pustule—above the level but contain pus.
8. *Evaculation of rash.* The rash appear at the 3rd day of fever then—	Rashes are appear in the 1st day of fever.
macule within 2—3 days,—	macules changes to papule within few hours.
papule within 24 hours,—	papules within few hours vesicule.
vesicule 2—3 dyas,—pustule.	vesicule with in 24hrs. pustule and with in a few days
pustule 8—9 days,—scab.	scab formed from pustule 4
scabs begin to form 10—14 days after the rash appears and after seperation of scab, scab fall off 14to28 days after the rash begins and there remains permanent scars marks.	to 7 days after the rash appears and subsequently there is seperation of scab living no scar mark. Scab fall off with in 14 days after the rash begins.
9. Temperature. Withthe appearance of rashes temp. falls and it may even	No such special features of temperature.

touch the normal level and there is subsequently second rise of temp. in pustule stage.

10. *Association of Herpes zoster.*
It never shows.

It may be associated with Herpes zoster.

N.B.1. Chickenpox is due to a filter passing virus, the virus being probably identical with the virus of herpes and the contactants with one disease may develop the other or the dermotropic virus of chicken pox may become neurotropic to cause herpes or vice versa.

2. The incubation period is 10-14 days and the spread is by droplets, dired scabes or through rupture skin lesions.

3. Children and young adults of both sexes are the common subjects.

4. A second attack is very rare.

5. Vaccination affords no protection.

8.2. COMPLICATIONS OF SMALLPOX

1. Secondary infection of the skin producing boils, abcesses and generalised eczema (Eczema vaccinatum)

2. Conjunctivitis, keratitis and Panophltalmitis producing complete loss of eye.

3. May produce corneal opacities.

4. Bronchopneumonia.

5. Heart failure and perpheral circulatory failure.

6. Septicaemia and toxaemia.

7. Delerium, convulsions and encephalopathy.

8. Urinary inflammation of the kidneys causing albuminuria.

9. Glands enlargement and enlargement of the parotid glands.
10. Otitis-media.

8.3. SEQUALAE

The following are the after effects of sequala of smallpox.

1. Deep pitted scarring of the face and body
2. Permanent blindness
3. Corneal opacity.
4. Otitis-medias leading on to deafness.
5. Alopecia/diffuse or patchy falling of hair.

8.4. PRECAUTIONS

1. Prophylactic vaccination of the rest of the family.
2. Isolation of the patient.
3. Quarantine of the contacts.
4. The room should be well ventillated, cool and dark and unnecessary furniture should be removed.
5. The patient should be confined to bed.
6. Disinfection regularly of all the discharges, excreata and clothes of hte person.
7. To protect the eruption from excoriation and injuries.
8. Nose adn mouth to be washed some mild antiseptic lotion.
9. Eyes to be bathed with boric acid lotion.
10. Frequent baths during convalerence to help loosen the scabs.

8.5. TREATMENT

A Prophylactic—

Prophylactic vaccination properly done usually protects an individual for about three years, so re-vaccination should be done in every 4th year (with freezedried vaccine).

Vaccination is contra-indicated—

(a) If there is eczema and skin disease or pyoderma.

(b) In pregnancy, during the first six months.

(c) In fever, Occular infection or extreme general debility.

Post vacing enchephalitis is an unfortunate but rare complication of vaccination. It usually occurs between the 7th and 10th day. There is malaise, rise of temperature, vomiting, consulsion or coma.

B. General Managements—

(a) Isolation of the sick, till all scabs disappear.

(b) Disinfection of the room and formites, and

(c) Quarantine of contacts for 16 days are essential.

C. Medicinal Treatment—

As a prophylactic during an epidemic, *Variolinum 200 or Malandrium 200 or Vaccinaunum 200* is useful. If this is given as a precaution, the regular attack may be avioded in most cases. If howevwer, an attack appears in any case, it will be mild and can be managed with a few remedies and complications will rarely occur.

If it starts with fever, even after the use of above medicine, Aconite 30, Belladonna 30 or Bryonia 30 may be given according to symptoms and it is followed by Antim tart 30. if there is nausea and vomiting, or Thuja 30 if there are no special symptoms, further development may be arrested or mitigated. If however, the eruption appears Variolinum 30 or Malindrium 200, if continued for a few days, will reduce trouble completely.

LEADING REMEDIES:

1. Aconite It is especially called for during the chill and first few hours of the fever, and when there is severe pain in the head full bounding pulse; thirst; intolerance of light and delirum.

2. Balladonna It may follow Aconite, especially when there is severe headache and delirium; also when there is intolarance of noise.

3. Bryonia It is used for the several backache, pains in the bones, soreness of the chest, and constipation.

4. Variolinum/Malindrinum Probably the most potent of all, having the complete picture of the disease from which it is prepared, Dulness of head. Severe pains in back and limbs, which become quite numb. Chills, followed by high fever. Voilent headache, white coated tongue, Great thirst. Severe pains and distress in epigastric region with nausea and vomiting, mostly of greenish water. In many cases, profuse diarrhoea.

Given steadily, the disease will return a milder course. It changes imperfect pustules into regular ones, which soon dry up. Promotes suppuration and desiccation. Prevents pitting.

9. MUMPS (Parotitis)

• What is mump? What are its clinical features, complications, Lab diagnosis and treatment?

Def.—Mumps is an acute infectious desease caused by Paramyxoma virus (muxovirus parotitis) which enters trhe circulatory system via upper respiratory tract by droplet infection and then commonly settles down the gladular tissues, particularly the salivary glands, and rarely in nervous tissues. particularly the salivary glans, and rarely in nervous tissues.

The children and young adults are the usual subjects. The incubation period is 2—3 weeks with extremes of 12 to 35 days and the disease rapidly spreads amongst the contacts by droplets.

Mumps may relapse after a distinct interval.

9.1. CLINICAL FEATURES:

1. Fever, sorethroat, pain and stiffness of the masseter muscle and swelling of hte parotid region are complained of; the swalling is at first unilateral and is commonly on the left; after 1–5 days the other gland is involved; great pain during chewing and clenching teeth or swallowing and excess salivation may be caused.

2. Headache, malaise and epistaxis may precede the prarotid swelling.

3. Abdominal pain due to pancreatitis or cophoritis may be added.

4. The temperature varies between 37.8°–38.9 °C (100°–102 °F), and settles down in a few days. Most cases have slight temperature for two or three days only.

5. Bradycardia is usual.

6. Tenderness below and behind the angle of the jaw, illdefined swelling of the parotids and rarely tenderness and swelling of the submandibular, sublingual, cerical glands may be elicited.

7. There may be high fever (104–105 °F) delirium oircula-tory, depression and meningeal reaction.

8. The swelling does not usually suppurate nor persists.

9. The temperature comes down in 3–5 day but may rise again due to complications like orchitis, pancreatitis etc.

10. Lymphocytosis and albuminuria may be present.

9.2 COMPLICATIONS

1. *Epididymo*–Orchitis It is the commonest complication and, if bilateral, may lead to sterility. Fortunately, unilateral testicular atropy is more common. Recurrence of temperature with painful swelling of inflamed testical is diagnostic.

2. *Oophoritis*—It is suspected if there is relapse of tempera-

ture with bilateral pelvic pain. Vaginal or rectal examination gives the clue.

3. *Mastitis in women*—It is rare but may be a troublesome complications.

4. *Acute pancreatitis*—It is rare but severe complication which may occur at about the weekend of the initial parotitis. Vomiting, upper abodominal pain, fever and toxaemia are suggestive. Destruction of islet cells of pancreas may lead to diabetes mellitus.

5. *Neurological complication*, e.g. meninngo enchaphatitis, facial palsy, deafness due to VIIIth craninal nerve involvement etc. may occur. Polyneuritis is very rare.

6. *Rarely nephritis*, arthritis and thyroiditis have been recorded.

• [Prognosis*f* Favourable. A single attack confers life long immunity].

9.3. DIAGNOSIS

Diagnosis is clinical. Typical swelling of parotids with mild to moderate constitutionl symptoms draws attention. Submaxillary and sublingnal glands aer also involved. In severe cases swellings on the sides of face and below the mandible make the face like rounded like a fulmoon. Tems perature varies between 100 °F and 103 ° F and settles down in a few days. Most cases have slight temperature for two or three days only. It is usually a self-limiting disease unless one or more complications occur.

Laboratory tests—are rarely required except to confirm doubtful cases like suppurative parotitis due to obstruction (Salivary calculi), tuberculous parotitis, saecoidosis etc. Mikulicz's syndrome (parotid and lacrymal gland enlargement) or mixed parotid tumors can be readily recognised. When there is doutbt, serum antibody titre against the paramyxoma virus or biopsy of tissue may require.

9.4. GENERAL MANAGEMENT

1. "Mild cases require isolation and rest for about ten days with medicine (like Belladonna), and semi liquid diet, as chewing may be very painful.
2. Bed rest in initially necessary.
3. Mericulous attention to oral hygiene is mandatory.
4. Gragling with warm normal saline after every meal should be ensured.
5. Antiseptic gargles may kill normal flora and are not recommended.
6. Symptomatic treatment is compulsary.

9.5 TREATMENTS

A. Preventives

Mumps live virus vaccine is now available and is quite effective, but inactivated vaccine is satisfactory. Mumps hyper immunoglobin may be given to highly suscetible cases after one year of age. Homoeopathically pilocarpus 30 is used as a prophylactics.

• B. Homoeopathic Medicines used.

1. Belladonna. Inflammation of parotid glands especially right sided. Inflamed area is swollen, hot, tender and red.

Intense throbbing pain burning sensation. Pain is by jark motion, chewing, talking and by touch. Patient cannot lie on the affected side. Difficulty in deglutition. Dryness of oral cavity with thirstlessness. Tongue dry, strawbery.

Associated with high fever, throbbing headache. Congestion of head and face. Complaints come on suddenly from taking cold.

2. Mercurius sol. It is the principle remedy, as it has a specific action on the salivary glands. The special symptoms are tenderness, salivation, offensive breath and threatening suppuration. Inflammation is especially right sided, with

profuse offensive saliva, tongue large, flabby and showing imprints of teeth. Foetid odour from couth. Pain in the affected part at night, damp, weather, warmth of bed, from cold or warmthings, *during rest.* Thirst increased with moist mouth.

3. Pilocarpus (Jaborandi)-Dr. Burnett's homoeopathic remedy for mumps seems to surpass all the rest i.e. pilocarpus. It acts quickly, and also relieves the the pain.

Moreover it has a reputation for the metastases in which mumps excels, whether to testes or mammae, when the swelling suddenly subsides, as the result of a chill, and worse troubles supervene. Pilocarpus also acts as a prophylactic.

4. Phytolacca-Inflamation of sub-maxillary and parotid glands with stony hardness. Pain shoots into ear when swallowing. Worse cold and wet.

10. JAPANESE ENCEPHALITIS

• What are the Causes of Japanese encephalitis? Give the clinical manifestations, complications, and Treatment of this disease. What are the sources of Infection?

The synonyms of *Japanese encephalitis are Japanese B encephalitis* (JBE) and *Russian autum encephalitis.*

The *Causative organism* is a 'togavirus' which belongs to group B arbovirus. During recent epidemics repeated isolation of the virus was done in nature from *Culex vishunt* mosquitoes. So this mosquito is suspected to be the most important vector of JBE in our country. Recently this virus also has been isolated from *Anopheles hyrcanus, Anopheles barbirostris and culex epidesmus.* The mosquitpoes become infective 10–20 days after imbibing viraemic blood.

• 10.1. EPIDEMIOLOGICAL CONSIDERATIONS

• SOURCES OF INFECTION–

The virus is probably a parasite of wild birds. Young night hermons act as amplifiers; in them increase of the amount of virus occurs. Domestic pigs also serveas amplifiers. In most

of the species of birds viraemia may remain 1–7 days, and these birds do not show signs of illness. Bats can also maintain viraemia for 6 days and they also do not became sick. Nearly 100% of pigs in an endemic area develop antibodies against JE virus. Viraemia in them is sufficient to infect the mosquitoes, and viraemic has been demonstrated in pigs following the bites of infected mosquitoes.

Vertebrates like pigs, sparrows, herons plumed egrets, swines, certain lizards and possibly bats get infection and maintain it continuous chains.

The perod of viraemia in man is very short. History of contact between cases is rare. Man to man spread of the disease does not occur. Children under five years of age are mostly affected and in them many sequelae may be left.

10.2. PATHOGENCITY

1. Oedema and congestion of CNS occur.
2. There are neuronal degenerations and necrosis of the cerebral cortex, the cerebellum and the cord
3. Perivascular cuffing in general and adjacent tissue is infiltrated with round cells.
4. Degeneration of purkinje cells in the cerebellum is particularly noteworthy and there is often severe involvement of spinal cord also.

10.3 CLINICAL MANIFESTATIONS

Incubation period in man is 6–8 days.
Onset may be acute or gradual.
It may begin with a mild febrile illness.
The first phase of the disease may be absemt or it may be unnoticed, and there may be sudden onset of high fever, headache and there is bradycardia.
The patient is weak and lethergic, and face remains expressionless.
There may be confusion, delirium and convulsioon (this may be the first symptom in children), which may progress

to coma. Motorand sensory disturbances may be present. Speech may be affected. Upper motor neurone type of paralysis may part of the body.

Neck rigidity is usually present, and kernig's sign is positive. Reflexes are disordered.

After initial leucocytosis, leucopoenia follows. The CSF is usually clear, but it is under pressure, and there may be 400 or more cells/mm^3. Protein in CSF is moderately raised.

10.4 PROGNOSIS

The duration of the illness is quite variable sudden improvement andsurprising recovery may occur incertain patients who remain unconcious for days. Convalesence is usually prolonged. Fatal cases usually die within 10 days.

10.5. SEQUELAE

Sequelae are common in children and severe in infants. There are nervousness, emotional or personality changes, tremors, incoordination and mental impairment, Residual paralysis, aphasia, cereballar ataxia, decerebrate rigidity and psycosis may precipitate in certain cases.

9.6. TREATMENT

(i) There is no specific treatment. Accordingly, supportive care is of paramount importance.

(ii) Blood should be examined for malaria and other parasites.

(iii) Devoted attention should be given to air way.

(iv) Bladder and bowel functions should be watched.

(v) Fluid and electrolyte balance should be maintained. Glucose should be given orally or, if indicated,intravenously 25% solution or as 5% drip to tone up liver.
Adequate nutrition should be ensured.

(vi) Measures against bed sores, secondary pulmonary

infection and hyperpyrexia should be taken.

(vii) Specific antiviral agents are still in the area of experimental medicine.

10.7. CONTROL

A Vaccination—Vaccination is usually done with 2 doses formalin inactivated mouse-brain vaccine (1ml/dose, at interval of one week).

No vaccine is prepared in India. Imported vaccine is a bit costly. It is to be had from Government source.

A new line of approach is to infect man with an avirulent B arbovirus, i.e. West Nile (WN), followed by a booster with inactivated Japanese B. Activity of WN virus is reported to be present in India.

According to some, vaccination of amplifier hosts, pigs, is worthy of consideration as a means of breaking the infection link to man. This should be tried in our country.

B. Mospuito control—*Culex vishnui* mosquitoes are probably resistant to the insectides belonging to organochlorine compounds, such as DDT and dieldrin. Organophosphorus compounds such as malathion, fenthion etc, are still considered to be effective in killing them. Ultra low volume (ULV) malthion spray from aeroplane is worthy of trial.

C. Personal protection—Personal protection against mosquito bites by wearing adequate garments from evening and or by applying repellents on exposed parts of the body and by using mospuitonets should be stregthened.

D. Homoeopathic Medicinal treatment

• Belladonna 30 is one of the most important remedies in the beginning, and should be given, in watery solution, one tea-spoon every 2 hours, and continued. In most cases as decided improvement will be noticed after even 24 hours. If this does not help sufficiently, it may be followed by Bryonia 30, one globuledry on the tongue every 2 hours and then Sulphur 30 also in the same manner. Other medicines are

Gelsemium, Glonoine, Apis, Helleborus, Hyoscyamus, Stramonium and Pulsatilla.

(1) ***Bryonia***—Constant inclination to sleep; sudden starting from sleep; with delirium; stars, cries burning and shooting pains through the head; cold sweat on the forehead. It is very often indispensable after Belladonna when the pain is—Violent, lancinating pains shoot through the head from one side to the other.

(2) ***Balladonna***—When the pain is more throbbing and hard, aching, pressing, pressing pains use Bell.

(3) ***Sulphur***—is very useful in desperate cases, and

(4) ***Helleborus***—painful heaviness of the head, with heat in the head, and cold extremities. The pain is less violent when the patient lies still and quit. The pain in the head is os a stupefying nature, as if the brain were bruished. There is a predisposition to bury the head in the pillow. The child sleeps with its half open eyes. The child shrieks and cries when roused up, or taken from its cradle.

11. RABIES (HYDROPHOBIA)

• What is Rabies? What are it's signs and symptoms and treatment.

Rabies is a disease of the central nervous system of dog and wild carnivorous animals. which is accidentlly transmitted by the the bite of an infected animal.

It has been estimated that more than 15,000 people die of rabies in India every ear and more than 3,000,000 persons receive antirabies vaccine.

The *causative organism* is a filterable virus. In sylvatic type of rabies the virus is maintained by various animals, such as jackals, mongoose, foxes, skunks, wolves, racoons and vampire bats.In the urban type dog alone is responsible. Cats and cattle may also be infected.

11.1 INCUBATION PERIOD

Varies from 30 days to 1 year, to many years (average 1–2 months).

11.2. MODE OF TRANSMISSION

By licking of an abraded surface of skin or bite or scratches of an infected dog. Aerosol transmission (specially in cases of vampire bats) is also possible.

11.3. SYMPTOMS AND SIGNS

There are three Stages 1. **Prodromal stages.** May last for 1–4 days, There is fever (102 ° F), headache, malaise, restlesness, sore throat, non-productive cough, and pain in hte scar (in 80%).

II. **Stages of excitation.** This starts within 24–48 hours. Restlessness is intense; there is excitation, agitation, hallucination, mental confusion, hyperesthesia with strong light; loud sound, slight touch, strange smell or even gentle breeze. Hydrophobia means sudden spasm of muscles of mouth, pharynx and larynx. In this condition if a glass of water is offered, a series of spasmodic jerks would be noticed, water from the mouth would be ejected, patient is afraid to taking water. This is known as furious rabies

There is excessive lacrimation, salivation and perspiration, Foaming at the mouth is seen. Spasms of respiratory muscles start. Deep tendon reflexes are increased, ther eis extensor planter response, pupils are dilated, temperature is 105 ° F.

III. **State of paralysis.** If the patient survives long enough, diplopia, facial motor neurone paralysis, paraplegia and hemiplegia occur. Patient is exhausted, helpless and ultimately he lapses into coma. Death is due to respiratory failue. This is also known as *dumb rabies.*

11.4. PROGNCSIS

Rabies in man is 100% fatal; time of survival after onset of symptom is about 4 days, maximum 20 days.

Infected animals may survive. Neuropathological changes are supposed to be relatively minor in man. Considering

these facts it has been suggested that if prolonged cardi-orespiratory services were employed to councteract the dys-function of brain-stem, survival may be possible. As a result of this mode of treatment, a man has survived from rabies. This has created new hope in this dreaful disease.

11.5. TREATMENT

1. Treatment of established cases.

(1) Isolation of the patient in a quiet dark room.

(2) To relieve muscular spasm medicines are used.

(3) Violent spasms may be controlled by inhalation of chloroforms.

(4) Tracheotomy, if facilities are available.

(5) Salivary glands of man are rarely infected. But there are records that human bite can transmit rabies So reasonable care should be taken by the nursing staff while attending the patient.

II. treatment of persons exposed to infection.

A. Treeatment of a bite by a suspected animal.

1. The wound is to be thoroughly washed with soap and water.
2. If the patient comes within 30 minutes the bleeding should be encouroged.
3. The wound should then be washed with potash per-manganate solution.
4. Each tooth mark should be probed and touched with pure phenol.
5. Stitich should be avoided, whenever possible, par-ticularly on face.
6. Tetanus toxoid or ATS should be given.
7. Topical application of antirabies serum, if available, is optional.

The animal (a) if recognised, should be watched for 10 days. If it is almost certain that the bitten person has not been infected and antirabies vaccine is not required.

The vaccine must be taken, if the animal dies with in 10 days or if it remains untraced.

(b) If the bitting animal is dead, its brain (region of Corpus callosum, 2 samples, one in formal saline and the other in glycerine) should be sent to an experienced pathologist or to the veterinary college for the examination of *Negri bodies.*

Antirabies Vaccine.

Nerve tissue derived vaccine (NTV) is available from Pasteur institutes and hospitals. It is given subcutaneously into the different areas of abdominal wall, daily for 14 days(2ml.*f* 10ml. daily).

In head and neck injuries, class III treatment (i.e. children below 10 years should receive 5ml. daily for 14 days and adults should receive 10ml. daily for 10 days) is done. This vaccine in rare cases produces allergic enaphalomyelitis. *Duck embryo derived vaccine* (DEV) is relatively safer. It is not available freely.

When risk is severe, antirabies antiserum of either equinine (40 units/kg.) or human (20 units/kg.) origin can be given IM, part of which may be inflicted around the wound. This is also not available in primary health centres. Subsidiary health centres obtain this vaccine from Primary health centres.

- **Homoeopathic Medicine used.**

The chief are *Belladona, Stramonium, Hydrophobinum* and *Scutellaria lateriflora Cantharis, Lachesis and Fagus sylvatica*

1. *Belladonna*—Throbbing headaches from within outward, stabbing in brain; pains over scalp, as after violently pulling the hair; distorted features; pale face, with thirst; sweat only on face; sensitiveness of hearing; spasmodic

distortion of mouth; head drawn backward and buried in pillow; dysphagia for water; violent, small, frequent, anxious respiration; convulsive covements of limbs, with lassitude and anxiousness; extreme sensibility to cold ari; delirious prattle about dogs which surround him; desirous of dying when free from rage, wants to bite those around him, bites spits.

2. *Cantharis—Alternate paroxysm of rage and convulsions* excited by touching larynx, by making pressure pon abdomen by the sight of water; burning and dryness of mouth; excessive sexual nisis with constant painful erections and continual itching and burning of internal sexual organs;inflammatory symptoms prevail over convulsive ones.

3. *Hydrophobinum* (Lyssin)—Slight dizziness and nausea; severe headache with stiffness of jaws and numb hands; twitching of face and hands; face pale, yellow, nearlybrown; mouth full of salive and total disinclination to drink; saliva viscid with constant spitting; sensation of inability to swallow, but can do so when trying; violent spasm of throat with sense of suffocation; constrictive sensation in throat, much worse when swallowing liquids.

CHAPTER—II

TROPICAL DISEASES

DISCUSSION FOR LEARNING

1. PYREXIA (Fever)
2. MALARIA (MT Malaria).
3. KALAAZAR.
4. GIARDIASIS.
5. FILARIASIS.
6. TROPICAL EOSINOPHILIA.
7. CHOLERA.

1. PYREXIA

Q. 1.1. What is pyrexia? What are the different types of pyrexia?

Pyrexia (fever) is a disease condition in which there is an elevation of the body temperature above the normal level.

NORMAL TEMPERATURE

Axillary—98F [generally 97°F]

Oral—98.4°F [0.5 to 1°F higher than the axillary temperature].

Rectal—99.6°F [0.5 to 1°F higher than the oral temperature].

• **Different Types of Pyrexia**

Pyrexia

Intermittent	**Remittent**	**Continued**
[In which temperature touches the normal line at least once in 24 hours i.e. complete remission daily.]	[In which temperature never touches the normal line within 24 hours and the variation of temperature between maximum and minimum is more than 1.5°F] • Seen in cases of Subteration malaria, Subacute kalaazar, paratyphoid, Influenza, Acute tonsilitis. Bronchopneumonia. Diptheria, Eruptive-fever.	[Like remittent but variation of temperature is not more than 1.5°F] Seen in cases of Lobar pneumonia Cerebrospinal fever Rheumatic fever, Plague, Second week of enteric fever, Acute kalazar. Miliary tuber culosis, Septicemia, Erysipelas etc.

Quotidian	**Tertian**	**Quartan**
[Every day rise of temperature again touches the normal line within same day. e.g. Malaria, Tuberculosis.	[Temperature rises and falls every other day seen in case of Benign tertian malaria.]	[Temperature rise and falls every third day, Seen in case of malaria.

Q.2.2. What are the types of onset, course and termination of pyrexia?

TYPES OF ONSET

According to *Savill's system* of *Clinical Medicine*, the types of onset of pyrexia are of following types—

1.*Sudden*—seen in case of Influenza, Tonsilitis, Pneumonia.

2.*Gradual*—seen in case of Tuberculosis and sometimes Typhoid fever.

3.*Recurrent*—i.e. a fresh rise of temperature after the initial period of pyrexia subsiding e.g. seen in relapse of Enteric fever or in Acute kalaazar.

THE COURSES OF PYREXIA

This can be divided into 3 stages

1.Initial stage,—"Stadium incrementi"

In this stage, temperature rises either abruptly as in lobar pneumonia, insidiously as in typhoid (ledder like rise).

2.Stage of maximum rise or "stadium fastigium"

In this stage, the temperature reaches its maximum and then runs continuously or settles to normal after lasting for few hours only (intermitten).

3.Stage of decline or "stadium decrementi"

In this stage, temperature falls to normal either by *crisis or lysis.*

TYPES OF TERMINATION

1.*Sudden i.e. in crisis*—This means sudden fall of temperature to normal or below normal within 6 hours or so. This is usually associated with profuse sweat. This is found in Lobar pneumonia, Influenza, B. coli infection etc.

2. *Gradual i.e. in lysis*—This meant gradual fall of temperature and may take few days to settle to normal e.g. in Typhoid.

Name some Protozoal infections causing Pyrexia.

1. Plasmodium vivax

2. P. falciparum Malaria
3. P. ovale
4. P. malarial
5. Leishmania donvovani—Kala azar.

2. MALARIA

• What is Malaria? What are the causes of Malarial fever? What vector is responsible for transmission of malaria? Whar are the various types of malaria? Describe a case of cerebral malaria or malignant tertian (MT) malaria. How will you diagnose a case of malaria. What are the typical signs and symptoms of malaria?

Introduction:

Malaria has once again raised its head in India, and cases are occuring all over the country. Medical practitioners should be watchful, as some of the cases will be missed in the early stage, unless this possibility is remembered. The parasites enters human body during bits of infective female *Anopheles mosquito*; hence presence of this mosquito in the area is the real warning. Infection may also occur via blood transfusion, improperly sterilised syringes or defective placental circulation.

2.1. WHAT IS MALARIA?

Malaria is a Protozoan disease transmitted to man by the bite of Anopheles mosquitoes (female), *causative organism is genus Plasmodium.*

Four species known to infect man are—

1. *Plasmodium vivax* causes Benign tertian—(BT) malaria.
2. *Plasmodium malaria* causes quartan—(QT) malaria.
3. *Plasmodium falciparum* causes malignant tertian—(MT) malaria.

4. *Plasmodium ovale* causes tertian malaria, is relatively rare and mild illness.

2.2. WHAT ARE THE CLINICAL FEATURES OF DIFFERENT TYPES OF MALARIA?

* ***General***—there is some variation in malaria produced by different *plasmodium* but in all—chills, fever, headaches, muscle pains, splenomegaly and anaemia are common. *Harpes labialis* may sometimes appear.

* ***The hallmark of this disease***—is malarial paroxysm which recurs in all, except falciparum infection. The typical paroxysm begins with rigor for 20 to 60 minutes followed by high temperature for 3 to 8 hours and then defervescence with profuse diaphoresis leaving the patient exhausted.

**The febile paroxysms are of following stages—

(a) the *cold stage* (20 minutes to one hour.)

(b) the *hot stage* (1-4 hours.)

(c) the *sweating stage* (2-3 hours).

The paroxysm recurs every 3rd day in tertian fever and, every fourth day in Quartan fever.

(a) In Tertian Malaria—

The prodrome of myalgia, headache, chilliness and low grade fever for 48 to 72 hours heralds the onset of typicals paroxysms which occur on alternate days. If there has been double infection with two maturation cycles in which case daily chills can occur; such double infection synchronize within a week and then follows the classic tertian pattern.

(b) In Quartan Malaria—

In this infection paroxysms occur every third day, unless multiple infections alter the cycle initially and chills occur on 2 out of 3 or even daily until the cycles synchronize.

Quartan malaria is a more disabling infection than tertian. Oedema, albuminuria and haematuria, a clinical state similar to acute haemorrhagic nephritis may appear occasionally.

(c) In Estivoautumnal malaria—

Severe disease where agglutinated masses of parasitized erythrocytes block capillaries through out the body. There is asynchronization of the cycle of multiplication. Typical malarias paroxysms occurs but continuous remittent or irregular fever is present in many cases.

• CEREBRAL MALARIA
[Malignant Tertian (MT) Malaria]

Q.2.3. Describe a case of Malignant Tertian Malaria (Cerebral malaria).

Clinical Signs and symptoms related to central nervous system, due to MT malarial parasite (Plasmodium falciparum) *are collectively classified as cerebral malaria.* Hyperpyrexia and peripheral ciculatory failure may also occur in some cases. When there is severe collapse and shock, temperature may not be raised and this is known *as aglid* malaria.

(a) Pathology.

Blockage of capillaries and pre-capillaries occurs by masses of parasitized agglutinated red blood corpuscles. Occasionally intravascular coagulation takes place. The endothelium of brain vessel is affected causing local leakage of protein and fluid.

(b) Clinical features:

Neurological symptoms usually appear during the course of an untreated attack in a person, who is non-immune. But involvement of central nervous system may be the first indication of infection. Malnutrition, specially in children, predisposes to cerebral malaria.

The symptoms are hyperpyrexia (41.6°C=107°F or more), headache, drowsiness, convulsion, specially in children, incontinence of faces and urine, delirium and coma. Pupils are often unequally contracted. There may be localized muscular twitching. Splenomegaly may be present.

In some patients, consciousness is retained, the patient responds to questioning or physical stumuli. In others, mental disturbances may be seen. Behaviour is changed. In subordination in early stage, thereafter excitement and mania are observed. Coma follows. In children there may be diarrhoea and vomiting.

Hemiplegia may occur. Mortality is nearly 50 per cent, if diagnosed and treated late.

MT malaria, if not properly treated, may be serious and some of the cases may present with cerebral symptoms, G1 tract symptoms or cardiovascular symptoms.

(C) Differential Diagnosis of MT Malaria.

1. Heat hyperpyrexia.
2. Encephalitis.
3. Typhoid fever.
4. Meningitis.
5. Uraemia with pyelonephritis.
6. Brain abscess.
7. Cerebrovascular accidents.

Other causes of coma i.e. diabetes and narcotic poisoning, when associated with fever due to secondary infection, should also be excluded.

(d) How can we Diagnosis a case of malaria?

1. Diagnosis is done by the intermittent nature of fever, bilious vomiting, coated tongue, hepato-splenomegaly anaemia, Jaundice, and by demonstration of malarial parasites and gametocyte in a thick

and thin blood films should be properly stained to settle the diagnosis. If these are negative routine leucocyte count, haemoglobin estimation and urine examination.

2. Lumbar puncture should be done to exclude viral or pyogenic meningitis.

3. Other investigations, e.g. serum bilriubin, blood urea, blood culture, even X-ray skull may be considered, when necessary.

2.3. (e) TREATMENT

Malignant malaria is a medical emergency. Prompt, energetic and effective treatment is required to save the life of the patient.

* Homoeopathic Medicine used for Treatment

1.Ipecac—When there is any doubt in regard to choice of a remedy especilly at the commencement of the disease, this remedy may be given. Ipecac is specially indicated if *large doses of quinine had been given*, or if the fever commences with an internal chill which gets worse in the warmth; little or nothing in the cold stage, but a great deal during the hot stage; clean or slightly coated tongue; nausea and vomiting, and oppression of the chest immediately before the attack, or during the cold and hot stages.

2.Nux vom—It is very frequently used after *Ipecae* more particularly if, at the onset of the paroxysm, the extremities feel as if paralysed and *chill and heat are mingled*, one being felt externally, the other internally, *with dread of being uncoverd in the least even during the hot and sweating stages* external warmth affords no relief. Giddiness with a feeling as if drunk, cramps in the muscles of the abdomen or calves of the legs; stitches in the sides; heat and pain in the head; buzzing in the ears; thirst and anxiety during the hot stage constipation.

3.Arsenic—It is always useful in all cases suppressed by large quantities of quinine.

Time of fever—1 to 2 P.M. or 12 to 2 A.M. In this remedy the different stages are not distinctly marked, but the chilliness, heat and sweat occur simultaneously, or when there are frequent changes from chilliness to heat, and vice versa, or internal heat; also when the paroxysm is imperfectly developed; when there is little or no sweating, or at least not for some time after the heat has subsided; great prostration of strength; burning pains in the stomach, insupportable pains in the limbs, or all over the body; *anxiety and restlessness; excessive thirst*, dringking often but little at a time; uneasiness about heart or oppression and spasms of chest; nausea or sickness and vomiting; bitter taste in the mouth; violent headache; continuing after the hot stage; buzzing in the ears during the sweating. *All the sufferings* of the patient as, the headache, pain in the limbs etc, are *increased during the attack.*

4.China—The paroxysm begins generally towards midday, with intense thirst long before the chill, but no thirst as chill increases or during heat, the thirst begins after the fever subsides, and increases when the sweat begins; exhausting sweat, after the fever subsides; exhausting night sweats followed by ringing of the ears, with sensitiveness in the regions of the liver and spleen; scanty urine, loss of appetite and bloated abdomen are present as after effects of the fever.

What are the typical signs and symptoms of Malarial Fever?

The patient is having 3 stages in the development of the fever.

1. *Cold stage or Stage of rigors*—patient shivers from head to feet, his teeth chatter and he covers himself with blankets. The temperature goes on rising.

2. *Hot stage*—Shivering abates and give place to feeling of intense heat. Flushed face; headache, vomiting, dry burning skin, temperature rises up.

3. *Sweating stage*—Patient breaks into profuse perspiration and the temperature rapidly declines with feeling of relief.

This is a typical history of malarial fever. If the paroxysm occurs on every fourth day's of short duraiton; this will be a *Quartan Malaria* due to malarial parasites of *plasmodium malariae.*

3. KALA—AZAR (VISCERAL LEISHMANIASIS)

• What is Kala-Azar? What is the cause for Kala Azar? What vector is responsible for its transmission? How will you diagnosis Kala azar? What are it's complications? What are its pathology? Name three homeopathic medicines with their indications.

3.1. WHAT IS KALA-AZAR?

Kala-azar is a tropical and subtropical disease of all ages and both sexes, caused by protozoon *Leishmania donovani* which is transmitted by female sadfly *Phlebotomus argentepes* from man to man in India.

In other countries dogs and several other animals may also harbour the parasite. Rarely it may be transmitted through blood transfusion. Congential infection in infants is also known to occur. Incubation period is difficult to determine but may be weeks to months.

3.2. PATHOLOGY.

The parasite is ovoid, about 4x2 in size, having a large lateraly placed nucleus and a small rod-shaped nucleus almost tangentially. In culture on N N N (Nicolle, Novy, McNeal) an elongated Promastigote (flagellated) form is seen. This form also develops in the gut of the insect vector after it sucks the infected blood from a patient.

Generally proliferation of the organism takes place in the liver, spleen, bone marrow and lymph nodes. Changes may appear in the skin, as hypopigmented macules or papillomatous nodules. Bone marrow depression may take place resulting in leucopoenia, thrombocytopoenia and anaemia. Liver functions may be affected. This is gross nutritional

deficiency of the liver due to compression of the sinusoids by the swollen kupffer's cells. In the long run this may possibly result in cirrhosis of liver in untreated or inadequately treated chronic cases.

There is a significant rise in fraction of serum gammaglobulins with reversed albumin-globulin (AG) ratio.

* 3.3. CLINICAL FEATURES:

1. Incubation period is usually one to two months but may exceed one or two years.
2. Onset is insidious.
3. Some patient may remain asymptomatic and detected only accidently, due to loss of health and splenomegaly.
4. The fever may be irregular or may show malarial or typhoid like onset. It may be confused with tuberculosis, brucllosis, Hodgkin's disease and even subactute bacterial endocarditis.
5. Wasting and weakness are progressive.
6. In about 20% cases four hourly temperature chart may show double or triple rise in 24 hours.
7. Pulse is rapid and of good volume.
8. Tongue is clear.
9. Anaemia is present.
10. Abdominal discomfort due to huge splenomegaly may be the presenting feature. Later on cirrhotic features may develop.
11. Jaundice, oedema and hypoproteinaemia may appear.
12. Low Platelet Count may result in epistaxis, bleeding from the gums or from any other source. Contrary to this the women patients may have amenorrhoea.
13. Some may complain of arthalgia.
14. Skin may be dry, rough, hypo or hyper-pigmented leading to blackening befitting the name Kala-azar.
15. Hair may fall out.

16. Stomatitis and diarrhoea occur.
17. Hookworm and other helminthic infestation are common associations.
18. Intercurrent infections, such as bronchopneumonia, pulmonary tuberculosis and dysentery are not uncommon, if patient goes untreated. Death is certain in such cases, if not treated.

3.4. DIAGNOSIS:

(1) Patients with irregular fever, splenomegaly, emaciation and in late cases skin pigmentation particularly from endemic areas, deserve consideration of kala-azar as the first diagnosis.

(2) Malaria and kala-azar may co-exist. So a therapeutic trial of chloroquine to exclude a malaria has some value.

(3) Typhoid like onset may rarely occur, but then absence of prostration, clean tongue and fairly rapid pulse in kala-azar will be significant.

(4) *The formal gel test* by adding a drop of commercial formalin to 1ml. of patients serum and looking for opacification with or without coagulation instantaneously or within 20 mm., 2 hours or 24 hours may give a clue towards the possibility of kala-azar.

(5) *Peripherial blood picture* shows leucoponeia with low neutrophil count and increases of monocytes. The eosinophil count may be high due to associated helminthiasis.

(6) The diagnosis is established by detecting the amastigate forms of *L. donovani* (L.D. bodies) in the materials obtain from sternal or splenic puncture stained with Leishman's stain. Parasites may also be found in the scrapings from the skin lesions.

3.5. COMPLICATIONS

1. ***Respiratory***—epistaxis, bronchitis, broncho-pneumonia pulmonary tuberculosis etc.

2. ***Alimentary***—cancrum oris, bleeding gums, diarrhoe.
3. ***Cardiac***—enlargement of the heart.
4. ***Cutaneous***—dermal leishmanoid.
5. ***General***—Oedema, anasarca, cachexia, haemorrhage from various sites.

3.6. HOMOEOPATHIC MEDICINE USED FOR TREATMENT.

1.Nat mur—Patient anaemic, upper part of the body emaciated, inclined to take cold; patient cravessalt. Swollen spleens resulting from kala-azar. *Nat mur* produces stitches, pressure and congestion in the spleen. Much quinine taken is an addtional indication.

2.China—This remedy corresponds to congestion, pain and stitches in the region of spleen and liver with their swelling. Dull aching in region of spleen and liver. Hyperaemia of spleen. Nervous system is very sensitive, physical or mental effort aggravates.

3.Ceanothus—Pain in whole left side, with shortness of breath. Chronic pains in the spleen, worse in damp weather. Pigmentation of skin due to prolonged exhausting attack of fever.

4. GIARDIASIS.

• What is Giardiasis? Give it's clinical manifestations and treatment?

Giardiasis is a common disease of the gastro-intestinal tract with the flagellated parasite, Giardia lamblia which is often found in this continent. It requires adequate attention of the physician concerned as because sometimes it is misdiagnosed. In other cases vague gastro-intestinal complaints are described as symptoms of giardiasis.

* **GIRDIA LAMBLIA**—This symmetrical pear shaped parasite has both trophozoite and cystic forms. The length of the trophozoite is 10-25 mm, it is rounded anteriorly and tapered posteriorly. At its flattend ventral side there is a shallow sucking disk on the anterior portion. It has two oval nuclei and 4 pairs of flagella. Cyst measures 8-0m. in length, oval or elliposid, contains 2-4 nuclei and 4 pairs of bristle shaped curved axonemes containing retractile flagella.

4.1. CLINICAL MANIFESTATIONS:

(a) When the infection is light, the patient may remain asymptomatic.

(b) In moderate infestation the following symptoms may occur—Chronic recurrent nausea, mild cramp in the abdomen, distention of abdomen, flatulence and mild diarrhoea. There may be 2-10 motions/day. The faeces may contain mucus, but blood and pus are absent.

(c) In *severe infection*, in addition to the above symptoms vomiting and fever occur. Chronic patients may lose weight. Other symptoms of mal-absorption may supervene. Persons suffering from hypo-gamma globunopathies are susceptible to *G. lamblia* infection. Giardia is the most common cause of diarrhoea, steatorrhoea and morphological abnormalities of the small intestine in these patients. The parasites can invade gall bladder. The trophozoites of *G. lamblia* are sometimes found in the bile taken from the gall bladder. The significance of this finding is not well understood.

4.2. DIAGNOSIS

When diarrhoea or mal-absorption is unexplained, giardiasis should be excluded first.

4.3. PROGNOSIS:

As it has been pointed out, in some persons infection may not produce any symptom. In others there may be waxying

and waving of the symptoms. Treatment may cure 90% of infections and sometimes more than one course of treatment is required.

4.4. PREVENTION

(i) Protective immunity does not develop.

(ii) Re-infection often occurs.

(iii) So prophylactic medicine is not usually recommended.

(iv) Treatment of infected persons.

(v) Personal Hygiene, and

(vi) Environmental hygiene should be maintained adequately.

• Homoeopathic Medicine used for Treatment:

1. **Sulphur**—For persistent or chronic cases of Giardiasis **Sulphur** is the remedy; the tenesmus continues, in fact there is a sort of tenesmus all the time, the stool are slimy and there is frequent sudden urging to stool. Sometimes this condition is present without tenesmus.

2. **Aloes**—The stools are of a jelly-like mucous, and covered with slimy white mucous and accompained by griping in the epigastric region, the amount of mucous expelled is large.

3. **Rhus tox**—Tearing pains down the thighs as an accompaniment of Giardiasis would indicate *Rhus Tox.*

5. FILARIASIS

• What is Filariasis? Give its clinical features and treatment.

Filariasis is cuased in our country due to (a) *Wuchereria*

bancrofti and b) **Brugia malayi**. It is estimated that at least 250 million people throughout the world are infected with these worms. In India, about 5 million people are estimated to be suffering from *Bancroftian flariasis.*

In India, periodic nocturnal form occurs. Microfilariae circulate in the blood at night (peak hours 10 p.m. to 2 p.m.). Mosquitoes imbibe the microfilariae from infected persons. The microfilariae develop into infective stage in the throax of the mosquitoes. A healthy person acquires the infection through the bite of the infective mosquito. *Here Culex pipiens* (Culex fatigans) mosquitoes transmit W. bancrofti. The vectors of *B. malayi* are *Mansonia* spp. and *Anopheles barbirostris* mosquitoes.

5.1. CLINICAL MANIFESTATIONS

Some persons having microfilariae in the blood remain asymptomatic. In others the following manifestations may occur.

(1) *Lymphangites* mainly involving the lymphatics of testes and epidydimis, accompained by high fever (103°-104°F), chill and rigor.

(2) *Lymphadenitis* (mainly groin, sometimes exilla)

(3) *Dilatation of lymphatics*, i.e. lymphangio varix.

(4) Dilated lymphatics may rupture and lymph hydrocale an lymphuria may occur. Varicose chyle vessels running through mucus membrane of the urinary passage may rupture and chyle through the urine may come out (chyluria). Similarly, chylous diarrhoea, chylothorax, chylous ascites etc. may occur. These are rare manifestations.

(5) *Lymphoedema and elephantiasis* Elephantiasis is the end result of filariasis. This is hyperplasia of skin and connective tissues of the affected part.

(6) Super-infection with *Staphylococcus aureus* or *Streptococcus pyogens* may lead to septic lymphangitis or abscess formation.

5.2. TREATMENT (Homoeopathic Medicine used.)

(1) **Merc sol 30**—For this fever with the attendant symptoms, chill, glandular swellings, painful swelling on the calves of the legs, high fever *Merc sol 30 acts as a specific.* It may be given in the 200th potency at fortnight intervals, to prevent a recurrence of the trouble. In chronic cases when the fever recurs periodically use 1 M or higher even when chronic swellings of the legs remain after the fever.

(As this is a trouble some complaint, prolonged treatment with the very high potencies is necessary for a case which is possible in recent cases).

2. Bell and Apis—Lymphangitis especially in women suffering from tearing pains in scalp and nape of necks, with high fever, followed immediately by the appearance of nodes and hard cords about the head, soon disappearing. Only to be followed by another attack *Ars Iod, Car carb, Calc Iod etc.*

6.TROPICAL EOSINOPHILIA.

[**Synonym**—Eosinophilic lung, Pulmonary tropical eosinophilia, Weingarten's syndrom]

• What is meant by Tropical Eosinophilia? Give its aetiology pathology, clinical features, differential diagnosis and Treatment.

Def. *It is syndrome characterised by spasmodic cough, paroxysmal dyspnoea, absolute eosinophilia and is sometimes associated with systemic manifestations like fever, loss of weight and anaemia. Rarely haemoptysis also may occur.*

6.1. AETIOLOGY

1.All age groups, right from infancy, may suffer, but the majority are between 20 and 50 years of age. Males are more affected than females and there is some genetic predisposition.

2. Infection by some form of *filaria* is commonly associ-

ated with its aetiology. Larval introduced into the body by mosquito develop into adult worms, somewhere in the tissue. These adult worms produce macrofilarae which are rapidly killed in the tissue exciting an eosinophilic response at various sites as well as in blood.

3. *Aspergillus fumigatus* may also cause asthma and eosinophilia, as well as pulmonary infiltration.

4. *Ascaris lumbricoides* may cause similar syndrome.

5. Idiopathic case are seen where no aetiology can be demostrated.

6.2. PATHOLOGY

The cut surface of the lung may show irregularly scattered whitish nodules 3-5 mm in diameter. Microscopic examination shows groups of alveoli distended with macrophages, eosinophils and fibrin. In the centre of some nodules lung tissue gets destroyed leading to formation of eosinophilic abscesses, in some of which microfilariae may be seen, surrounded by hyaline material.

6.3. CLINICAL FEATURES

A. Symptoms:

(a) **In acute variety**—Sudden onset of high fever with cough and rapid breathing.

(b) **In chronic variety**—onset is gradual with malaise, anorexia and low grade fever. Later on hacking cough in paroxysms at night. At the end of about of coughing, sticking mucus is expectorated. Dyspnoea on exertion may occur.

B. Signs:

Hyper-resonance, prolonged expiration and a few crepitations. Spleen may be palpable. Liver and lymph glands may enlarge.

C. Radiology:

Chest X-ray may be normal. The commonest change is acentuation of bronchovascular markings. Other findings might be miliary mottling, pulmonary opacities, hilar gland elargement and pleural effusion. Occasionally mottled shadows became confluent to produce a patch of pneumonitis.

D. Investigations:

(a) **Blood—**

Total white cell count is 10,000 to 80,000 or more. The hall-mark of diagnosis is 'absolute eosinophilia'. The total eosinophil count should be at least 2000/c.mm.

(b) High E.S.R.

(c) *Bone marrow* shows proliferation of the eosinophilic series of cells.

(d) *Sputum*—presence of eosinophils may be a clue.

(e) Filaria Complement Fixation Test—Postive (Test becomes negative after successful treatment).

(f) *Serlogical findings*—Raised serum globulin levels.

(g) *E.C.G.*—Ist degree Heart Block, Corpulmonale sometimes.

6.4. DIFFERENTIAL DIAGNOSIS OF EOSINOPHILIA

(a) Parasitic infections, fungal infections specially aspergillosis, drugs, serum therapy and chemical agents, penicillin, sulphonamides, P.A.S., mephenesin, nickel salt etc.

(b) Collagen disorder, malignant neoplastic disorder.

(c) Other infections like tuberculosis, brucellosis, coccidiomycosis.

6.5. PROGNOSIS

(a) Good, if adequately treated.

(b) Pulmonary fibrosis occurs in inadequately treated cases. Ventilation is restricted and diffusion impaired. This may result in cor-pulmonale.

6.6. HOMOEOPATHIC MEDICINE USED FOR TREATMENT

1. **Dulcamara**—When excited by exposure to cold or damp, when occuring in wet weather, or attended with some fever, bitter taste in the mouth, diarrhoea at night, foul tongue, and violent itching and burning in skin. Even in cases of much sneazing with bronchitis.
2. **Arsenic**—If caused by eating unripe fruit, or in severe cases, worse at night, followed by a croup-like cough; also after the skin disease has been suddenly suppressed forming Asthma.
3. **Nux Vom**—when there is considerable gastric derangement with constipations and when the skin eruption is excited by indulgence in spiritous liquors.

7. CHOLERA.

• What is cholera? Whar are its aetiology and clinical features? How can we cure, manage and prevent a case of cholera? What would be the indications for the use of saline? Describe about oral rehydration are the therapy. What is meant by cholera infantums?

Det: *Cholera is an acute infectious disease characterised by frequent purging with copious rice water stool and vomiting followed by dehydration, electrolite loss, resulting collapse, muscular cramps and suppression of urine; collapse attended with high mortality, if untreated.*

7.1. AETIOLOGY

The disease is caused by both by classical *vibrio cholerae and bio-type El Tor vibrio*—of *O sub-group I of Inaba, Ogawa and Hikojima* sub-types but only first two sub-types are common in our country. There are three toxins in the cell free filtrates of cholera vibrio, viz, endotoxin, heat stable exotoxin and heat labile exotoxin. It has been detected that heat labile exotoxin (enterotoxin) mediates movements of water and ions from tissues into the lumen and hence it is responsible for pathogenesis of cholera.

Biochemically, these vibrios ferment sucrose, and manose, with production of acid only but lactose and arabinose are not fermented.

El Tor bio-type vibrio is morphologically, biochemically and seriologically identical with true cholera vibrio. But El Tor is resistant to Mukerjee's cholera phage IV and polymixin. Moreover it agglutinates chicken R.B.C.

El Tor produced cholera Epidemic in 1397, and pandemic of 1961.

7.2. CLINICL FEATURES

The severity of cholera is dependent on the rapidity and duration of fluid loss. Epidemiological studies have shown that more than 90 percent of *El Tor chloera* cases mild and Clinically indistinguishable from other acute diarrhoeas. However, a typical case of cholera show 3 stages—

(a) Stage of evacuation.
(b) Stage of colapse.
(c) Stage of Recovery.

(a) Stage of evacuation.

The onset is abrupt with profuse, painless watery diarrhoeas followed by vomiting. The patient may pass as many as 40 stools in a day. The stools may have a *'rice water'* appearance.

(b) Stage of collapse.

The patient soon passes into a stage of collapse because of dehydration. The classical signs are—

Sunken eyes,
Hollow cheecks,
Scaphoid abdomen,]
Washerman's hand and feet,
Absent pulse,
Un-recordable blood pressure,
Loss of skin elasticity,
Shallow and quick respirations.

The out put of urine decreases and may ultimately cease. The patient becomes restless, and complaints of intense thirst and cramps in legs and abdomen. Death may occur at this stage, due to dehydration and acidosis resulting from diarrhoea.

(c) Stage of Recovery.

If death does not occur, the patient begins to show signs of clinical improvement. The blood pressure begins to rise, the temperature returns to normal, and urine secretion is re-established. If anuria persists, the patient may die of renal failure.

The classical form of severe cholera occurs in only 5—10-percent of cases. In the rest, the disease tends to be mild characterised by diarrhoea with or without vomiting or marked dehydration. As a rule, mild cases recover in 1-3 days.

• **Epidemiologically**, cholera due to El Tor biotype differs from classical cholera in the following respects—

(i) a higher incidence of mild and asymptomatic infection. This implies that the characteristic picture of rice-water stools and other signs of classical cholera described above may not be seen often,

(ii) fewer secondary cases in the affected families;

(iii) occurance of chronic carriers, and

(iv) Since El Tor vibrois are more resistant than classical Cholera vibrios, they survive longer in the extra-intestinal enviornment.

7.3. PREVENTIVE MEASURES

1. Cholera inoculation.
2. Isolation of the patient or immediate hospitalisation.
3. Vomitus and excreta of the patient to be destroyed.
4. Sterilisation of the linen and clothing.
5. Milk and water to be boiled.
6. Food to be protected from flies.
7. Salads and raw or over ripe fruits to be forbidden.
8. Hands must be well washed before eating..
9. Drinking vessels and eating utensils should be cleaned with boiling water and kept away from flies.
10. Starvation to be avoided during cholera epidemic.

7.4. CURATIVE MEASURES

(1) Warmth to be maintained with the help of hot water bottles, warm; blankets etc.
(2) Replacement of the fluid and salts by giving rectal subcutaneous, intervenous saline or oral rehydration.

• 7.5. HOMOEOPATHIC MEDICINAL TREATMENT

Camphor Tincture should be given in all cases in the beginning.

When cholera is fully developed—

1. **Veratrum Album—30** is the Principal remedy with the following characteristic symptoms—

   (a) Sharp cutting pains in the abdomen.
   (b) Profuse watery stools like rice-water, with nau-

sea, vomiting, with a cold forhead and great weakness.

(c) Violent thirst for large quantities of very cold water and acid drinks, which is vomited soon after.

(d) Great sinking and empty feeling in the abdomen and great exhaustion after each stool.

(e) Moderate cramps in hands, feet and calves, with suppression of the Urine.

(f) Excessive coldness; skin withered and wrinkled.

2. Cuprum met—30 after **Veratrum alb.** when that remedy has not relieved the cramps, which is very violent and extend all over the body.

When the evacuations are not very copious, but the spasms in the chest and stomach are very painful, with great tenderness to touch, the spasm coming on in peroxysms, both in the stomach and in the extremities; the thirst is moderate the vomitings is allayed for a time, by drinking water; the face is blue and cold; the respiration is short and laboured, the voice is husky; and the Urine suppresses *Cuprum met—30* is the remedy.

3. Arsenic album.

When the purging and vomiting become very frequent; the evacuations from the bowels being thin, watery and of dark colour and very offensive smell, or light coloured and almost without smell, especially when accompained by intense buring pains, or cramps in the stomach and bowels, with violent thirt, and great prostration of strength; also burning in the anus and rectum with tenesums. It is useful also in the last stage of the disease.

During prevalence of Cholera as an epidemic Hahnemann recommended Cuprum met (ars) 30, and Veratrum alb 30, in a rotation of every six or seven days.

7.6. MANAGEMENT

Management of cholera and other dehydrating diar-

rhoeas is almost the same. The patho-physiological changes in cholera are due to rapid loss of a large amount of isotonic fluid from the gut. Acidosis occurs as result of base-deficit. The object of treatment is to correct water and electrolyte loss and the acidosis by administration of fluids containing electrolytes and alkali, the mode of administration depends on the severity of a particular case.

The most rapid way of correction of dehydration is by the intravenous route and several types of IV fluids are used for the purpose. No hypertonic fluids are used intravenously.

The different IV fluids commonly used are—

(a) **Normal Saline and isotonic alkali solution (2:1)** may be used. Iso-alkali solution. i.e. 1.9% sod-bi-carb solution is recommended.

(b) **5.4:1 solution contains 5 gm of sodium chloride,** 4 gm Sodibicarb and 1 gm,. of Potassium chloride per liter of water,

(c) A specially prepared **diarrhoea treatment solution** which contains glucose 8 gm, Sodi-chloride 4 gm. Sodi acetate 5.6gm, and Pot. Chlor 1 gm. in 1 litre of water, may also be given.

(d) **Riger-lactate or Hartmann's solution** is commercially available, ready for use intravenously. (Most suitable for children).

N.B.

• How can we use saline?

The essence of treatment lies in rapid correction of biochemical changes and attempt should be made to correct this within 2 hours in an adult and 6-8 hours in children. In a severely dehydrated patient the rate or administration is one litre in 15 minutes (for an adult) until an easily palpable radial pulse appears. There after the rate should be slowed down to 1 litre in 30-35 minutes. The use of a large bore needle (No.18) is important.

• **The initial quantitity of IV fluid can be determined** from the body weight of the patient. A severely dehydrated patient with shock, has lost 10% of his body-weight. Thus a patient weighing 40 kg. will require four litres of fluid for initial correction. Careful record of pulse rate and Urine is a good guide, if no facility for weighing as patient exists. IV fluids is started at the rate of one litre in 15 minutes. As soon as an easily palpable radial puls appears, the rate of IV administration is slowed down to a litre in 30-35 minutes. When the pulse rate is 90 per minute in adult and 100-105 per minute in children below 5 years of age, IV administration is to be stopped. Thereafter the patient is to be maintained with oral fluid only.

• **Fluid requirement**—can also be determined with the help of Plasma Sp. gravity (PSG). A patient will require 4 ml. of fluid per kg. of body **wheight** for a rise of PSG of 0.001 above 1025 (Normal vlue). **PSG** is best determined with **TS Meter.**

• **Maintenance**—After initial correction, maintenance is done by replacing the volume lost in stool by *IV route or orally.*

• 7.7. ORAL REHYDRATION

Glucose is actively absorbed through the gut, and in the process carries water and electrolyte along with it. This absorption continues even in the face of diarrhoea. Glucose solution containing electrolyte has therefore been used for rehydration. It has been extensively used and found to be highly effective. Most of the mild and moderately severe cases of dehydration respond to oral therapy alone.

Composition:

Sodium chloride	3.5 gms.
Sodium bicarb	2.5 gms.
Potassium chloride	1.5 gms.
Glucose	20 gm.
Water	1 litre.

Any drinking water will also serve the purpose. The fluid should be given orally; quantity in an hour should not exceed 1 liter for an adult and 200 ml for child (below 5 years).

Larger quantity excites vomiting. However, if any case fails to improve with oral fluid (which occurs rarely), IV fluid should be started immediatly.

• Present Market Name—Electroral Powder.

•• SPECIAL NOTE:

1. Criteria for adequency of treatment of the cholera.

(a) Return of pulse rate and blood pressure to normal.
(b) Normal skin turgor.
(c) Sense of well being of the patient.
(d) Normal Urinary out put; this may require 12-24 hours following initial correction of dehydration.
(e) Normal plasma Sp. gravity.

2. Indications for saline:

(i) When Sp. gravity of blood rises to 1060 or above.
(ii) Blood pressure below 80 mm. of Hg.
(iii) Pulse low in volume or tension or no pulse.
(iv) Marked signs of dehydration.

3. Contra indications for I.V: Salina:

(i) Cardiac asthenia.
(ii) Lung complications.
(iii) Rectal temperature high or over 100°F.
(iv) Obstinate tympanitis.
(v) Fair pulse.

4. Advantages of Oral Fluid.

(a) It is effective in correcting dehydration. in most cases. Patient may not pass into a state of shock if instituted early.
(b) The ingredients are cheap and easily available.
(c) No sterilization is necessary.

(d) Over hydration is impossible.
(e) It can be administered by paramedical staff or even patient's relatives.

7.8. WHAT WOULD BE THE DIETIC MANAGEMENT IN CASE OF CHOLERA?

If there is no vomiting, oral feeding should be encouraged even in presence of diarrhoea. Semifluid diet may soon be changed to light, easily digestible, low residue meal till full normal diet is tolerated. If the stool is vibro free, hospital stay should not be more than 24 hours, once the diarrhoea has stopped.

*[Generally for the first 2 days only boiled cold water or green cocoanut water or iced saline 0.5% with 2% glucose. Frequent drinks not exceeding, 2 ozs at a time to be given. Later barley water, whey, skimmed milk etc. are allowed].

In conclusion, it may be said that the treatment of cholera has been made simpler so that it can reach even the remotest corner of the country. Introduction of oral fluids has made it more simple and much less costly.

7.9. What is cholera infantum ? How is it known these days ? Mention homoeopathic medicine for treatment.

This is an epidemic or sporadic gastro-intestinal infection in infants, often during the summer months and characterised by fever, vomiting, enterocolitis with toxaemia and a high morality rate.

It has been variously named as summer diarrhoea, infective or infections diarrhoea, acute gastro-intestinal infection or epidemic diarrhoea in children.

HOMOEOPATHIC MEDICINES ARE USED IN THIS CASE-

(1) Aethuja. (2) Podophyllum. (3) Arsenic, (4) China, (5) Antim crude etc.

Leading Remedies and their indications

1. Aethuja Cynapium are used

(i) During dentition, (ii) Due to intolerance of milk, (iii) Hot summer weather, (iv) Due to improper feeding in babies, (v) From constant feeding in adults.

Child cannot bear milk in any from. It is vomited in large amounts of hard curds. Vomiting is preceded by deathly nausea. Excessive weakness prostration and deep sleep follow the vomiting. Appearance of child is as if it were dying, pale hippocratic face, whitish blue pallor aroud lips and nose, eyes sunken.

In babies vomiting is generally accompanied by thin, yellow, greenish, slimy stool. Patient is hungry after vomiting. Complete absence of thirst.

2. Podophyllum-Cholera infantum in summers. Diarrhoea of children, during teething, after eating while being bathed or washed. Diarrhoea of long standing, early in morning, continues through forenoon, followed by natural stool in evening.

Stool—green, watery, offensive and profuse, gushing out, chalk like, jelly like, undigested.

Stool with sensation of weekness or sinking in abdomen and rectum. Prolapse of rectum before or with stool.

3. Arsenic alb.-Cholera infantum. When there is extreme prostration, restlessness, sinking sensation, cadaveric appearance, cold sweat, extremities cold. Foul, pungent, penetrating odor from faces and urine, and of vomit also. Vomiting of everything taken, sips of water cold fluid are vomited immediately. Stools are watery, acid scanty and offensive with vomiting and burning in abdomen. With all the complaints is associated with *fear of death:*

CHAPTER III

CARDIO-VASCULAR DISEASES

DISCUSSION FOR LEARNING

1. NAME OF THE COMMON DISEASES OF HEART AND PERICARDIUM.
2. VALVULAR DISEASES.
   - A. MITRAL STENOSIS.
   - B. MITRAL RE-GURGITATION (Incompetence)
   - C. AORTIC RE-GURGITATION.
3. ANGINA PECTORIS.
4. MYOCARDIA INFARCTION (Syn. Coronary Thrombosis)
5. ACUTE CORNONARY FAILURE.
6. HEART BLOCK.
7. ADAM-STROKES SYNDROME.
8. HEART FAILURE.
9. HYPERTENTION

1. NAME OF THE COMMON DISEASES OF THE HEART AND PERICARDIUM

Q.3.1. What are the common diseases of the heart and Pericardium? what are their characteristic features?

For practical purposes diseases of the heart and pericardium may be classified under five prominent differential features-

1. Disorders with PYREXIA.
2. Disorders in which PAIN is a characteristic symptoms.
3. Disorders which are accompanied by a ENLARGEMENT of the AREA OF CARDIAC DULLNESS.
4. Disorders in which an ALTERATION of the CARDIAC SOUNDS or a MURMUR, forms the diagnostic feature, and
5. Conditions which are recognised by an ALTERATION of the RHYTHM or RATE of the PULSE.

• MAIN FEAUTES AND NAME OF THE RESPECTIVE DISEASES:

MAIN FEATURES	NAME OF THE DISEASES
1. Pyrexia	1. Pericarditis 2. Ecute Endocarditis
2. Pain	1. Angina of Effort. 2. Spasmodic Angina. 3. Coronary Thrombosis 4. Angina Innocens (Pseudo-Angina) 5. Pericarditis.
3. Enlargement of the Area of Cardiac Dullness	1. Cardiac Hypertrophy. 2. Cardiac Dilatation. 3. Chronic pericardial Effusion. 4. Adherent pericarditis
4. Altered Heart Sounds	1. Mycoardial Degeneration 2. Endocarditis. 3. Congenital Heart disease 4. Pericarditis.
	1. Sinus Arrhythmia. 2. Premature Beats (Extra systoles)

5. Alteration of Rhythm or Rate of pulse	1. Sinus Arrhythmia. 2. Premature Beats (Extra systoles) 3. Paroxysmal Tachycardia. 4. Auricular Fibrilation. 5. Bradycardia. 6. Heart Block.

2. VALVULAR DISEASES

Q.3.2. What is meant by Valvular diseases? Give examples.

Valvular disease most commonly occurs in early life due to endocarditi is (acute or chronic) and in elderly persons due to chronic degenerative changes. The effect is a thickening or puckering of the valves and rings which results in one or both of two conditions-

(a) *Stenosis or narrowing of the orifice.*
(b) *Regurgitation in which the valves are incompetent and allow a reflux of the blood to take place from imperfect meeting and closure of the cups.*

According to these, there are 4 major valvular lesions involving the left side of the heart, and two rare types.

1. Mitral Stenosis.
2. Mitral Regurgitation.
3. Aortic Stenosis.
4. Aortic Regurgitation.
5. Pulmonary incompetence and stenosis.
6. Tricuspid valvular disease.

A. MITRAL STENOSIS.

• What is Mitral Stenosis? What are it's aetiology, clinical features and complications? Describe it's treatment in general and surgical point of views.

Def. *This is a narrowing of the mitral value, that interferes with the current of blood passing from the left auricle to the left ventricle.*

1.1. WHAT ARE IT'S ETIOLOGY?

Etiology—are of two primary varities-

(a) A congenital form (extremely rare)
- (i) results from either malformation of the mitral ring, or
- (ii) a foetal endocarditis.

(b) An acquired form-
- (i) rheumatic infection.
- (ii) a slow degeneration, sclerosing process,—part of a generalised cardiovascular degeneration.

(c) Age-commonly starts between 6 to 20 years.

(d) Sex-females commonly preponderant.

1.2. CLINICAL FEATURES

(What are it's signs and Symptoms?)

In case of mild stenosis the subject may be asymptomatic for prolong time and in severe stenosis and in moderate case the symptoms will develop.

• A. CLASSICAL SYMPTOMS

1. ***Effort intolerance***—sudden or acute. Sudden when caused by pulmonary oedema.
2. ***Haemoptysis.***
3. ***Angina pectoris.***

   Causes — (a) Rt. Ventricular hypertrophy-imbalance in blood supply,

   (b) Atrial fibrilation due to cardiac ischaemia.

   (c) Discomfort (not pain) due to costo-chondritis.
4. ***Easy fatiguability.***
5. ***Palpitation.***

   Causes— (a) Rt. ventricular hypertrophy.

   (b) Atrial fibrillation.
6. ***Anasarca.***

***B. SIGNS:**

1. **Malar flush.**
2. **Small volume pulse.**
3. **Blood pressure-low.**
4. **Neek veins engorged, but if there is pulmonary hypertention—giant 'a'** waves are found.
5. **Apex beat**—Normal in position. Character is *slaping or tapping. Ther is a diastolic thrill.*
6. **Para-sternal heave and diastolic shock-**if there is pulmonary hypertension.
7. **Auscultation**
   - (i) ***First heart sound***—short and sharp (not if the valve is calcified or if there is a prosthetic value)
   - (ii) ***Second heart sound***—audible in mitral area.
   - (iii) ***Opening snap***—(of Potain)-better heard after deep expiration.
   - (iv) ***Murmur***—mid-diastolic rumbling, low pitched, localised to the mitral area, best heard after expiration, on turning the patient to left lateral position and best heard with the bell stethoscope. There is a pre-systolic accentuaiton which disappears if there is atrial fibrillaition.
   - (v) **Second heart sound in pulmonary area is spilt.**

8. **Investigations**

(a) X-ray findings:

(i) *Dilatation of the pulmonary artery and conus.*
(ii) *Enlargement of the left auricle.*
(iii) *Small or absent aortic knucle.*
(iv) *No left ventricular enlargement.*

In R.A.O. view with Barium swallow, there is delay at the level of bifurcation of the trachea and abrupt deviation of the barium bolus in the oesophagus to the right where as in general enlargement of the heart, this deviation is gradual, not a abrupt.

(b) E.C.G. findings:

(i) *'P' waves widened and notched (P mitrate),*
(ii) *QRS complex shows varying degrees of right axis deviation in pulmonary hypertension.*

It is a book meant for quick reference. In presenting this modest volume to the Student and the Profession, hoping that it will specially serve as a guide to the student and family physician who are first to see and treat the patients suffering from various medical disorders. The author has not intended that the Chapters serve as complete reviews of the subjects. Instead he has dealt with the practical aspect of only common problems that a Student as well as Family Physicians come across in his day to day classes and practice. With this view in mind, the author has described in details the office procedures employed in diagnosis, and treatment of various ailments with general management, prophylactic treatment, Homoeopathic medicinal treatment, dieting and any other accessary measures which generally lack in various books of this subject.

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