PART-I

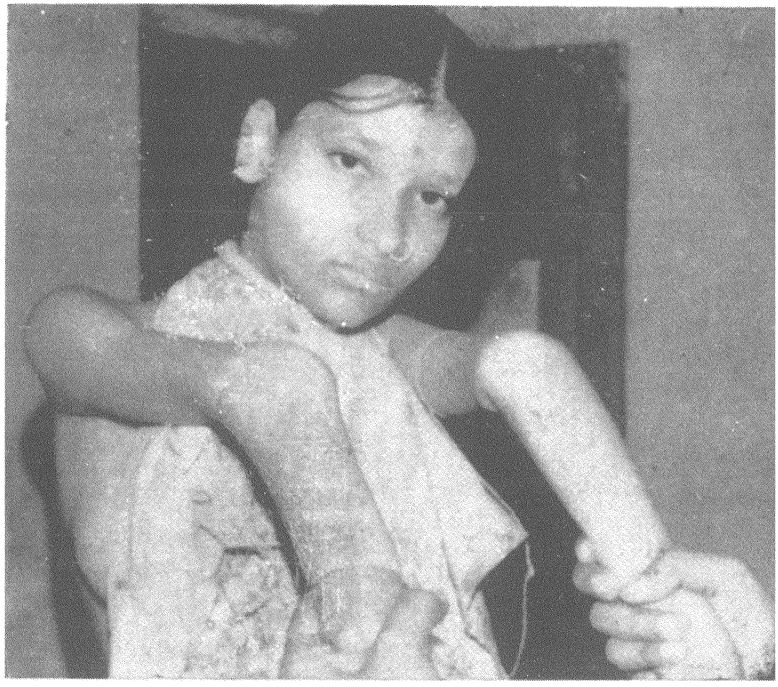


Fig. A : Ehlers Danlos Syndrome ; note the hyperextensible joints.

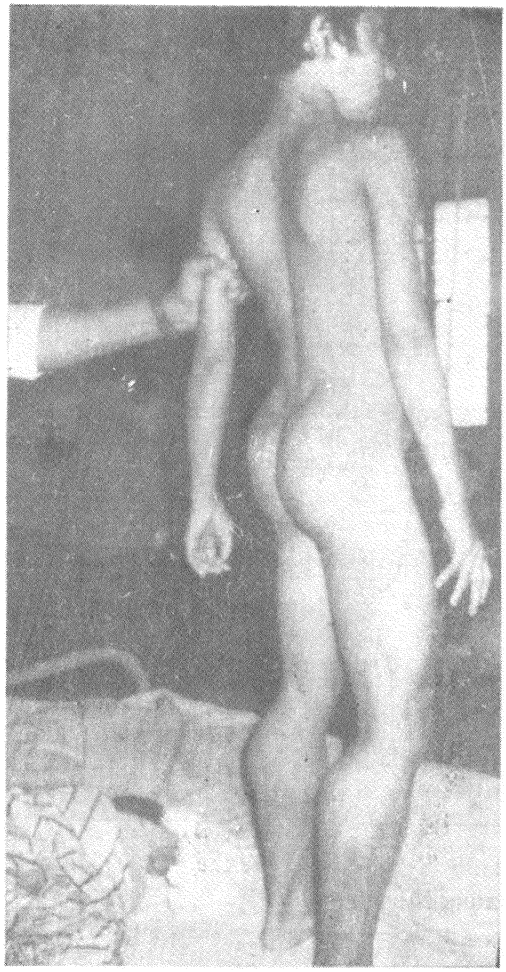


Fig. B : Duchenne's Pseudohypertrophic muscular dystrophy.

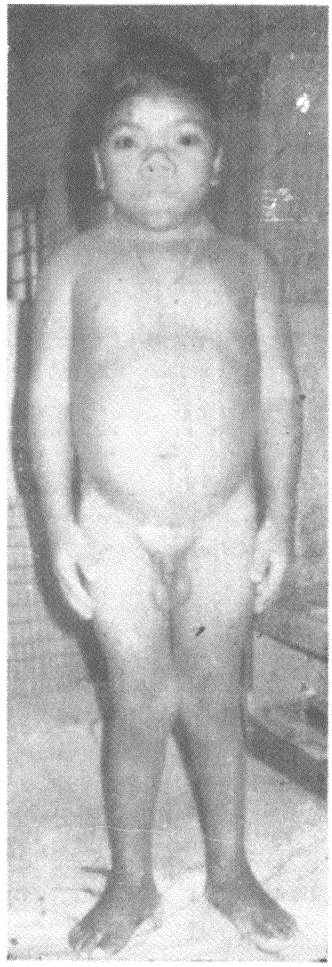


Fig.C: A cretin.

CHAPTER 1

GENERAL EXAMINATION

Young man, go to the bedside of the patient, there only can you learn medicine—Thomas Sydenham

PRINCIPLES OF HISTORY TAKING

The patient's complaints as regards his illness is the most important part in history taking. A detailed history from the patient gives clue to the correct diagnosis of the disease. Sympathetic attitude and keen observations are essential to properly elicit the history and get to the real malady of the patient. It is preferable to note down the history of the illness in the same way as expressed by the patient as far as practicable. Repetition of same complaint or irrelevant facts should be ignored intelligently. Leading questions are to be avoided during enquiry except where the patient is confused or is unable to express himself intelligently because of, say, mental dullness. It is imperative to exercise the restraint everytime a history is taken as leading questions suggest to the patient the symptoms the examiner expects to find or brings about an unconscious distortion of the patient's history. Making bedside notes is very essential.

The following points in the history should be noted in a routine way :

A. Name – This is asked as a rule for identification of the particular individual for future references.

B. Age – It is a very important clinical data in history -taking as some diseases are prevalent in particular age

groups. For example–arteriosclerosis, ischaemic heart disease, menopausal syndrome, malignancy etc. are maladies of older age group whereas acute rheumatic fever, congenital heart diseases, acute nephritic syndromes, congenital haemolytic anaemias, infectious diseases like measles, diphtheria are found in infancy and childhood. Chronic leukaemias, gall-bladder diseases, peptic ulcers are the diseases common in middle life.

C. Sex– Diseases like mitral regurgitation, cirrhosis of liver, chronic duodenal ulcer are more common in males whereas atrial septal defect, chronic cholecystitis, hypo and hyperthyroidism, anaemia of nutritional origin, are most commonly seen in females. Haemophilia and Duchenne muscular dystrophy are found almost exclusively in males.

D. Occupation– Enquiry about the patient's occupation may give some hints to the diagnosis of many diseases. Examples :

(i) A person long employed in a printing press having blue line in the gum and suffering from abdominal colic, paraesthesias in the extremities and revealing punctate basophilia in the peripheral blood smear has certainly been suffering from chronic lead poisoning.

(ii) In an individual working in a coal mine and suffering from breathlessness, cough with expectoration for a long time and clinically showing features of pulmonary emphysema,—the most possible diagnosis is coal miner's pneumoconiosis.

(iii) A sewer of paddy-field worker who suddenly develops high temperature with rigor, muscle pain, nausea and vomiting with jaundice, evi-

dences of anaemia and haemorrhagic episodes with leucocytosis, positive guineapig inoculation test (with blood in the first week or with urine in the second and third weeks) and revealing leptospira in smears from liver, kidney and blood culture–is a patient of Weil's disease.

(iv) Chances of coronary arterial diseases are more in businessmen, persons of sedentary habit, and those with type A personality.

(v) Men working in plastic and rubber industries are prone to develop bronchial asthma and cancers of genitourinary tract, particularly those who handle chemicals like benzidine.

(vi) Workers in mines (gold, copper, coal) pottery and sand blasting may develop silicosis, a type of pneumoconiosis. Pulmonary tuberculosis is very often a complication of silicosis.

(vii) Workers in printing industry who handle gum acacia often develop bronchial asthma.

(viii) Asbestosis is a type of pneumoconiosis that is common in persons working in textile industries and in persons engaged in construction work. Pleural plaques, bronchogenic carcinoma and pleural mesothelioma are often associated with asbestosis.

Persons suffering from these occupational chronic debilitating illnesses may legally claim for compensation and physicians are likely to be called into diagnose the condition and assess the severity.

E. Marital Status–The matrial status of the patient must be noted– diseases like haemophilia A, nephrogenic diabetes insipidus, Duchenne muscular dystrophy, glucose 6–phosphate dehydrogenase deficiency are X-linked reces-

sive and hence are usually carried asymptomatically by females while male offsprings are unfortunate victims. Marriage in extremes of age results in babies with congenital cardiac defects, abortion etc. and this is exemplified by the common occurrence of Down's syndrome (Trisomy 21) in children of elderly mothers and increasing frequency of Marfan's syndrome with increasing paternal age.

Psychiatric disturbances are sometimes precipitated by marital disharmony.

Marriage between close relatives or consanguineous marriages result in more frequent expression of genetic defects in the offsprings.

F. Address–Diseases like malaria, kala-azar, blackwater fever and filaria are common in West Bengal and parts of Orissa, whereas pernicious anaemia and subacute combined degeneration are rarely encountered in the tropics, but quite commonly seen in temperate countries.

Intestinal and extraintestinal (e.g. hepatic) amoebiasis. giardiasis, ascariasiss, hookworm infection are very common in West Bengal, and other regions of Eastern India particularly along the Gangetic plains.

G. Chief complaints–It is essential to note down three or four most important symptoms of the patient in chronological order along with their durations.

H. History of present illness– The patient should be allowed to narrate the history of the present complaints from the beginning of its development in sequences, without any leading questions being put in as far as practicable.

Unnecessary elaboration and repetition of symptoms should be tactfully avoided. Intelligent and educated patients sometimes narrate their complaints in medical terms e.g. acidity, rheumatism etc. In such cases, they should be asked to describe what discomfort they actually feel.

Details of previous treatments should be enquired about particularly the name and duration of drugs taken and any adverse reactions out of them (e.g. hypersensitivity to penicillin), previous surgical procedures, irradiation or psychotherapy. These information may have influenced the presenting symptoms or disease. For example, a female patient presenting with symptoms suggestive of diabetes mellitus may have history of long continued intake of corticosteroids for her rheumatoid arthritis.

The patient should also be asked to produce the previous records, if possible. In case of children and very old or mentally sick patients, close relatives should be asked about the previous treatment.

I. Past illness–The history or relevant diseases from which the patient has suffered in the past should be elaborated; e.g.

(i) Rheumatic fever; (ii) Malaria and kalaazar; (iii) Infectious diseases e.g. diphtheria, scarlet fever, small pox; (iv) Infective hepatitis; (v) Syphilis and gonorrhoea etc.

There might be some correlation of the above mentioned diseases with the present clinical signs–e.g. hepatomegaly, hepatosplenomegaly, features of portal hypertension due to cirrhosis, rheumatic valvular heart disease, cardiomyopathy, tabes dorsalis, syphilitic aortic incompetence, gonococcal arthritis etc.

J. Family history and Personal history–(i) Enquiry about the health of parents and causes of death if they are not alive should be made because diseases like diabetes mellitus, hypertension, coronary arterial disease etc. run in family and they are usually multifactorial.

(ii) As regards brothers and sisters, it is of same value because hypertension, congenital heart diseases, diabetes

mellitus, ischaemic heart diseases, congenital haemolytic anaemias may be frequent amongst the brothers and sisters of the same family, In such cases a family tree should be constructed.

(iii) Food habit is very important because malnutrition, iron deficiency anaemia, avitaminosis etc. are very common in the tropics.

(iv) Chronic alcoholism may lead to diseases like cirrhosis of liver, coronary arterial disease, peripheral neuritis whereas excessive smoking may lead to chronic bronchitis with emphysema, Buerger's diseases, bronchogenic carcinoma etc.

(v) Regular habits of physical exercise may prevent obesity, coronary arterial disease etc.

K. Psychological history– Psychological disturbances should be elicited very carefully while taking the history as psychoneurosis and psychosomatic disorders like peptic ulcer, hyperthyroidism, bronchial asthma, ulcerative colitis, irritable bowel etc. are encountered daily in the practice of medicine.

Type of work, social and sexual relationships should be asked when neurotic and psychosomatic disturbances are clinically suspected. Common psychoneurotic symptoms like anxiety, depression, obsessive thoughts should be elicited. History of delusions or false beliefs and hallucinations or false sensation or perceptions should be carefully enquired about if severe mental disorders like schizophrenia is suspected.

L. Menstrual History–In females, menarche or age of onset of menstruation, duration of menstruation and quantity of blood loss in each menstrual cycle or any abnormalities thereof should be noted.

Number of birth and complication during pregnancy or/ and birth (too early birth, sectio etc.) should be asked.

History of amenorrhoea, if any, should be asked for. Onset of menopause or stoppage of menstruation and symptoms attributed to premenopausal syndrome should be enquired about in elderly females.

Excessive blood loss due to gynaecological causes like fibroid uterus or cancer cervix may be a clue to the search for a cause of anaemia.

Perimenopausal bleeding, endometrial carcinoma, dysfunctional uterine haemorrhage are sometimes associated with excessive bleeding.

History of taking oral contraceptives with its duration should be noted. Obesity, hypertension, deep vein thrombosis, jaundice and sometimes breast carcinoma may occur as complications of these drugs.

M. Social Background—The living condition should be noted :

Patient is living on street, in a hut, house, flat.

Patient has to climb e.g. three stages to get at home.

There is freshwater available, or not; electricity or not. Note, if necessary situation how patient is coming with other people e.g. relatives, friends etc.

Is there any other social pressure or environmental disorder which might cause unwell feeling or even disease (eg home near a big and busy street, lot of noise, no proper sleep possible etc.

There could be much more of interest, which you find out after a second or third talk with the patient. The social and environmental sorrounding is one of the basic items. This conditions decides at last whether we were able to cure the patient or only "repair" a symptom.

PHYSICAL EXAMINATION

GENERAL SURVEY (Decubitus, build, nutrition, facies, anaemia, cyanosis, jaundice, pigmentation, oedema, clubbing, temperature, pulse, respiration, blood pressure).

1. DECUBITUS

Definition– The posture of the patient in bed. The different types are—

(i) Decubitus of choice— The patient does not feel uncomfortable in any particular position.

(ii) Propped up, which may be grossly generalised as follows :

45"–mild dyspnoea

60°—moderate dyspnoea

90° —Severe dyspnoea (also known as orthopnoea). However, even a mildly dyspnoeic patient may be most comfortable at 60° or even at 90° elevation. This is because dyspnoea is a subjective phenomenon and patients vary from one to another in tolerance or endurance.

Causes of propped up decubitus :

(a) *Cardic*–Left ventricular failure due to mitral incom petence. aortic valvular disease. hypertension, myocardial infarction, cardiomyopathy, patent ductus arterisous, coarctation of aorta etc. Left atrial failure due to mitral stenosis, myxoma of left atrium, ball valve thrombus in the left atrium, mitral atresia etc. Massive pulmonary embolism (acute corpulmonale). Chronic corpulmonale.

(b) *Respiratory*–Bornchial asthma, pleural, effusion, pneumothorax and hydropneumothorax, bronchogenic carcinoma, chronic bronchitis with or without emphysema, pulmonary arteriovenous fistula, acute pulmonary oedema due to over transfusion, carbon monoxide poisoning etc.

(c) *Gastrointestinal*—Huge ascities—mechanically pushing the diaphragm upwards causes a de crease in vital capacity and leads to reduced air entry into the lungs.

(d) *Neurological*–Any lesion below the level of pons that causes paralysis of respiratory muscles (including Guillain-Barre syndrome), recurrent laryngeal nerve palsy causing stridor and maysthenias and myopathies causing respiratory muscle weakness or paralysis.

(iii) *Trepopnoea*—Patient feels comfortable lying on the affected side and becomes dyspnoeic if he lies on the opposite side e.g. pleural effusion, hydropneumothorax, pneumothorax etc.

(iv) Curled up decubitus is seen in acute renal colic, acute biliary colic meningitis etc.

(v) Opisthotones—seen in tetanus, strychnine poisoning spinal meningitis, maple syrup urine disease (leucinosis) or may occur as a hysterical manifestation.

(vi) Listless attitude— is seen in unconsciousness, peritonitis, parkinsonism, hysteria.

COMMON CAUSES OF UNCONSCIOUSNESS

Neurological λ Cerebral haemorrhage λ Cerebral embolism λ Subarachnoid haemorrhage λ Cerebral thrombosis λ Postepileptic coma and status epilepticus λ Encephalitis and encephalomyelitis λ Meningitis.

Endocrine λ Diabetic coma (hyperglycaemic diabetic ketoacidosis or hyperosmolar nonketotic) λ Addisonian crisis λ Hypothyroidism (Myxoedema coma) λ Pituitary coma (Pituitary apoplexy)

Cardiovascular λ Adams-Stokes syndrome λ Syncopal attack due to severe aortic stenosis, pulmonary stenosis, aortic, stenosis Fallot's tetralogy etc. λ Hypertensive encephalopathy.

Respiratory λ Carbon dioxide narcosis, respiratory failure.

Gastrointestinal λ Hepatic coma (encephalopathy associated with hepatic failure)

Renal λ Terminal stage of uraemia.

Poisoning λ Barbiturate, morphin etc.

Traumatic λ Head injury.

Psychogenic λ Hysteria.

2. BUILD

Definition — Build is the skeletal structure in relation to age and sex of the individual as compared to a normal person. Build is short in those in whom it is below the 3rd centile of a normal population of same age and sex and tall when the height is above the 97th centile for normal population.

Types—(1) Short—the common causes are—

(a) Normal genetic variation.

(b) Chromosomal abnormalities e.g. Down's syndrome, Turner's syndrome, Noonan's syndrome.

(c) Childhood diseases e.g. marasmus, kwashiorkor, rickets, tuberculosis, fibrocystic disease, gluten, enteropathy, hookworm infestation, congenital cyanotic or acyanotic heart diseases, thalassaemia major, chronic renal disease, congenital syphilis glycogen storage diseases etc.

(d) Skeletal abnormalities, e.g. Achondroplasia, gargoylism, Ellis van Creveld syndrome, rickets, poliomyelitis, Still's disease.

(e) Endocrine disorders, e.g. craniopharyngiomas or other pituitary tumours, idiopathic hypopituitarism. Frohlic's syndrome, Laurence-Moon Biedl syndrome, hypothroidism, sexual precocity, Cushing's syndrome, congenital adrenal hyperplasia.

(f) Intrauterine maldevelopment e.g. foetal alcohol syndrome, progeria etc.

(g) Drug induced e.g. glucocorticoids, androgens, anabolic steroids, oestrogens, antithyroid drugs, vitamin D etc.

(2) Tall—This is mostly inherited from tall parents and pathological causes are not common. The different causes are—

(a) Constitutional

(b) Overnutrition causing tallness before fusion of epiphyses

(c) Chromosomal abnormalities e.g. Klinefelters syndrome (47 XXY). Supermale (XYY) and superfemale (XXX)

(d) Miscellaneous causes include Marfan's syndrome, homocystinuria, cerebral gigantism, lipodystrophy and Beckwith Wiedemann syndrome.

3. NUTRITION

Definition— It means nourishment of the body which is assessed by

(i) Subcutaneous fat,

(ii) Bulk of muscle

(iii) Features of vitamin deficiency, if any.

However, in a recent study logistic regression analysis has shown that mid-upper arm circumference is as effective as other nutritional indices and combinations of different indices have not been shown to be better especially in predicting death in malnourished children (BMJ. 1986, 293, 373).

Types (1) Subnutrition—Body weight 10% less than that of a standard person in relation to age and sex. It is due to quantitative dietary deficiency.

(2) Starvation–Body weight 25% less than that of a standard person in relation to age and sex.

(3) Malnutrition Deficiencies of vitamins, minerals and essential amino acids are characteristic. It is due to qualitative dietary deficiency. But overnutrition is also a form of malnutrition.

(4) Cachexia–A cachectic shows anaemia, evidences of vitamin deficiency and features of starvation.

(5) Emaciation—Loss of subcutaneous fat and diminution of muscle bulk.

CAUSES OF PATHOLOGICAL SUBNUTRITION :

(i) Pulmonary tuberculosis (due to anorexia as well as defective utilisation of nutrients).

(ii) Diabetes mellitus (loss of calorie due to glycosuria and malabsorption may be associated due to various gastrointestinal disorders).

(iii) Thyrotoxicosis
(iv) High fever
(v) Malignancy
] due to increased metabolic rate

(6) Obesity–It means an excess of adipose tissue that imparts a health risk. The degree of excess adiposity is difficult to quantity; but the Framingham study has shown that a 20% excess over ideal weight imparts a definite health risk.

Causes of obesity : These may be either (a) primary or idiopathic where the precise mechanism remains unknown, or (b) secondary, which may be due to (i) hypothyroidism, (ii) Cushing's syndrome, (iii) hypothalamic disorders, (iv) pinealomas, (v) pseudohypoparathyroidism, (vi) Pancreatic insulinomas, (vii) testicular hypogonadism, (viii) polycystic ovarian disease, (ix) induced by drugs e.g. insulin, oral hypoglycaemic agents, glucocort-

icoids oestrogents etc. (x) abnormal distributions of fat e.g. lipodystrophies, (xi) or may be associated with syndromes or unknown aetiology e.g. Laurence- Moon-Biedl syndrome, Prader-Willi syndrome, Alstrom syndrome etc.

(7) Nutritional oedema–This includes
- (i) Wet beriberi.
- (ii) Famine oedema with hypoproteinaemia
- (iii) Famine oedema without hypoproteinaemia (isohydraemic famine oedema)
- (iv) Epidemic dropsy.

(8) Protein Energy Malnutrition (i) *Kwashiorkor* : It is a clinical condition characterised by retardation of growth, wasting of muscles, grey or lustreless hair, pigmentation, desquamation and ulceration of skin, oedema and signs of vitamin deficiency occurring in infants and children due to dificiency of protein with adequate calorie intake. Commonly, it occurs due to prolonged breast feeding.

(ii) *Nutritional marasmus* : Here also infants usually under one year of age suffer from retardation of growth, loss of weight, wasting of muscles and subcutaneous fats but the precipitating factor is the severe restriction of all foods, reproteins, calorie, vitamins etc. One-fifth to one-third of the body weight is lost and the face is pinched and has a curiously senile expression with sunken eyes. The thorax is particularly wasted with prominent ribes. Early weaning and rapid succession of pregnancies are common causes in our country.

(iii) *Marasmic Kwashiorkor* : In this variety, the children have some clinical features of both the above disorders.

4. FACIES

Definition—Expression in the face of patient.

Types —

(i) Anxious— It indicates awareness or apprehension of the patient about the disease. It may be found in nervous individuals.

(ii) Dull and vacant look of mentally retarded children Mongoloid facies is characterised by slanting eyes, epicanthic fold, small nose with a small oral cavity. This is characteristically seen in Down's syndrome (trisomy 21).

(iii) Masked facies—Parkinsonian syndrome. This facies is characterised by wide palpebral fissures, infrequent blinking, and spontaneous ocular movements are lacking. This is also seen in bilateral facial paralysis, facial myopathies, myasthenia, gravis, progressive systemic sclerosis etc. The face remains devoid of any expression.

(iv) Hepatic facies—This is characterised by shrunken eyes, hollowed temporal fossa, pinched up nose, parched lips, muddy complexion, icteric tinge.

(v) Tabetic facies—Features are persistent wrinkling of the forehead in an attempt of compensate for the drooping of eyelids due to pseudoptosis caused by paralysis of Muller's muscle.

(vi) Thyrotoxic facies—Staring look and exophthalmos are the characteristic features.

(vii) Coarse facial appearance, wrinkling of eyebrows and thick tongue are the characteristic features of cretinism. Apathy of hypothyroidism should draw the attention of the clinician.

(viii) 'Moon' facies the bloated appearence of the face and rounding of the features are caused by–
Cushing's syndrome and disease,
Steroid therapy for a prolonged period,

Acute (proliferative) glomerulonephritis, Minimal lesion and membranous glomerulonephritis, Myxoedema etc.

Superior mediastinal syndrome and pulmonary stenosis.

(In glomerulonephritis the lower eyelids become puffy because of less subcutaneous fat and low tissue tension).

(ix) Facies of bilateral facial palsy—a face devoid of any expression with loss of nasolabial and other promiment furrows (vide supra).

The commonest cause is Guillain-Barre Syndrome of acute infective polyneuritis (when bulbar nuclei are affected. Other rare causes are sarcoidosis, leukaemias and lymphomas, Melkersson's syndrome. In infants forceps delivery in an important cause.

(x) Flushed facies—(a) Malar flush–High colour of the cheek as seen in mitral stenosis particularly with severe pulmonary hypertension in fair skinned individuals. Another cause is myxoedema.

Severe flushing of the cheeks hectic flush is encountered in pulmonary tuberculosis. Facial flushing is characteristic of patients with Cushing's syndrome, polycythaemia, emphysema (pink puffers) and carcinoid syndrome. Postprandial facial flushing occurs in rosacea. In congestive cardiac failure the cheeks may be red and high coloured and a bluish tint may be evident.

(b) Generalised flush—May be caused by–(i) High fever (ii) Severe hypertension (iii) Thyrotoxicosis (iv) Chronic alcoholism (v) Carcinoid syndrome (vi) Pheochromocytoma (vii) Drugs like atropine,

nicotinic acid (niacin) and percutaneous absorption of monosulfiram, (viii) Systemic mastocytosis (ix) Toxic erythemas (x) Measles rubella, scarlet fever (xi) Lupus erythematosus etc. Localised flushing is seen in erythromelalgia and also in systemic lupus erythematosus (SLE).

(xi) Pallor—It is seen in shock where blood flow through the capillaries diminished, in syncopal attack, left heart failure, peripheral vascular diseases like Raynaud's phenomenon and arterial spasm on exposure to cold. Generalised pallor is found in severe anaemia.

(i) Pallor with anaemia—found in all types of severe anaemia, infective endocarditis, acute rheumatic fever etc.

(2) Pallor without anaemia—The cardiovascular causes are—Tight mitral stenosis, Severe aortic stenosis, Acute myocardial infarction, Acute left ventricular failure, Acute peripheral circulatory failure and other causes are—

(i) Nephrotic syndrome, (ii) Causalgia, (iii) Acute alcoholic coma, (iv) During paroxysm of vertigo in Meniere's disease and (v) Acute nephritic syndrome especially when there is anaemia.

(xii) Risus sardonicus—The eyebrows are raised and the angles of the mouth drawn out due to tonic spasm of the muscles of the face in tetanus.

(xiii) Photophobia–in meningitis and also seen in meningisms.

(xiv) Elfin facies —Found in supravalvular aortic stenosis (William's syndrome) that may be associated with hypercalcemia.

5. ANAEMIA

Definition — Qualitative or quantitative reduction of circulating RBC and/or of the percentage of hae-

moglobin concentration in relation to standard age and sex. Normal blood count–

RBC — Male, Female — 5 to 6 million /cmm; 4.5 to 5.5 million/cmm

WBC— Male, Female — 4,000 – 11,000 cmm

Haemoglobin—

Male–14.6 to 15.5 gm per 100 ml (100.11%)

Female–13.3 to 14.6 gm per 100 ml (90.100%)

[100% = 14.6 gm per 100 ml]

Reticulocyte — o to 1%

Platelet — 1.5 to 4 lacs/cmm.

Anaemia is said to be severe when heamoglobin is less than 40% moderate when 40.50% mild when 50.60%

Sites to be looked for—

(i) Lower palpebral conjunctiva,

(ii) Tip and dorsum of tongue,

(iii) Soft palate,

(iv) Nail beds—

(v) General skin, palm and sole.

Colour of the mucous membrane of the conjunctiva and the tongue is more reliable than the general skin. Estimation of percentage of haemoglobin and examination of stained films indicate the severity and aetiology of anaemia.

Symptoms referable to anaemia :

General—Fatigue and lassitude.

Neurological—Giddiness vertigo, dimness of vision, headache, insomnia, tingling and numbness of extremities.

Cardiovascular—Palpitation, dyspnoea, anginal attack.

Gastrointestinal—Indigestion diarrhoea, anorexia,

Signs due to anaemia :

General–Pallor, oedema.

Cardiovascular—Water-hammer pulse, pistol shot sound, capillary pulsation forceful apex, haemic murmur over the pulmonary area in left second of third space or in the apical region, ejection click and nonrumbling soft diastolic murmur in mitral area. This is due to relative stenosis of mitral or tricuspid valve secondary to greatly increased blood flow. This murmur is encountered in sickle cell anaemia where the anaemia is very severe and chronic.

(Noncardiac cuses of water-hammer pulse are thyrotoxicosis, high fever, Paget's disease, arteriovenous fistula, wet beriberi, chronic annoxic, corpulmonale, hepatic coma etc.)

Respiratory—Crepitations in lung bases.

Neurological features of polyneuritis or subacute combined degeneration of spinal cord and papilloedema.

Renal—Albuminuria.

Gastrointestinal—Enlarged liver and spleen due to proliferation of reticuloendothelial cells.

Types of anaemia—

I. Iron deficiency (Hypochromic microcytic) (a) Haemorrhagic acute post haemorrhagic anaemia following trauma or intestinal bleeding and chronic post haemorrhagic anaemia in bleeding from haemorrhoids, from peptic ulcer, due to hookworm infestation or chronic menorrhagia;

(b) Nutritional deficiency :

(c) After gastrectomy ;

(d) Malabsorption syndrome.

II. Megaloblastic—This is due to vitamin B12 and/or folate deficiencies leading to arrest to maturation of the cells. The causes are Nutritional; Pregnancy; Liver diseases; Malabsorption syndrome; Drugs e.g. (a) Folate antagonists e.g. methotrexate and pyrimethamine, (b) Anticonvulsants eg Phenytoin, (c) Cytosine arabinoside

by interfering with DNA synthesis. The causes that are rare in our country are (a) Pernicious anaemia (b) In Leukaemias and haemolytic anaemias–due to excess utilisation of folate; (c) Diphyllobothrium latum infestation.

III. Dimorphic anaemia—Presence of the picture of both iron deficiency and megaloblastic anaemia in peripheral blood. This type is quite common in our country. The causes are – (i) Hookworm infection with nutritional deficiency state. (ii) Pregnancy.

IV. Anaemia of scurvy.

V. Anaemia of hypothyroidism.

VI. Haemolytic anaemias—

(1) Hereditary disorders of RBC :

(a) Congenital spherocytosis;

(b) Haemoglobinopathies like sickle cell anaemia, thalassaemia, haemoglobin C disease and haemoglobin E disease;

(c) Glucose-6-phosphate dehydrogenase (66PD) and other enzyme deficiencies in RBC.

(2) *Due to antibody formation against erythrocytes :*

(i) Haemolytic disease of newborn;

(ii) Autoimmune (acquired) haemolytic anaemia;

(iii) Symptomatic haemolytic anaemia;

(iv) Paroxysmal haemoglobinuria;

(v) Rh incompatibility :

(vi) Mismatched blood transfusion.

(3) *Due to infective or toxic factors :*

(i) Organisms like haemolytic streptococci, staphylococci, Clostridium welchii etc.

(ii) Malaria : Blackwater fever.

(iii) Arsenic, lead and other heavy metals and drugs like sulphonamides, potassium chlorate, methyldopa and chemicals like napthalene etc.

(vi) Anaemia due to bone-marrow depression–causing pancytopaenia or aplastic or hypoplastic anaemia.

(a) Idiopathic

(b) Secondary to :

(I) Drugs like chloramphenicol, trinitrotoluene, gold, anticonvulsants (troxidone), arsenic, following use of cytotoxic drugs—due to idiosyncratic reaction.

(II) Idiosyncrasy to certain chemicals and insecti cides–benzol and its derivatives like trinitrophenol.

(III) Repeated exposure to X-rays and radioactive substances.

VII. Myelosclerosis, myelofibrosis, myelophthisic anaemia, multiple myeloma lead to simultaneous pres -ence of myelocytes and normoblasts in the peripheral blood and is referred to as *leucoerythroblastic* blood picture and is due to bone-marrow infiltration.

VIII. Anaemia of uncertain origin :

(a) Uraemia (partially due to deficiency of erythro - poietin in chronic renal failure);

(b) Malignancy, chronic infections, hepatic cirrhosis, rhuematoid-arthrities etc. lead to *anaemias of chronic disorders.*

IX. Sideroblastic anaemia — It is a type of dyshaemopoietic anaemia where peripheral blood picture is hypochromic microcytic or dimorphic in type but the bone-marrow contains ringed sideroblasts. Sideroblasts are nucleated red blood cells having excess iron containing granules in the cytoplasm. Here utilisation of iron is impaired due to defect in erythropoiesis. These are either hereditary (sex-linked partially recessive) or acquired. The latter may be (i) primary (idiopathic)

or (ii) secondary. The secondary anaemias may be due to— (a) Drugs like anti-tuberculous, paracetamol, phenacetin; (b) nutritional disorder e.g. chronic alcoholism, nutritional megaloblastic anaemia, malabsorption; (c) increased haemopoietic cell proliferation e.g. myeloproliferative disorders, leukaemias haemolytic anaemias. Nearly one-third of the sideroblastic anaemias respond to large (e.g. 100 mg) daily doses of pyridoxine.

In all cases of severe anaemia apart from routine examination of blood, bone-marrow study should be carried out, Coomb's test may have to be done in some forms of haemolytic anaemia. In hereditary spherocytosis, the test is negative; it is positive in immune haemolytic anaemias.

6. CYANOSIS

Definition — Bluish discolouration of the skin and mucous membrane due to excessive amount of reduced haemoglobin in the blood (more than 5 gms/100 cc.) Clinical cyanosis is present when oxygen saturation is below 85%. Types :

1. *Peripheral cyanosis*—This occurs in the presence of normal arterial oxygen saturation and is due to pronounced oxygen unsaturation of the venocapillary and capillary blood.

Sites—Tip of nose, fingers and toes, ear lobule, palm and sole.

Mechanism—

(a) Vasconstriction (peripheral),
(b) Low cardiac output,
(c) Sluggish circulation in extremities.

Causes—

(i) Acute left ventricular failure or acute left atrial failure with peripheral stasis.
(ii) Shock from severe burns or severe haemorrhage.
(iii) Exposure to cold.
(iv) Cryoglobulinaemia–Cryogobulin is an abnormal plasma protein (globulin) which forms get at low temperature; may be found in lym--phoma, nephrosis, multiple myeloma, collagen diseases, kala azar etc.
(v) Raynaud's phenomenon–characterised by bluish colouration of the digits due to excessive vasoconstrictor response to cold to mechanical stimuli.
(vi) Venous obstruction due to any cause and local vasomotor disturbances may give rise to local cyanosis.

II. *Central cyanosis*—Due to excessive oxygen unsaturation of the arterial blood.

Sites—Tongue, inner surface of lip and also sites for peripheral cyanosis.

Causes—

(a) Pulmonary–Corpulmonale, pleural effusion, pneumothorax, respiratory failure, pneumonia, absorption collapse etc.
(b) Cardiac–Congenital cyanotic heart diseases with right to left shunt–e.g. Fallot's tetralogy, Eisenmenger complex and syndrome. The syndrome is characterised by pulmonary hyper tension with reversal of shunt, the term Eisenmenger's complex is used when the reversed shunt is at the ventricular level. Cyanosis tardive or late cyanosis may be found

in A S D with reversal of shunt due to increased pulmonary and right ventricular resistance because of heart failure of pulmonary compli cations.

(c) Vascular—Pulmonary arteriovenous fistula.

III. *Enterogenous*—This group includes—

(i) Sulphaemoglobinaemia–55–68 band in spectroscopy.

(ii) Methaemoglobinaemia—Cyanosis is due to formation of methaemoglobin where ferrous iron of the haem of haemoglobin is converted into ferric ($Fe^{++} \rightarrow Fe^{++}$) form in excessive amounts.

Causes :

(a) Inherited defects like haemoglobin M or deficiency of cytochrome b5 reductase.

(b) Poisoning with chemicals e.g. aniline, nitrobenzene etc.

(c) Drugs e.g. phenacetin, dapsone, sulphonamides etc.

(iii) Cherry red colouration of skin is produced in carbon monoxide poisoning.

Clinical effects of cyanosis—

(1) Hypertrophy of and bleeding from gum,

(2) Recurrent arthritis, gout and tophi formation,

(3) Plumonary osteoarthropathy.

(4) Secondary polycythaemia—e.g. in corpulmonale or cyanotic congenital—heart diseases,

(5) Embolic manifestation–e.g. pulmonary embolism.

IV. *Differential cyanosis*—

(a) Hands blue but feet red—in coarctation of aorta with transposition of great vessels.

(b) Hands red but feet blue–in patent ductus arteriosus

with reversal of shunt due to pulmonary hypertension.

7. JAUNDICE

Definition—Yellowish discolouration of the skin and mucous membrane due to excessive bilirubin in the blood. [Normal range is 0.2 to 0.8 mg/100 ml serum]

Latent jaundice [1 mg–1.9 mg/100 mm serum] can be detected only by serum analysis.

Sites— (i) Upper bulbar conjunctiva,
(ii) Soft palate,
(iii) Undersurface of tongue,
(iv) Skin,
(v) Palm and sole.

Internal tissues are also stained when the jaundice is severe.

Types—(a) Obstrcutive, (b) Haemolytic, (c) Toxic or Hepatocellular or combination of any of these.

Obstructive Jaundice : This is due to a block in the pathway between the site of conjugation of bilirubin in the liver cells and entry of bilirubin in the intestine.

Clinical effects—

(i) Greenish-yellow bulbar conjunctiva.
(ii) Petechial haemorrhage (due to vit K defi ciency which being a fat soluble vitamin is not absorbed from the intestine and hence the bleeding disorder).
(iii) Sinus bradycardia—increase in the vagal inhibitory tone due to circulating bile salt.
(iv) Marks of scratching due to pruritu–possibly a reflex; bile acids acting as irritants on the nerve endings.

(v) Enlarged liver.

(vi) Gall bladder may or may not be palpable depending upon the cause. (According to Courvoisier's law, gall bladder is usually not palpable in jaundice due to a stone in the common bile duct whereas in carcinoma of the head of pancreas gall bladder becomes distended).

(vii) Splenomegaly due to associated biliary cirrhosis (rarely).

(viii) Mustard oil coloured urine.

(ix) Clay coloured stool.

(x) Xanthelasma, xanthoma tuberosum etc. in about 20% of cases.

Liver function tests which are mainly dependent upon the patency of the bile ducts are impaired. In obstructive jaundice. serum alkaline phosphatase level varies between 30 and 100 KA units. [Normal serum value in adult is 3 to 13 King-Armstrong (KA) Units per 100 ml or 40–100 i u l.]

Causes of obstructive jaundice :

(A) Intrahepatic—

(a) Viral infection—infective hepatitis;

(b) Drugs like (i) Chlorpromazine, (ii) Para aminosalicylic acid, (iii) Sulpha drugs—Sulphadiazine, (iv) Chlorpropamide, (v) Methyl testosterone and other anabolic steroids, (vi) MAO inhibitors, (vii) oral contraceptives, (viii) Alcohol, (ix) INH etc.

(c) Active chronic hepatitis :

(d) Cirrhosis of liver

(e) In pregnancy—due to cholestasis;

(f) Lymphoma e.g. Hodgkin's disease;

(g) Secondary carcinoma of liver;

(h) Sometimes in severe bacterial infection;
(i) Pericholangitis in chronic ulcerative colitis.

(B) Extrahepatic—
(i) Impacted gall stone;
(ii) Enlarged glands at porta hepatis;
(iii) Carcinoma of head of the pancreas;
(iv) Carcinoma of ampulla of Vater or bile duct;
(v) Carcinoma of gall bladder;
(vi) Rarely a duodenal ulcer involving the commonbile duct;
(vii) Stricture of common bile duct, viz after surgery;
(viii) Sclerosing cholangitis complicating ulcerative colitis.

Haemolytic Jaundice: This is due to excessive destruction of red blood cells– resulting in increased bilirubin load on the liver.

Clinical features :
(i) Lemon-yellow bulbar conjunctiva.
(ii) Anaemia, the degree of which varies with the severity of haemolytic process and power of bone-marrow to regenerate.
(iii) Splenomegaly—due to excessive activity of the reticuloendothelial system.
(iv) High-coloured stool containing large amount of stercobilinogen and stercobilin.
(v) Freshly voided urine is of normal colour since no bilirubin is present but oxidation of excess urobilinogen to urobilin quickly turns the urine dark.
(vi) Examination of blood reveals reticulocytosis.
(vii) Liver function tests dependent upon meta bolic activities of the parenchymal cells are normal.

Causes :

(i) Congenital

(a) Hereditary spherocytosis;

(b) Haemoglobinopathies (sickle cell anaemia, thalassaemia etc.);

(c) Glucose-6-phosphate dehydrogenase (G6PD) and pyruvate kinase deficiencies.

(ii) Acquired

(a) Mismatched blood transfusion;

(b) Rh-incompatibility;

(c) Following poisonous snake bite;

(d) Drugs e.g. primaquin, phenacetine, sulphonamides causing haemolysis due to G6PD deficiency in RBC;

(e) Infection by parasites–malaria and kala azar;

(f) Acquired immune haemolytic anaemia.

(g) Marchiafava Micheli syndrome or paroxysmal nocturnal haemoglobinuria (PNH)–due to unusual sensitivity of RBC to complement;

(h) Paroxysmal cold haemoglobinuria (PCH) secondary to syphilis (congenital syphilis);

(i) March haemoglobinuria—due to external trauma to small vessels.

Toxic or Hepatocellular Jaundice : This is due to damage of liver cells by toxic or infective agents.

Causes :

(A) Infective :

(i) Viral—Hepatitis A, B, C, D, E, Cytomegalovirus, Epstein Barr and Yellow fever virus.

(ii) Spirochaetal—Leptospira icterohaemorrhagia (Weil's disease).

(iii) Protozoal—Toxoplasma gondii.

(iv) Rickettsia—Coxiella burnetti (Q fever agent).

(B) *Toxic—(1) Drugs :*

(i) Chlorpromazine and other phenothiazine derivatives;

(ii) MAO inhibitors;

(iii) Imipramine, amitryptiline;

(iv) Erythromycin, tetracycline (in high doses particularly in pregnancy and in impaired renal function), rifampicin.

(v) Isoniazid and para aminosalicylic acid;

(vi) Methyldopa

(vii) Phenylbutazone, indomethacin and gross overdosage of paracetamol.

(viii) Halothane, the anaesthetic agent (idiosyncratic reaction).

(2) *Poisons and Toxins :*

(i) Carbon tetrachloride,

(ii) Yellow phosphorus,

(iii) Copper Sulphate,

(iv) Alcohol.

(v) A fungal toxin used as poison (amanita phalloides).

(C) *Other conditions*– In pregnancy (besides cholestasis) jaundice may be due to acute fatty liver, toxaemias or hyperemesis, both the latter being rare causes.

Some other important causes of jundice :

(a) Primary defect in bilirubin transport of conjugation in the liver cells particularly in premature infants is known as *physiological jaundice of the newborn.* The defect in conjugation is due to immaturity of the enzyme mechanism.

(b) Gilbert's syndrome–the fault is in the uptake

of bilirubin by liver cells and a partial defici ency of glucuronyl transferase enzyme. This is an inherited autosomal dominant disorder.

(c) Dubin-Johnson syndrome is an autosomally inherited benign defect in the transport of bilirubin glucuronide from the liver cells into the bile canaliculi. Rotor syndrome is some what similar.

(d) Crigler-Najjar syndrome–An autosomal recessive condition, consists of the two types :

Type 1–Total absence of the enzyme glucuronyl transferase. It is the severe form. Unconjugated hyperbilirubinaemia may lead to kernicterus in newborn and ultimately to death.

Type II–Partial deficiency of the enzyme glucuronyl transferase is present. The patient may survive to adult life.

Jaundice in cardiovascular disorders

(a) Congestive cardiac failure–hepatic conges tion and hepatocellular hypoxia is congestive cardiac failure are associated with disturbance in the function of the liver. This may give rise to icteric tinge.

(b) Recurrent and/or multiple pulmonary infarctions.

(c) May be associated with repeated myocardial infarction.

(d) Latrogenic.

8. PIGMENTATION

Usual sites that are to be examined are –

(i) Face,

(ii) inside the cheek,

(iii) Creases of palms,
(iv) Skin particularly pressure points and areas exposed to light.

Causes of pigmentation—

(A) Physiological
(i) Pregnancy–e.g. chloasma, linea nigra, sec -ondary areola etc.
(ii) Racial.
(iii) Bluish black pigmentation of the mongols.

(B) Pathologial
Congenital–(a) Von Recklinghausen's disease–typical *cafe-au-lait* pigmentation.
[N. B.–other causes of cafe-au-lait pigmentation are–(i) tuberous sclerosis or epiloia (ii) Polyostotys tic fibrous dysplasia – Albright's syn-drome; (iii) Watson syndrome–pulmonic stenosis with cafe-au-lait patches, (iv) normally in 10% of all people, (v) infective endocarditis etc.
(b) Xeroderma, pigmentosum.
(c) Peutz-Jeghers syndrome.
(d) Multiple polyposis of colon.
(e) Acanthosis nigricans juvenile variety.
(f) Blooms syndrome characterised by typical facies, retardation of growth and photosensitive telangiectatic erythema on face.
(g) Nevus.
Acquired– (a) Physical agents :
(i) Exposure to radiation;
(ii) Exposure to sun-rays and heat;
(iii) Erythema ab igne–caused by local heating in domestic conditions.
(b) Skin diseases :
(i) Lichen planus, (ii) Exfoliative dermatitis,

(iii) Pityriasis versicolor, (iv) Dermatitis herpetiformis, (v) Patchy pigmentation may alternate with white patches in leucoderma, (vi) Lichen simplex chronicus, (vii) Psoriasis, (viii) Discoid lupus erythematosus, (ix) Acanthosis nigricans–associated with diabetes mellitus, gross obesity or internal malignancies e.g. carcinoma stomach.

(c) Poisoning :
  (i) Chronic arsenical poisoning
  (ii) Argyria–diffuse slaty grey colouration due to deposition of silver in the skin.

(d) Endocrine :
  (i) Addison's disease–increased pigmentation in skin and mucous membrane varying from light to dark brown in colour.
  (ii) Pituitary tumours—particularly ACTH and MSH producing tumours; Nelson's syndrome.
  (iii) Thyrotoxicosis;
  (iv) Diabetes mellitus;
  (v) Prolonged steroid therapy.

(e) Parasitic–chronic malaria, kala-azar.

(f) Nutritional deficiency :
  (i) Malabsorption syndrome, chronic cachexia, chronic liver disease kwashiorkor, etc :
  (ii) Pellagra :

(g) Metabolic :
  (i) Haemochromatosis (bronze diabetes)–generalised, bronze colouration ;
  (ii) Prophyria (congenital, variegate and cutanea tarda);
  (iii) Willson's disease (Hepatolenticular degeneration).

(h) Drugs : Busulphan, fixed drug eruption as may occur with sulphur drugs; steroids etc.

Causes of hypopigmentation :

Congenital–

(a) Albinism–Congenital absence of pigment in the skin either localised (piebaldism) or generalised;

(b) Vitiligo;

(c) Fanconi's syndrome;

(d) Phenylketonuria.

Acquired–

(a) Infections :

(i) Hypopigmented anaesthetic patches in tuberculoid leprosy (associated nerve thickening confirms the diagnosis);

(ii) Pityriasis alba;

(iii) Tinea versicolor—a fungal infection;

(iv) Eczematous dermatitis;

(v) Psoriasis.

(b) Endocrine factors :

(i) Hypopituitarism; (ii) Hypogonadism.

(c) Others :

(i) Alopecia areata; (ii) Chloroquine.

Varieties of skin eruptions :

(a) Macule– Abnormal colour of the skin in a localised area without elevation of depression, e.g. haemorrhages into the skin, rose spots of typhoid fever, secondary syphilis.

(b) Papule–A raised area from the surface, size about 5 mm. e.g. acne lichen.

(c) Vesicle–Raised from the surface and contains milky or serous fluid (size about 5 mm) eg chicken pox, small pox.

(d) Pustule–Raised from the surface, size about 5 mm. contains pus e.g. chicken pox, small pox.

(e) Bigger than papule involving epidermis and dermis e.g. leprosy.

Diets :

(f) Wheal–Raised from the surface of the skin with a pale centre and red periphery. Mainly type II.

(g) Blebs–Area of the epidermis raised from the surface, size more than 5mm; contains milky or serous fluid e.g. herpes, impetigo pemphigus vulgaris.

Haemorphagic spots (Purpura) :

(1) Petechiae : The spot is less than 1 mm in diameter and does not disappear on pressure by a glass slide.

(2) Suggillations : These are larger macules, more than 2 cm, in diameter and does not disappear on pressure.

(3) Ecchymoses : These are extensive purpuric macules.

(4) Haematoma : These are large haemorrhages in the skin causing elevation of the skin.

Causes of haemorrhagic spots (purpura) in the skin :

(A) Capillary endothelial defect :

(a) Vascular purpura (Sympatomatic)–

(i) Anaphylactoid purpura e.g. Henoch–Scchoenlein, purpura simplex.

(ii) Infections : infective endocarditis, septicaemia, meningococcal meningitis, typhoid fever etc.

(iii) Chemical agents and drugs (e.g. phenylbutazone, aspirin, indomethacin. Phenobarbitone, penicillin, sulphonamide, snake venom.)

(iv) Metabolic : uraemia, hepatic failure.
(v) Other symptomatic vascular purpura (e.g. Cushing's diseases, scurvy, dysproteinaemias like cryoglobulinaemia multiple myeloma, etc)

(b) Miscellaneous :
(i) Systemic disorders like collagen diseases e.g. polyarteritis nodosa, allergy;
(ii) Mechanical ;
(iii) Orthostatic.

(c) Congenital :
(i) Hereditary haemorrhagic telangiectasia (Osler-Rendu Weber disease);
(ii) von Willebrand's disease;
(iii) Hereditary capillary fragility;
(iv) Ehlers Danlos syndrome.

(B) Due to deficiency of blood platelets :
(1) Primary, idiopathic thrombocytopenic purpura
(2) Secondary :

(I) Common causes :
(i) Drug and chemicals–Cytosin arabinoside, busulphan, methotrexate, vincristine–all cause purpura by depressing the bone-marrow.
(ii) Aplastic anaemia.
(iii) Leukaemias, Lymphomas, myelofibrosis. disseminated carcinomas – cause thrombocytopenia by infiltrating the bone-morrow.
(iv) Hypersplenism–platelets are sequestered in the spleen.
(v) Systemic lupus erythematosus–autoimmunity
(vi) Liver diseases;
(vii) Infective endocarditis;

(viii) Deficiency of vit B_1–defective maturation;
(ix) After massive blood transfusion.
(x) Disseminated intravascular coagulation–excessive consumption of clotting factors.

(II) Rare causes :
(i) Wiskott-Aldrich syndrome–hereditary defect in maturation of platelet;
(ii) Trombotic thrombocytopenic purpura;
(iii) After prosthetic valve replacement;
(iv) Food allergy etc.

9. OEDEMA

Definition–A local or generalised condition in which the body tissues and/or the serous sacs contain an excessive amount of tissue fluid. In a restricted sense it means an increase in the extravascular component of the extracellular body fluid.

Sites–

( ) Dependent oedema or pitting oedema is classically found in congestive cardiac failure in ambulent patients; (a) at the ankle (b) on the dorsum of the foot, (c) and gradually ascends upward along the leg, thigh and trunk with increasing severity of the failure.

In patients confined to bed. examine the skin over the sacrum (small of the back). Other places like eyelids, abdomen (parietal oedema) should also be looked for. Oedema of the face, particularly puffiness of the lower eyelids is found in acute nephritic and nephrotic syndromes.

(II) Solid oedema of myxoedema of chronic lymphostatic disorders e.g. filariasis does not

pit on pressure.

Method of demonstration :

Firm pressure for 5 seconds over the medial malleolus, medial surface of lower end of tibia and sacral region will produce a relatively persistent dimple.

Types of oedema : (A) Symmetrical and (B) Asymmetrical. Symmetrical oedema may be either generalised or localised.

Causes of generalised symmetrical oedema :

(a) Congestive cardiac failure–Possible mechanisms are—

(i) Impairment of renal blood flow leading to a fall in the glomerular filtration rate resulting in excessive reabsorption of water and salt by the renal tubules.
[Normal renal blood flow–1.3 litres/minute. Glomerular filtration rate–180 litres/day of which 1.5 litres are excreted as urine.]

(ii) Increased central venous pressure→increased capillary pressure→transudation of fluid into interstitial space.

(iii) Secondary hyperaldosteronism and sodium retention. (ADH is found in the urine of patient with congestive cardiac failure)→increased retention of water in distal tubules.

(iv) Lymphatic factors like lymphangiectasis, incompetent valve, poor drainage also play important roles.

(v) Chronic passive congestion of liver (so reduction of albumin synthesis in the liver), poor appetite and loss of protein into the oedema fluid and in urine cause hypoalbuminaemia

leading to interstitial fluid accumulation.

(b) Renal :

(i) Nephrotic syndrome–Due to excessive loss of protein in the urine leading to diminished colloidal oncotic pressure (ii) Acute nephritis–inflammatory swelling of the glomeruli causes fall of glomerular filtration rate leading to relative increase in tubular reabsorption with consequent reduction in urine volume and expansion of intravascular fluid volume.

(c) Hypoproteinaemia : Oncotic pressure is mostly maintained by serum albumin, fall in concentration of which predisposes to oedema etc. The causes are–(i) Inadequate protein intake–e.g. (kwashiorkor, famine, pyloric obstruction with vomiting). (ii) Failure of digestion or absorption e.g. malabsorption syndrome, chronic pancreatitis, resection of considerable length of small intestine etc. (iii) Reduced synthesis in liver diseases like cirrhosis or chronic active hepatitis. (iv) Excessive loss of protein in the gastrointestinal tract (e.g. gluten enteropathy, ulcerative colitis, Crohn's disease, chronic gastrointestinal infection) (v) Excessive loss of protein in urine e.g. nephrotic syndrome.

Generalised symmetrical oedema may rarely be due to– (d) angioneurotic oedema, (e) idiosyncratic reaction of aspirin, potassium iodide, (f) excessive arsenic ingestion, (g) beriberi etc.

Localised symmetrical oedema may be caused by– (i) Obstruction of the superior vena cava or its main branches by mediastinal neoplasms, chronic mediastinal fibrosis, thoracic aneurysms, thrombosis etc. (ii) Erysipelas, cellulitis,

Ludwig's angina, (iii) Angioneurotic oedema, (iv) Dermatoses of various aetiologies, (v) inferior vena cava obstruction of various aetiologies, (vi) Milroy's disease (vii) Pretibial myxoedema, (viii) Epidemic dropsy etc.

Asymmetrical oedema is mainly due to (a) Local causes e.g.–(i) arteriovenous aneurysms, (ii) Milroy's disease, (iii) superficial or deep tissue infections, (iv) venous obstruction, (v) lymphatic obstruction e.g. by filariasis, metastatic carcinoma etc., (vi) Stings, bites and other causes of local inflammation; and rarely due to (b) General causes e.g. toxins, drugs angioneurotic oedema etc.

10. TEMPERATURE

Normal 98°F to 99°F, (normal body temperature shows a diurnal variation of 1.5°F with an increase towards evening, reaching the peak between 6 pm and 10 pm).

Subnormal–Below 98°F. (36. 7°C)

Pyrexia–Above 99°F. (37.2°C). An increase in the diurnal variation of more than 1.5°F is the rule, but the pattern of diurnal variation is commonly maintained.

Hyperpyrexia–Above 106°F (41.1°C)

Hypothermia–Below 95°F (35°C).

Causes of hyperpyrexia :

(1) Septicaemia (2) Lobar pneumonia (3) Heat stroke (4) Malaria (5) Pontine haemorrhage (6) Encephalitis (7) Pyelitis (8) Thyroid storm (9) Malignant hyperthermia (e.g. caused by halothange or succinylcholine) (10) Neuroleptic Malignant syndrome or NMS (caused by potent neuroleptics in therapeutic doses).

Causes of hypothermia :

(1) Myxoedema coma.

(2) Peripheral circulatory failure, congestive cardiac failure.
(3) Enteric fever, when there is haemorrhage or perforation.
(4) Accidental prolonged exposure to cold.
(5) Hypoglycaemia.
(6) Acute respiratory failure.
(7) Renal failure.
(8) Extreme wasting as in malignancy or starvation.
(9) Coma due to alcohol, barbiturates, chlorpromazine, tricyclic antidepressants, morphine etc.

Types of fever :

Continuous–The daily fluctuation is less than 1·5°F and temperature does not touch the baseline. This is commonly encountered in pneumococcal pneumonia, in second week of enteric fever, lobar pneumonia, rheumatic fever, miliary tuberculosis, meningitis etc.

Remittent—The diurnal fluctuation exceeds 2°F and does not touch the base line. This is commonly found in pulmonary tuberculosis, amoebic liver abscess, urinary tract infection etc.

Intermittent—Fever continues for several hours (usually 104°–105° F), and returns to normal sometime during the day, as occurs in vivax malaria, Types–(a) quotidian, (b) tertian, and (c) quartan.

(a) Quotidian–The paroxysm of intermittent fever occurs daily, as in septicaemia, double infection with p. vivax etc.
(b) Tertian–The paroxysm occurs on alternate

days as in benign tertian malaria due to p. vivax or malignant tertian due to p. falciparum.

(c) Quartan–The interval between the two consecutive paroxysmal attacks is two days as in quartan malaria due to p. malariae.

A combination of the above three types of fever may be found in a case of typhoid, i.e., remittent in the first week, continued in the second week and fluctuation, of 3° or 4°F in the third week.

Hectic–Temperature is characterised by a great swing e.g. a rise by 5°F during the night returning to normal in the morning accompained by sweating. This type is commonly found in septicaemia, empyema, advanced tuberculosis etc.

Shivering, commonly referred to as rigor (due to constriction of the skin vessels) occurs in infection by parasites (e.g. malaria); E coli infection, occasionally following transfusions or infusions due to some pyrogen reaction, and may be produced and perpetuated by intermittent administration of aspirin or other effective antipyretics.

The rise of temperature in acute E coli pyelitis and in pneumonia is very abrupt whereas in typhoid fever and in miliary tuberculosis the process in a gradual one. In typhoid fever, the temperature rises in a series to steps classically known as 'staircase phenomenon.'

The fall of temperature may occur by *crisis* or by *lysis.* The temperature falling quickly 6 to 12 hours is known as a crisis and is found in lobar pneumonia. Alternately the fever subsides gradually over several days by lysis infections untreated by antibiotics (as occurred in typhoid fever before the introduction of chloramphenicol) and in bronchopneumonia.

The course of fever may be regular (as in lobar pneu-

monia and malaria) or irregular (as in tuberculosis or bronchopneumonia).

Regular alternation of recurrent bouts of pyrexia with a period of apyrexia is known as Pel-Ebstein temperature and is sometimes seen in lymphomas (e.g. Hodgkins' disease) and an irregular alternation may sometimes be noticed in infective endocarditis.

Relapsing fever–This comprises a group of acute infectious diseases clinically characterised by cyclic periods of fever and apyrexia. Malaria is a good example, but the term is classically used for the fever caused by spirochaetes of the genus Borrelia recurrentis.

Pyrexia of unknow origin : (PUO or FUO)

I. Definition : When a fever of more than 101°F, persists for 2-3 weeks with the cause remaining obscure despite intensive study for 1 week, the fever is called pyrexia of unknown origin.

II. Causes : Some of the common causes are Hodgkin's and non-Hodgkin's lymphoma, carcinoma of lung, liver and other sites with or without metastasis, connective tissue disorders like systemic lupus erythematosus, polyarteritis nodosa, infections like tuberculosis brucellosis, subacute infective endocarditis subphrenic abscess, pyeloneph itis and hypersensitivity to a drug.

III. Investigations : PUO requires systematic and thorough clinical examination and intensive laboratory investigation to detect the cause.

Preliminary investigation (Step I) —

(i) History (including drung history) and thor ough clinical examination etc.

(ii) Routine haemogram including differential leu cocyte count and ESR to exclude abnor malmononuclear cells suggestive of glan

dular fever, leucopenia for enteric fever, viral fever and brucellosis, high ESR for collagen disease and paraprotinaemia, tuberculosis etc.

(iii) Midstream urine for routine examination microscopy, culture and sensitivity test for 6 consecutive days.

(iv) Liver function tests

(v) Plain X-ray of chest, (both P–A and lateral views)–to exclude tuberculosis, sarcoidosis and other chest infections.

(vi) For female patient–a high vaginal swab for culture and sensitivity test.

(vii) Venous blood culture, 3, taken at intervals of 1 hour, when temperature is > 101, 3°F(38.5°C) or more usually suffice, but for those who have received antimicrobials within the last 2 weeks and in whom endocarditis remains a possibility a total of 6 cultures should be taken over a 2 day period. And in emergent conditions 2 cultures are to be taken simultaneously from different anatomical sites. The femoral vein should preferably by avoided.

(viii)Plain X-ray of abdomen – so exclude subphrenic abscess enlargement of organs like kidney, liver, spleen, appendicular abscess etc.

(ix) Mantoux test.

(x) Throat swab for culture and sensitivity.

(xi) Routine examination of stool–for Entamoeba histolytica etc.

Step II :

(i) Stool culture and sensitivity for enteric fever and other salmonella infection.

(ii) Widal test–for enteric fever.

(iii) Intravenous pyelogram–when pathology in kidney is suspected or where size of the kidney is enlarged (perinephric abscess etc.)

(iv) Paul-Bunnell-test–for infectious mononucleosis, cytomegalovirus complement fixation test and test for toxoplasmosis–if atypical lymphocytosis or mononucleosis found in peripheral blood.

(v) Test lupus erythematosus or LE cell, antinulcear factor, rheumatoid factor or latex fixation tests if ESR is above 50 mm in first hour and clinically collagen disease is suspected.

(vi) Antistreptolysin (ASO) titre–if throat swab shows β-haemolytic streptococci and rheumatic fever is suspected.

(vii) Study of plasma proteins particularly immunoglobulins–when ESR is > 50 mm and multiple myeloma or paraproteinaemia is suspected.

(Viii) Liver scan–for primary or secondary liver cancer or amoebic liver abscess. If primary cancer is suspected it can be confirmed by alpha fetoprotein (AFP) level of blood.

(ix) Coomb's test for autoimmune haemolytic anaemia.

(x) Wassermann Reaction–for secondary syphilis.

(xi) Amoeba complement fixation test.

xii) Complement fixation test for Q–fever.

Step III :

(i) Arterial blood culture have shown to have no additional advantage over venous blood cultures. Bone-marrow cultures may help (when blood culture is negative) in patients with disseminated salmonellosis. tuberculosis, deep mycoses etc.

(ii) Cholecystogram.

(iii) Plain X-ray of skull and paranasal sinuses.

(iv) Liver biopsy–for primary liver cancer cryptic form of miliary TB.

(v) Screening of diaphragm when plain X-ray of abdomen suggests a subphrenic abscess.

(vi) Bone marrow–stained film to exclude multi ple myeloma and marrow culture for AFB and Brucella.

(vii) Kveim test–when plain X-ray chest and nega tive Mantoux test suggest sarcoidosis.

(viii) Biopsy of lymph gland–for tuberculosis; lymphoma etc.

Step IV :

The following tests are to be done only for rare disorders and for unusual presentation of common diseases :

(i) Dental X-ray – for dental abscess which may be painless and is overlooked.

(ii) Bipedal Lymphangiogram–for lymphoma.

(iii) Fibrin degradation product in plasma and urine–for disseminated intravascular coagulation.

(iv) Weil-Felix-reaction–for Rickettsial diseases.

Step V :

(i) Laparotomy–This is done when the above

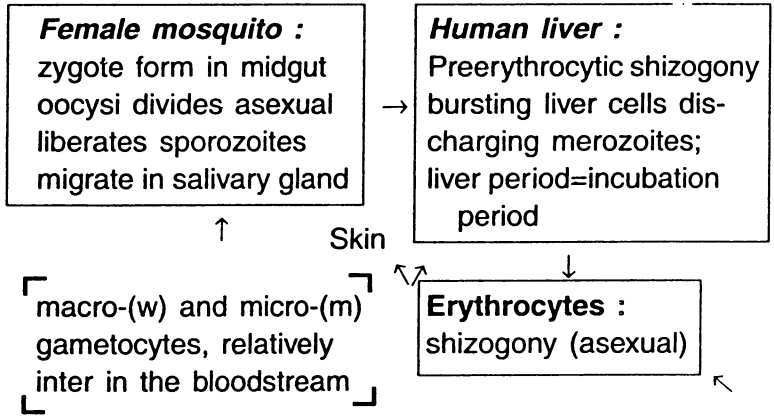
procedures fail and when factitious fever is excluded. Lymphoma, granuloma retroperitoneal abscess or arteritis may be con firmed and staged if required after biopsy.

MALARIA

Malaria is the most common infectious disease in the world (300–500 mio newly infected people per year India 2.3 Mil in 1991). There are three different types of malaria, all transmitted by female anophelese mosquitos (see fever period pages before) :

Benign type (1/3) : λ Malaria quartana (Plasmodium malaria)
λ Malaria tertiana (Pl. vivax and ovale)

Malignant type (2/3) λ malaria tropica (Pl. falciparum)
Double infections are possible, too.



[release of 6-24 merozoites]

Clinical features : Malaria is an acute febrile illness with a periodic picture. The dangerous, life threatening

malaria is caused by pl. falciparum, with the shortest incubation period of about 7 days. Relapses may occur in malaria tertiana as a result of reactivated liver hypnozoites. The general development is shown below :

Cold stage : Feeling of cold and apprehension, chills, malaise, headache, fatigue, shakes of the whole body (15 min-1h). chest, back abdominal and joint pain nausea vomiting clinically anaemia possible, mild jaundice with soft hepato/splenomegaly, skin cold, cyanotic and dry. pulse, low volume tachycardia.

Hot stage (2-6th) flush fever>40°C(<104°F). tachypnoea, nausea, vomiting weakness, confusion, delirious skin dry burning and flushed; pulse : high volume tachycardia.

Sweating stage (2–4th) : immense sweating temperature weak and sleepy, but more or less O.K.

Malaria	Fever perioidicity
–quartana	quartan (72h)
–tertiana	tertian (48h)
–tropica	irregular : sub-bi-tertian, (24/36/48h), quotidian

Thrombocytopenia is a marker of the severity of malaria.

Complications due to a disorders of the microcirculation is severe cases of malaria tropica (seldom in others) :

- λ Cerebral malaria → consiousness, generalized convulsion up to persisting coma (open-eyed), opisthotonos
- λ Cerebellar syndrome (common. in India) → taxia, Intention tremor, hypotonia, dysarthria and nystogymus
- λ Pulmonary oedema → possible at any stage, dyspnoea. crepitations ARDS (adult respiratory distress syndrome)

- λ Cardiovascular shock → collapse, skin extremely cold temp.
- λ Renal dysfunction → combined with jaundice, pro longed coma, hypoglycaemia and hypovolaemia.

Chronic malaria : large spleen and anaemia.

Malaria in infants appear with anaemia and paleness within a day or two children being breast fed and less likely to get malaria.

Diagnosis : History and examination

Blood examination :

Thick film : taken from the side of the fingerball. You should be able to read through the patch. if posssible use Field's stain.

Thin film : smaller drop than needed in thick film. Stain with Giemsa. In doubtful cases repeat blood examination every four hours. One negative slide does not mean no infection. Every 'or should be able to carry out this two blood examinations.

Fluorescent stain : (a) quantified Buffy coat technique (QBC),

(b) RNA-specific fluorochrome-benzothiocarboxypurine (BPC).

Serological tests (a) indirect fluorescent antibody test (IFAT).

Positive 6-10 days after beginning of sease (wildly used).

(b) indirect haemoagglutination test (IHA); only simple equipment necessary, but less sensitive.

(c) enzyme-linked immunosorbent assay (ELISA) small quantity of antigen required.

(d) radioimmunoassay (RIA); for research items.

Other tests : (a) Pl. falciparum-histidin-rich protein 2 (PFIHRP-2) paper-strip test prepared with monoclonal

antibodies to detect malaria tropica, but less sensitive than microscopic examination.

(b) polymerase chain reaction (PCR) and transcript amplification system (TAS) to detect DNA and RNA.

Start immediatly after diagnosis if the species in not known or you face a mixed infection treat as falciparum malaria.

Generally keep in mind :

1. Select falciparum drug treatment according to your local resistant pattern!
2. There are chloroquine-resistant species in some regions, so be aware of if success stays away!
3. Do not treat with chloroquine when used as prophylaxis!
4. Reexamine patient after some weeks for signs of recrudescence !

Drug groups	**Effectivity**	**Side effect**
Arylaminoalcohols	blood schizontocides	
λ Quinine/Qinidine (i.v.,p.o.)	severe m. tropical and in pregnancy	hypoglycaemia, allergy
λ Mefloquine (p.o.)	prophylaxis	bradycardia, acute brain syndrome not in pregnancy!
λ Halofantrine (p.o.)	multiresistent malaria tropica (MRPE)	prolongation of ECG QT interval !not in pregnancy!
4. **aminoquinolines:** λ Chloroquine (i.v.,p.o.)	blood schizontocides benign malaria uncomplicated m. tropica (not everywhere)	i.v. hypotension. longterm : blurring of vision, pruritus
λ Pyronaridine	MRPF	dizziness, headache GI-disorders

Drug groups	Effectivity	Side effect
8. **aminoquinolines:**	hypnozoites, gameto-cytocides	
λ Primaquine	redical cure of P. vi vaxlovale, eleminates sexual cycle in p. falciparum	haemolysis in G6PD deficiency methaemoglobin-aemia
λ WR-238.605	ten times more effective	new drug with less experience
Biguanides :	blood schizonticides	
λ Pyrimethamine	in combination with sulfonamides for acute treatment and prophylaxis of falciparum malaria	not in pregnancy and 6 weeks child Steven-Johnson syndrome
Sulfonamides :	blood schizonticides	
λ Sulfadoxine	in combination with	Urticaria, hepatitis,
λ Cotrimoxazole	pyrimethamine and proguanil	agranulocytosis; ! not in pregnancy!
Peroxides :	blood schizontocides	
λ Artemisinin	severe complicated falciparum; cerebral malaria	experimental stage new drugs; erytrotoxic bone-mar-
λ Artemether	severe falciparum	row, cardiotoxic
λ Artesunate	uncomplicated falciparum; better combined with mafloquine	Feticide
Naphthoquinones :	all growing stages	
λ Atavaquone	experimental drugs in	
λ BW566C80	research stage	

1997 the WHO announced that malaria becomes a major problem in the future. The scientific research dominated by the rich countries (USA and Europe) have no further interest in developing new effective drugs or vaccination against malaria. There are some ideas about future treatment, but

not yet in research state. The most difficult matter in malaria is the fast development of resistance especially plasmodium falciparum, which causes malaria tropica.

Treatment Table

Falciparum malaria

11. PULSE

(i) Definition–Pulse is the lateral expansion of the

Drug	Dosage
Quinine salt (not bisulphate type)	600 mg/8h po for 7 days in severe ill patients : 20mg/kg over 4th iv (max. 1.4g).
Next	followed by 10 mg/kg iv over 4h after 8-12h interval (max. 0.7g) this repeated untill pat can swallow to complete 7 days treatment 3
Pyrimethamine plus Sulfadoxine or in Fansidar-resistant areas Tetracycline	tablets as a single dose 250 mg/6th for 7 days
Mefloquine	20 mg/kg (base) divided in two doses with 6-8h interval po
Halofantrine	3 × 500 mg 6h in between and repeated after one week (on empty stomach)

Benign malaria

Drug	Dosage
Chloroquine base 150 mg = phosphate (po) 250 mg =sulphate (iv) 200 mg in P vivax and ovale cradieation therapy is to be added after above course with Primaquine	initially 600 mg (base) followed by 300 mg single dose 6-8h later next two days 300 mg single dose total amount of 3 days should be 25 mg/kg chloroquine base. 15 mg/d for 14-21 days

arterial wall imparted by the column of arterial blood due to contraction of the left vantricle.

(ii) Rate–Beats per minute, normal 60 to 100 per minute, average 72 beats per minute in adult and 130 per minute at birth.

Bradycardia–Rate less than 60 per minute.

Causes : (1) Physiological–in trained athletes, during sleep etc.

(2) Pathological
Vasovagal attacks.
Myxoedema.
Obstructive jaundice.
Raised intracranial tension.
Drug e.g. digitalis propranolol.
Different types of heart block; the rate is 20 to 40 beats per minute in complete heart block.
Sick sinus syndrome.
Other causes include hypothermia, acute nephritis, phaeochromocytoma, aortic stenosis, carotid slnus synciope etc.

Tachycardia–Rate more than 100 per minute, found normally in infants and in anxiety state.

Relative bradycardia–When with per degree rise of temperature [F] the pulse rate increase is less than 10 beats per minute, the condition is called relative bradycardia. For example, when temperature rises to 100°F pulse rate should be about 82 per minute normally, but less than this if there is relative bradycardia. Relative bradycardia is found in first week of enteric fever meningitis etc.

Relative tachycardia– Here pulse rate rises by more

than 10 beats per minute per degree rise of temperature and is seen in rheumatic carditis, tuberculosis etc.

Sinus tachycardia–Pulse rate varying between 100-160 per minute, It indicates that the cardiac impulse arises from the sino atrial node.

Physiological causes of sinus tachycardia : Exercise, emotion, belladonna group of drugs amyl nitrite or other vasodilators etc. are known to produce sinus tachycardia.

Pathological causes : Shock acute haemorrhage, fever, thyrotoxicosis, severe anaemia, congestive cardiac failure, cardiac neurosis, toxaemia, phaeochromocytoma, severe myocardial disease etc.

Paroxysmal tachycardia–more than 160 beats per min, usually between 180 and 200 per minute. The ventricles almost always respond to each atrial beat, rarely there may be 2 : 1 atrioventricular block.

Causes of paroxysmal atrial tachycardia (PAT) : Rheumatic carditis, mitral stenosis, ischaemic heart disease, hypertensive heart disease, thyrotoxicosis, atrial septal defect. Wolf-Parkinson-White (WPW) syndrome (characterised by paroxysmal atrial tachycardia short P-R interval, wide QRS complex, a slur on the initial phase of the QRS complex–the delta wave), cardiomyopathy functional e.g. anxiety state etc.

Exertion, emotion, coffee, alcohol etc. are the aggravating factors. PAT with block is sometimes encountered in digitalis intoxication with hypokalaemia.

(iii) *Rhythm*–Spacing of successive pulse waves in time. The rhythm may be regular or irregular, the latter may be completely irregular (irregularly irregular) or the rhythm is

occasionally interrupted by slight irregularity or a recurring pattern of irregularity (regularly irregular). Thus the pulse in atrial fibrillation, multiple extrasystoles, partial heart block with dropped beats, atrial flutter with varying degrees of A-V block are *irregularly irregular* whereas in premature beats, sinus arrhythmia and atrial flutter with 2 : 1 block it is *regularly irregular*

Sinus rhythms : Impulse originates in the SA node. Types–(a) sinus bradycardia, (b) sinus tachycardia, (c) sinus arrhythmia in which the heart rate increase with inspiration and slows down in expiration. It is increased by deep breathing and abolished by exercise. This variety is normally encountered in children and young people and usually absent in elderly individuals large ASD, heart failure sick sinus syndrome etc.

Ectopic rhythms : Here the impulse arises from a site other than the S-A node e.g. in the atria, A-V node or ventricles. The rhythm may be regular or irregular.

Premature (Ectopic) beat or Extrasystole–It arises from some abnormal focus in the heart, occurs prematurely, is small and followed by a compensatory pause. It may be caused by overindulgence in coffee, tobacco and alcohol; dyspepsia, anxiety or organic heart disease like rheumatic carditis, ischaemic heart disease, hypertension or cardiomyopathy etc. When a premature beat follows each normal beat, the pulse is said to be coupled and designated as *pulsus begeminus.*

Atrial fibrillation, atrial flutter and atrial tachycardia may be regarded as similar ectopic rhythms which differ only in the rate of the ectopic atrial focus. A rate of 140 to 220 per minute result in atrial tachycardia; a rate of 250 to 350 per minute is atrial flutter and rates above 350 per minute is atrial fibrillation.

Paroxysmal tachycardia may be supraventricular (atrial, atrial tachycardia with A-V block nodal) or ventricular. Ventricular tachycardia (VT) is commonly encountered in ischaemic heart disease, digitalis toxicity, electrolyte and or acid base imbalance etc.

Atrial flutter–Rapid regular generation of impulse occurs at a rate of 250-350 per minute in the atria, all of which cannot traverse the A-V node and so usually there is 2 : 1 physiological heart block. The block may increase to 3 : 1 or 4 : 1 when carotid pressure is applied, (due to increased vagal tone) and so the ventricular rate diminishes transiently but the sinus rhythm is not restored. Vagal stimulation in a case of paroxysmal supraventricular tachycardia (PSVT) will either terminate the paroxysm or have not effect. Rheu-matic heart disease, ischaemic heart disease and thyrotoxicosis are the common causes of atrial flutter.

Atrial fibrillation–Rapid fibrillation waves (f waves in ECG take the place of normal atrial contractions and ventricles respond at random. The atrial rate is between 350 and 600 per minute. The A-V node cannot conduct so many impulses and varying degrees of heart block always exist in an untreated patient as a result of concealed conduction. The ventricular rate is usually 100–150 per minute. It is recognised clinically by complete irregularity of the pulse both in rate and volume and the varying intensities of the irregular heart sounds. pheumatic, ischaemic, hypertensive and thyrotoxic heart diseases are the common causes of atrial fibrillation.

Heart block–It is either S-A of A-V block. In S-A block a complete cardiac cycle is missed so that a gap appears in the pulse and is due to increased vagal tone or intrinsic SA nodal disease. Conduction between the atria and the

ventricles is impaired in A-V block, which may be of three types :

(a) First degree block : detected most commonly by the ECG which reveals P-R interval more than 0.2 second.

(b) Second degree of partial heart block : Some impulses from the atrial do not reach the ventrical. It may be of three types :–

(1) Mobitz Type I or Wenckebach type : Gradual prolongation of P-R interval followed by a dropped beat–the cycle is repeated; better prognosis.

(2) Mobitz Type II (periodic block)–The P-R or P-P intervals remain unaltered and any one P-wave is not followed by QRS complex e.g. 6 : 5 A-V block.

(3) Second degree constant block e.g. 2 : 1, 3 : 1 or 4 : 1 A-V blocks.

(c) Third degree or complete heart block : In this type no impulse from atria reaches the ventricles and hence the atria and ventricles contract independent of each other and the ventricular rate is usually between 20 to 40 per minute. Ischaemic heart disease, calcific aortic stenosis rheumatic cardiovascular diseases, syphilitic heart disease, congenital cardiac lesions, digitalis, infectious diseases like diphtheria etc. are the common causes of heart block.

Ventricular fibrillation (VF) : This arrhythmia is characterised by rapid, irregular, uncoordinated and ineffective contractions of the ventricles. It may occur in acute myocardial infarction as a complication of general anaesthesia by chloroform of cyclopropane, after toxic doses of digitalis or quinidine etc. Clinical presentation may be as Stocks-Adams syndrome with syncope and convulsion.

(iv) *Volume* : It is defined as amplitude of pulse wave. It signifies left ventricular output per beat.

Causes of high volume pulse : Hyperkinetic circulatory

states eg, fever severe anaemia, thyrotoxicosis, aortic incompetence and corpulmonale, atheros clerosis of aorta, complete heart block or gross bradycardia from any cause etc.

Causes of low volume pulse : aortic stenosis, tight mitral stenosis, pulmonary stenosis, severe pulmonary hypertension, obstructive cardiomyopathy, pericardial effusion, shock due to any cause etc.

(v) *Tension* : Pressure required to obliterate the pulse is known as systolic tension. Optimum pressure exerted by the proximal finger to have the maximum thrust felt by the middle finger is said to be diastolic tension. Sphygmomanometry being such a simple, easy, and accurate procedure, estimation of tension had become obsolete.

(vi) *Condition of arterial wall* : It should be noted for evidence of arterial thickening or undue mobility. Using 3 fingers, exsanguinate the artery and then roll over the bony surface. Sufficient pressure is applied on the brachial artery to abolish pulsation in the radial artery which should then be rolled over the bony surface. Normally the arterial wall is not palpable. It may be palpable in old age due to arteriosclerosis (tortusity and cord like thickening).

(vii) *Equality* : Comparison of volume of pulse of two upper and lower extremities. *Causes of inequality between the radial pulses are—*

(i) Anatomical variations.
(ii) Thoracic inlet syndrome.
(iii) Pre-subclavian coarctation.
(iv) Pressure over axillary artery.
(v) Volkmann's ischaemic contracture.
(vi) Aortic arch syndrome.
(vii) Supravalvular aortic stenosis etc.

N.B.– Radial artery and femoral artery should be palpated simultaneously to detect coarctation of aorta. Delayed pulsation of femoral artery compared to that of radial artery, i.e. radio femoral delay suggests coarctation of aorta. Difference of timing of radial and dorsalis pedis pulse is 0.02 to 0.03 second. Inequality of brachial pulse may be due to thrombosis, embolism or atherosclerosis of aorta.

Bounding pulse : A large pulse wave signifying a high pulse pressure, associated with increased blood flow, seen in hyperkinetic circulartory states.

12 RESPIRATION

The rate, rhythm and type of breathing are determined by placing the hand over the epigastrium and noting the features of respiration without the patient's knowledge.

(a) *Rate :* 18-20 per minute in adults.

Increased rate (tachypnoea)

(i) Fever
(ii) Exertion
(iii) Excitement and emotion
(iv) Pulmonary diseases e.g. pneumonia pulmonary embolism etc.
(v) Hypoxaemia from cardiac or pulmonary causes as in interference with reflex control of respira tion by structural changes in lung e.g. fibrosing alveolitis.
(vi) Shallow and frequent breathing in pleurisy and peritonitis.

(b) Rhythm : Varies considerably even among nor mal individuals

Biot's breathing : This is a type of periodic breathing where periods of apnoea are interrupted by a phase of hyperpnoea consisting of four or five breaths only all of

which are of the same amplitude and the beginning and the end of the phases are abrupt but no waxing and waning of respiration is seen. This is commonly found in children suffering from meningitis, but may occur in primary brainstem lesions and in increased intracranial tensions.

Inspiration may be unduly prolonged in laryngeala or tracheal diseases, whereas expiration may be prolonged in bronchial or pulmonary disease.

Cheyne-Stoke breathing : This is the commonest variety of periodic breathing (independently described by John Cheyne in 1818 and William Stokes in 1846) in which the respiration becomes gradually deeper until a peak is reached and this is followed by a complete pause of breathing or apnoea. The pause lasts for 10 to 30 seconds while the hyperpnoeic phase of 30 or more breaths lasts for 1 to 3 minutes. The patient may remain asymptomatic; it is usually prominent at night.

Causes of Cheyne-Stokes respiration :

(i) Left ventricular failure : Commonest cause, par ticularly in those with degenerative arterial diaeases.
(ii) Renal failure.
(iii) Morphine poisoning.
(iv) Bronchopneumonia or other respiratory infections in the elderly.
(v) Occasionally during recovery from Stokes-Adams attacks.
(vi) Incresed intracranial tension, cerebral haemorrhage, thrombosis cerebral tumours and severe head injuries.
(vii) In sleep in apparently healthy elderly subjects.
(viii) In normal subjects at high altitudes and after hyper ventilation.

(c) *Types* :		
(i) Thoracic, e.g. In women*	(ii) Abdominal e.g. young children	(iii) Abdominc-thoracic e.g. In men
Anxiety states Hysteria, Diphragmatic palsy. Acute peritonitis. Huge ascites.	Pneumothorax, chest movement due to losing spondylitis.	Pleurisy (restriction of severe pain). Anky-Intercostal paralysis.

13. BLOOD PRESSURE

Blood pressure is measured by the sphygmomanometer. The width of the cuff is 12 cm for an adult, 3 inches for young children. 1 inch for infants and the length should be no less than 25 cm. To avoid a falsely high blood pressure in the leg a wider (8 inch) cuff should be used as this compresses the thigh more effectively than the narrower one which is used for the arm.

Casual recording of the blood pressure may not give the true figure due to exercise, fear of emotion and thus it varies from time to time. So the blood pressure should be recorded with the patient at rest in a comfortable position.

To measure blood pressure in the arm the patient should lie flat, on his back, whereas for that of the leg the patient should lie prone (as the systolic pressure will be much higher if the patient sits or stands up). Before commencing the recording of blood pressure, the patient should remain at rest in supine (or prone) positions for 5 minutes.

In recording the blood pressure, particular care should be taken to wrap the cuff firmly and evenly around the base of the arm about 2.5 cm above the elbow joint, with the middle of the rubber bag over the brachial artery. The arm

* in last trimen on of pregnency

should not be hyperextended as it may introduce error in recording the diastolic pressure. The cuff should then be inflated till the radial pulse disappears and then deflated slowly. The point at which radial pulse first reappears indicates the systolic pressure. As the cuff is further deflated, pulsation of radial artery gradully assumes a water hammer character and then all on a sudden resumes its normal character, the reading corresponding to the sudden change represent the diastolic pressure.

In auscultatory method the diaphragm of the stethoscope should be placed over the brachial artery close to or under the edge of the sphygmomanometer cuff. Care should be taken that the diaphragm is not pressed heavily over the artery as it may give wrong diastolic pressure. The cuff is then inflated quickly to 20 mm above the systolic pressure recorded by palpation and slowly deflated.

The highest level at which successive clear, tapping sounds (Korotkoff's phase I) are heard is the systolic pressure. As the pressure is further lowered in the cuff, the point at which louder and sharper sounds suddenly become muffled (Korotkoff's phase IV) or inaudible (Korotkoff's phase V) indicate the diastolic pressure. Normally sounds disappear few mm below the change over/but in aortic incompetence sounds may be audible even at zero pressure. Experimentally it has been shown that direct recording of intraarterial diastolic pressure more closely correlates with mufling than when the sounds completely disappear.

To measure the blood pressure in the thigh the sphygmomanometer cuff should be adjusted around the lower part of the bare thigh with the patient in prone position. The diaphragm of the stethoscope is to be placed over popliteal artery.

N. B. – If anaeroid gauges are used, they must be calibrated every 6 months against a mercury manometer.

Ausculatory (silent) gap–

Sometimes as phase of silence may separate the first appearance of Korotkoff's sounds from their second appearance at a lower pressure and this the auscultatory gap. The phenomenon tends to occur in–(i) venous distension, (ii) reduced arterial flow velocity into the arms as in severe aortic stenosis. In such cases the diastolic pressure will be overestimated if recorded at the point of first muffling of sounds and the systolic one will be underestimated if recorded at the point of second appearance of sounds. The auscultatory gap is of obvious clinical importance in systemic hypertension.

In an arrhythmia, the higher pressure of the beat following an ectopic beat should be ignored.

In atrial fibrillation the systolic pressure should be taken at the point where the majority of beats come through and the diastolic pressure where the majority of beats become muffled.

When there is a differnece of pulse volumes between the two arms as in presubclavian coarctation, aortic arch syndrome. supraclavicular aortic, stenosis, pressure over the brachial artery by enlarged lymph gland etc. blood pressure in both the arms should be recorded. Slight disparity (less than 5 mm Hg) between reading from each arm is common in atherosclerotic and hypertensive patients and is not of much clinical significance.

Blood pressure should also be recorded in both arms and legs when there is feeble or delayed pulsation in femoral, popliteal, posterior tibial or dorsalis pedis arteries as may occur in saddle shaped embolism at the bifurcation of the abdominal aorta and more significantly in coarctation of aorta.

Normal Blood pressure :

In infancy the systolic pressure is 75 to 90 mm Hg; in childhood 90 to 110 mm Hg : and in puberty 100 to 120 mm Hg. The diastolic pressure varies from 50 to 70 mm Hg. till puberty. Blood pressure varies widely in healthy adult subjects. Systolic pressure varies from 100 to 145 mm Hg and diastolic from 60 to 90 mm Hg. The upper limit of the normal blood pressure for adults below 45 years is 130/80 mm Hg, for adults above 45 years 140/90 mm Hg; the systolic pressure in legs is up to 20 mm Hg above that in the arms in a normal individual in the horizontal position, but the diastolic ones are almost identical.

Pulse pressure is the difference between systolic and diastolic pressures. Normally it is 30 to 60 mm Hg.

In children below 15 years, systolic pressure over 130 mm Hg and diastolic pressure over 80 mm Hg is considered hypertension.

In adult individuals, if the blood pressures are in excess* (160/100 MHg) of the above mentioned values after several controls, hypertension should be diagnosed.

Divergent blood pressures (eg 160/20 mm Hg) are found in arteriosclerosis, aortic incompetence, pheochromocytoma etc.

Systolic hypertension is encountered in atherosclerosis, aortic incompetence, complete heart block with severe bradycardia etc. Diastolic hypertension is encountered in essential hypertension, renal diaseases, eclampsia, Coushing's syndrome, pheochromocytoma etc.

N.B.– Very recently a simple method has been devised that records only the systolic blood pressure. The instruments is known as finger sphygmomanometer and can be used on any of the four fingers. Compared to the conven-

*(>160/100 mm Hg)

tional device, it is claimed to be a more reliable indicator for making diagnostic and therapeutic decisions and in one recent study it had a specificify of 98.5% during routine screening compared to the conventional mercury column device that had a specificity of 97.6% (BMJ, 1986, 293; 775). To sum up a few recent recommendations of the British Hypertension Society (BMJ, 1986, 293 611) are outlined here with some modifications.

(i) An anaeroid type loses accuracy over time and so it should be checked at different pressure levels by connecting with a Y piece to the tubing of a standardised mercury manometer.

(ii) Bladder lengths should be 80 cm of arm circumference; 35 cm for a normal and lean arm, 42 cm for a muscular and obese arm, < 12 cm for children below 5 years.

(iii) Sitting and supine pressures make little difference in BP and arm must be at heart level and supported during measurement otherwise the BP reading will be falsely high.

(iv) The mercury manometer must be vertical, at eye level and not more than 3 feet from the observer.

(v) A digit preference bias is best avoided by recording to the nearest 2mm Hg.

(vi) The auscultatory (silent) gap occurs when Korotkoff sounds disappear between the systolic and diastolic pressure and may lead to underestimation of the systolic pressure unless first recorded by palpation and if a silent gap is present, it must be clearly recorded.

(vii) The BP of children below 5 years cannot be measured easily by the usual BP instrument and in them the BP measured on different occasions varies considerably. Thus only when clinically indicated measurement of BP in them should be undertaken and moderate deviations from normal should be ignored.

REGIONAL EXAMINATION

HEAD AND NECK

(a) Inspection and palpation. Look for any abnormalities in or for position, size, shape, any asymmetry, deformity, irregularity and depression or elevation. in Kippel-Feil syndrome, the neck is short mobility restricted, a characteristic posture of the neck is seen and there may be mirror movements of the limbs. Slight bending may be noticed in a defect of vision (an attempt by the patient of compensate as occurs in head tilting to the opposite side with *superior oblique paralysis* of one side) or in abscesses or localised infections of the neck avoid discomfort. Hypertrophic changes in bones as with an underlying *meningioma* without any defect in the scalp may occasionally be found. Bossing of skull bones and a square shape may be found in *rickets*. The frontal eminences are very much exaggerated, the bridge of the nose is depressed and the forehead is vertical in *congenital syphilis*. The skull assumes a globular form in *hydrocephalus*. The sutures are opened up and imperfectly ossified areas are seen in *craniotabes*. The skull is bigger particularly in the transverse diameter in Paget's disease. Skull appears large in contrast to short stature in *achondroplasia*. Skull appears small compared to prominent supraorbital ridges and lower jaw in *acromegaly*. Long headed skull is known as *dolicocephaly* where as a bulletheaded skull is called *brachycephaly*. In *oxycephaly* or "steeple head" the skull is conical or tower shaped because the coronal and saggital sutures undergo premature synostosis and is seen in *gargoylism*,

Apert's syndrome etc. Oxycephaly is also known as *acrocephaly* and shows overgrowth of the vertex, exophthalmos, optic atrophy and a divergent squint.

Localised swelling of the skull may be present due to sebaceous cyst, tumours like osteoma, fibroma, sceondary deposit, acute leukaemias, dermoid cyst, encephalocele etc. *Leontiasis ossea* is a progressive irregular enlargement of the cranial and facial bones resulting in asymmetry and the superior maxilla is especially prominent. In Parry-Romberg disease there is facial hamiatrophy.

(b) Occasionally a bruit may be audible on auscultation over the temporal parietal and forntal regions, and may be due to–

(i) Carotido-cavernous fistula (thrills and bruits over the eyes).

(ii) Intracranial aneurysms.

(iii) Intracranial angiomas e.g. Struge-Weber syndrome (haemangiomas of leptomeninges with mucocutaneous haemangiomas along the distribution of trigeminal nerve and facial naevus one on side).

(iv) Angiomas of scalp.

(v) Brain tumours.

(vi) Paget's disease.

(c) Neck rigidity (must be seen as a routine).

(d) Any evidence of exophthalmos should be looked for.

(e) Involuntary movements of the head in diseases e.g.– (i) Park-insonism–constant tremor. (ii) Habit spasms–sudeen jerky movement with facial grimace. (iii) de Musset's sign–jerky head movements

with each heart beat seen in severe aortic regurgitation (iv) Chorea–sudden, jerky movements.

(f) *Neck Veins :* The patient should be propped up by a back rest to any angle that best and *maximally* demonstrates the jugular venous pulsation (the angle is that between the trunk and the level of the bed at the hip joint). The top to the oscillating venous blood column should be identified and the different waves determined. Normally the pulsations of the neck veins should not be above the root of the neck at an angle of 45% If it is significantly above that level most possibly the patient is in congestive cardiac failure. If the vein is engorged but not pulsatile *superior mediastinal syndrome* is suspected where there is obstruction to venous return in the SVC.

The internal jugular vein is ideally examined in preference to the external jugular and venous pulsations both sides of neck should be seen as a routine, but that of the right side is a better indicator of the activities of the right heart.

Hepato Jugular reflex : Increased jugular venous pulsations can be demonstrated incongestive cardiac failure by applying pressure on the liver or the anterior abdominal wall. This increases the intraabdominal venous pressure leading to increased venous return to the right heart which cannot be compensated in heart failure and even early congestive cardiac failure can be diagnosed. It is better termed as abdominojugular reflux.

Carotid artery : Normally the carotid artery pulsation is seen anterior to the sternomastoid muscle. The pulsation is exaggerated in nervous individuals, in normal persons during excitement, aortic incompetence, thyrotoxicosis,

fever severe anaemia pregnancy, patent ductus arteriosus, coarctation of the aorta and kinking of the carotid artery (kinked carotid) due to elevation of the aortic arch as a result of hypertension or atherosclerosis, particularly in females, on the right side. Carotid kinking may be confused with the pulsating aneurysm at the base of the right common carotid artery.

The pulsation is *diminished* in carotid artery stenosis, aortic arch syndrome, severe aortic stenosis, mitral stenosis and pulmonary stenosis.

The exaggerated carotid pulsation in aortic incompetence is known as Corrigan's sign. Inspection of carotid pulsation may reveal atrial or ventricular premature beats as also atrial fibrillation. Enlargement of the carotid artery with each pulsation is seen in aneurysm of the carotid artery as well as on the right side in kinked carotid. Silent carotid pulsation is a common feature in severe aortic stenosis and severe pulmonary stenosis.

(g) *Thyroid Gland*: The gland should ideally by palpated from behind after well exposure of the neck which is slightly flexed. Patient must be in the sitting posture. The patient is asked to swallow and note whether the gland moves up and down. The gland should also be examined from the front by Kocher's method with the patient supine and the neck adequately extended by placing a pillow in the nape of the neck. The isthmus and the two lobes are to be palpated. Thrills or bruits over the gland may be found on routine examination.

(h) *Lymph Glands*: Cervical lymph glands are examined from behind with the neck slightly flexed. Glands should be palpated in a definite order starting from the occipital lymph glands and gradually proceeding to postauricular, anterior auricular, tonsillar, submandibular and submental lymph

glands, glands of the anterior and posterior triangles of the neck both on the upper and lower parts and lastly the supraclavicular lymph glands.

After the examination of the cervical lymph glands, the axillary, the epitrochlear and the inguinal lymph glands are palpated as a routine. The group or groups of glands affected, their consistency, tenderness and whether these are discrete or matted are to be noted very carefully to arrive at a clinical diagnosis.

Cervical lymph glands may be enlarged in–

1. *Infections e.g.–*

(a) Infections from the surrounding area, e.g. aural infection, throat infection, scalp infection. Usually caused by pyogenic streptococci and staphylococci.
(b) Tuberculosis (Cervical adenitis or scrofula).
(c) Infectious mononucleosis.
(d) Syphilis.
(e) Rubella.
(f) Viral (e.g. cytomegalovirus infection and pharyngoconjunctival fever caused by adenovirus).
(g) Toxoplasmosis.
(h) Histoplasmosis.
(i) Brucellosis.

II. *Neoplastic condition e.g.–*

(1) Hodgkin's and non-Hodgkin lymphomas.
(2) Chronic lymphatic leukaemia.
(3) Acute leukaemias.
(4) Metastases from solid tumour e.g. carcinoma of head, neck, thyroid, lung, stomach, breast etc.

III. *Miscellaneous conditions e.g.–*

(1) Sarcoidosis.
(2) Connective tissue diseases like systemic lupus erythematosus (SLE) rheumatoid arthritis, dermatomyositis etc.

CAUSES OF GENERALISED LYMPHADENOPATHY

(Involving more than two separate node groups)

I. Hodgkin's and non-Hodgkin lymphomas,
II. Acute and chronic lymphatic leukaemias,
III. Miliary tuberculosis,
IV. Infectious mononucleosis,
V. Sarcoidosis,
VI. Toxoplasmosis and histoplasmosis,
VII. Secondary syphilis,
VIII. Brucellosis,
IX. Histiocytosis,
X. Infectious hepatitis,
XI. Immunoblastic lymphadenopathy,
XII. Persistent generalised lymphadenopathy. (PGL) and
XIII. Acquired Immune Deficiency Syndrome (AIDS).

When there is generalised lymphoglandular enlargement, routine clinical examination of liver and spleen should be done and any tenderness in the sternum or iliac crest or other bones (due to marrow hyperplasia) is to be found out. In leukaemia, sarcoidosis and tuberculosis chest X-rays should be taken. When leukaemia and lymphomas are suspected, examination of peripheral blood for the detection of abnormal cells and examination of bone-marrow by sternal puncture of iliac crest biopsy are essential for confirmation of the diagnosis. Next procedure is to remove a lymph gland (gland biopsy) for histological examination. The gland which is most superficial and isolated should be selected. This procedure may be helpful in differentiating tuberculosis from lymphoma at a certain stage of the disease. Lung tomography (to demonstrate mediastinal and hilarlymphadenopathy), lymphangiography (to exclude involvement of abdominal lymph glands) and

bone scan (to note whether any skeletal structure is involved or not), are done in a suspected case of lymphoma. Even laparotomy and splenectomy are needed for confirmaion of the diagnosis and staging of lymphomas.

CLUBBING : This is physical sign characterised by bulbous changes and diffuse enlargement of the terminal phalanges of the fingers and toes and is due to an increase in the volume of the soft tissues as well as increased curvature of the nails in both longitudinal and transverse plains. The angle between the nail and nail bed (the Lovibond's angle), gets obliterated and hence clubbing is also known as *Lovibond's sign.* The phenomenon of clubbing is also termed *Hippocratic fingers.* Hypertrophic osteoarthropathy is usually considered as a further extension of the clubbing process and in this the changes are in the larger bones and joints of distal parts of limbs.

Pathology–Proliferation of subungual connective tissue.

Degree : (a) First degree–There is only increased fluctuation of the nail bed.

(b) Second degree–There is an increased anteroposterior and transverse diameter in addition to fluctuation.

(c) Third degree–Combination of the changes described above and increased pulp tissue.

(d) Fourth degree–Combination of the above changes and subperiosteal thickening in bones of wrist and ankle.

In hypertrophic osteoarthropathy there is thickening of the periosteum of radius, ulna, tibia and fibula in addition to the clubbing of fingers, Hypertrophic osteoarthropathy is also known as secondary hypertrophic osteoarthritis. It is frequently associated with disorders of the lungs particularly bronchial carcinoma. It also occurs in association with pleural tumours but is quite rare in secondary tumours of lung. It may also be either familial or idiopathic. When

sufficiently progressed, ribs, clavicles and scapulae may be involved. Any disorder causing clubbing may cause hypertrophic pulmonary osteoarthropathy. The most reliable sign for mild clubbing is the obliteration of the Lovibond's angle.

Mechanism : It is known that clubbing mostly occurs first in the thumb and index fingers, subsequently spreading to the other digits but the exact mechanism is not definitely known. Some theories have been postulated. These include–(i) Toxic e.g. infective endocarditis. (ii) Anoxia e.g. congenital cyanotic group of heart diseases (e.g. Fallot's tetralogy (iii) Reflex phenomena–Improvement of clubbing in bronchogenic carcinoma after vagotomy supports reflex theory. (iv) Metabolic–clubbing in thyrotoxicosis and in acromegaly may probably be due to abnormal metabolic process. (v) Alteration of the pressure gradient between the radial artery and digital artery may play some role in the formation of clubbing.

Lobectomy in Pancoast's tumour and decortication in empyema may produce some improvement of clubbing. All these also may improve hypertrophic pulmonary osteoarthropathy.

Causes of Clubbing : These may be remembered by the following mnemonic :

C	L	U	BB	I	N	G
Cardiac diseases like congenital cyanotic heart diseases, infective endocarditis, cardiac tumours.	Lung disease (a) Bronchiectasis (b) Lung abscess (c) Empyema. (d) Fibrocaseous also type of tuberculosis (e) Bronchogenic carcinoma.	Ulcerative colitis	Biliary and other types of cirrhosis	Intestinal (Steatorrhoea) Regional enteritis intestinal tuberculosis, abdominal Hodgkin's chronic bacillary and amoebic dysentery	Normal in some people	Genetic factors

Miscellaneous Causes

(1) Occupational (2) Thyrotoxicosis (3) Acromegaly (4) Carpal tunnel syndrome (5) Sarcoidosis (6) Asbestosis

Unilateral clubbing may be found in presubclavian coarctation, bronchogenic carcinoma and in aneurysm of right subclavian artery.

Painful clubbing may be in bronchogenic carcinoma.

In the presence of hypertrophic osteoarthopathy the most important condition to be excluded is an intrathoracic neoplasm and it has been said that osteoarthropathy militates against a diagnosis of tuberculosis.

UPPERLIMBS : EXAMINATION OF ARMS AND HANDS :

(A) Length of both arms and arm span is compared with the trunk;–this may help in the diagnosis of Marfan's syndrome.

(B) Following points are noted in examination of hand:

(i) Clubbing--Vide supra.

(ii) Digital throbbing–It signifies vasodilatation, it can be demonstrated by holding the fingers of the patient's right hand with the fingers of the examiner's right hand in the position of flexion.

(iii) Capillary pulsation–This is classically demonstrated on the inner surface of the lower lip which is everted and a glass slides is pressed firmly on the mucous membrane to produce an area of blanching. The blanched area becomes pink with each systole in aortic incompetence. This can be demonstrated in the nails also.

Nails may show significant *pitting* in psoriasis.

(iv) Splinter haemorrhages under the nails may be

present in infective endocarditis, scurvy, rheumatic heart disease, occasionally in normal subjects trichinosis, rheumatoid arthritis SLE etc.

(v) Osler's node–It has a pale centre with surrounding erythema due to arteriolar embolism. The lesion is characteristic of infective endocarditis and is found in fingers or toes, in the palms or in the sole.

(vi) Hand may be warm and signifies thyrotoxicosis, wet beri-beri, chronic respiratory failure and Paget's disease of bone. Cold hand may be due to exposure to cold, neurosis, low cardiac output states and Raynaud's phenomenon.

(vii) Flat (even concave), fragile or spoon shaped nail (koilonychia) may be found in iron deficiency anaemia, thyrotoxicosis or it may be congenital; white nails (leuconychia) may be found in chronic liver diseases (like portal cirrhosis) including cardiac cirrhosis.

(viii) Palmar erythema may be found in physiological conditions like pregnancy and in pathological conditions like fever, thyrotoxicosis, rheumatoid arthritis, anoxic corpulmonale, hepatic failure, chronic alcoholism etc.

(ix) Any wasting of muscle is to be noted. This is commonly found in motor neurone diseases, thoracic inlet syndrome, carpal tunnel syndrome, lead neuritis and leprosy etc.

Any atrophy of the skin is to be noted which is commonly found in old age, reumatoid arthritis and in osteoarthritis.

(x) Presence of only one crease in palm (simian

crease) is found in mongolism 21.

(xi) Tremor : This may be physiological due to anxiety, fear and in old age. Pathological causes of tremor are as follows : familial toxic (chronic alcoholism or tobacco), thyrotoxicosis, uraemic twitchings flapping tremor of portocaval (hepatic) encephalopathy, static tremor of parkinsonism, dynamic tremor of cerebellar disorder, tremor due to belladonna poisoning.

(xii) Involuntary movements like chorea, athetosis, hemiballismus must be noted. Similarly other abnormal movements like tics, myoclonus and fasciculation may be present in different neurological disorders.

(xiii) Joints may show either evidence of osteoarthritis, rheumatoid arthritis, psoriatic arthropathy, features of gout and pulmonary osteoarthropathy.

(xiv) Congenital deformities : (a) Arachnodactyly– characterised by elongated spider fingers and toes (found in Marfan's syndrome), (b) Syndactyly and polydactyly : These may be hereditary or familial and may be found in association with ventricular septal defect, Fallot's tetralogy, Laurence -Moon-Biedl syndrome, Apert's syndrome, Chondroectodermal dysplasia (Ellis-van Creveld syndrome) etc.

(xv) Heberden's nodes : These are hard bony nodules found at the base of the distal phalanges or at the distal ends of middle phalanges. These are found in osteoarthrosis of finger joints and in women as a hereditary condition.

(C) Different groups of lymph nodes of axilla (apical, medial and lateral groups, central anterior and posterior groups) should be examined by palpation.

(D) Nutrition of the muscles of the upper limb is assessed. It may be altered in disorders of nervous system and myopathies.

THORAX :

The type of chest, obvious deformity if any, rate and depth of respiration are noted. Prominent veins in the chest wall and different pulsation accessory nipple and sinuses, if any, are to be looked for. Routine inspection of the posterior aspect of the chest must be done for the detection of any spinal deformity like kyphosis, scoliosis or gibbus.

ABDOMEN :

Movement of the abdominal wall, distension either localised or generalised any scar mark or operation mark, dilated vein, shape and position of the umbilicus, any visible peristalsis and the hernial orifices are to be noted in the regional survey of the abdomen.

Visible peristalsis may be seen in pyloric stenosis. small or large gut obstructions. Prominent veins may be due to portal vein or inferior vena cava obstruction. Generalised distension of the abdomen may be due to intestinal obstruction or paralytic ileus or acute dilatation of stomach. Umbilicus may be everted in huge ascites.

LOWER LIMBS :

Legs are examined for clubbing, oedema, varicose veins, varicose ulcer, vascular disturbances like diminished pulsation of dorsalis pedis artery, phlebitis or a gangrene of the toe. Bony deformities such as bowing of the legs may

be noticed in Paget's disease of bone and in rickets; *locked knee* and *sabre tibia* may be found in syphilis. There may be evidence of post polio contracture.

Pes cavus : In which there is a fixed deformity of the foot with a high arc is associated with Friedreich's ataxia, muscular dystrophy, peroneal muscular atrophy, poliomyelitis, spastic hemiplegia etc. or may be idiopathic.

Measurement from the highest point of iliac crest to the medial melleolus should be done–as a routine and compared to that of the opposite side.

CHAPTER 2

CARDIOVASCULAR SYSTEM

IMPORTANT ANATOMICAL LANDMARKS

Borders :

The base of the heart is represented by a line joining the right 3rd sternocostal articulation and a point at the level of the left 2nd intercostal space just internal to the parasternal line.

The *right border* extends from the right 3rd sternocostal articulation above up to the right 7th sternocostal articulation below. This is a slightly curved line with convexity to the right side. The *left border* is traced by a line joining the point at the level of the left 2nd intercostal space above just internal to the parasternal line and the apex beat below. This border is concave to the left in the upper 1/3 while the lower 2/3 is slightly convex.

The apex beat is normally situated in the left 5th intercostal space $^1/_2$" inside the midclavicular line. In children it is in the left 4th space on the midclavicular line.

Position of valves

(1) Mitral valve is obliquely placed behind the inner end of the left 4th costal cartilage and the adjoining part of the sternum.
(2) Tricuspid valve is situated obliquely behind the right 5th costal cartilage.
(3) Pulmonary valve is placed horizontally at the upper border of the left 3rd costal cartilage.
(4) Aortic valve corresponds to a line drawn obliquely

across the left half of the sternum at the level of the lower border of the left 3rd costal cartilage.

Auscultatory areas

The heart sounds and murmurs are best heard in these areas.

(1) Mitral area– It corresponds with the apical area situated in the left 5th space 1" inside the midclavicular line. It varies with the size of the heart i.e., it is not fixed.

(2) Tricuspid area–It is situated in the left 4th intercostal space near the sternal edge and also in the lower part of the sternum.

(3) Pulmonary area–The pulmonary murmurs are best heard in the 2nd left intercostal space close to the parasternal line.

(4) Aortic area–The aortic murmurs are best audible over the right 2nd costal cartilage.

Sometimes an aortic murmur is heard only in the left 3rd space close to the sternum (*Erb's point*).

EXAMINATION OF THE CARDIOVASCULAR SYSTEM

GENERAL EXAMINATION

(a) *Feel the temperature of the extremities.* Warm hands are observed in hyperkinetic circulatory states such as pregnancy, thyrotoxicosis, chronic anoxic cor pulmonale, beriberi, Paget's disease of bones aortic regurgitation etc.–

Hands may be cold due to exposure to cold, anxiety neurosis, low cardiac output (as occurs in mitral stenosis, aortic stenosis, left ventricular failure, peripheral circulatory failure and severe pulmonary hypertension).

(b) *Look for dilated veins over the dorsum of hand.* These are found in chronic cor pulmonale, beriberi, Paget's disease, pericardial effusion, old age etc.

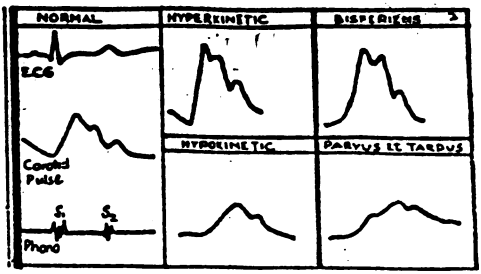


Fig. 3A : Normal Carotid and 4 abnormal Carotid Pulses.

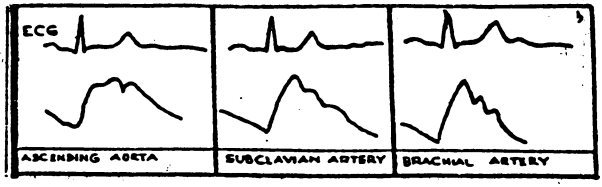


Fig. 3B : Changes in the Contour of the arterial pressuae pulse during a Pullback of a micro manometer catheter from the central. aorta to the brachial artery.

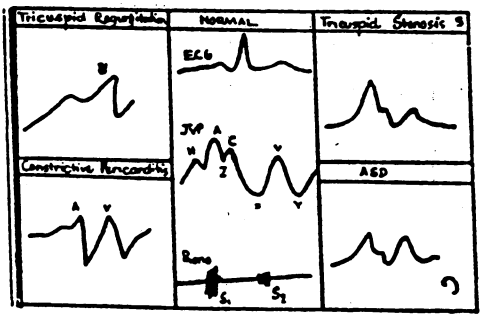


Fig. 3C : The normal and 4 abnormal types of Jugular venous pulse (JVP).

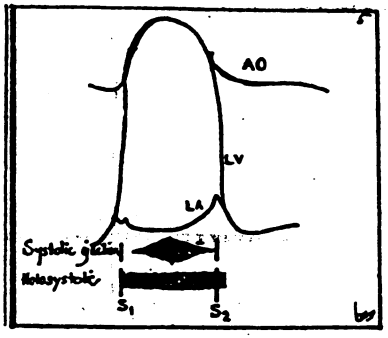


Fig. 3D: Characteristics of a systolic ejection and a pansystolic regurgitant murmur. AO- Aortic pressure tracing. LA and LV: Left atrial and left ventricular pressure tracing, s_1 and s_2 : First and second Heart Sounds.

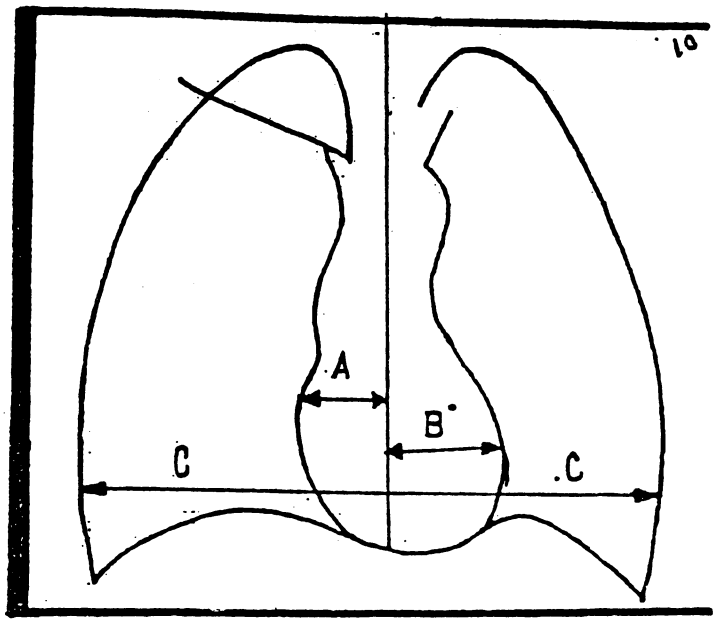


Fig. 3E: Diagrammatic representation of the measurements of the cardiothoracic ratio. A+ B= Maximum transverse diameter of the heart. C=Maximum transverse diameter of the chest. **C T** Ratio = A+B/C (Normal Value: about 0.5)

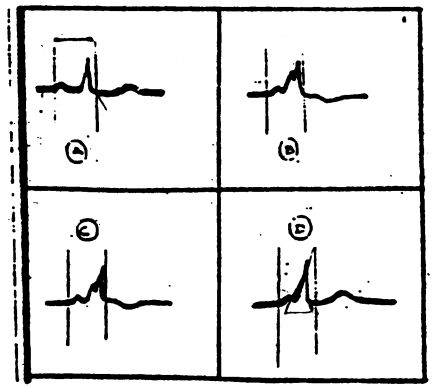


Fig. 3F: Variations in the ECG pattern of preexcitation. A- Normal sinus Beat. B- usual preexcitation beat. Normal PJ but short PR interval. C- short PJ interval. D- short PR and PJ interval with normal QRST complex. Note the Delta waves in B and C.

Fig. 3G : Left lateral view with Barium Oesophagus showing sickle shaped pattern of the oesophagus due to left atrial hypertrophy.

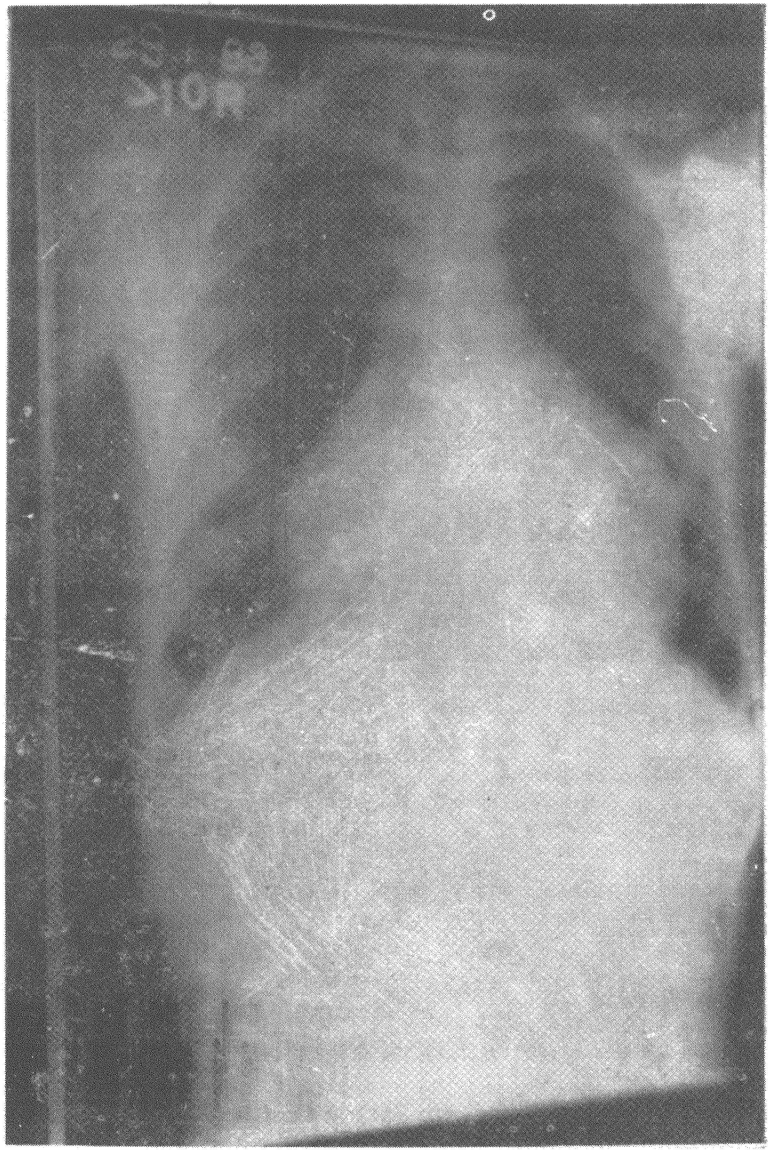


Fig. 3H : Globular appearance of the heart due to pericardial effusion.

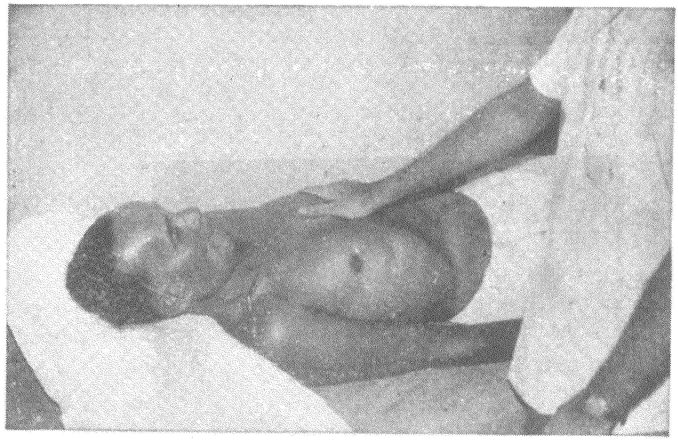


Fig. 3I : Demonstrating Palpation of the apical Impulse

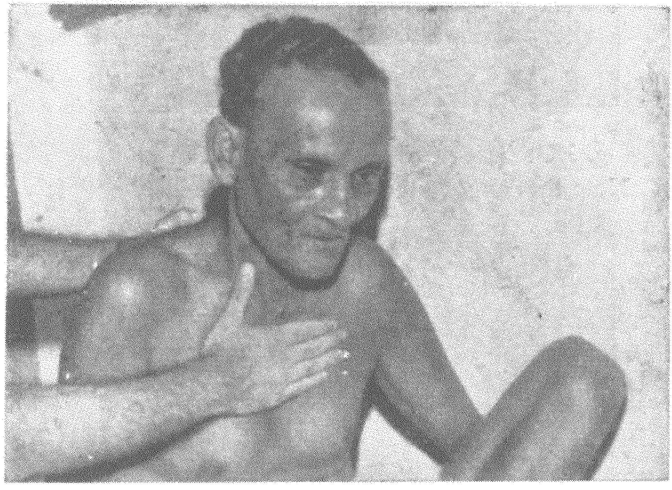


Fig. 3J : Demonstrating Palpation of the base of heart. Note that the patient is in a Sitting posture.

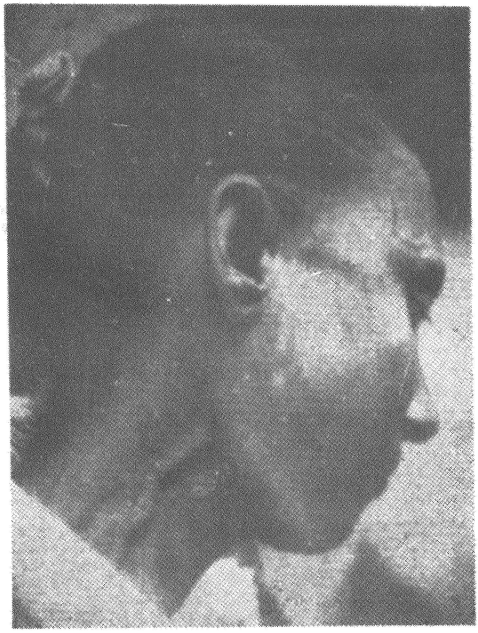


Fig. 3K : Case of congestive heart failure. Note the engorgement of the neck veins.

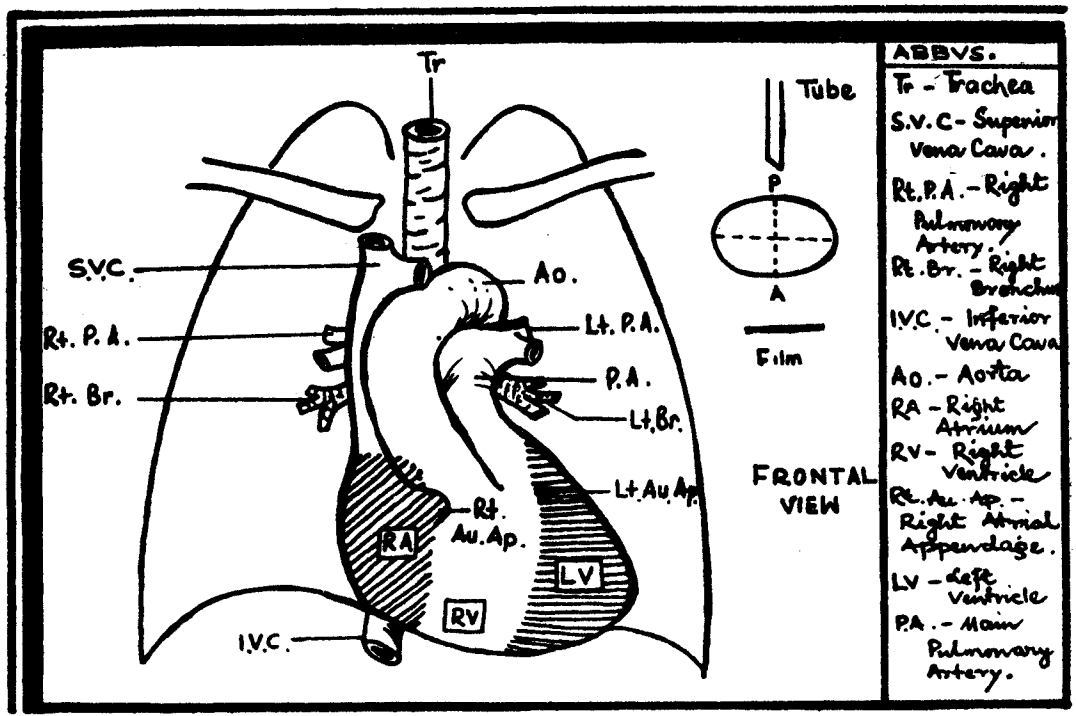


Fig. 3L : X-ray chest PA View-diagrammatic representation of the different Cardiovascular structures

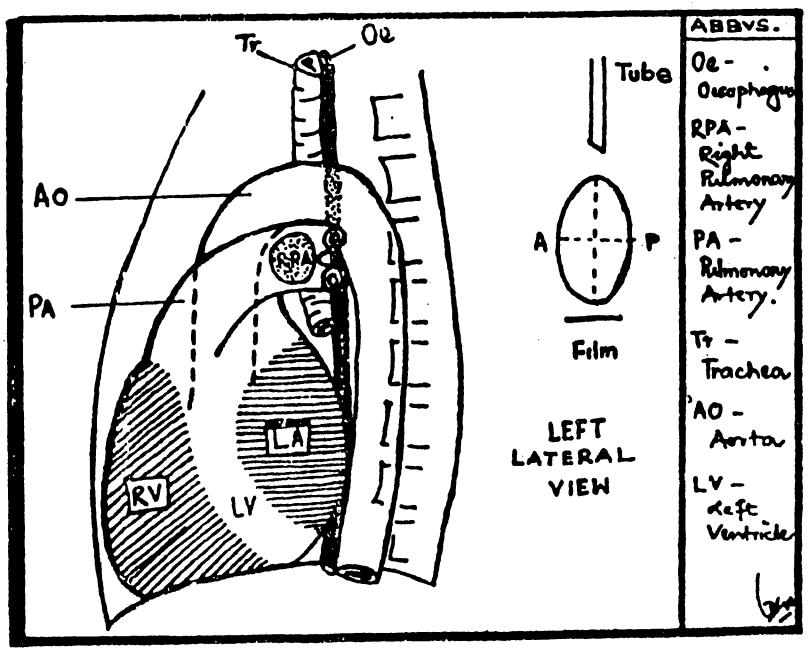


Fig.3M : X-Ray chest left lateral view diagrammatic representation of the different cardiovascular structures.

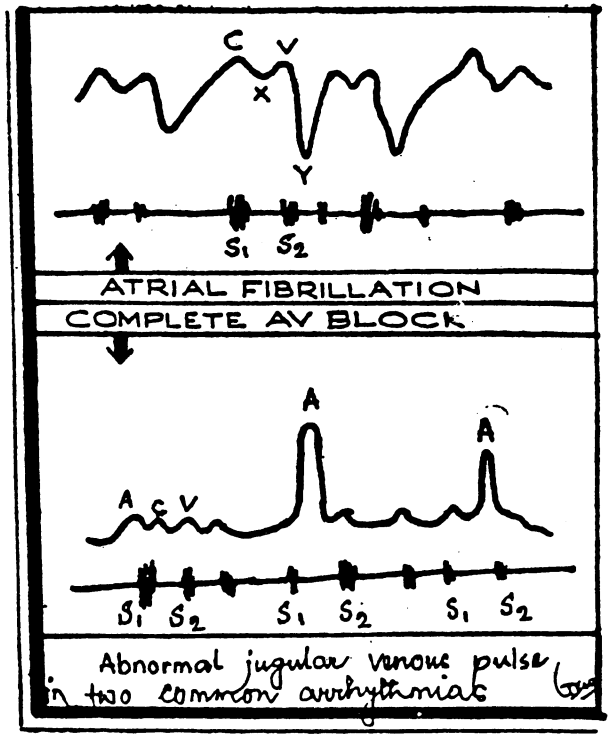


Fig. 3N : Two abnormal JVP's.

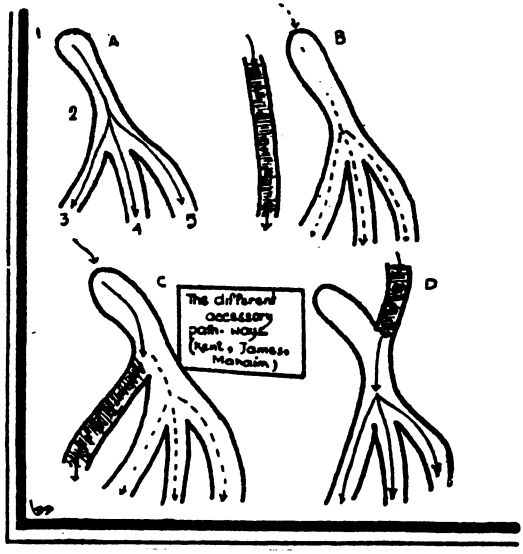


Fig. 3O : B-Kent; C- james; D- Mahaim.

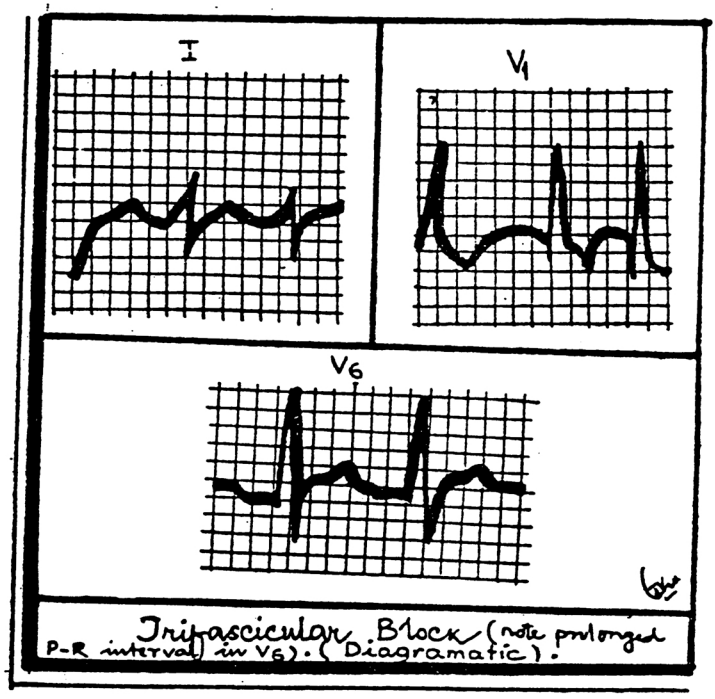


Fig. 3P : ECG of Trifascicular Block.

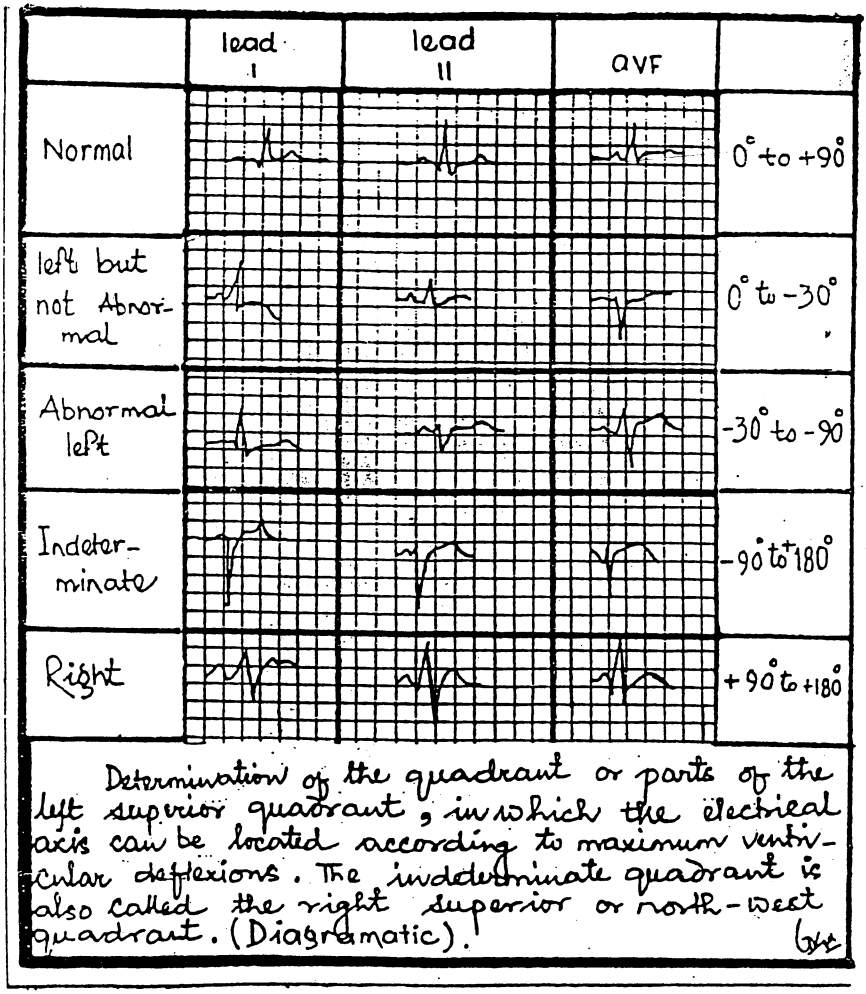


Fig. 3Q : ECG showing different types of axis deviations.

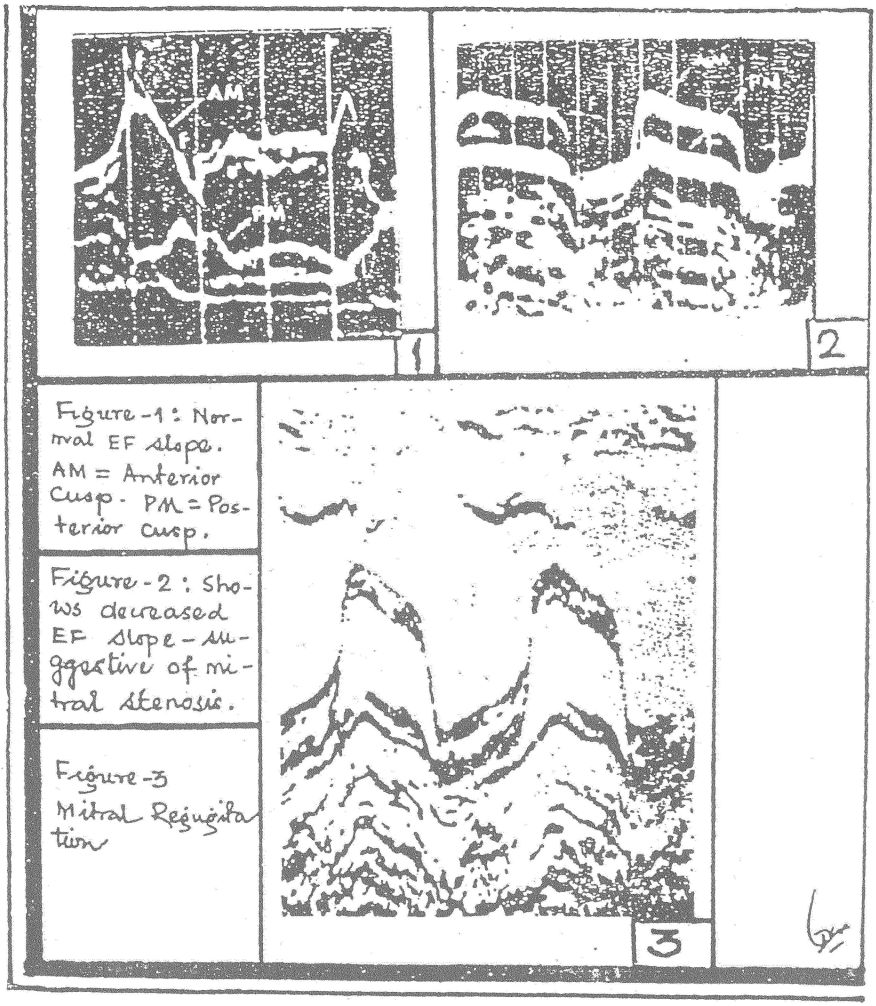


Fig. 3R : Echocardiogram of the mitral valve.

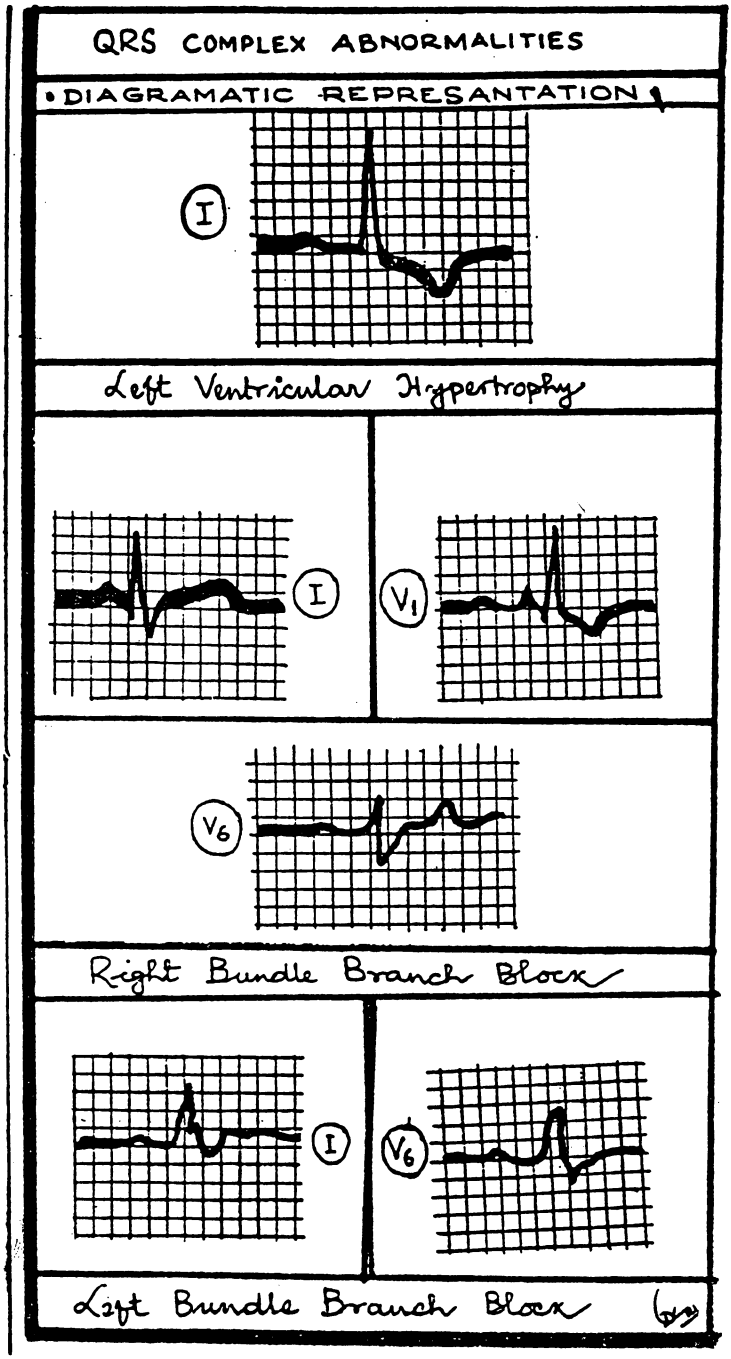


Fig. 3S : ECG showing Different abnormal QRS complexes.

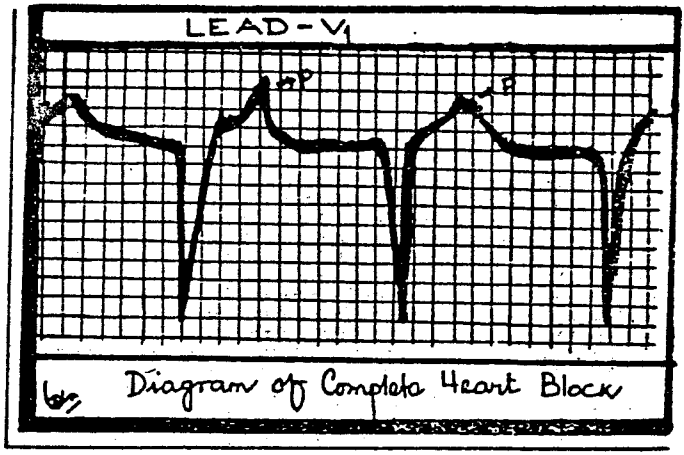


Fig. 3T : Another abnormal but common ECG finding.

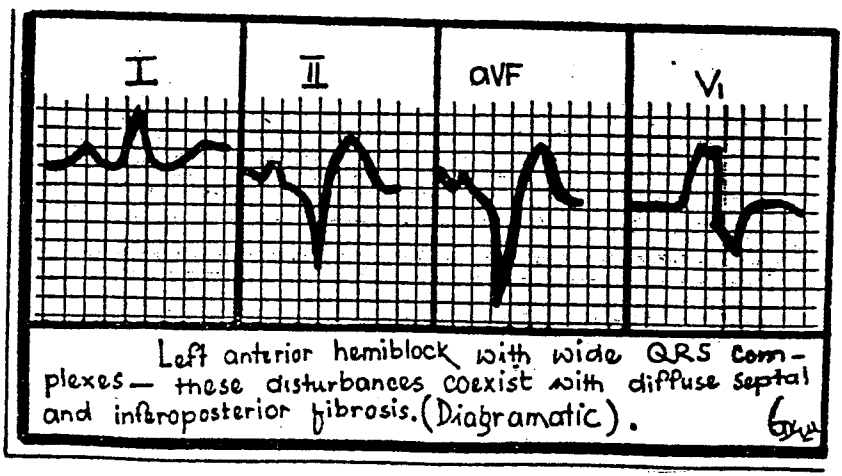


Fig 3U : Another abnormal ECG.

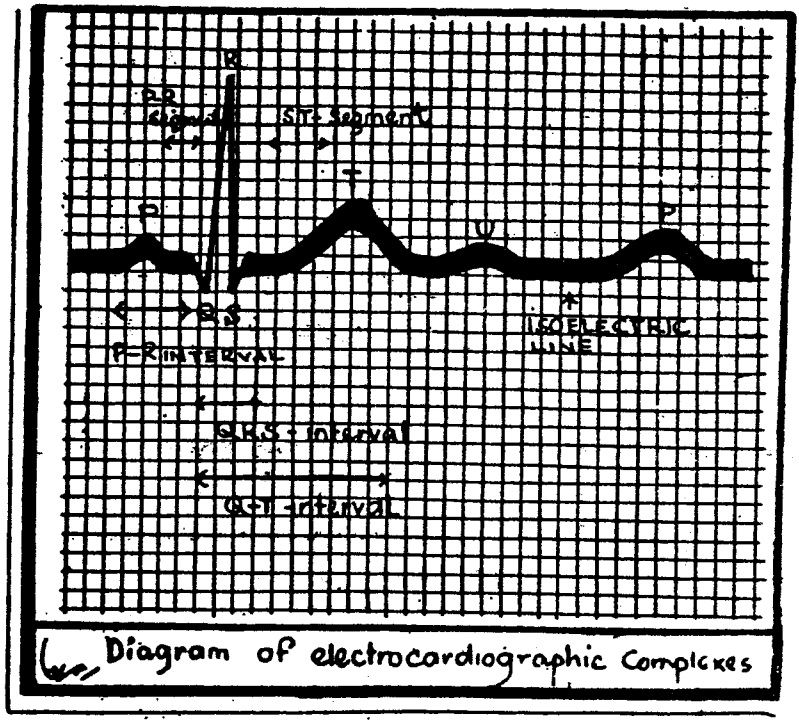


Fig 3V : The Normal ECG Tracing (Diagrammatic).

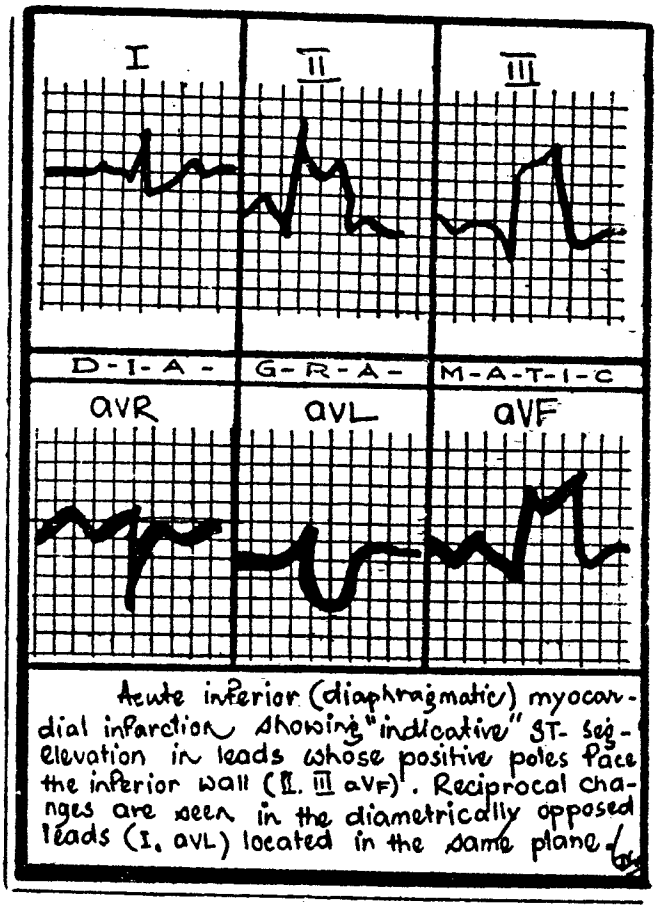


Fig 3W : ECG showing Inferior Interior Wall AMI.

(c) *Clubbing of the fingers and toes should be searched for*–they are seen in infective endocarditis, congenital cyanotic heart diseases, cardiac tumours e.g. left atrial myxoma and in post operative infections particularly in open heart surgery. Clubbing may be present only in the left hand in presubclavian coarctation and also in aneurysm of the arch of the aorta.

(d) *Note whether the patient is cyanosed or not.* Cyanosis due to cardiac causes in usually associated with clubbing of the fingers (i.e. central cyanosis). Peripheral cyanosis is found in acute left ventricular failure (e.g. due to acute myocardial infraction).

Differential cyanosis is a very important clinical finding–*Blue hands with red feet* are diagnostic of coarctation of the aorta with transposition of great vessels. *Blue feet with red hands* are diagnostic of patent ductus arteriosus with reversal of shunt due to pulmonary hype tension.

(e) *Examine the nail beds* for linear splinter haemorrhages and also look for tender Osler's node in the pulp of the fingers. These are found in infective endocarditis.

(f) *Examine the palms*, they may look pale and see whether they are moist or dry. Pallor is found in infective endocarditis and acute rheumatic carditis. Palmar erythema is one of the clinical features of hyperkinetic circulartory states. Warm an moist hands is found in thyrotoxicosis. Cold and moist hand is four l in anxiety neurosis. Dry hand with thick and rough skin is found at times in hypothyroidism. Janeway lesions may be found in infective endocarditis.

(g) *Note digital pulsations* by gently holding the pulps of the patient's fingers with those of your own. Prominent digital pulsations (Quincke's pulse) is commonly found in

aortic regurgitation, thyrotoxicosis, patent ductus arteriosus, beriberi etc.

(h) *Look for the webbing of fingers, hypermobility of different joints* and arachnodactyly or long slender gracile spider-leg like fingers, as are found in Marfan's syndrome. Aortic regurgitation, dilatation of the aorta and various other congenital cardiac anomalies may be found in Marfan's syndrome.

Look for evidences of arthritis which might be rheumatic or rheumatoid or may be due to gout. In gout, gouty tophi may be found on hands, periarticular tissues and cartilage of the ear. Gout may be associated with pericarditis. Rheumatoid nodules may be the clue to apparently idiopathic pericarditis. In *active* rheumatic myocarditis rheumatic nodules may be found over the occiput, elbow or on the tendon sheath in front of the wrist.

Scleroderma affects the skin giving rise to a diffuse indurated feel of the skin of the fingers, hands and eyelids. It may be associated with pericarditis, cardiomyopathy, heart block, aortic valve diseases and systemic and pulmonary hypertension.

Xanthomata should be searched for over the eyelids, as nodules over the tendon sheaths or as orange yellow streaks on the palms of the hand. It is an important sign of hyperlipidaemia which is often associated with ischaemic heart diseases.

(i) *Search for syphilitic stigmata* in the pupil (Argyll Robertson pupil), condyloma late, features of tabes dorsalis etc.

(j) *Examine the arterial pulse*–its rate, rhythm, volume, tension, condition of the arterial wall, character, equality of the two sides and presence or absence of radio-femoral delay.

Method of palpation–Pulse is usually felt over the radial artery at the anterior aspect of the wrist with the tips of 3 fingers while the patient's forearm is slightly pronated and wrist slightly flexed. The method is known as trisection. To note the character of the pulse either the brachial or the carotid artery pulsation is preferable.

PULSE

(A) RATE– It means the number of beats per minute. Normal rate varies from 60 to 100 per minute (average being 72 per min.)

Pulse rate may be less than 60 per minute–when it is known as *bradycardia*. It occurs physiologically in trained athletes and pathologically in myxoedema, obstructive jaundice, raised intracranial tension, starvation, different degrees of heart block, digitalis toxicity, sick sinus syndrome etc.

Bradycardia in athletes disappears after exercise while it persists if it is due to complete heart block.

Pulse rate in complete heart block is usually 40 per minute or less and may fall to as low as 20 per minute.

Pulse rate when goes above 100 per minute is known as *tachycardia*. It is normal in a child up to the age of five.

If pulse rate is 100-140 per minute, it is known as *sinus tachycardia* and is found in shock, haemorrhage, high fever, thyrotoxicosis, severe anaemia, toxaemia, after administration of belladonna group of drugs etc.

Paroxysmal tachycardia means pulse rate more than 160 per minute. It may be *supraventricular* (PSVT) or *ventricular* (VT). Supraventricular paroxysmal tachycardia may dain be *paroxysmal atrial tachycardia* or *paroxysmal nodal tachycardia*. Ventricular tachycardia is almost always due to organic lesions of the heart.

PSVT is regular in time and constant in rate as long as

the attack lasts. This is due to spontaneous development of an ectopic pacemaker in the atrium resulting in abnormally rapid atrial contractions which are visible in the form of 'a' waves in the neck vein. In this type, the pulse rate and the number of 'a' waves of the neck vein per minute are equal.

In VT the ectopic focus is in the ventricle. The atria contract at a normal rate and there is a varying relationship between atrial and ventricular contractions. Irregular cannon waves are found in the neck veins. The differential diagnosis of atrial, nodal and ventricular tachycardia has been listed below :

Atrial tachycardia	*Nodal tachycardia*	*Ventricular tachycardia*
(1) *Pressure over the carotid sinus*– Very little change.	The heart rate is reduced to normal	No change,
(2) *Pulse rate*– Constantly above 160/minute,	Constantly above 160/minute.	Variable,
(3) *'a' waves of neck* veins– Number of 'a' waves per minute is equal to pulse rate.	Regular cannon waves.	Irregular cannon waves.
(4) *Auscultation*– No change except accentuated 1st sound due to tachycardia.	Changing intensity of the 1st heart sound.	Changing intensity of the 1st heart sound.
(5) After injection of mecholine– Little change.	Tachycardia disappears.	No change
(6) *E.C.G.*– It is helpful for distinction of the 3 types with certainty, 'p' waves are upright in leads II, III and a VF.	'p' waves are inverted in same leads (i.e., II III and a VF).	Bizarre pattern of QRS complex

Important causes of paroxysmal tachycardia are rheumatic carditis, ischaemic heart disease, hypertensive heart disease, thyrotoxicosis, cardiomyopathy, after thoracic or any major operation etc.

(B) RHYTHM–It is the spacing of successive pulse waves in time.

Types–

(a) Irregularly irregular–It means dissimilarity between any two pulse beats in all aspect. This is commonly encountered in atrial fibrillation, multiple extrasystoles, atrial flutter with varying degrees of heart block.

(b) Regularly irregular–In this type, irregularity of the pulse waves come at a regular interval or the irregularity may continue in a regular manner. This is found in second degree heart block, extrasystoles etc.

Pulse deficit–It is the difference between the ventricular contraction rate and the arterial pulse rate.

To detect pulse deficit, separately count the heart and pulse rates for 1 minute. Accuracy can be ensured if simultaneous counting of heart and pulse rate is done by two examiners.

Pulse deficit is usually more than 10 in untreated atrial fibrillation. Pulse deficit is less than 10 in slow atrial fibrillation, atrial fibrillation with digitalised heart and multiple extrasystoles.

Atrial fibrillation and flutter

(a) Atrial fibrillation is a common cardiac disorder. Though its exact mechanism is not known. It is thought to be due to wavelets of excitation pursuing an irregular and erratic course through the atria. Normal atrial contractions are replaced by rapid fibrillary waves (represented by f-waves in the ECG). The ventricles respond slowly and randomly due to inability of the AV node to conduct such

a high rate of atrial discharges. The atrial rate is around 350 to 600 per minute, the ventricular rate is about 100 to 150 per minute. The pulse is irregularly irregular due to varying degrees of A-V block and the first heart sound is of varying intensity. It may be paroxysmal or permanent. Though an irregular pulse in atrial fibrillation is the traditional teaching, It has been recenlty found that about 1/3 to 1/2 of the patients have regular beats for prolonged periods (*Br. Heart J. 56, 4, 1986*).

(b) Atrial flutter is much less common than atrial fibrillation. Here the atria contract regularly but rapidly (usually 250 to 350 per minute) and the ventricles usually respond to every alternate beat because of a regular 2 : 1 A-V block. Occasionally this A-V block may be 3 : 1 or 4 : 1 and then the ventricular rate is slower, but bears a regular relation with the atrial rate. Rarely the heart block may be variable producing an irregular ventricular rate and rhythm.

Causes of atrial fibrillation and flutter are–

(i) Rheumatic heart disease (mostly affecting the mitral valve).
(ii) Ischaemic heart disease.
(iii) Hypertensive heart disease.
(iv) Cardiomyopathy.
(v) Pericarditis.
(vi) Atrial septal defect, Ebstein anomaly etc.
(vii) Thyrotoxicosis.
(viii) Excessive tea, coffee, tobacco, alcohol consumption.
(ix) After cardiac surgery.
(x) Lone atrial fibrillation (without any apparent cause).
(xi) Rarely after acute infection like diphtheria.
(xii) Rarely associated with W.P.W. syndrome.

It must be remembered that atrial fibrillation can develop

on top of atrial flutter if the latter is being treated with digitalis.

Signs of atrial fibrillation–

(i) Ventricular rate is usually about 100–150 per minute.
(ii) Irregularly irregular pulse with a pulse deficit of more than 10.
(iii) Varying intensity of the heart sounds.
(iv) Absence of 'a' waves is the jugular venous pulse in the neck.
(v) A systolic murmur may appear in the mitral area.
(vi) Accentuated second sound at the pulmonary area, (P_2) due to left atrial failure.
(vii) ECG–'P' waves are replaced by fibrillary 'f' waves.
(viii) Echocardiographic gives a view on the irregular contraction of the atria.
(ix) Fluoroscopy shows irregular contractions of the atria.
(x) Features of embolism may be present.

Signs of atrial flutter—

(i) Regular ventricular rate of about 100 per minute.
(ii) Abrupt drop of ventricular rate to half or one-third or doubling or tripling of previous rate due to abrupt change of A-V block pattern between 2 : 1 and 3 : 1 types. There is usually no pulse deficit and the pulse is regular.
(iii) There may be a slight variation of the intensity of the first heart sound due to slight variation of the P-R interval.
(iv) Regular, rapid, small "a" waves may be seen in the jugular veins.
(v) Pressure on the carotid sinus may suddenly reduce the pulse rate. It remains reduced as long as

the pressure is maintained. On release of the pressure, the pulse rate returns to the previous level.

(vi) ECG–'Saw-tooth' appearance of the atrial impulse (F waves) with the ventricular response of a regular rate of 2 : 1 or 3 : 1 (may be up to 8 :1).

(vii) Echocardiographic shows atrial contraction as regular and a ventricular response (2:1:3:1) etc.

(viii) Rerely used :

Fluoroscopy shows regular atrial contractions.

Sinus arrhythmia–This is characterised by slowing of the pulse rate in expiration. This is a physiological phenomenon found in children. *Mechanism*–During the expiratory phase of respiration excess blood comes to the left atrium due to squeezing action of the lungs. The stroke volume of the left ventricle, therefore, increases and in turn stimulates the baroreceptors leading to reflex slowing of the heart rate. When a large atrial septal defect is present, the stroke volume does not go up as the right atrium shares the blood from the left atrium. So sinus arrhythmia may be absent or minimum in a child with atrial septal defect.

N.B. – (i) Sinus arrhythmia may also be absent in heart failure, sick sinus syndrome, autonomic neuropathies etc.

(ii) The brainbridge reflex causes acceleration of the heart rate when the venous return is so excessive as to over distend the right atrium.

(C) VOLUME–This is estimated by assessing the amplitude of a pulse wave. *High volume pulse* is characteristic of hyperkinetic circulatory states e.g. hyperthyroidism, after exercise, high fever, severe anaemia, atrerio-venous fistula, severe anoxia, emphysema of the lungs etc. *Low volume pulse* is found in states of low cardiac output e.g. shock,

aortic stenosis, tight mitral stenosis, severe pulmonary stenosis, pericardial effusion, constrictive pericarditis, paroxysmal tachycardias, congestive cardiac failure and obstructive cardiomyopathy etc.

(D) Tension–This is the lateral pressure exerted by the column of blood on the walls of the arteries.

Tension is a rough estimation of blood pressure within the vessel. Its estimation by palpation is unreliable and so the blood pressure should be determined with the help of sphygmomanometer.

At first the artery is pressed by the distal finger to prevent regurgitation; gradually pressure is applied by the proximal finger while the middle finger is used to feel the pulse wave. The pressure required by the proximal finger to obliterate the pulse wave is the systolic tension. The optimum pressure exerted by the proximal finger to have the maximum thurst felt by the middle finger is the diastolic tension. Hypotension may be postural or may be due to organic diseases of the heart e.g. acute myocardial infarction, left ventricular failure, severe pulmonary hypertension, severe aortic stenosis, severe mitral stenosis and acute peripheral circulatory failure.

(E) CONDITION OF THE ARTERIAL WALL–Exsanguinate the artery and roll the empty artery against the surface of the radius. The artery is felt like a cord with tortuosity in Monckeberg's medial sclerosis (may be associated with *locomotor brachialis* and tortuous temporal arteries etc.

(F) QUALITY OF PULSE–Inequal pulse on two sides may be due to *anatomical variations* or due to *organic conditions* e.g. thoracic inlet syndrome, presubclavian coarctation, pressure over axillary artery by lymph nodes. Volkmann's ischaemic contracture and aortic arch syn-

drome. The pulse of the upper extremity should also be compared with that of the lower extremity. Normally radial and femoral arteries pulsate at the same time but the radial artery pulsation is 0.02 to 0.03 seconds earlier than the dorsalis pedis artery pulsation. The pulsations of the lower extremities may come after the pulsations of the upper extremities in the coarctation. Femoral pulse may be absent in saddle embolism and aortic arch syndrome. Delay in the formal artery pulsation compared with the right radial artery pulsation is found in coarctation of the aorta.

(G) CHARACTER–The different types include the following :

(1) *Water hammer pulse of high volume collapsing pulse*– In this type an abrupt rise and a sharp fall of the pulse wave is felt. This is produced by sudden regurgitation or flow of blood away from its normal course e.g. regurgitation into the left ventricle in aortic incompetence and into the pulmonary artery from the aorta in PDA. Other causes are high fever, severe anaemia, thyrotoxicosis, chronic anoxic cor pulmonale, complete heart block, Paget's disease and arteriovenous fistula. The increased cardiac output stimulates the baroreceptors in the aortic arch which in turn causes reflex dilatation of the peripheral vessels into which the arterial blood rapidly flows out. The sudden collapse is most important because it indicates sudden reflex vasodilatation of peripheral vessels and also the amount of regurgitation of blood into the left ventricle. It is important of remember that the collapse is a systolic event.

There is another type of collapsing pulse–the low volume collapsing pulse found in mitral regurgitation and ventricular septal defect. The duration of the pulse wave is shorter as the left ventricle empties quickly into the left atrium and the right ventricle respectively in addition to the aorta.

(2) *Low volume pulse (pulsus parvus)*–This is characteristically found in tight mitral stenosis, severe aortic stenosis, severe pulmonay hypertension, acute left ventricular failure due to any cause, obstructive cardiomyopathy, pericardial effusion etc. In aortic stenosis the upstroke may also be delayed–pulsus parvus et tardus.

(3) *Anacrotic pulse*–The upstroke of this pulse is slow with an anacrotic notch. The anacrotic notch is in the ascending limb of the pulse wave. It is found in aortic stenosis.

(4) *Pulsus paradoxus*–This is a misnomer and should better be called pulsus normalis exaggeratus as it is nothing but exaggeration of a physiological phenomenon.

The pulse volume decreases with inspiration and increases with expiration significantly in this condition. In fact it reflects the exaggeration of the normal respiratory variation in systolic pressure. Though the systolic pressure is normally decreased by 5 to 10 mm Hg with inspiration, this is rarely perceptible by palpation of the radial pulse in a healthy person and if suspected, pulsus paradoxus must be confirmed by using a sphygmomanometer. The systolic pressure is always higher in expiration than in inspiration. If the difference between the readings of systolic pressure in expiration and inspiration is more than 10 mm Hg pulsus paradoxus is confirmed.

Causes of Pulsus paradoxus–

(i) Rapidly developing pericardial effusion and cardiac tamponade.
(ii) Chronic constrictive pericarditis (in about one-third to half of all patients).
(iii) Restrictive cardiomyopathy.
(iv) Respiratory obstruction causing wide variations of intrathoracic pressure, e.g. severe bronchial asthma, chronic obstructive lung disease.

(v) Severe cardiac failure
(vi) Haemorrhagic shock
(vii) Pulmonary embolism
(viii) Pregnancy
(ix) Extreme obesity
(x) Superior mediastinal syndrome.

The physiological basis–In inspiration there is a fall in intrathoracic pressure causing an increase in the right ventricular filling as the systemic veins are extrathoracic, while the pulmonary vessels dilate and entraps a significant amount of blood. The left atrial and ventricular filling is marginally decreased leading to marginal decrease in stroke output of the left ventricle. Since blood pressure is the product of cardiac output and peripheral resistance, the systolic pressure marginally decreases during inspiration as the former falls and the latter remains constant.

This normal phenomenon is accentuated in the above conditions causing pulsus paradoxus because–

(a) During inspiration the intrapericardial pressure rises further due to traction on the pericardium, which causes obstruction to venous return and thereby reduce the cardiac output, and also (b) there is a competition between two ventricles for a fixed total diastolic volume within the fixed space of the filling of the right ventricle which occupies most of the available intrapericardial space resulting in a further decrease in the left heart filling and the left ventricular stroke output.

Pulsus paradoxus is contributed to by the transmission of the negative intrathoracic pressure to the aorta.

The finding of pulsus paradoxus is critical diagnosing cardiac tamponade as most patients with slowly developing tamponade do not have the classical features.

Total paradoxus is the state of complete disappearance

of the palpated pulse during inspiration and occurs in severe tamponade combined with hypovolaemia.

Pulsus paradoxus may be absent in cardiac tamponade when there is–(i) unequal compression of the two ventricles (ii) associated ASD and (iii) an associated aortic incompetence.

(5) *Pulsus alternans*–This is characteristically found in severe left ventricular failure. It should be searched for by carefully palpating the radial, brachial, carotid and femoral arteries. The alternate pulse waves are weak because all the muscle fibres cannot contract in one beat as some are healthy and others degenerated. Thus there is an alternation in the left ventricular contractile force often associated with an alternation of left ventricular end diastolic volume and stroke volume. Therefore, a small volume beat is delivered alternately with a normal beat and the diagnosis is best made with the help of a sphygmomanometer.

While recording the blood pressure with the sphygmomanometer, at first the strong beats are heard and the rate remains half of the pulse rate. With gradual lowering of pressure suddenly all the beats, both strong and weak are heard alternately and the rate becomes equal to the pulse rate. The pressure difference between the first appearance of only the strong and both strong and weak beats varies between 10 and 40 mm of Hg

Occasionally patients may have 'latent; *pulsus alternans* which may be unmasked by erect posture of vasodilators e.g. nitroglycerine. Besides severe depression of myocardial function alternans may be seen in acute aortic regurgitation, aortic stenosis, infectious myocardities and rarely in the beats following PSVT or extrasystoles.

(6) *Pulsus bisferiens (Double Kicking pulse)*–This is best demonstrated in the brachial and carotid arteries. There

are two distinct palpable peaks. The first lift (percussion wave) is greater than the second lift (tidal wave) if aortic incompetence is the predominant lesion. If the second lift is greater than the first lift, the stenosis is predominant. The bisferiens pulse thus is diagnostic of *combined aortic stenosis* and *incompetence.* but may also be seen in *isolated florid aortic incompetence.* The pulsus bisferiens is produced as a result of ventury effect inside the left ventricle during ventricular systole. Pulsus bisferiens may also be produced by *idiopathic hypertrophic sub-aortic stenosis* (IHSS) : – the initial rapid systolic wave is called *percussion wave* and the second, slow rising. late positive pulse wave is the (reflected) *tidal wave.*

Pulsus bisferiens with a small volume pulse is known as dicrotic pulse where anacrotic peak is absent but dicrotic peak is exaggerated and hence the name dicrotic pulse. It is typically found in typhoid fever, but usually occurs in severe cardiac failure, cardiac tamponade, hypovolaemic shock and rarely may be seen in healthy adolescents or young adults. A systolic pressure of more than 130 mm Hg militates against a dicrotic pulse.

(7) *Pulsus bigeminus*–It is a particular type of pulse where two beats and a long pause recur in a regular manner. If three beats and a pause recur regularly, it is known as pulsus trigeminus. Pulsus bigeminus is commonly found in 2 : 1 heart block and in digitalis toxicity. It is produced by a small ventricular premature beat occurring regularly after a larger normally conducted beat, and the larger pulse occurs after the long diastolic filling phase that follows the premature beat. But in those with IHSS the postpremature ventricular contraction is weaker than normal because of increased obstruction to left ventricular outflow.

Estimate the blood pressure with the help of a sphygmomanometer. The technique of measurement and its importance in clinical diagnosis have been discussed in chapter I. The blood pressure of lower limbs may be lower than that of the upper limbs in coarctation of aorta. Buerger's disease, nonspecific arteritis of lower limbs, peripheral arterial embolism etc. and may not be recordable at all in lower limbs due to saddle embolism at the bifurcation of the common iliac artery.

Carotid artery pulsation is an important clinical sign to be looked for. The carotid pulsation is exaggerated in any condition giving rise to hyperkinetic circulatory state. The exaggerated pulsation of carotid arteries seen in aortic regurgitation is known as *Corrigan's sign*. Unilateral carotid pulsation due to kinked carotid is found in atherosclerosis.

Note the suprasternal notch for any pulsation. Suprasternal pulsation is present gross aortic regurgitation, left presubclavian coarctation, unfolding of aorta, aneurysm of the arch of aorta etc.

Examination of the Neck Veins :– Internal jugular veins reflect most reliably the haemodynamics of the right atrium. The neck veins should be examined with the patient reclining at a suitable angle (say, 45°) and the neck muscles are relaxed. The top of the venous column does not normally exceed 2 cm vertically above the *sternal angle* with the patient reclining at 45º. *The sternal angle is taken as the reference point because the centre of the right atrium is at a depth of 5 cm from it in any position of the body.* This means in a healthy person reclining a 45° the mean level will be invisible. When the subject is at 90° angle, the venous level should not be visible beyond the clavicle normally. If the venous engorgement is found in the middle of the neck or more at 45° angle or if the venous

engorgement is seen at the root of the neck at 90° angle, it indicates increased jugular venous pressure. The venous engorgement will be exaggerated normally but not beyond the lower one-third of the neck when firm and gentle pressure is applied over the abdomen. This is known as *hepato jugular* or abdomino jugular reflux.

Examine the internal jugular vein and not the external jugular one. External jugular vein is a superficial one and may be engorged if the patient wears a tight coller or bends his neck to one side. External jugular vein never drains directly into the superior vena cava–therefore, any alteration of pressure in the right atrium will not be immediately reflected in the external jugular vein. Besides, it frequently shows some anatomical abnormalities e.g. It may drain into the axillary vein.

Note the vertical hight (from the sternal angle) of the pulsation, its character, the waves, relationship with respiration and heart sounds relationship with pulse rate etc.

Venous pulsation are (i) soft and undulating, lie (ii) lateral to the arterial pulsation and are (iii) neither jerky nor abrupt. They are (iv) better seen than felt whereas arterial pulsations are better felt than seen. Venous pulsations (v) alter with inspiration and expiration and (vi) also with change of posture. The venous pulse (vii) has a definite upper level though it may be necessary to make the patient sit up or lie down to find it out. This level falls during inspiration when blood is drawn into the heart. Venous pulsations are usually more sinuous and less sharp than arterial pulsations. Venous engorgement occurs in the following conditions :

(i) Cardiac causes–Congestive cardiac failure, pulmonary embolism, pericardial effusion, chronic constrictive pericarditis etc. The jugular veins are pulsatile in the above

mentioned conditions. However, jugular veins may be engorged but nonpulsatile (vide infra).

(ii) Extracardiac causes–Superior mediastinal obstruction (due to bronchogenic carcinoma, mediastinal lymphadenopathy, aneurysm of the arch of the aorta, retrosternal goitre etc). The neck and face are puffy and the upper limbs are oedematous in superior mediastinal syndrome and hence the name reversed congestive cardiac failure;–the engorged veins are non-pulsatile.

(iii) The neck veins may be engorged by increased intravascular volume as occurs in massive infusions and transfusions.

In healthy persons and in congestive cardiac failure the jugular venous pressure falls on deep inspiration due to the sucking in of blood into the right heart because of increased negativity of the intrathoracic pressure. On deep expiration the reverse happens. But in constrictive pericarditis, and pericardial effusion there is a *paradoxical rise* in the jugular venous pressure on deep inspiration, as the increased venous return to the right side of the heart cannot be accommodated in the rigid, unyielding confined space. This is known as *Kussmaul's sign.*

Pulsation of the jugular vein : The venous pulsation is composed of 3 waves and 2 troughs.

(a) The 'a' wave is produced by active atrial contraction in the last part of the atrial systole. It disappears if there is no active atrial contraction as in atrial fibrillation.

Gaint 'a' waves are visible when the right atrium contracts actively against the outflow tract obstructions e.g. tricuspid stenosis and tricuspid atresia and also when it contracts forcefully against the *high right ventricular enddiastolic pressure* in

pulmonary hypertension, pulmonary atresia and pulmonary stenosis. The auscultatory counterpart of giant 'a' wave is the *presystolic* gallop (4th sound). Giant 'a' wave is also known as "venous corrigan".

(b) 'x' descent–It is due to atrial relaxation and downward displacement of the tricuspid valve towards the apex of the right ventricle in systole. A small or absent 'x' descent is characteristic of tricuspid incompetence, 'x' descent may be inconspicuous if there is atrial fibrillation.

(c) The 'v' wave indicates the passive rise in pressure as venous return continues while the tricuspid valve is closed. The abnormal (C–V) wave found in tricuspid incompetence is really not an exaggeration of the normal v-wave but occurs earlier in systole, either directly after or replacing the x-descent. It should better be called an "s"-wave (systolic wave).

(d) 'c'-wave–A third or 'c'-wave occurs shortly after the 'a'-wave, coinciding with the 1st heart sound. Though described by physiologists it is rarely clinically visible. It is due to the closure of the tricuspid valve rather than to a transmitted carotid impulse.

(e) 'y' descent–It indicates the opening of the tricuspid valve and subsequent pouring out of blood from the right atrium into the right ventricle. This 'y' descent may be steep in tricuspid incompetence and chronic constrictive pericarditis and is always accompanied with a loud 3rd heart sound (or diastolic gallop) due to sudden rush of blood into the right ventricle from the right atrium.

A slow 'y' descent is characteristic of right atrial outflow tract obstruction e.g. tricuspid stenosis. Huge pathological 'a' waves known as *cannon waves* appear when the atria contract against closed A-V valves. Irregular connon waves associated with varying intensity of first heart sound are seen in complete heart block, paroxysmal ventricular tachycardia etc. while regular cannon 'a' waves may occur with 1 : 1 atrioventricular relationship during VT or SVT or in junctional rhythms.

II. EXAMINATION OF THE HEART

Inspections :

General inspection of the chest with special reference to the shape. superficial veins, obvious bulgings, width of the subcostal angle, movement with respiration etc. should be done before proceeding to a detailed examination of the heart. The precordium is the area of the anterior surface of the chest that overlies the heart.

(i) Bulging of the precordium is very common in congenital and rheumatic heart diseases (but not in hypertension and coronary arterial diseases.) This is due to overaction of the heart, as a result of altered haemodynamics or due to enlargement of any chamber, inside a soft bony cage (i.e., before ossification). The chest becomes barrel shaped in chronic cor pulmonale (e.g. in emphysema), –the sternum becomes prominent and the anteroposterior diameter is increased due to the presence of the voluminous lung inside the chest.

(ii) Look for dilated and engorged superficial veins which may be present in superior vena caval obstruction. The direction of blood flow will be from above downwards.

(iii) Look for any retraction of the lower part of precordium e.g. in adherent pericarditis.
Systolic retraction of the back of the chest on the left side in the regions of 11th and 12th ribs is sometimes visible in adherent pericarditis (*Broadbent's sign*).

(iv) A depressed sternum may be the cause of a right ventricular outflow tract obstruction without any organic lesion in the heart (e.g. pectus excavatum).

(v) An accessory nipple (petolythelia) is found associated with some cases of systemic and pulmonary hypertension, cardiomyopathy and congenital heart diseases.

(vi) Next look for any pulsations over the precordium. Normally the apical impulse is visible in thin built persons in the left 5th intercostal space just internal to the midclavicular line. The apical impulse may be seen on the right side in dextrocardia. It may be displaced outward and downward due to left ventricular hypertrophy as occurs in aortic regurgitation, aortic stenosis, mitral regurgitation, patent ductus arteriosus, obstructive cardiomyopathy, ischaemic and hypertensive heart diseases, atrial septal defect (septum primum type) etc. Parasternal pulsations are seen in the left 4th and 5th space near the left parasternal line.
Pulmonary arterial pulsation in the left space is normally found in children with thin chests. In adults such pulsations are *pathognomonic* of hyperdynamic circulation through the pulmonary circuit, as occurs in atrial septal defect, patent ductus arteriosus and ventricular septal defect. Exaggerated pulsations of the *pulmonary artery* is

characteristic of pulmonary hypertension. A diffuse pulsation on the *right sternal edge* is suggestive of an aneurysm of the ascending aorta, that in the *suprasternal notch* suggests hyperkinetic circulatory state, aneurysm of the arch of the aorta, coarctation of the aorta and unfolded aortic arch in hypertension. Pulsations on the *lower right sternal edge* is due to enlarged right atrium, tricuspid stenosis, tricuspid atresia and Ebstein's disease. Abnormal pulsations to the left of the sternum may be caused by right ventricular or left atrial hypertrophy as occurs in mitral stenosis.

In this connection, it is worthwhile to mention that pulsations over the *epigastrium* may be found in hyperkinetic right ventricle or right ventricular hypertrophy; aneurysm of the abdominal aorta, transmission of the aortic pulsation by a tumour e.g. gastric carcinoma or due to the *pulsatile* liver of tricuspid incompetence. It might also be found at times during excitement in normal individuals. Carotid pulsations may be exaggerated and prominently visible in aortic regurgitation, thyrotoxicosis, aneurysm of the aorta, kinked carotid (more so on the right side) and hypertension and also during excitement, emotion and exertion.

(vii) Inspect the back below the level of the scapular angle for any visible pulsation (Suzman's sign) which is expected in coarctation of the aorta. The spine should be observed carefully for scoliosis or kyphosis as they may be responsible for the development of chronic cor pulmonale.

(viii) In massive pericardial effusion, a bulging is some-

times seen in the epigastrium. This is known as *Auenbrugger's sign.*

(ix) Systolic retraction : With each systole there is a retraction of the intercostal space at the apex. This is found in normal persons. In chronic constrictive or adhesive pericarditis there is retraction of ribs at the apex along with the retraction of the intercostal space.

(x) Diastolic heart beat : In constrictive pericarditis a sudden thurst is seen and felt at the apex during diastole but no systolic impulse is there. This is known as diastolic heart beat. This is accompanied by a diastolic sound (pericardial knock) on auscultation and systolic retraction of the precordium. This is caused by sudden emptying of cervical veins is diastole. This is a pathognomonic sign of constrictive pericarditis.

Palpation

The patient should lie flat on the bed and the right palm is placed over the precordium Palpate the individual areas separately. The patient should be turned to the left to note the character of the *apex beat* more accurately, but never for its site. If the apex beat is not palpable in the dorsal position, *allow* the patient to sit up and lean forward and then locate the site. The patient should be turned to the left side to note three things–character of the apex beat, diastolic thrill and diastolic murmur.

(a) *Apex beat*–It is the downmost and outermost point over the precordium where a definite (and not necessarily the maximum) thrust can be felt.

During an examination of the apex beat the following points should be noted.

(a) Site–In a lean and thin person who has a tubular

chest, the apex beat may be in the left 6th space. Impalpable apex may be due to the apex beat lying under the rib, pulmonary emphysema, obesity, pericardial effusion and in elderly women with pendular breast. The apex beat may be displaced to the right in (congenital) dextrocardia or in acquired conditions of dextroversion. The extracardiac causes of shifting of apex beat to the right side are left sided pleural effusion, pneumothorax and hydropneumothorax and /or right sided pulmonary fibrosis and collapse.

The acquired dextroversion and congenital dextrocardia can be clinically differentiated by the fact that the trachea is deviated to the right in the former and remains central in the latter.

(b) Character–*Hyperdynamic* apex is a forceful and ill sustained one found in diastolic overloading of the left ventricle. This is observed in mitral regurgitation, aortic regurgitation, patent ductus arteriosus and ventricular septal defect. In these cases, the left ventricle receives the normal quota of blood together with the regurgitated blood of previous systole. The increased amount of blood dilates the left ventricle which forcibly pumps the blood into the systemic circulation. But the contraction is ill sustained because the left ventricle finds on obstruction to its outflow tract (because of patent valves or reflex vasodilatation of peripheral arterioles).

Heaving apex beat is a forceful and well sustained one, the finger being distinctly lifted for a moment. In this case the left ventricle is more hypertrophied than dilated (concentric hypertrophy) and is due to either (i) outflow tract obstruction as in aortic stenosis or obstructive cardiomyopathy or (ii) high peripheral vascular resistance as in systemic hypertension and coarctation of the aorta. All these result in overloading of the left ventricle.

The apical impulse may be tapping or slapping with a palpable loud first heart sound and is characteristically found in mitral stenosis and in any condition giving rise to hyperkinetic circulatory state with tachycardia.

Hypokinetic apex–The apical thrust is diminished and it is found in constrictive pericarditis, pericardial effusion, acute myocardial infraction, shock, myxoedema etc.

(2) Feel for thrills in the mitral area. It is detected best when the breath is held in expiration. Thrills are purring sensations or vibrations felt on the precordium or over arteries and are caused by torrential and turbulent blood flow through a diseased valve or an abnormal opening. In other words, it is nothing but the palpable vibrations of low frequency associated with murmurs. If the thrill coincides with the apex beat, it is systolic and if it precedes the apex beat, it is diastolic. The thrill should always be differentiated from a friction fremitus which will disappear on holding the breath and also from fine contractions of pectoralis muscle in cold weather which will cease in a warm environment.

Thrills over the mitral area may be–

(a) Systolic as in mitral regurgitation, atrial septal defect of septum primum type; some cases of obstructive cardiomyopathies and ventricular septal defect.

(b) Diastolic as in mitral stenosis, best palpable with the patient in left lateral position, holding the breath after deep expiration. Presystolic thrills of organic mitral stenosis are found even in an early stage. It is best palpable *after* the patient performs some exercise (when the heart contracts more

actively) and with the patient turned to the left. The diastolic thrill of Austin-Flint murmur is rarely palpable as also that of left artial myxoma.

(3) Next palpate the lower left sternal edge with the fingers of the hypothenar eminence and–

(a) Observe the character of the cardiac impulse. If it is heaving, it indicates right ventricular systolic overload. Right ventricular systolic overload may be produced by (i) right ventricular outflow tract obstruction as found in pulmonary stenosis and pulmonary atresia; or (ii) increased pulmonary vascular resistance in pulmonary hypertension of any aetiology. A hyperdynamic (forceful and illustained) right ventricular impulse indicates an eccentric hypertrophy of the right ventricle as it is dealing with an excessive volume of blood against a low peripheral resistance. This is typically found in atrial septal defect. In chronic cor pulmonale due to chronic bronchitis and emphysema, palpate the epigastrium for a right ventricular heave.

(b) Search for thrills over the lower left sternal edge (usually systolic) and the possible causes are ventricular septal defect, infundibular pulmonary stenosis, tricuspid regurgitation, atrial septal defect of primum type etc. A continuous systolodiastolic thrill over this region indicates (i) coronary arteriovenous fistula or (ii) rupture of sinus of Valsalva into the pulmonary artery or into the right ventricle.

(4) Palpate the pulmonary area for a detection of (i) the pulsations of the pulmonary artery, (ii) thrills and (iii) palpable 2nd heart sound.

(i) Pulmonary artery pulsation may be palpable in

atrial septal defect, ventricular septal defect, patent ductus arteriosus, total anomalous pulmonary venous drainage, severe pulmonary hypertension (known as pulmonary heave) and idiopathic dilatation of the pulmonary artery.

(ii) A systolic thrill over the pulmonary area is found in isolated pulmonary stenosis, two-thirds of the cases of atrial septal defect, high ventricular septal defect and Fallot's tetralogy.

A continuous thrill over the pulmonary area is characteristic of patent ductus arteriosus, aortopulmonary window and bronchopulmonary fistula or anastomosis.

(iii) A palpable 2nd heart sound over the pulmonary area is known as *diastolic shock*–characteristic of pulmonary hypertension of any aetiology.

(5) Place the flat of the hand over the base of the heart and note whether there is any expansile pulsation. This indicates an aneurysm of the arch of the aorta.

(6) Palpate the aortic area with the flat of the hand placed horizontally below the right clavicle, also covering a part of the sternum. A thrill over the aortic area is almost always systolic, indicative of congenital or acquired aortic stenosis.

Diastolic thrills over the base of the heart is not rare. It may be palpable in syphilitic or other varieties of aortic regurgitation and ruptured aortic cusps (e.g. in infective endocarditis). Systolic thrills over the carotid artery are found in aortic stenosis and is known as *carotid shudder*. It is very prominent in supravalvular aortic stenosis and may even be felt in the brachial arteries.

(7) Place your finger tips in the suprasternal notch. A suprasternal pulsation may be indicative of coarctation of the aorta, gross unfolding of the aorta, aortic regurgitation and aneurysm of the aorta. Also look for a tracheal tug.

(8) Place the ulnar border of the palm longitudinally over the right sternal edge. A pulsation in this region may be due to an aneurysm of the ascending aorta or rarely due to a huge right atrium caused by tricuspid atresia of Ebstein's disease.

(9) Feel for a pulsation over the back in the interscapular and infrascapular region. It is pathognomonic of dilatation of the collaterals in coarctation of the aorta (*Suzman's sign*). At times a systolic thrill over these collaterals may be present.

(10) *Pericardial* rub may be palpable over the precordium. It corresponds with heart beat and is neither influenced by nor related to respiration. It is found in various clinical conditions causing acute fibrinous pericarditis where the visceral and the parietal layers of the pericardium rub against each other with each contraction. It usually disappears when pericardial effusion develops. It is commonly found in tuberculous, acute rheumatic, viral pericarditis etc.

PERCUSSION

Although percussion of the heart become obsolete since the advent of the non-invasive investigative procedures, it may be used for the diagnosis of pericardial effusion, emphysema of the lungs and aneurysm of the arch of the aorta etc.

The surface marking of the borders of the heart has been described previously.

Percussion of the right border–Find out the level of the upper border of hepatic dullness along the right midclavicular line. Go one space above the liver dullness and keep the pleximeter finger parallel to the right border of the heart and apply light percussion on the right 3rd and 4th spaces. Go on percussing medially and put a mark on the point of dullness in each space. Join the skin marking with a slightly curved line with convexity towards the right.

The right border of the cardiac dullness may be increased in pericardial effusion, tricuspid stenosis, Ebstein's disease, aneurysm of the ascending or the descending aorta etc.

Percussion of the left border–First localise the apex by palpation and put a mark there; now start percussing the left border.

(i) Percuss the left 2nd, 3rd and 4th spaces from above downwards with the pleximeter finger placed 1/4 inch outside the lateral border of the sternum and note any change of resonance.

(ii) Now percuss along an arbitrary line that runs obliquely from the tip of the acromion process of the left side to the xiphisternum. The dullness will be normally obtained in the left 4th space.

(iii) Lastly percuss the chest, space by space from the anterior axillary line obliquely towards the apex from below upwards. This is the left border of the heart. The left border of cardiac dullness may cross the normal limit in left ventricular hypertrophy, pericardial effusion, ventricular aneurysm and in any condition giving rise to pulmonary arterial segment dilatation, such as in pulmonary hypertension, atrial septal defect, ventricular septal defect, patent

ductus arteriosus poststenotic dilatation of the pulmonary artery, idiopathic dilatation of the pulmonary artery etc.

Percussion of the base of the heart–Percuss the right second intercostal space, the midsternum and left second intercostal space starting from the right side. A dull note over the left 2nd space is observed in aneurysm of the arch of the aorta, massive pericardial effusion etc. Increase in the retrosternal dullness is also found in tumours of the anterior mediastinum.

Rotch's sign :– Dullness of the right 5th intercostal space and change of the cardiohepatic angle from a right angle to an obtuse angle are found in massive pericardial effusion.

AUSCULTATION

Place the chest piece over the apical impulse, then in turn over the pulmonary, aortic and tricuspid areas; then over the lower end of both sides of the sternum and also over other sites like the femoral artery, carotid artery etc. The anatomical landmarks of the different valves and the auscultatory areas have been already described.

During auscultation of the different areas. note the followings :

(a) First and second heart sounds :–their intensities, qualities and rhythms.

(b) Adventitious sounds e.g. murmurs, friction rubs or any added heart sound.

The murmurs are heard carefully to ascertain their timing (in *relation* to systole or diastole of the ventricles), intensity, character, site (whether localised or radiated) and alteration with respiration etc.

HEART SOUNDS : These are the sounds heard in one cardiac cycle and are –

I. *Valve closure sounds*–The first and second heart

sounds are produced by the closure of the atrioventricular and semilunar valves respectively.

II. *Valve opening sounds*–The opening snap of stenotic lesions of the atrioventricular valve is produced by the opening of the mitral valve (in mitral stenosis) or the tricuspid valve (in tricuspid stenosis).

III. *Vascular vibration sounds*–They are known as ejection clicks and may be pulmonary and aortic. Nonejection systolic click (s) may be heard in mitral valve prolapse.

IV. *Ventricular filling sounds*–The third heart sound, (or the protodiastolic or ventricular gallop) is classically a ventricular filling sound.

V. *Atrial contraction sound*–The fourth heart sound (or the presystolic or atrial gallop) is a classical atrial contraction sound.

1. THE VALVE CLOSURE SOUND–

The first heart sound is *dull* and *prolonged*, best heard at the apex. It indicates the beginning of the ventricular systole. It is produced by the closure of the bicuspid (mitral) and tricuspid valves.

The second heart sound is best heard in the pulmonary and aortic areas and indicates the beginning of the ventricular diastole. It is *short* and *high pitched*, produced by the closure of the aortic and pulmonary valves, The aortic and pulmonary components are heard best over the aortic and pulmonary areas respectively.

Normally the mitral component of the first heart sound and the aortic component of the second sound are better heard than the tricuspid component of the first sound and the pulmonary component of the second sound respectively because the mitral and aortic valves are working

against a high left ventricular pressure (100/0mm Hg) whereas the right sided valves are working against a low right ventricular pressure (20/0mm Hg).

Abnormalities of the First Heart Sound :

(A) Alteration in the intensities of the first heart sound which depends upon ·

(1) Position of the valve cusps at the beginning of systole–

(a) If the valve cusps remain wide apart at the beginning of the systole due to a high pressure gradient between the atria and the ventricles (as in mitral and tricuspid stenosis) the first heart sound will be loud. First heart sound tends to be loud in *tachycardia* from any cause such as emotion. exercise, fever, anaemia, hyperkinetic circulatory states such as thyrotoxicosis, pregnancy etc. because of the same mechanism–

(b) If the valve cusps remain close to each other at the beginning of the ventricular systole or if they cannot appose closely on each other, the intensity will be muffled such as in mitral and tricuspid regurgitation. The sound tends to be faint in myocardial infraction, myocarditis, myocardial failure cardiomyopathy and also in hypothyroidism.

(2) P. R. Interval–If the P. R. interval is short, the first heart sound becomes loud such as in tachycardia due to any cause.

First heart sound becomes muffled if the P. R. interval is prolonged as occurs in rheumatic carditis, digitalis toxicity etc. In heart block and Wenckebach's phenomenon, and in premature or ectopic beats, the first heart sound is of variable intensity.

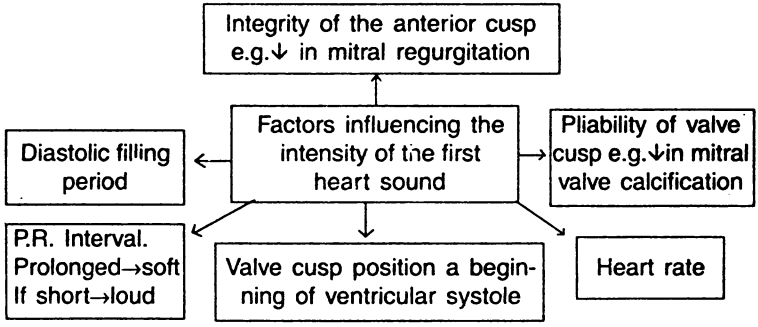
(3) Pliability of the valve cusps–If the pliability is lost,

the heart sounds will be diminished in intensity as occurs in calcification, fibrosis and rigidity of the anterior leaflet of the mitral valve even in presence of mitral stenosis.

(4) Muscle mass of the ventricular pump–First sound *is booming* in intensity in hypertensive patients because of the increased muscle mass. If the rate of the left ventricular pressure pulse is rapid, S_1 will be accentuated.

(5) Presence of fluid in the pericardial sac hinders the normal conduction of heart sounds and, therefore, intensity of the first heart sound is diminished in pericardial effusion. Presence of an emphysematous lung and increased thickness of the chest in obesity are also responsible for diminution of the intensity of the first heart sound.

Factors influencing the intensity of S_1 may be summarised as follows :



(B) Splitting of the first heart sound

Normally there is a gap of 0.02 to 0.06 seconds between the closure of the mitral (M_1) and the tricuspid valves (T_1), *which cannot be clinically detected.* If the gap exceeds 0.06 seconds, it becomes detectable and is suggestive of *right*

bundle branch block.

Normally M_1 precedes T_1 because the impulse starts from the left side and traverses towards the right. In mitral stenosis as the left atrium takes more time to empty, the *mitral valve* closes late and, therefore, M_1 and T_1 will fuse or M_1 will follow T_1 This is known as *reversed splitting* of the first heart sound. The tighter the mitral stenosis, the wider is the splitting. Physiological splitting of the first heart sound from slight asynchrony in ventricular contraction is a normal phenomenon. It can sometimes be detected on healthy individuals by listening at the lower end of the sternum with the breath held in expiration. The importance of the split first heart sound lies in its proper recognition and in differentiation from other extra heart sounds. *Pathological splitting of the first heart sound occurs in complete right bundle branch block.*

Splitting of the first heart sound must be distinguished from (i) The presystolic triple rhythm produced by atrial contraction sound (4th heart sound) and (ii) from a first heart sound followed by an ejection click as found in cases of aortic stenosis and pulmonary stenosis.

Presystolic triple rhythm (S_4) is found in patients with left heart disease with left ventricular hypertrophy, ischaemic heart diseases, hypertrophic cardiomyopathy, acute mitral regurgitation, delayed AV conduction even in the absence of clinically detectable heart diseases etc. It is absent in atrial fibrillation.

ABNORMALITIES OF THE SECOND HEART SOUND

(A) *Alteration in the intensity of character–*

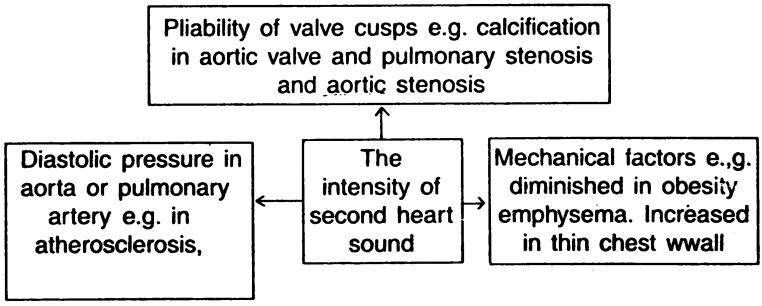
The 2nd heart sound may be accentuated if there is a high pressure gradient between the ventricles and the great vessels as for example, the P_2 is accentuated in pulmonary hypertension and the A_2 is accentuated in systemic hypertension. Second sound has two components : – the aortic (A_2) and the pulmonary (P_2). A_2 is loud in systemic hypertension, aortic atherosclerosis, aortic dilatation caused by syphilis or atheroma, and is also accentuated in unfolding of the aorta because of the close proximity of the aorta with the chest wall.

A_2 may be ringing in syphilitic aortic aneurysm because the aortic cusps open against a dilated root and ascending aorta.

P_2 is loud in pulmonary hypertension due to mitral stenosis, primary pulmonary hypertension, pulmonary hypertension secondary to atrial septal defect, ventricular septal defect and patent ductus arteriosus. Loud P_2 may also be present in chronic lung diseases such as emphysema and massive pulmonary fibrosis and also in dilatation of the pulmonary artery.

Both A_2 and P_2 may be loud in a patient with thin chest wall or in co-existence of both systemic and pulmonary hypertension.

Factors influencing the intensities of A_2 and P_2 may be summarised as follows–



If there is calcification of the semilunar valve cusps, A_2 and P_2 will be of diminished intensity. This occurs in calcific, aortic and pulmonic stenosis.

In Fallot's tetralogy, the aorta is situated anteriorly and the pulmonic valve is stenosed and so the pulmonary second sound is *soft* or *inaudible*–thus the 2nd sound is single in this condition. Second heart sound is often single in moderate aortic stenosis because the two components (A_2 and P_2) merge and fuse with each other. In severe pulmonary stenosis the P_2 may become inaudible because it is very faint.

In common truncus arteriosus there is only one functioning valve and hence there is only one component of the second heart sound (i.e, the second heart sound is single).

In Eisenmenger's VSD or single ventricle, second heart sound is single as P_2 becomes synchronous with A_2.

Causes of single second heart sound : A single S_2 is heard when either the A_2 or the P_2 becomes inaudible or merge together and so the causes include :

(A) *When the A_2 is inaudible :*
- (i) Diminished intensity in calcific aortic stenosis.
- (ii) Synchronous with P_2 in Eisenmenger's VSD or single ventricle.
- (iii) Concealed by
- (a) Loud P_2 in pulmonary area when there is pulmonary hypertension.
- (b) Loud holosystolic murmur of mitral incompetence or VSD.
- (c) Loud and prolonged ejection systolic murmur of pulmonary stenosis.

(B) *When the P_2 is inaudible :*
- (i) Diminished intensity in Fallot's tetralogy, pulmonary atresia and rarely in pure pulmonary stenosis.

(ii) Diminished intensity may also be due to poor transmission caused by hyperinflation of the lungs.
(iii) Synchronous with A_2 {as in (ii) of (A) above.}
(iv) May be concealed by systolic murmur of aortic stenosis.

(C) When A_2 and P_2 fuse : vide supra.

(D) *Splitting of the 2nd heart sound*

Normally there is a gap of 0.03 second between the closure of the aortic and the pulmonary valves. If a normal person takes a deep breath and holds it, more blood comes to the right ventricle due to physiological suction. The right ventricle takes more time to eject this extra amount of blood into the pulmonary circulation. So the pulmonary component goes late and a small split of the 2nd sound is audible during inspiration, being known as *physiological splitting* of the 2nd sound.

Effect of posture on splitting of the second heart sound:–

In normal children and young adults expiratory splitting may sometimes be heard in recumbent posture. It should normally disappear on sitting or standing up. If it persists even after sitting or standing up it is *definitely pathological.* Normal physiological splitting may not be audible in presence of tachycardia, thick chest wall or emphysema lung.

Pathological splitting–Abnormally wide splitting of the second heart sound is due to either an abnormal delay in the pulmonary valve closure or a premature closure of the aortic valve.

Delay in *pulmonary valve closure* (P) may be due to (i) delayed activation of the right ventricle (as in right bundle branch block) or (ii) due to prolongation of the right ventricular systole. Prolongation of the right ventricular systole may be due to (a) ralative increase in right ventricular stroke volume (as in left to right shunt e.g. atrial septal

defect) or due to (b) right ventricular outflow tract obstruction (as in pulmonary stenosis) or due to (c) impaired right ventricular function.

Early or premature *aortic valve closure* (P_2) may result from decreased left ventricular outflow into the aorta as occurs in mitral regurgitation and ventricular septal defect.

If the 2nd heart sound shows splitting both in inspiration and expiration it is known as pathological splitting. It occurs when the right ventricle deals with an enormous amount of blood both in inspiration and expiration. The P_2 will be significantly late (causing wide splitting) and this is found in (i) *atrial septal defect and* (ii) *total anomalous pulmonary venous drainage.* If the atrial spetal defect is a big one, an already overdistended right ventricle cannot accommodate the extra amount of blood from the left atrium via the right atrium and as a result, wide and fixed splitting of the 2nd sound occurs. Fixed splitting of the second heart sound is also audible in (a) severe right heart failure, (b) massive pulmonary embolism and (c) in conditions with impaired right ventricular function as in cardiomyopathy.

Close splitting of the 2nd sound is an important sign of *ventricular* septal defect as right ventricle receives an extra amount of blood from the left ventricle through the septal defect.

Reversed splitting of the 2nd sound is said to be present when splitting is audible during expiration but absent during inspiration. This happens when (i) the left ventricle takes more time to eject the blood into the aorta or (ii) there is a delay in the activation of the left ventricle. In these conditions the aortic valve closes after the pulmonary valve giving rise to audible splitting of the second sound on expiration. During inspiration the increased venous return to the right side of the heart causes a delay in the

pulmonary valve closure; so the A_2 and P_2 come very close to each other resulting in a single sound. This is the reverse of normal and hence called reversed or paradoxical splitting of the second heart sound. The common causes are (a) aortic stenosis, (b) systemic hypertension, (c) coarctation of the aorta, (d) left bundle branch block (e) myocardial infraction, (causing impaired left ventricular myocardial function) (f) severe aortic regurgitation and (g) patent ductus arteriosus.

Splitting of the second heart sound in expiration should be differentiated from (i) the opening snap of mitral stenosis, (ii) triple rhythm due to a third heart sound and (iii) from a pericardial knock which is found in early diastole in constrictive pericardities.

Causes of pathological splitting of the second heart sound

(A) Due to delayed pulmonary 2nd sound :

(i) Atrial septal defect.
(ii) Right bundle branch block.
(iii) Left ventricular pacing or ectopic.
(iv) Anomalous pulmonary venous return.
(v) Pulmonary stenosis.
(vi) Right ventricular failure.

(B) Due to abnormalities of left heart, causing early A_2 and / or delayed P_2 :

(i) Ventricular septal defect.
(ii) Mitral *incompetence.*

(C) Due to reversed splitting of the second sound :

(i) Electrical delay of A_2 :
(a) Left bundle branch block,
(b) Wolff-Parkinson-White syndrome.
(c) Right ventricular pacing or ectopic.

(ii) Mechanical delay of A_2 :
(a) Aortic stenosis (outflow tract obstruction).
(b) Systolic hypertension.
(c) Aorto-pulmonary communication.

(d) Left Ventricular failure.
(e) Ischaemia of the left ventricular myocardium.

II. VALVE OPENING SOUNDS

The valve opening sounds are not heard in normal subjects because the atrioventricular pressure gradient is not so high as to open the mitral and tricuspid valves loudly. So an opening snap will be produced only when there is a *high pressure gradient between* the atrium and the ventricle. The best example is mitral stenosis. The opening snap is a sharp high pitched sound; best heard with a diaphragm chest piece, after deep expiration and is usually loudest between the apex beat and left sternal border. Its timing is early in diastole 0.06 to 0.10 second after A_2.

The opening snap is thus diagnositc of high atrioventricular pressure gradient as occurs in (i) organic lesions e.g. mitral stenosis, tricuspid stenosis, etc. or (ii) high flow across the AV valves e.g. posterior cuspal type of mitral regurgitation, ASD, VSD, PDA etc. The tricuspid opening snap is best heard on holding a deep breath. The absence of an opening snap in mitral stenosis may be due to rigidity of the anteromedial cusp caused by calcification, gross destruction of the valve cusps by fibrosis or infective endocarditis, or prevention of opening of the mitral valve cusps by the regurgitant blood coming from the aorta into the left ventricle in acrtic valve incompetence.

Presence of an opening snap and a loud S_1 indicate a pliable mitral valve that would be readily amenable to mitral *comissurotomy* (valvulotomy), but experience has shown that although it signifies a mobile anterior cusp it by no means indicate that separation of the cusps will be easy.

The opening snap also denotes a significant mitral stenosis; tighter the stenosis, closer is the opening snap to the S. The opening snap. does not disappear after a

successful mitral valvulotomy. It also denies the presence of significant aortic regurgitation or an anterior cuspal type of mitral regurgitation. Development of severe pulmonary hypertension in a case of tight mitral stenosis reduces the left atrioventricular pressure gradient and then the opening snap may be absent.

Opening snap must be differentiated from (i) wide splitting of the second heart sound, (ii) a third heart sound and (iii) pericardial knock. Though an opening snap may be present in tricuspid stenosis it is very difficult to hear and differentiate from a mitral opening snap; it is audible during deep inspiration and diminished after expiration.

III. VASCULAR VIBRATION SOUNDS (ejection click)

These are produced during the ejection phase of the ventricles when a sharp jet of blood comes out through a narrowed opening and strikes against the wall of the great vessels or when a jet of blood after crossing a high vascular resistance impinges on the wall of the great vessels. These sounds coincide with the maximal opening of the aortic or pulmonary valves.

An ejection click is a sharp, high pitched sound heard *immediately* after the S_1.

Aortic ejection click is best heard over the aortic area or in the apex. It is classically found in severe systemic hypertension, mild to moderate aortic stenosis, bicuspid aortic valve and gross aortic regurgitation.

Pulmonary ejection click is best heard over the pulmonary area and is found in early pulmonary stenosis, pulmonary hypertension, idiopathic dilatation of the pulmonary artery and Fallot's tetralogy. Some belive that the ejection click in Fallot's tetralogy is aortic in origin since an enormous amount of blood from both the ventricles enter the dextroposed aorta. A pulmonary click differs from an

aortic click in *becoming quieter* or *absent with inspiration*, in contrast to all other right sided auscultatory events,–they accentuate with inspiration.

Non-ejection systolic clicks may be present in mitral regurgitation caused by a prolapse of the posterior leaflet of the mitral valve;–and may be multiple. Generally they occur later than systolic ejection sounds, but may be heard any time during systole. They are best audible at the apex as well as in the lower left sternal border. Such systolic clicks are the *commonest* physical findings in mitral valve prolapse (*Barlow's syndrome*) and may be associated with systolic murmurs;–hence also known as *click murmur syndrome*.

IV. VENTRICULAR FILLING SOUNDS OF THE THIRD HEART SOUNDS (S_3)

These are heard during ventricular diastole. The third heart sound (S_3) is heard during the rapid filling phase.

The S_3 is the vibratory sound produced as a result of sudden distention of the left or right ventricle by the rapid flow of blood from the respective atria during the early rapid filling phase of ventricular diastole. It occurs between 0.12 and 0.16 second *after aortic valve closure* (A_2). When the ventricle distends, the mitral valve cusps become taut and stretched by the papillary muscles and chordae tendinae between the mitral ring and the apex and the vibration sound is produced. S_2 is absent in significant mitral stenosis or tricuspid stenosis which precludes rapid ventricular filling.

Physiological S_3 is heard in children and young adults. If it is heard in persons over the age of 40 years it should be considered pathological and when triple rhythm is associated with tachycardia, it is called gallop rhythm.

Pathological S_4 (*or ventricular gallop, rapid filling gallop*

or Protodiastolic gallop) may be heard loudest at the apex or near the sternum;–left sided S_3 is heard in systemic *hypertension* myocardial infraction, mitral regurgitation, thyrotoxicosis or cardiomyopathy causing left ventricular failure while the right sided S_3 is heard in massive pulmonary embolism, cardiomyopathy, tricuspid regurgitation etc.

A third heart sound in left ventricular failure will be heard in the middle of the diastole and hence the name diastolic gallop. The term gallop was given to this diastolic sound because of the usually associated tachycardia though." a gallop remains a gallop irrespective of the heart rate. "The diastolic gallop indicates diastolic overload and decreased myocardial contractility in a failing ventricle, and, therefore, is a bad prognostic sign.

V. ATRIAL CONTRACTION SOUND OR THE FOURTH HEART SOUND (S_4)

It results from and is simultaneous with the active atrial contraction against a raised and diastolic pressure of the right or the left ventricle and is either due to pulmonary hypertension and pulmonary embolism (causing right atrial S_4) or systemic hypertension and myocardial infraction (causing left atrial S_4). In a few normal persons an S_4 may be due to delayed atrioventricular conduction (i.e., prolonged P-R interval).

In complete heart block independent irregular S_4 may be audible, in the late part of diastole or in presystole. Addition of S_4 to the normal sounds also produce triple rhythm or (if associated with tachycardia produce) gallop rhythm called presystolic gallop rhythm. Often with the onset of cardiac failure S_4 *may be replaced* by S_3 or both may become audible.

Left sided S_4 are also audible in IHSS, aortic stenosis, other conditions associated with left ventricular outflow tract obstruction, coarctation of the aorta etc.

Left sided S_3 and S_4 should be searched for by the bell of the stethoscope, at the apex, with the patiern in the left lateral position and breath held in expiration.

Quadruple gallop– Occasionally the third and the fourth heart sounds are heard separately giving rise to four audible sounds in one cardiac cycle. This is known as quadruple gallop. It is heard in some cases of hypertensive and thyrotoxic heart failure, and also in Ebstein anomaly.

Summation gallop–In some gallop rhythm the extra sound may be produced by superimposition of the S_3 on the S_4 i.e., by summation of the 3rd and 4th heart sounds. Such a cadance is called summation gallop. It is found most commonly in left ventricular failure due to systemic hypertension or acute myocardial infraction.

The *pericardial knock* is a sharp third heart sound which indicates the termination of the ventricular filling, occurring 0.10 to 0.12 second after A_2, and often audible in patients with constrictive pericarditis and is due to the restrictive effect of adherent pericardium on diastolic expansion of the ventricles.

VI. PERICARDIAL FRICTION SOUNDS

These are present both in systole and diastole. The pericardial rub is best heard inside the apex and over the pulmonary area. The character is rough and grating. It is better heard on holding the respiration. It may have three components, *presystolic, systolic* and *early diastolic*. The rub may be best elicited with the diaphragm pressed firmly to the chest wall with the patient upright and leaning forward and the intensity in increased by forced inspiration.

The pericardial friction rubs are heard in the following conditions :

(i) Acute fibrinous pericarditis of rheumatic origin.
(ii) Acute pericarditis of tuberculous origin.

(iii) Acute nonspecific pericarditis of viral origin.
(iv) Chemical pericarditis due to chronic renal failure.
(v) Reactionary or haemorrhagic pericarditis due to myocardial infarction.
(vi) Pyogenic pericarditis.
(vii) Malignant pericarditis in association with bronchogenic carcinoma.
(viii) Pericarditis associated with acute pulmonary embolism.

MURMURS

Definition–Murmurs are adventitious or abnormal cardiac sounds produced by circulatory sequences through the valves, great vessels of abnormal openings when there is an abnormal turbulence in the flowing blood. Bruits are sounds produced similarly in the peripheral arteries.

Classification

(A) (1) Ventricular regurgitant murmur.
(2) Ventricular ejection murmur.
(3) Ventricular filling murmur.

(B) Murmurs may be organic, where there is a structural defect in the heart; innocent, where the heart is normal and functional, where the murmur is due to an enormous blood flow in an already diseased heart.

(C) Murmurs are classified also according to their sequence with the systole and diastole i.e.
(i) Systolic–when it coincides with the apex beat,
(ii) Diastolic or (iii) Systolodiastolic or continuous.

(1) *Ventricular regurgitant murmurs.*

They are always systolic. If the blood regurgitates from the left ventricle into the left atrium through the incompetent mitral valve or from the left ventricle into the right ventricle

through a ventricular septal defect, the murmurs are known as murmurs of mitral regurgitation and ventricular septal defect, respectively. Similarly, when blood regurgitates from the right ventricle into the right atrium through incompetent tricuspid valve during systole, the murmur is known as tricuspid regurgitation murmur. The regurgitant mumur starts immediately after the first heart sound and usually continues up to the second heart sound–hence they are known as *pansystolic murmurs*. They are usually associated with systolic thrills and coincide with the apex beat. In mitral regurgitation the murmur is conducted towards the back as the blood is flowing from an anterior (left ventricle) to a posterior chamber (left atrium). In ventricular septal defect the murmur is conducted towards the right side of the chest as the jet of blood is going from a left sided (left ventricle) to a right sided chamber (right ventricle). In tricuspid regurgitation the murmur is conducted from the left sternal edge obliquely upwards towards the right sternal edge because right ventricle is situated along the left sternal edge, whereas the right atrium along the right sternal edge.

The regurgitant pansystolic murmurs are always associated with a 3rd heart sound which is usually loud.

(2) *Ventricular ejection murmurs*

These murmurs are produced by the ejection of blood from the left or right ventricle into the aorta or pulmonary artery repectively during the rapid ejection phase of the ventricular systole. They are loudest during midsystole, start slightly after the 1st sound (following the ejection click) and stop before the onset of 2nd sound. Hence they are known as *midsystolic ejection murmurs*. They are usually associated with systolic thrills at the base of the heart and over the carotid or pulmonary arteries.

(a) In aortic stenosis (congenital or acquired), obstructive cardiomyopathy, conditions giving rise to enlarged left ventricle with normal aortic ring (i.e., relative stenosis). Hyperkinetic circulatory states where an enormous amount of blood passes through a normal left ventricular outflow tract, an aortic midsystolic ejection murmur is produced.

(b) A pulmonary ejection systolic murmur is produced in pulmonary stenosis and may be associated with a systolic thrill in the pulmonary area. In atrial septal defect an enormous amount of blood passing through the normal right ventricular outflow tract will produce a similar ejection systolic murmur of lesser intensity. This is known as functional ejection systolic murmur of atrial defect. The same mechanism explains the pulmonary ejection systolic murmur due to an anomalous pulmonary venous drainage.

(c) An ejection systolic murmur in pulmonary hypertension with right ventricular enlargement is produced by flow of blood from the right ventricle into a dilated pulmonary, artery and may be heard over the pulmonary area.

(3) *Ventricular filling murmurs*

These murmurs are produced by (i) right or left atrial outflow tract obstruction or (ii) regurgitation of blood from the aorta or the pulmonary artery into the respective ventricles.

(a) The murmur of atrial outflow tract obstruction is best heard in the *middle of diastole* as well as in *presystole*, There will be accentuation of the presystolic murmur because of the active atrial contraction in the last rapid filling phase of the

ventricle. These are classically found in mitral stenosis and tricuspid stenosis. The murmur in the former is *low pitched rumbling* in character. It is *localised* as the blood flows from a posterior to an anterior structure. It is usually accompanied by a diastolic thrill. The murmur of tricuspid origin increases with inspiration and this is known as *Carvallo's sign.*

Functional middiastolic murmurs occur when an enormous quantity of blood flows through a normal mitral valve, as occurs in PDA, VSD, hyperkinetic circulatory states or when a large amount of blood passes through the tricuspid valve as in ASD.

(b) When blood regurgitates back into the ventricles from the aorta or the pulmonary artery, a diastolic murmur is heard in *diastole* when the ventricles are in isometric relaxation phase. In this phase the atrioventricular valves are closed and so no blood is coming from the atria into the ventricles. They are less audible after middiastole as the blood coming from the artria into the ventricles will prevent the pulmonary and aortic regurgitations. However, in long standing aortic regurgitation the murmur may be long or *even holodiastolic.* The murmur is best heard over the left third intercostal space along the left sternal edge, which overlies the outflow tract of the left ventricle. This area is known as *Erb's point.* To differentiate between the diastolic murmurs of pulmonary and aortic regurgitation, look for the peripheral signs of aortic regurgitation (e.g. water hammer or high volume collapsing pulse, wide pulse pressure, dancing

carotid arteries or Corrigan's sign capillary pulsation, Hill's sign, pistol shot sound or Traube's sign, Duroziez's diastolic murmur and de-Musset's sign) and try to find out if there is a right ventricular heave with accentuated P_2 suggestive of pulmonary incompetence. The murmur of pulmonary regurgitation caused by pulmonary hypertension (e.g. that due to mitral stenosis) is known as Graham-Steel murmur.

Hill's sign–Increase in the femoral arterial pressure of more than 20 mm Hg above the brachial artery pressure is known as Hill's sign. The normal difference is 20 mm Hg. The greater the incompetence the higher is the gap.

de-Musset's sign–To and fro nodding of the head along with carotid pulsation is known as de-Musset's sign.

Muller's sign–It is the rhythmical pulsatory movement of the uvula, found in florid aortic incompetence, caused by forceful arterial pulsation.

(4) *Continuous murmurs*

These are produced by the flow of blood from high pressure to low pressure vessels.

Causes–(1) Continuous murmur over the left 2nd intercostal space along the left sternal edge is audible in patent ductus arteriosus, pulmonary arteriovenous fistula, aortopulmonary window and bronchopulmonary anastomosis. The blood flows from the aorta into the pulmonary artery or from the bronchial artery into the pulmonary artery.

(2) If it is heard below the left 3rd space along the left sternal edge. it is suggestive of coronary arteriovenous fistula or rupture of a sinus of Valsava into the right atrium, pulmonary artery.

The murmur of coarctation of the aorta is vascular in origin, and is late systolic in timing with spilling over into the diastole. When blood passes through the constriction, a late systolic murmur is produced and by this time the ventricle goes into the diastolic phase so that the murmur spills over into the early phase of diastole. When blood passes through the collaterals in coarctation of the aorta, a murmur can be heard over the dilated intercostal arteries in the interscapular and infrascapular regions. This is known as *Suzman's sign.*

N.B. – A continuous murmur e.g. that of PDA should be indentified carefully to differentiate it from a to and fro murmur e.g. that of AS and AR or VSD and AR.

(5) *Functional and innocent murmurs*

(a) *Functional murmurs*–These occur in the absence of an organic heart disease at the site of production of the murmur. The examples include–

(i) Systolic murmurs in the pulmonary area in ASD which is produced by an increased flow of blood through the normal pulmonary valve and not by the flow of blood through the septal defect.

(ii) A middiastolic murmur at the lower sternal edge in ASD due to an increased flow of blood through the tricuspid valve.

(iii) An apical, soft, low pitched, presystolic or middiastolic murmur in some cases of aortic regurgitation, when the regurgitant flow from the aorta along the ventricular wall impinges against the anteromedial mitral valve cusp–it produces a relative or functional mitral stenosis. This is the mechanism by which this *Austin Flint murmur* is produced. However, there are other theories about the genesis of the Austin Flint murmur.

(iv) In mitral stenosis with severe pulmonary hypertension an early diastolic blowing murmur is heard along the left sternal border, produced by a functional pulmonary regurgitation, It is known as *Graham Steel* murmur.

(v) An apical functional diastolic murmur may also be found in ventricular septal defect, patent ductus arteriosus, and mitral regurgitation because of torrential flow across the mitral valve.

(b) *Innocent murmurs* are usually systolic murmurs arising in a normal heart and the causes of these are unknown. These are found in almost all children. The possible mechanisms are – (i) passage of a normal amount of blood through the pulmonary artery to the pulmonary vascular bed whose resistance is more than what is found in adults; and (ii) hyperkinetic circulatory states in children due to persistently high heart rate. As such, these murmurs disappear as age advances when the heart rate and pulmonary vascular resistance fall. At times innocent murmurs may only be heard after exercise. Innocent murmurs are best heard over the apical and pulmonary areas.

Haemic murmurs–This term is best avoided. It only indicates a murmur associated with hyperkinetic circulatory state producing increased amount of pulmonary blood flow in most cases. In some cases of severe anaemia there may be dilatation of the valve ring producing functional mitral regurgitation.

The following points are to be noted carefully when a murmur is detected on auscultation :

(i) *Timing in the cardiac cycle* : Note carefully whether the murmur is systolic. diastolic or systolodiastolic. The timing is easily done during a slow heart rate but may be difficult during tachycardia. For timing of a murmur the apex beat or the carotid pulsation may be used. Apex beat coincides with S_1 and carotid pulsation occurs 1/10th second after S_1. A systolic murmur appears immediately after S_1 although there is a small gap if the murmur is midsystolic. A diastolic murmur appears immediately after S_2 or after an appreciable time interval but immediately before the S_1 (if it is presystolic). One should also note carefully whether the murmur is heard throughout the whole systole (pansystolic). or in the middle of diastole (middiastolic) and so on and so forth. At times it may be difficult to time a murmur. In such cases the timing of the murmur in the cardiac cycle should be found out by noting its. character. In most difficult circumstances phonocardiographic help is needed.

(ii) *Character of the murmur* : Various types of murmurs are heard of which the following are characteristic :

(a) Low pitched rumbling murmur of mitral stenosis.

(b) Soft blowing, high pitched early diastolic decrescendo murmur of aortic regurgitation.

(c) Continuous machinery (train through tunnel; Gibson's) murmur of patent ductus arteriosus.

(d) High pitched blowing murmur at the apex radiating towards the axilla in mitral regurgitation.

(e) Loud, rough and harsh systolic murmur beginning a little after S_1 with midsystolic accentuation

(diamond shaped) and stoping just before S_2 heard in the aortic area in aortic stenosis. It is conducted upwards towards the carotid arteries, and sometimes best heard in the mitral area.

(iii) *Conduction of the murmur*: The direction in which the murmurs are conducted should be carefully noted. The murmur of mitral regurgitation is selectively conducted to the axilla and left infrascapular area of the chest. The murmur of mitral stenosis is localised to the apex. The direction of conduction or the absence of conduction characterises certain murmurs. The conduction depends on the loudness of the murmur; conductive nature of adjacent tissue, the direction of the blood flow that produces the murmur, and at times on the state of hypertrophy of the papillary muscles.

(iv) *Intensity of the murmur* : Murmurs have a wide variety of intensity ranging from very faint to very loud. The area of maximum intensity should be found out. It must be remembered that the intensity of a murmur is not always proportional to the haemodynamic disturbance. Thus a murmur of a small VSD may be much more intense than that due to large one. Arbitrarily the intensity of murmurs have been divided into SIX different grades, grade I to VI (vide infra). When a murmur is produced at a valve, the maximum intensity of the murmur will be heard at the particular point where that particular valve sound is best heard. For example, mitral stenosis murmur is best heard at the mitral area, aortic stenosis murmur is heard with maximum intensity at the aortic area etc.

(v) *Alteration of murmur with respiration* : The stroke

out put of the right heart increases during inspiration while that of the left gets reduced. So the murmurs originating in the right heart becomes more intense during inspiration, and accentuation of a tricuspid murmur with inspiration is known as *Carvallo's sign*. The murmur which becomes louder on expiration is of the left heart. A sustained Valsalva manoeuvre results in intensification of the murmurs of IHSS and prolongs or brings out that of mitral valve prolapse.

(vi) *Change of murmur with change of posture* : Murmurs should be studied both in the recumbent and in the upright (i.e., sitting or standing positions). The murmur of mitral stenosis is best heard in the mitral area with the patient recumbent and turned to a left lateral position; on the other hand, the murmur of aortic regurgitation is best heard in the aortic area of Erb's point with the patient sitting upright and leaning forward and the chest being held in a position of full expiration. The murmurs of mitral valve prolapse and IHSS are intensified on standing and *decreased* or *abolished* by prompt squatting.

(vii) *Behaviour of murmur during exercise* : Exercise increases the intensity of the faint murmurs or at times bring out an otherwise faint murmur to a prominence. Thus the diastolic murmur of mitral stenosis becomes louder and prominent after exercise. Isometric hand grip exercise accentuates the murmur of aortic regurgitation, but diminishes that of aortic stenosis and IHSS.

Remember that in children a transient systolic murmur may be heard after exercise–in most cases it is innocent.

Auscultation of special regions other than the heart

The results of the detailed examination of the heart, performed as advised above may direct the clinician to look for special signs specific for some diseases or conditions e.g. :

(1) Auscultation of the lung bases for crepitations should be done in cases with left heart enlargement. The crepitations are due to pulmonary venous congestion which leads to interstitial pulmonary oedema : and if sufficiently progressed, to alveolar oedema.

(2) The abdomen should be auscultated for systolic bruits over the renal artery, diagnostic of renal artery stenosis.

(3) Pistol shot sounds are present over the femoral artery in patients with high pulse pressure.

(4) Duroziez's murmur--It is the diastolic murmur heard over the femoral artery when one presses the femoral artery distally with the edge of the chest piece. This indicates arterial reflux in aortic regurgitation. *Duroziez's sign* : When the chest piece is placed over the femoral artery, a diastolic murmur is heard if the artery is pressed distally and a systolic one if the artery is pressed proximally. This is Duroziez's sign, one of the peripheral signs of aortic regurgitation.

(5) Auscultation over the lumbar region on the back may reveal a murmur due to blood flow through the constricted aorta in a subphrenic type of coarctation of the aorta.

(6) Auscultation over the shin bone may reveal a continuous murmur indicative of Paget's disease of the bones or peripheral arteriovenous fistula.

(7) A continuous murmur over the jugular vein is known as *venous hum;* it is a common normal finding in children in an upright posture.

(8) Systolic bruits may be heard over the thyroid in thyrotoxicosis.

(9) A murmur may be heard over the closed eyes in a carotido-cavernous fistula.

ELECTROCARDIOGRAPHY

The electrocardiogram is the recorded magnified galvanometric deflections which reflect the electrical events of the heart. The waves produced are known as P. Q. R. S. T and U. This helps us (i) to understand the pathway of conduction of the excitatory wave, (ii) to detect hypertrophy of the ventricles even when its clinical or rediological detections remain elusive, (iii) to diagnose myocardial ischaemia or infraction and (iv) to record and analyse the arrhythmias. For a detailed account of electrocardiography the students are advised to consult specialised text-books, but if they comprehend the basic principles of electrocardiography they will be able to easily interpret the electrocardiogram.

In routine practice a 12 lead electrocardiogram is recorded; these leads are–

(a) Edberg lead .....

(A) Six limb leads which are again divided into–

(a) 3 standard or bipolar limb leads.

Lead I–indicates the potential difference between the left and the right arms.

Lead II–Potential difference between the left leg and the right arm.

Lead III–Potential difference between the left leg and the left arm.

(b) Goldering leads.....

(b) Augmented unipolar limb leads :

aVR–The exploring electrode is placed on the right arm.

aVL–The exploring electrode is placed on the left arm.

aVF–the exploring electrode is placed on the left leg.

NB–Remember that the limb leads are affected by the position of the heart within the chest.

(c) Wilson leads .....

(B) Six unipolar chest leads designated V_1 to V_6–

V_1–Right sternal border in the 4th intercostal space.

V_2–Left sternal border in the 4th intercostal space.

V_3–Between V_2 and V_4

V_4–Left 5th intercostal space on the midclavicular line.

V_5–Left 5th intercostal space on the anterior axillary line.

V_6–Left 5th intercostal space on the midaxillary line.

THE NORMAL ELECTROCARDIOGRAM

The waves produced in the ECG are P, QRS, T and U and indicate the depolarisation of the atria (P) and the ventricles (QRS) and repolarisation of the ventricles (T). The wave of atrial repolarisation remains buried in the QRS complex. The U wave is possibly caused by slow repolarisation of the Purkinje fibres.

The ECG is recorded on a specially prepared graph paper. The thin vertical lines or thin horizontal lines are 1 mm apart and represents horizontally 0.04 seconds and vertically 0.1 mV.

Normal Patterns

Normally in the standard limb leads, P, R, and T are positive waves and Q and S are negative waves.

It must be remembered that the deflection of waves in any lead will depend on the direction of the impulse relative to that particular lead.

P wave–First wave of electrocardiogram and represents the spread of impulse through the atria (atrial depolarisation). Its amplitude should not exceed 2.5 mm and width should be less than 0.11 sec. in duration.

P-R interval–P-R interval is atrioventricular conduction time and is measured from the beginning of the P wave to the beginning of the QRS complex. Normally its duration is 0.12 to 0.20 seconds.

QRS interval–QRS interval denotes ventricular depolarisation and normal duration is 0.05 to 0.10 second.

Q wave–Q waves are absent in V_1 and V_2 and where present, it is less than 0.04 sec. in duration and less than 25% of R.

J point–This is the point where the QRS ends and ST begins.

ST segment–From the J point to the beginning of the T wave. Usually isoelectric but may be elevated up to 2 mm or depressed by 0.5 mm. Look for its shape; normally it curves smoothly and joins the proximal limb of the T wave. Its elevation or depression are in comparison to the T-P segment.

S wave–Represent ventricular repolarisation and usually rounded and slightly asymmetrical. It is inverted in a VR, may be upright, diphasic or inverted in III, aVL, V_1 and upright in all other leads. Normal height should not exceed 5 mm in standard leads and 10 mm in any precordial leads.

Q-T interval–It is measured from the beginning of the QRS to the end of the T wave. It represents the duration of the electrical systole. This interval is dependent on age, sex and heart rate.

U wave–Occassionally this is seen after the T wave. Its

significance is uncertain. U-wave coincides with the phase of super normal excitability during ventricular recovery. It is in this phase that most of the ventricular premature beats are seen to occur.

Electric heart axis

The normal electric. heart axis is between 30° to 90° You look in which standard limb lead. QRS in most positive, normally in 1 or 11.

Is e.g. III most positive, the axis is over, 90°, it is a right axis deviation.

Is e.g. III or aVF most negative the axis is under, 30°, it is a left axis deviation.

A slight right heart deviation might be seen physiologically in tall and thin but healthy people.

Abnormal Patterns

One should observe the shape, breadth and the height of each of the waves of PQRST complex.

(1) P wave :

(a) Broad and bifid–Left atrial hypertrophy (P-mitrale) ('P' more than 0.11 seconds).

(b) Tall and peaked–Right atrial hypertrophy (P-pulmonale ('P' more than 3 mm).

(c) Absent–AV nodal rhythms, S-A block, hyperpotassaemia (severe), atrial fibrillation (replaced by f wave).

(d) Inversion–Dextrocardia nodal rhythm.

(e) Difficult to identify–in extreme tachycardia e.g. paroxysmal atrial tachycardia.

(2) P-R interval :

(a) Prolonged–(i) Partial A-V block; first and second degrees.

(ii) Giant left atrium in ASD of the septum primum type and mitral regurgitation.

(b) Shortened–(i) A-V nodal rhythm.
(ii) Atrial ectopic rhythm of lower origin.
(iii) Normally in infants.
(iv) WPW syndrome.

(3) QRS complex – Abnormalities of this complex may be found in ventricular hypertrophy, myocardial infraction, conduction defects etc.

(a) Prolonged QRS–intraventricular conduction defects, hyperpotassaemia, quinidine therapy etc.

(b) Low voltage QRS (height less than 5 mm in all three standard leads) found in–

Obesity, emphysema, anasarca, myxoedema, ischaemic heart disease, cor pulmonale, constrictive pericarditis etc.

(c) High voltage QRS–Seen in ventricular hypertrophy.

(4) ST segment–This segment is influenced by-Ischaemic heart disease, digitalis toxicity, myocarditis, potassium intoxication, acute pericarditis (concordant ST elevation), ventricular hypertrophy, intraventricular conduction defects etc.

(5) T wave–Normally the T wave is upright in leads I, II, V_2 to V_6; inverted in a VR and variable in leads II, aVL, aVF V_1 and V_2.

(a) Tall and peaked T waves are found in hyperkalaemia.

(b) Inverted T waves may be–
(i) Symmetrical in myocardial infraction (nontransmural).
(ii) Asymmetrical in L V-strain.

(6) Prolonged QT interval may be seen in–
Myocardial infraction, hypocalcaemia, quinidine and procainamide toxicity, myocarditis, congestive cardiac failure etc.

(7) Shortened QT interval may be found in– Digitalis therapy, hypercalcaemia hyperpotassaemia.

(8) U wave– Pronounced in potassium deficiency. In myocardial inschaemia and left ventricular strain its polarity is reversed i.e. inverted.

ECG changes in certain important clinical conditions

A. Left atrial enlargement

Broad an notched P wave (P-mitrale) usually best seen in leads I and III. Notching is significant when the distance between the two peaks exceed 0.04 sec.

In V_1, the P wave is characteristically wide, slurred and diphasic in which the downward component is most prominent.

B. *Right atrial enlargement*

(i) Tall slender, peaked P wave in II, III and aVF (P- pulmonale).

(ii) P wave prominent, peaked diphasic or inverted in V_1.

C. *Left ventricular hypertrophy*

(i) R in V_5 or V_6 is 27 mm or more.

(ii) S in V_1 and R in V_5 or V_6 together 35 mm or more.

(iii) Ventricular activation time (intrinsicoid deflection or VAT) over 0.05 second in V_5 and V_6.

(iv) QRS greater than 0.13 sec. in V_5 and V_6.

(v) ST segment depressed and T wave inverted in V_5 and V_6.

(vi) R in a VL more than 13 mm.

(vii) R in a VF more than 20 mm.

(viii) Left axis deviation (– 15° or more).

D. *Right ventricular hypertrophy*

(i) R taller than S in V_1.

(ii) Ventricular activation time (intrinsicoid deflection) over 0.03 second in V_1.
(iii) Persistent S in V_5 and V_6.
(iv) S-T segment depressed and T wave inverted in V_1 to V_3.
(v) Right axis deviation (+110° or more); depressed ST segment and inverted T wave in II and III.

E. *Right bundle branch block (RBBB)*
(i) RSR or rSR pattern in V_1 and V_2.
(ii) Wide S wave in V_5 and V_6.
(III) QRS interval 0.12 second or more.
(iv) Ventricular activation time (intrinsicoid deflection 0.06 second or more in V_1 and V_2.
(v) ST depression and T inversion in V_1 and V_3.

F. *Left bundle branch block (LBBB)*
(i) Wide and slurred R in V_4 to V_6.
(ii) Q absent in V_4 to V_6.
(iii) QRS interval 0.12 second or more.
(iv) Ventricular activation time (intrinsicoid deflection) more than 0.09 second in V_4 to V_6.
(v) ST depression and T inversion in V_4 to V_6.
(vi) Absence of Q, abnormal R with S T depression and T inversion in lead I.
(vii) In a VL a pattern similar to that seen in V_4 to V_6.

G. *Acute myocardial infraction (AMI)*
(i) Within a few hours after the infraction the ST segment becomes elevated with a convexity upwards.
(ii) Next to appear is the symmetrical T wave inversion ie. the peak of the T is midway between the beginning and the end. It occurs in hours to days.

(iii) Abnormal Q waves appear. They are found usually before any gross T wave changes occur. At times instead of appearance of Q waves, there may be only a reduction of the voltage of R. Abnormal Q waves are usually permanent.

The above mentioned changes are found only in those leads which represent the area of the myocardium involved in infraction.

Following leads are to be examined for exact localisation of the site of infraction :

Anterior wall–I, aVL, V_1 to V_6.

Anteroseptal wall–I, aVL, V_1 and V_2.

Anterolateral wall–I, aVL, V_5 and V_6.

Inferior wall–II, III aVF.

Inferolateral wall–II, III aVF, V_5 and V_6.

H. *First degree A-V block*

The P-R interval is prolonged to more than 0.2 sec. It indicates a delay in conduction through the A-V node or bundle of His.

I. *Second degree A-V block*

It may be of two types–Mobitz type I or Wenckebach type and Mobitz II. *Wenckebach or Mobitz type I A-V block*–The PR interval is usually normal in the first beat. Then with each successive beat it gradually becomes longer and longer (while the PR interval becomes shorter and shorter) until one QRS is dropped indicating a complete failure of ventricular response to atrial activation. After that the next P-R interval is again normal and the same phenomena occurs over and over again. The site of block in proximal to the His bundle and within the A-V node. *Mobitz type II A-V block*–Here the PR and PP intervals are constant throughout

but the ventricles fail to respond to atrial stimulation periodically either in a regular or an irregular fashion. The dysrrhythmia shows dropped beats regular e.g. with 2 : 1, 3 : 1 or 4 : 1 block or it may be irregular. Here the block is either in the His bundle or distal to it. Mobitz type II block is usually a more serious disorder than Mobitz type I block.

J. *Bifascicular Block.*

In this usually there is RBBB with left anterior or posterior hemi blocks (LAH or LPH) and the ECG shows–(i) Left (with LAH) or right (with LPH) axis deviations and (ii) Features of RBBB.

K. *Trifascicular Block.*

It is bifascicular block with PR prolongation.

L. *Complete Heart Block*

No impulse can pass through the A.V. node; the atria and the ventricles beat independent of each other. There is no definite relation between the P waves and the QRS complexes which are independent of one another. The artial rhythm is usually regular and the atrial rate is usually the average sinus rate. The ventricular rate is usually 40 per minute (varying between 20 and 60) and ventricular rhythm is also regular because the ventricle continues to beat in response to a pacemaker situated either in the A-V junction (nodal) or in the ventricular myocardium, producing idionodal or idioventricular rhythms respectively.

EXERCISE ECG–ECG in ischaemia is classically characterised by ST depression in leads with dominant R waves. Since the resting ECG may remain entirely normal in patients with IHD it may be necessary to document the ECG changes

during and after exercise to demonstrate the ischaemia. During exercise the ECG may reveal *J point depression* increasing as the exercise progresses, with the ST *becoming entirely flat for* the first 80 msec of its duration and with further change may even be *negative or downsloping.* An important criterion is that 1mm or 0.10 mv or more of flat ST displacement in a standard lead indicates myocardial ischaemia. Where ST displacement is transient improving with cessation of exercise it is known as type I response and is of minor prognostic abnormality. Type II is a protracted ST depression provoked by mild exercise and constitutes a major prognostic abnormality, such as caused by severe multivessel disease or left main coronary artery disease.

TECHNIQUE OF PERICARDIOCENTESIS AND THE STUDY OF THE PERICARDIAL FLUID

A pericardiocentesis is done to–(i) attempt at establishing a diagnosis of pericardial disease, (ii) relieve acute cardiac tamponade, (iii) aid anaesthetic management of the perioperative decompensated patient needing pericardiectomy and (iv) study elevation of venous pressures.

If performed at the bedside, ECG monitoring essential.

Approaches are – (i) subxiphoid (ideal) and (ii) parasternal.

The patient is adequately sedated and 0.5 mg to 1 mg atopine given IV to prevent vasovagal reaction.

With the patient propped up to 45° " a point 2 cm below the tip of the xiphoid and just to the left of midline is locally anaesthetized. The needle (about 6 inch long and of small guage) is introduced under ECG monitoring and cautiously

advanced towards the left shoulder. The resistance of the diaphragm and pericardium suddenly yields and as the needle touches the epicardium a scratch sensation is felt.

In the lateral thoracic approach the needle should be inserted just beyond the apex beat or in the left fifth intercostal space just internal to the lateral border of the cardiac dullness and directed backward and inward, towards the spine.

At first about 5 cc fluid is drawn out and allowed to clot if it is grossly haemorrhagic to exclude right ventricular puncture in which case the fluid readily coagulates.

When the pericardial space has been properly localised and LP needle of wider gauge is introduced along the same path and all possible fluid is removed.

In diagnostic pericardiocentesis air may be injected a the completion of the procedure to aid in follow up radiological studies.

Following the procedure most patients should be observed for 24 hours in an intensive care unit.

Possible complications are–(i) laceration of a coronary artery, (ii) laceration of the right ventricle, (iii) right atrial or ventricular perforation, (iv) pneumothorax, (v) gastric or colonic perforation, (vi) arrhythmias, (vii) tamponade, (viii) systemic hypotension etc.

Assessment of the case is very important and should be done by carefully taking the history and performing the clinical examination and radiological studies because diseases like cardiomyopathy and fibroelastosis, may stimulate pericardial effusion. After pericardiocentesis, the pulse and the blood pressure are recorded at hourly intervals and any irregularity of the pulse is to be considered seriously.

The main therapeutic indications of pericardiocentesis are (i) cardiac tamponade–clinically characterised by the triad of low volume pulse, engorded neck veins and a quiet

heart and (ii) evidences of considerable quantity of fluid in the pericardial cavity.

Similarly for diagnostic purposes in polyserositis, Meig's syndrome and if there is a strong suspicion of pericardial effusion on clinical grounds, this procedure is undertaken. Cases of amoebic abscess of the left lobe of liver bursting into the pericardium in the tropics have been diagnosed after a needle has been put into the pericardium.

The study of the fluid–macroscopic, microscopic, biochemical and pathological–is carried out as in cases of pleural and peritoneal fluids. Cholesterol crystals have occasionally been demonstrated in pericardial fluid of patients suffering from myxoedema.

RADIOGRAPHIC EXAMINATION OF THE HEART

Standard postero anterior and oblique views (both right and left) of X-ray chest are usually taken and if required, right anterior oblique (RAO) view with barium filled oesophagus is taken.

PA views–Cardiac silhouette is a flask shaped shadow between the two translucent lungs. Normally the cardiac apex is just internal to the midclavicular line.

The right border of the cardiac shadow is formed from above downwards by (i) the outer border of the superior vena cava with the ascending aorta and (ii) the convex outer border of the right atrium up to the diaphragm; the left border of the cardiac shadow is formed from above downwards by (i) the aortic kunckle, (ii) the pulmonary artery segment and then by (iii) the left ventricle up to the apex. If the left atrium is enlarged then in an overpenetrated film just internal to the right atrium another rounded border may be seen–that of the enlarged left atrium giving rise to a double contour of the right border of the heart. In left atrial enlargement an upward displacement of the left main bronchus is seen.

In the RAO view the barium filled oesophagus courses directly adjacent to the left atrium. This view is of particular importance for recognising compression of posterior displacement of the oesophagus by an enlarged left atrium.

In the left anterior oblique view of X-ray chest with the patient rotated to an angle of 50° or less the left ventricle is seen to overlap the spine when it is enlarged.

In the PA view an *enlargement of the ascending aorta* is seen in syphilitic aortitis with aneurysm, in aortic regurgitation and in aortic stenosis when poststenotic dilatation is present. Unfolding of the aorta is seen in atherosclerosis.

In mitral stenosis where the left atrial pressure is significantly high, engorged subpleural lymphatics are seen at the lung bases as horizontal lines. In radiographic finding these lines are called Kerley B lines. When the main branches of the pulmonary artery are engorged due to increased pulmonary blood flow it is called *pulmonary plethora.* When vascular markings in the lung fields are diminished it is called *pulmonary oligaemia.*

Fluoroscopy is used to see the (i) cardiac size (ii) pulsations of the different chambers and great vessels and (iii) to detect calcification of the cardiac valves, coronary arteries or pericardium etc.

Angiocardiography–The contrast media is injected via the catheter into the different chambers of the heart or into the great vessels and serial films are taken. When following an injection of the contrast media high speed X-ray motion pictures are taken it is called *cine angiography.* If the contrast media is injected selectively into a particular coronary artery and skiagrms taken, it is called *coronary angiography.* Angiocardiography, coronary angiography and

cine angiography have helped in better understanding of the dynamic anatomy of the heart, cardiac valves and coronary arteries in normal conditions as well as in a variety of cardiac disorders. These procedures are very important in selecting the appropriate cardiac patients for surgical management. They help in accurate structural and functional diagnosis of complex cardiac lesions.

PHONOCARDIOGRAPHY

A phonocardiogram is a graphic display of the cardiac sounds and murmurs. It helps in proper and objective assessment of the auscultatory events and allows a perfect detection and understanding of the sequences of cardiac events so far as the heart sounds and murmurs are concerned. Its most important application is in the precise timing of cardiac sounds and murmurs especially in complex cardiac lesions.

ECHOCARDIOGRAPHY

This is a noninvasive method of examining the heart by utilizing ultrasound.

The frequency of the sound used in echocardiography ranges from 1 to 7 mega Hertz, i.e. 1 to 7 million cycles per second. Frequencies usually used in adults are 2 to 3 mega Hertz and those used in children are 3 to 5 mega Hertz. Sound waves of this frequency are produced by intermittently exciting a piezzoelectric crystal electronically. The waves of ultrasound coming out of the transducer penetrate tissues and are reflected from the different structures of the heart back to the crystal in the transducer. A form of echo is provided by the reflected sound signals and the transducer receives if (when not transmitting) and sends out an electronic signal proportionate to the intensity of the echoes.

Echocardiography detects the motion of the solid structures of the heart and by the standard frequency used, structures up to 8 inches from the surface can be examined. Lower frequency ultrasonic beams are used in adults so that it penetrates well through the chest wall. Resolution of objects even only 1 to 2 mm apart is possible with this frequency.

The ultrasonic beams travel in a straight line through a homogeneous medium. But as it strikes the interface between two media of different acoustical properties it undergoes reflection and refraction akin to light. The reflected portion returns to the piezzoelectric element in the transducer and gives rise to an electric signal.

The different modes of display of the ultrasonic echoes received by the transducer are–

(i) **A Mode**–A stands for *amplitude*. Here the intensity of the reflected echo signal is displayed on the horizontal axis of the oscilloscope and the time required to travel from the transducer to the target and back is displayed on the vertical axis. With movement of the heart the echoes move up and down during the cardiac cycle. This mode of echo display system is now rarely used.

(ii) **B Mode**–B stands for *brightness*. This brightness is displayed on the z-axis of the oscilloscope and in this more practical method of recording echo motion the amplitude of the echo is converted to brightness and the returning *echoes* are displayed on the oscilloscope as dots rather than spikes. The distance from the transducer is plotted on the y-axis.

(iii) M Mode–M stands for *motion*. In this mode the element of time is introduced by sweeping the oscilloscope from the bottom to the top. In this mode of echocardiography time is recorded on the x-axis and the B mode echo signal is

recorded on the y axis and thus the amplitude and the rate of motion of the moving objects can be recorded with great accuracy. An electrocardiogram is recorded simultaneously. The transducer is placed usually along the left sternal border on the third or fourth space and the beam of the ultrasonic wave is directed to the part of the heart to be examined. Usually the structures through which the beam travels and are reproduced on the oscilloscope from above downwards are–the chest wall, the anterior wall of the right ventricle, portion of the right ventricular cavity, the interventricular septum, a portion of the left ventricular, cavity, the mitral valve apparatus and the posterior left ventricular wall with endocardial and epicardial echoes. Other structures of the heart can also be visualized by suitably directing the beam of ultrasonic waves from the transducer.

(iv) Real-time–Cross-sectional or two dimensional (2D) echocardiography–This is a more advanced technique that provides information e.g. cardiac shape and lateral motion that are not available by the M-mode. This is also known as sector, scanner, in this a rapid and repeated scanning of a B mode echocardiographic tracing is done across a sector field at a rate that provides continuous image. Cross-sectional echocardiography helps to display transverse, sagittal or coronal sections of the heart and thus increases the scope of ultrasonic examination.

Echocardiography helps in the diagnosis of almost all the different morbidities of the heart but is especially helpful for mitral valve diseases, calcifications of the valve cusps, pericardial effusion, left atrial myxoedema, infective endocarditis (detection of vegetations), congenital heart diseases and cardiomyophathies.

DOPPLER ECHOCARDIOGRAPHY :

Now-a-days there are three types of Doppler echo :

(a) The oldest one is the PULSED Doppler, which is only able to record slow velocities of the blood corpuscle. This slow velocities give a hint to areas with thrombatic risk.

(b) CW-Doppler (continous wave D.) is able to screen high velocities, but not their direction.

(c) COLOUR Doppler is one of the newest investigations, enables the recording of high velocities and its direction. If the blood flews in direction towards the transducer it occurs a different colour as if it flews in the opposite direction.

With this investigation you can predict : congenital heart failure, congenital cranial aneurysm (through open fontanella of the newborn), heart valve failure (gradient estimation) and control of prosthetic valves.

Doppler is a server alternative to invasive diagnosis.

Radionuclide ventriculography.

Laen invasive then catheterization and also a very precise method estimating ventricular function and anatomy. Erythrocytes or albumine is labcled with technetium 99 No aview of myocardial perfusion thallium 201 is injected a bit before maximum exercise on an ergometer. Those exercise scans together with resting time scans taken 2-5 hours later detect reparfusion (ischaemic) regions and fixed defects (infraction).

SOME COMMON CARDIOVASCULAR DISEASES

CARDIAC VALVE DISEASES

1. MITRAL STENOSIS

I. *Aetiology* : 85% rheumatic, rest are congenital or associated with cardiomyopathy and very rarely associated with rheumatoid arthritis and Hurler's syndrome.

II. *Symptoms :*

1. Effort intolerance– sudden or insidious. Sudden when caused by pulmonary oedema.
2. Haemoptysis.
3. Angina pectoris, rarely which mey be due to–
   (a) Right ventricular hypertrophy–imbalance in blood supply and myocardial oxygen demand.
   (b) Atrial fibrillation due to cardiac ischaemia.
   (c) Discomfort due to costochondritis.
   (d) Associated coronary artery disease.
4. Easy fatiguability.
5. Palpitation resulting from–
   (a) Right ventricular hypertrophy.
   (b) Atrial fibrillation.
6. Dependent oedema and even anasarca.

III. Signs :

1. Malar flush, especially in fair-skinned individuals.
2. Small volume pulse.
3. Blood pressure-low.
4. Neck veins not engorged, but if there is pulmonary hypertension–gaint 'a' waves are found.
5. Apex beat–Normal in position, character is slapping or tapping. There is a diastolic thrill.
6. Parasternal heave and diastolic shock–when there is pulmonary hypertension.
7. Auscultation–S_1– short, sharp accentuated (not if the valve is calcified or if there is prosthetic valve). S_2–audible in mitral area.
   Opening snap (of Potain) : better heard after deep expiration.
   Murmur : mid-diastolic rumbling low pitched, localised to the mitral area, best heard after

expiration, on turning the patient to left lateral position and with the bell, in sinus rhythm, after exercising the patient, presystolic accentuation which disappears if there is atrial fibrillation, but may be audible rarely with a strong atrial contraction. S_2–in pulmonary area is split.

IV. *investigation :*

1. X-ray chest. PA and left lateral views.
2. ECG : P waves widened and notched (P mitrale). QRS complex shows verying degrees of right axis deviation in pulmonary hypertension, or P replaced by f waves in atrial fibrillation.
3. Echocardiographic : Left atrial enlargement; mitral valve situation (calcification, thickened etc); Doppler : less left ventricular at a diastolic input (degree estimation).

V. *Complications :*

1. Acute left atrial failure leading to pulmonary oedema.
2. Atrial fibrillation.
3. Systemic embolism usually due to atrial fibrillation.
4. Severe pulmonary hypertension.
5. Right heart failure and congestive cardiac failure.
6. Severe haemoptysis due to pulmonary infraction.
7. Infective endocarditis in 0.5% cases only. Percentage is low, since endocardial erosion is practically absent.
8. Dysphagia.

9. Ortner's syndrome : Hoarseness of voice due to involvement of left recurrent laryngeal nerve by–
   (i) left atrial enlargement.
   (ii) pressure of hilar lymph nodes
   (iii) enlargement of pulmonary artery segment.

VI. *Treatment*

Mitral valvulotomy (comissurotomy)–closed or open types. The latter by open heart surgery.

Indications for mitral valvulotomy

(1) Age between 20 and 40 years.
(2) Increasing breathlessness–from grade 1 to grade III cardiac disability status (NYHA).
(3) Severe haemoptysis.
(4) Clinical, radiological and electrographical evidences of progressive pulmonary hypertension.
(5) Associated with insignificant mitral and aortic regurgitations.
(6) Controlled congestive cardiac failure.
(7) Digitalised heart, with atrial fibrillation under anticoagulant cover.
(8) Treated cases of infective endocarditis with tight mitral stenosis.
(9) Damped mitral stenosis.

2. MITRAL REGURGITATION

1. *Aetilogy* (a) Organic :
   (1) Rheumatic (commonest).
   (2) Traumatic during mitral valvulotomy.
   (3) Infective endocarditis.
   (4) Congenital.
   (5) Associated with collagen diseases, myocardial infraction and cardiomyopathies.

(6) Functional : Associated with left ventricular hypertrophy and dilatation (e.g. hypertension, severe anaemia, myocardial infraction complicated by left ventricular failure etc.)

II. *Symptoms*

1. Breathlessness on exertion ending in cardiac asthma due to left ventricular failure.
2. Palpitation due to an enlarged left ventricle.
3. Features of infective endocarditis e.g. (a) Fever, (b) Pallor, (c) Clubbing,(d)Splenomega -ly, (e) Microscopic haematuria, (f) Embolic episodes.

[Causative organisms are : Streptococcus viridans, Diphtheroids, Heamophilus.

A blood culture for these organisms is mostly positive. Clinically this endocarditis changes the murmur of mitral regurgitation into a musical one the *Seagull Murmur.* A ruptured chordae tendinae or a valve cusp acts as the string of the musical instrument.]

4. Features of congestive cardiac failure.

III. *Signs*

1. Low volume collapsing pulse.
2. Neck veins normal unless there is cardiac failure.
3. Apex shifted down and out, and usually forceful and illsustained.
4. Systolic thrill in the mitral area signifying an organic heart disease.
5. Right ventricular heave (rare).
6. Auscultation–S_1 muffled, S_2 normal S_3 due to left ventricular diastolic overload.

A pansystolic murmur, conducted towards the axilla (but to the base if the regurgitation is posterior cuspal).

IV. *Investigations*

1. Radiology–left ventricular enlargement with a prominent left atrium (Boat Shaped Heart).
2. Echocardiographic–Left atrial enlargement left ventricular enlargement (Echocardio Doppler is useful to foretell gradient of mitral regurgitation).
3. Electrocardiogram–Left axis deviation, left ventricular hypertrophy and rarely p-mitrale.
4. Cardiac catheterisation–Left atrium opacifies during the left ventricular contraction as shown by angiocardiography.

V. *Treatment*

Implantation of a valve prosthesis, under open heart surgery. It may be a cage prosthesis or a disc prosthesis. Also annuloplasty may be done.

VI. *Complications*

1. Acute left ventricular failure.
2. Infective endocarditis.
3. Congestive cardiac failure.
4. Arrhythmias e.g.–(a) Ventricular premature beat (b) Atrial fibrillation (c) Ventricular tachycardia.
5. There may be a giant left atrium with atrial fibrillation and pressure effects.
6. Prone to produce ball valve thrombus–leading to recurrent attacks of syncope.

3. AORTIC STENOSIS

I. *Aetiology*

Mainly congenital (unicuspid of bicuspid) and rheumatic. Atherosclerotic or calcific aortic stenosis

are mostly secondary to rheumatic aortic stenosis. Rest are due to obstructive cardiomyopathy. Rare causes are–SLE, rheumatoid arthritis, Hurler's syndrome.

II. Symptoms Mild or moderate aortic stenosis causes no symptoms.

Severe stenosis usually present with–

1. Angina pectoris (commonest) usually on effort.
2. Attacks of dizziness, blackout or even syncope due to reduced cerebral blood flow resulting from diminished cardiac output. Syncopal attacks may be severe and frequent. These symptoms appear with effort or even with a change of posture.
3. Dyspnoea and other features of left ventricular failure.

III. *Signs*

1. Facies–Dresden china pollar.
2. Cold extremities.
3. Anacrotic pulse, pulsus parvus et tardus.
4. Apex beat–shifted down and out, heaving in character.
5. A thrill (systolic) at the base of the heart conducted to the carotid artery. *Carotid shud der*–thrill in carotid artery with thrill at the base.
6. Auscultation–
   Aortic area : A_2 diminished, single S_2 or a para doxical split. Ejection systolic murmur–diamond shaped.
   Ejection click–due to a sudden opening of the semilunar valve (this sounds like split 1st heart sound).
   S at the mitral area.

IV. *Investigations.*

1. Radiology : Left ventricular hypertrophy, a poststenotic dilatation is common, an aortic valve calcification seen especially on fluoroscopy.
2. Echocardiographic : Left ventricular wall condition (thick enlarged, harmonious or nor har monious). Aortia valve cusps occurs as endease steretione eitheic ication and thickening of valve.
3. Electrocardiogram : Left ventricular hypertrophy usually with strain pattern.

V. Complications :

1. Sudden death due to ventricular fibrillation during physical in severe aortic stenosis; in 3 to 5% of patients during asymptomatic phase.
2. Acute left ventricular failure.
3. Congestive cardiac failure.
4. Infective endocarditis.
5. Arrhythmias (VT and VF).
   The mortality is 9% per year in adults with aortic stenosis.

VI. *Treatment*

Sp must be done heiere serious complication got in front.

1. Transventricular aortic valvotams.
2. Aortic valve prosthesia.
3. Infective endocarditis.
4. Atherosclerotic.
5. Aorta stenosis.
6. Marfan's syndrome.
7. Persistent truncus arteriosus.
8. Ankylosing spondylitis.

4. AORTIC REGURGITATION

I. *Aetiology*

1. Rheumatic,
2. Syphilitic,

3. Traumatic,
4. Infective endocarditis,
5. Aneurysm,
6. Atherosclerotic,
7. Persistent truncus arterious,
8. Marfan's syndrome and
9. Ankylosing spondylitis.

II. *Symptoms*

1. Throbbing sensation all over the body.
2. Palpitations.
3. Breathlessness.
4. Swelling of the body due to congestive cardiac failure.
5. Chest pain.
6. Excessive sweating.

III. *Signs*

1. de Musset's sign–bobbing of the head.
2. Quincke's sign or visible capillary pulsations.
3. Water hammer pulse.
4. Blood pressure–shows wide pulse pressure.
5. Neck–Corrigan's sign.
6. Suprasternal notch pulsation.
7. Pistol shot sound in the femoral artery.
8. Hill's sign : The blood pressure in tne lower limbs>60 mm Hg above that of the upper limb in severe aortic regurgitation.
9. Durozeiz's murmur and Duroziez's sign (i.e. a systolic murmur on proximal compression of the femoral artery and a diastolic murmur on distal compression).
10. Traube's sign is the double sound heard over the femoral artery with each cardiac systole.
11. Apex beat is down and out and forceful and illsustained.
12. A diastolic thrill in the aortic area and the lower left sternal area.

13. Rosenbach's sign is the hepatic pulsation witheach systole.
14. Gerhardt's sign is the pulsation of an enlarged spleen with each systole.
15. Landolfi's sign is the pupillary size changes with each systole.
16. Ascultation–S_1 audible and soft, S_2 (A_2) is loud and is followed by a diastolic decrescendo murmur which is high pitched, soft, blowing in character, heard in the left sternal border S_3 and Austin Flint murmur may be present (in severe regurgitation). S_4 in also often audible.

IV. *Investigations*

1. Radiology–Cardiac enlargement due to left ventricular dilatation. The aortic knuckle is prominent. An aortic valve calcification raises the possibility of a combined aortic stenosis.
2. Electrocardiogram–Left ventricular hypertro phy with tall precordial T waves.
3. Echocardiogram (and Doppler) : Degree pre diction of aortic regurgitation possible; left atrial ventricular dilation; mitral valve might fluttar in high frequency.

V. *Complications*

1. Acute left ventricular failure.
2. Infective endocarditis.
3. Congestive cardiac failure.
4. Cardiac arrhythmias.

VI. *Treatment*

Aortic valve prosthesis.

Protocol for the diagnosis of cardiac valve disorders and assessment of myocardial func tion

(i) History.
(ii) Physical examination.
(iii) Chest X-ray. PA view, left lateral view or other oblique views as may necessary.

(iv) Electrocardiogram
(v) Echocardiogram Doppler
(vi) Carotid pulse tracings especially in aortic valve disorders
(vii) Radionuclide ventriculography with or without echocardiography
(viii) Angiogram
(ix) Phonocardiography, if necessary
(x) Cardiac catheterisation in selected patients with pressure measurements, oxymetry and selective angiocardiography.

Points (vi) to (x) should only be done after serious reflection.

CARDINAL SIGNS OF SOME COMMON CARDIAC DISORDERS

I. Congestive cardiac failure
(1) Tachycardia and gallop rhythm.
(2) Engorged and pulsatile neck veins.
(3) Enlarged and tender liver.
(4) Dependent pitting oedema.

II. *Left ventricular failure*
(1) Gallop rhythm with tachycardia.
(2) Moist sounds at the lung bases.
(3) Pulsus alternans in severe failure.

Outline of treatment in left ventricular failure
(1) Bed rest in propped up decubitus.
(2) Morphine–15 mg IM stat for sedation and to cut off the Herring Breuer reflex. Other opioids e.g. pethidine or pentazocine, may be used. All these may be given IV if urgent *action is necessary.*
(3) O inhalation through a face mask if necessary.
(4) Aminophylline – 0.25 gm IV stat
(5) Furosemide–40-80 mg IV stat.
(6) If there is no history of digitalis in the previous 2 weeks digitalis intravenously followed by oral route.
(7) Antibiotics.

HYPERTENSION

I. *Definition*

This is a clinical condition in which the systolic blood pressure is more than 150 mm Hg and the diastolic one is more than 90 mm Hg in adult individuals.

II. *Types*

PRIMARY

1. Essential bengin (most common) : This is a multifactorial disorder. (If both the while if one of the perents is affected 35% risk of developing hypertension.
2. Essential malignant : Results from the untreated benign form. No organic cause has till date been found for primary hypertension. The possible theories are–

(a) Neurogenic : Due to sympathetic stimulation.
(b) Humoral : Due to liberation of renin resulting from renal ischaemia.
(c) Besides neurohumoral theories, electrolyte disturbances may be the causes in the genesis of primary hypertension.

SECONDARY

The occurs usually below the age of 30 years and an underlying cause is always responsible which may be–

D. Renal :
(a) Acute glomerulonephritis,
(b) Chronic glomerulonephritis,
(c) Chronic pyelonephritis.
(d) Polycystic kidney disease.
(e) Renal tumours.

3. Endocrine :
(a) Thyrotoxicosis.
(b) Cushing syndrome.

(c) Phaeochromocytoma.
(d) Conn's syndrome.

4. Neurological :
   (a) Cerebral tumours.
   (b) Pseudobulbar palsy.
   (c) Bulbar poliomyelitis.
5. Vascular :
   (a) Coarctation of the aorta.
   (b) Renal artery stenosis.
6. Iatrogenic :
   (a) Use of oral contraceptives.
   (b) Prolonged steroid therapy (e.g. in bronchial asthma).
7. Pregnancy : Pre and post eclamptic renal cause.

III. *Symptoms*

Nothing typical : The symptomatic features are mostly psychogenic and follow the diagnosis of hypertension. These are – Headache, insomnia Irritability Lack of concentration, Weakness, Fatigue etc.

These may be due to cerebral artery sclerosis.

IV. *Signs*

1. Flushing of the face 2. Blood pressure; Systolic more than 150 mm Hg. Diastolic more than 90 mm Hg.
2. Initially this is intermittent but graually becomes persistent.
3. Opthalmoscopy reveals retinopathy which may be of the following grades :

Mild hypertension (Grade I) : Diastolic pressure : 90-110 mm Hg — Tortuosity of the 3rd branch of central artery with engorged veins; A : V = 1 : 2 (diam); veins mildly depressed at AV crossing.

Moderate hypertension (Grade II) : Diastolic pressure : 110-130 mm Hg — More tortuosity of the artery resembling cop per wire; there is arterio-venous nipping; A : V = 1 : 3.

Severe hypertension (Grade III) : Diastolic pressure : 130-150 mm Hg	Features of Grade II+Silver wire appearance, vein not visible at AV crossing, A : V=1 : 4 or the arterioles may become fine fibrous cord, vein is dilated distal to AV crossing. Cotton wool exudates present. Flame shaped haemorrhage rarely, Hard exudates.
Malignant hypertension (Grade IV) : Diastolic pressure : above 150 mm Hg	Papilloedema in addition to the above changes.

[Malignant hypertension is defined as marked elevation of blood pressure which is associated with :

(i) A diastolic pressure usually more than 150 mm Hg.
(ii) Persistent or progressive uraemia.
(iii) Papilloedema–confirmatory.]

5. Apex beat : shifted down and out, heaving in character.
6. Accentuated A_2, ejection click, ejection systolic murmur, S_2 paradoxically split and rarely aortic regurgitant murmur.
7. In severe hypertension there is an S_3 due to hypertensive heart disease.
   Hypertensive heart disease may lead to coronary atherosclerosis which in turn may ultimately lead to infraction.
   Hypertensive heart disease may lead to left ventricular failure; this is known as hypertensive heart failure.

Decapitated hypertension is a condition when there is a sudden fall of systolic pressure from, say, 160 mm Hg to 130 mm Hg due to acute left ventricular failure diastolic pressure ramaining at about 110 mm Hg.

Divergent hypertension a condition in which there is *high systolic* and low diastolic blood pressure e.g. in aortic regurgitation, thyrotoxicosis, patent ductus arteriosus etc.

Effect on other systems :

A. Cerebrovascular–Sudden convulsion with unconsciousness, papilloedema. Paresis of the limbs;– these are all due to hypertensive encephalopathy. Cerebral haemorrhage, Cerebral thrombosis etc.

B. Renal manifestations; Chronic renal failure, acute on chronic renal failure. Urine examination reveals– albumin, RBC Pus cells, Blood and granular casts etc.

V. *Investigations*

1. Radiology–Enlargement of the left ventricle and widening of the arch of the aorta.
2. Electrocardiogram–Left ventricular hypertrophy with or without strain pattern.
3. Blood–biochemistry reflects the aetiological factors, or, with end organ damage e.g. those of the kidneys, there will be features of uraemia with an increase of creatinine level and a decreased creatinine clearance.
4. Intravenous pyelography (rapid sequence IVP) and /or retrograde pyelography reveals renal pathology.
5. Special blood tests e.g. estimation of (i) adrenaline and (ii) non-adrenaline–for suspected phaeochro-mocytomas.
6. Urine–Excertion of vanillylmandelic acid (VMA),

metanephrines and catecholamines to confirm phaeochromocytoma.

7. Split renal function test : to reveal renal artery stenosis.
8. Plasma renin activity (PRA) and renal vein renin assay from both sides.

VI. *Complications*

Acute	***Chronic***
1. Cerebral haemorrhage.	1. Angina pectoris.
2. Hypertensive encephalopathy.	2. Congestive cardiac failure
3. Subarachnoid haemorrhage.	3. Coronary artery disease.
4. Acute myocardial infraction	4. Chronic renal failure.
5. Acute left ventricular failure	5. Acute on chronic renal failure.

VII. Management

1. Weight reduction possible?
2. Physical exercise (e.g. regular walking gymnastic, etc.)
3. Change of eating habit–salt reduced (0, 5 gm salt/d)
   –Cholesterol reduced (vegetable oil instead of animal oil etc.
4. Reduction of restriction of alcohol consumption and smoking. If there is still a high blood pressure systolic over 150 mm Hg/elder pts, over 160 mm Hg and diastolic over 100 mm Hg–add.
5. Antihypertensive drug treatment.
   Avoid to rescript more than three different drugs.
   E. g. for step care treatment :
   1 Step : diuretica (mostly elder pts.) of or e.g. B-blocker
   Regular BP measurement, it still hypertension, than,
   2 Step : –diuretica and B-blocker or

– diuretica and calcium antagonist or
– diuretica and ACE inhibitor or
– calcium antagonist and ACE inhibitor

ATTENTION : Never give B-blocker and calcium antagonists! Begain regular BP control, still hypertension present, than

3 Step : –diuretica, B-blocker and ACE inhibitor or – diuretica, calcium antagonists and ACE inhibitor control of BP regularly. There is still the possibility to try other combinations (see table).

Category	example	doses [mg/d]	side effect	contra-indication
a diuretics Thiazid	Hydrochlo-rothiazid	25-100	hyperglycaemia hyperuricemia hypokalemia	DIABETES renal failure, gout
Kalium saving	Amilorid	10-20	hyperkalemia, nausea vomiting diarrhoea	hyperkalemia severe liver-dysfunction, severe kid-neyinsufficiency
	Triam teren	100-200		
Henie-loop acting	Funosemid	20-40 (i.v.); 40-120(p.o.)	hypokalemia loss of Mg^{2+} Ca^{2+}, Cl hyperglycemia, hyperuricemia allergic reaction ototoxic	hypovolemia. renal failure with anuria, severe liver-dysfunction. hypokalemia
	Piretanid	3-9		
	Etacryn acid	50-150		
Aldosteron antagonist	Spirono-lactone	25-100	hyperkalemia gynecomastia impotence amenorrhoe.	renal failure hyperkalemia hypovolemia. natremia
		5-10	hyperkalemia	
B β - blocker	Atenolol (cardiose-lective	1× 50 200	bradicardia bronchospasm hyperglycemia sedation	bronchial asthma. congestive car-diac faiure.

category	example	doses [mg/d]	side effect	contra-indication
	metoprolol (cardioselective)	2-3x50 100	headache vertigo, gastrointestinal dysfunction rebound-effect	bradycardia. heart block shock situation/metabolic acidosis
	Pindolo ($\beta_1+\beta_2$; ISA)	3x5-10		
	Propranol of ($\beta_1+\beta_2$)	2-4x10-80		
c. calcium antagonist	Nifedipin	3x5-20	ventricular fibrillation, tachycardia oedema, headache flush	pregnancy, aortic stenosis, heart failure, hypotension
	Diltiazem	3x60-120		
	Verapamil	3x40-120	bradycardia AV-block ↓inotropie	!never combine with β blocker
d. ACE-inhibitor	Captopril	2+12.5 50	cough. hyperkalemia	renal arterial stenosis (both sides), renal dysfunction. pregnancy, nursing period
	Enalapril	1+5-40		
e. Angiotensin-II inhibitor	Losartan	25-50	vertigo, excessive BP reduction (when combined with diuretics). hyperkalemia	see ACE inhibitor
f.Vasodilator	Dihydralazin	3x12.5 50	angina pectoris, drug induced SLE (long term therapy >200mg/d), headache	! always combine with β -blocker and diuretics → reflactory tachycardia

category	example	doses [mg/d]	side effect	contra-indication
	Prazosin α_1 blocker	0.5-20	orthostatic collaps, vertigo	heart insuff, NYHAIV
g. central acting	clonidim (stimulates central α_2 receptor	3+75-300 µ g/d	bradycardia initial and rebound BP↑, sedation, dry mouth	sick sinus syndrome, bradycardia (Ibe cautious in combining with beta-blocker or Verapamil
	α -Methyl-Dopa (see Clonidin)	500-1000	orthostatic dysregulation, sedetion, Na^+-H_2O-retention	depression, pheochromocytoma, acute liver disease

VII. Management

a) Primary Hypertension :

1. Weight reduction possible?
2. Physical exercise→ regular walking gymnastic, etc.
3. Change of eating hahit→ salt reduced (max. 6g/d) try KCL based salt
   → cholesterol reduced (vegetable oil and food etc.)
   →in diabetes, diet for it
4. Leave hypertension promoting drugs.
5. Reduction or restriction of alcohol, coffee consumption and smoking. If there is still a high blood pressure start.
6. Antihypertensive drug treatment :
   Avoid to rescript more than three different drugs, think that the patient has to take them over a lot

of years perhaps the whole life. Routinious BP control is to be made, if possible by patient self control.

Step care treatment :

1st Step : ***Monotherapy*** with one of the basic drugs

β-blocker	Diuretics	Ca-antagonists	ACE inhibitor	$\alpha_1$blocker

Still no acceptable BP Extend to

2nd Step : **Doubletherapy**

Diuretics	and

β-blocker	Co-antagonists	ACE inhibitor	$\alpha_1$blocker

or

Ca-antagonists	and	β-blocker	ACE-inhibitor

ATTENTION : Never combine β-blocker with Verapamil!

3rd Step : Find out a combination which regulates the individual

hypertension of the patient best, for example ;

Diuretica	and	ACE-inhibitor	and	Ca-antagonists

In case of hypertensive emergencies :

(a) Nitroprussid, 0, 5 to 8, 0 mg/kg/min continous intravenuous (i.v.) ATTENTION ! Blood pressure might come down to fast, intensive BP control necessary.

or (b) Diazoxid 150-300 mg i.v.

or (c) Trmetaphan, 1 to 45 mg/min i.v.

Other measures that are of help : Mannitol infusion in jet or Furosemid i.v. These drugs are rapid acting and have to replaced by a management shown in step 2 or 3 for long term treatment.

Surgical management according to the aetiology. These include–

1. Bilateral adrenalectomy.
2. Removal of the tumour in a case of pheochromocytoma.
3. In coarctation–end to end anastomosis or graft ing.
4. Renal artery garfting in renal artery stenosis.

ACUTE MYOCARDIAL INFRACTION

1. *Incidence*

More in males, below the age of 50 yrs. Beyond that age the incidences are nearly equal in both sexes. Exceptions,– young females, if they suffer from juvenile diabetes, hypertension, hypothyroidism, hyperlipidaemia etc. or if there is a prolonged use of contraceptive pills.

II. *Clinical features*

1. Precordial pain (usually severe) may be–

(a) Retrosternal, radiating to the ulnar aspect of the left arm.
(b) Across the chest radiating to the ulnar aspect of both arms.
(c) Localised at the jaw.
(d) Below the shoulder blades radiating anteriorly.
(e) Epigastric.

Character of the pain–squeezing, constricting, heaviness over the precordium or there may be a sense of impending death.

2. Associated features include–

(a) Profuse perspiration (cold sweat).
(b) Giddiness or even syncope (due to sudden hypotension or associated heart block).
(c) Frank features of cardiac asthma–severe breath-

lessness with ratting sounds in the throat, bilateral moist sounds. In the lungs, gallop rhythm with pulsus alternans.

(d) Severe epigastric pain with vomiting, a sense of dysphagia and giddiness, resembling those of acute grastric perforation, acute heamorrhagic pancreatitis or acute pain of basal pleurisy.

3. May be painless with none of few of the associated features.

III. *Clinical diagnosis*

1. Patient is apprehensive, anxious and restless.
2. Pallor of the face or even ashen gry appearance (due to low cardiac output which may again be due to peripheral circulatory failure).
3. Rarely central cyanosis (if associated with left ventricular failure).
4. Decbitus propped up (if associated with left ventricular failure).
5. Extremities are cold and clammy.
6. Pulse : Usually tachycardia but sometimes bradycardia if associated with heart block or reflex vegal stimulation.

   Rhythm may be irregular due to frequent ventricular premature beats, which is an aminous sign as these may herald ventricular tachycardia and/or fibrillation.
7. Blood pressure : Both systolic and diastolic pressures are low. If systolic pressure is less than 90 mm Hg with cold and clammy extremities with marked cyanosis and oliguria–the condition is known as *cardiogenic shock*, which is a dreadful complication of myocardial infraction. However, sympathetic overactivity may lead to early transient hypertension.

Mechanism of cardiogenic shock–It is due to decreased coronary perfusion and peripheral circulatory failure. When the coronary perfusion is diminished, myocardial dysfunction occurs. This further reduces the perfusion and a vicious cycle sets in. Also peripheral circulatory failure produces splanchnic vasoconstriction giving rise to anoxia of the viscera which liberates lactic acid into the circulation resulting in metabolic acidosis, that is further responsible for the peripheral circulatory failure perpetuating the vicious cycle (Sodi-bi-carb prevents metabolic acidosis).

8. Jugular venous pressure : May be raised if associated with congestive heart failure.
9. Heart sounds : Usually muffled due to myocardial damage and an S_4 is very common and there may be an S_3 if heart failure sets in.
   After 1 to 2 days there may be a to and fro rough sound over the precordium mainly the left 4th intercostal space in the mid-clavicular line or over the pulmonary area. This is the *pericardial friction rub.* This is very common in an anterior wall myocardial infarction but rare in inferior wall infraction.
10. Body temperature within 36 hours the body temperature may rise to 102°F and this is due to a nonspecific reaction to the myocardial necrosis.

IV. *Investigation*

1, **ECG :** One of the basic diagnostic methods with 12 lead ECG.
   Peracute sign : high peak T wave is only seen during AMI (minutes) attack.
   acute sign : ST elevation in infract regions (hours).
   chronic sign : reduction of ST elevation and presence of (days to weeks) pathological Q wave.
   But be aware that ECG signs may be totally absent or minimized. Leads on the opposite of the infrac-

tion site may show ST depression, like in a mirror.

Infract region	Lead change in
anteroseptal	1, II, aVL, $V_1 - V_6$
inferior	II, III, aVF
septal	$V_1 - V_3$
lateral	$V_3 - V_6$.

2. Blood enzymes

(i) Creatinine phosphokinase (CPK) and CPK-MB (isoenzyme) rises after 6–10 hours of infraction.

(ii) Troponin T (and I), myocardial antigens rising earlier then CPK and are more specific, but not everywhere available.

(iii) Aspertal transaminase (AST) and lactte dehydrogenase (IDH) are released 2–4 days after infraction. There are not very specific.

(iv) Total and differential WBC counts : leukocytosis with a marked increase of polymorphs within 36 hours. Not AMI specific.

Mark if two of the following points are present start treatment without losing time !

1. characteristic clinical picture
2. ECG changes
3. characteristic enzyme increase in blood Fibrinolytic therapy

Several drugs are used today, the three most common are noted here.

Drug (Doses)	**Dosis and effect**
Streptokinase	1.5 mil units over 1h, with $t\frac{1}{2}$ 80 min; allergenic and anaphylactic reaction possible, reusing should be avoided. Fast injection causes rapid fall of BP.

Tissue type plasminogen activator (t-PA) or recombinant-PA (rt-PA); Alteplase	10 mg bolus, 50 mg over 1h, then 40 mg over 2h later on. Give always heparin : first as 5000 U bolus then 1000 U/hiv. t ½ 30 min; Due to the selective activity directly on the thrombos location these drugs are less likely to produce systemic co-agulation disturbances.
Drug (Doses)	**Doses and effect**
Anisoylated plasminogen streptokinase activator complex (APSAC) I Anistreplase	30 units over 5 min. t ½ 105 min; like streptokinase allergenic potence

Reperfusing the obstructed vessel, fibrinolytics have got their maximum effect within the first 4 hours. Still after 12-24 hours little benefit can be expected.

Because of the dangerous side effects as

(i) bleeding disorder
(ii) multiple microembolism
(iii) cardiac dyarithmias and
(iv) allergy

you have to follow strict **contraindications.**

1. haemorrhagic diathesis
2. Symptoms of peptic ulcer or GI bleeding
3. recent cerebral stroke
4. recent surgery and especially neurosurgery
5. malignant and uncontrolled hypertension
6. Prolonged cardiopulmonary resuscitation during this presentation.

V. *Management*

This is ideally done by admitting the patient in a coronary care unit (CCU) as early as possible where–

(i) The patient is put in a cardiac bed.

(ii) Oxygen is given through a nasal catheter (or ventimask or if necessary at a high pressure) at the rate of 4–6 litres i.e., 300 to 600 bubbles per minute.

(iii) To remove the anxiety and kill the pain–inj. morphine sulph–15 mg SC or IM to be repeated up to 60 mg or 2mg IV repeated as necessary.

or, Pethidine hydrocloride inj–100 mg stat IM with or without promethazine hydrochloride.

Or, inj, Pentazocine–which has minimum respiratory depressant effect, 40 mg IM. But this drug may increase LV. end diastolic pressure.

Or, Inj Diazepam–10 mg IV.

With this treatment the thrombotic obstracted coronarateria might be opened again. There is the chance to gain the stroken part of muscle back.

(v) Anticoagulant theraphy is started with–

Inj Heparin–10,000 units stat, 7,500 units SC 6 hrly for 48 hours. It prevents thrombo-embolic episodes but never prevents further extenstion of the infraction. Along with the above, phenindione (DINDEVAN)–40 mg tablets 5 tabs stat and I tab BD given orally. Each dose of heparin should be given after determining the bleeding and clotting times.

Dindevan takes 48 hours for its onset of action and so the dose should be regulated by prothrombin index which should be kept within 50%.

Before starting an anticoagulant therapy a through history is taken to exclude any haemorrhagic

disorders the presence of which contraindicates the use of the anticoagulants.

Heparin is withdrawn after 48 hours. Oral anticoagulant therapy is continued for 3-6 weeks and should be withdrawn gradually. A sudden withdrawal may precipitate another attack.

(N.B.–There is no uniform consensus of opinion regarding the merits of anticoagulant therapy in AMI.)

Oral anticoagulant therapy with aspirin acetylsalcylaci 100–160 m/d to lower the risk of a further myocardial infraction.

(vi) B-adrenoceptor blockade with 50 mg atenolol : first intravenous and then a second dosage per os. Reduction of mortality in AMI patients.

In routine practice a 12 lead electrocardiogram is recorded.

The Leads more detailed :

Six limb leads divided into

(i) 3 standard or bipolar limb leads (Edberg leads):

Lead I– indicates potential difference between the left and the right arm.

Lead II– potential difference between the left leg and the right arm.

Lead III– potential difference between left by left arm

(iii) 3 augmented unipolar limb leads :

aVR

aVL

aVF

(V) Complications of AMI e.g. (a) Cardiac arrhythmias, (b) Hypotension, (c) Cardiogenic shock etc. may appear and should be promptly diagnosed and treated detailed discussions of these are beyond

the scope of this book and students are advised to consult larger text-books. IV infusions started with a "Polarising fluid" which consists of (i) One bottle of 5% dextrose solution and to this is added (ii) 2 amp of inj. potassium chloride (20 MEq of K in each amp) and (iii) 15 units soluble insulin.

The rationality of infusing polarising fluid remains controversial, but at least it helps maintaining an IV channel to readily combat the emergency problems of early complications of AMI.

If there is a fall of BP, 24 mg *dexamethasone* (Decadron) is added into the bottle. The drip is continued for about 2 days (or more if necessary) at the rate of 6 to 10 drops per *minute* (or more, depending upon the haemodynamic variables).

In cardiogenic shock, sodi-bi-carb 3.4% 100 ml/day and *dopamine* (intropin) 5-15 μg per kg body weight per minute, both by IV infusion, should be given. Dopamine, however, should never be mixed with any other solution and is given only in 5% dextrose solution.

Ventricular premature beats or ventricular tachycardia should be treated with *lignocatine* 250 mg IV stat given through the drip, but lignocatine may cause further fall of pressure. So DC shock is the treatment of choice, especially with VT and when there is haemodynamic compromise.

If there is a heart block that cannot be combated by steroids and atropine, immediately a bedside floating electrode catheter should be introduced into the right ventricle via a peripheral arm vein and connected with an external *temporary pacemaker.*

If the signs of ventricular failure appear, furosemide 40-80 mg IV stat and inj. *aminophylline* 0.25 gm in 10 ml

distilled water IV are given and repeated if required. The role of digitalis in acute left ventricular failure complicating AMI is debatable, but may be given IV 0.5 mg stat and IV 0.25 mg every 6 hr after that.

Antibiotics may be needed for secondary infections.

Surgery–In pulmonary embolism (one of the commonest causes of death within 7 days) TERNDELENBURG'S OPERATION (pulmonary embolectomy(may be done in addition to the usual conservative measures.

Coronary dilators e.g. nitrates are now-a-days proving useful; 10 mg tab–1/2 tab given sublingually every 2 hours. (vi) Emergency coronary artery surgery are the current modalities of treatment becoming established at sophisticated centres.

(vii) Gradual rehabilitation is attempted as the patient improves; attention is given to the control of diabetes mellitus, obesity, systemic hypertension, hyperlipidaemias, etc. and smoking and consumption of alcohol and caffeine containing beverages are discouraged.

Diet : This should be preferably liquid or semisolid for the first day and then fat restricted and protein rich food with restricted calories are resumed. As the patient improves he should be given solid fat free diet with a calorie value not exceeding 1300 calorie daily. The cooking media should be vegetable oils like soya-bean and sunflower oils. Carbohydrates are to be restricted as it is converted to cholesterol in the body.

If congestive cardiac failure is present, daily salt intake should not exceed 1–1.5 gm.

Other measures include–

1. The patient should be instructed not to smoke for the rest of his life because an excess of nicotine causes coronary vasospasm in addition to the other adverse cardiovascular effects.

2. Alcohol consumption should be stopped but very restricted amounts may be allowed, if at all necessary.

3. Limited physical exercise like walking in empty stomach, playing golf or squash to be advised. No exertion to be done immediately (1 ½ hrs) after meals.

4. Mental stress being one of the most important aetiological factors in coronary artery diseases the patient should be asked to lead an easy life.

ANGINAPECTORIS

This means an attack of central retrosternal discomfort (that may be oppressive, constricting or crushing in nature and not necessarily always a pain) of the chest, caused by a transient myocardial ischaemia, and which comes with exertion, after a heavy meal or during mental excitement and is relieved immediately by rest and or coronary vasodilators e.g. sublingual administration of glyceryl trinitrate. It is almost always caused by atherosclerotic coronary artery disease on which there may be a superimposed coronary artery spasm.

In contrast to the patient with AMI, that of angina pectoris remains calm and quiet and relatively immobile as exertion causes and aggravates the pain and rest classically brings about relief.

Angina decubitus and its management

If the pain comes even at rest but persists for a very short time (5 to 10 mins) it is known as 'ANGINA DECUBITUS' and the management includes rest for 3 weeks and frequent administration of glyceryl trinitrate or isosorbide dinitrate sublingually at intervals of 2 hours.

A short course of anticoagulant therapy is justified to prevent thrombo-embolic phenomenon.

Modern methods of treatment in angina pectoris and

angina decubitus consist of (i) localisation and assessment of the degree of obstruction (s), (ii) followed by coronary artery bypass grafting (CABG) employing a reversed saphenous vein graft, provided the myocardial function remains normal, or, (iii) sometimes a percutaneous transluminal coronary angioplasty (PTCA) may help in dilating the constricted segment. Of late, *nitroglyceine ointment* applied locally to the precordium has proved effective, especially in angina decubitus. IV *nitroglycrine* is also proving helpful in severe cases.

Beta blockers and calcium antagonists are also effective therapeutic against.

Administration of *Carbimazole*–15 to 30 mg daily in divided doses is one of the treatments of persistent angina which helps by reducing the BMR, lessening the need of tissue perfusion and this diminishes the oxygen demand of the myocardium. This is rarely used β adrenergic blockers e.g. *Propranolol* is also used in angina in a dose of 40 to 480 mg daily in 2 to 4 divided doses; the mechanism of action being–1. Reducing the oxygen demand of the cardiac muscle. 2 Slowing of the heart rate and thus preventing the ischaemia and cardiac arrhythmias. 3. Hypotensive effect particularly in hypertensive heart disease.

Sophisticated investigations for ischaemic heart diseases

(i) Coronary angiography.
(ii) Ventriculography.
(iii) Radio nuclide (e.g. thallium 201, technetium 99 m etc) uptake tests. These may be used in exercise testing too.

(iv) Echocardiography.
(v) Systolic Time Intervals (STI) and Apexcardiography.
(vi) Haemodynamic and angiographic measurements–with ballon tipped (Swan-Ganz) catheter introduced into the pulmonary artery. Some of these have been discussed briefly earlier; but a detailed discussion of all remains outside the scope of this book and students are advised to consult text-books of medicine or cardiology for further details.

The most modern treatment of acute Angina with impending acute myocardial infraction are :

1. Sublingual administration of GTN Tablet under tongue.
2. If no relief sublingual administration of Nefedepine to be crushed and under tongue.
3. If there is no peptic ulcer Aspirin (300 mg) to suck.
4. If no relief administration of Cyclomorphine or dimorphine injection sub-cut.
5. Remove the patient to the nearest hospital with an ICCU where urgent administration of I.V. fibronolytic drugs like Streptokinase followed by I.V. Heparin Therapy.
6. If there is no complication and no arrhythmia and heart failure, oral ACE inhibitor and Beta-blocker and oral Aspirin to be continued for six weeks.

APPENDIX TO CHAPTER III
GRADING OF MURMURS

The grading of murmurs, based on their intensities is as follows :

GRADE I–The murmur is very faint, audible only when carefully searched for with a good stethoscope in

a quite room and with the patient holding his breath. Even then it is barely audible.

GRADE II–A faint murmur, still readily recognized.

GRADE III– Prominent, exactly not a loud murmur, but there is no thrill.

GRADE IV–A loud murmur, usually, associated with thrills.

GRADE V–Very loud murmur.

GRADE VI–Exceptionally loud mumur, such that it is audible even with the stethoscope just removed from its contact with the chest; without the stethoscope and with the examiner getting closer to the patient; and also when the chest piece is placed over the head of the patient or any part of the patient's body.

By convention, the murmur is usually recorded as, say, Grade 2/6, 3/6 or 5/6 etc. the denominator indicating the total number of grades.

CHAPTER 3

GASTROINTESTINAL AND URINARY SYSTEMS

The gastrointestinal system should be routinely examined in the following order–(i) the mouth, throat and oesophagus, (ii) the abdomen and (iii) ano-rectal examination.

EXAMINATION OF THE MOUTH

Lips–(a) Look for *herpes simplex*–These are painful vesicles on the outer surface of the lips commonly found in–

(i) Common cold, (ii) Influenza, (iii) Lobar pneumonia, (iv) Weil's disease, (v) Meningococcal meningitis, (vi) Malaria, (vii) After blood transfusion.

The herpes labialis often gives clue to the side and stage of lobar pneumonia e.g. right sided vesicles indicate a right sided lobar pneumonia. The vesicular stage of the herpes may indicate grey hepatisation and the denuded herpes suggests the stage of resolution of pneumonia.

(b) *Cheilosis*–It means cracking of the mucocutaneous junction of the lip. Caused by deficiencies of iron, niacin, riboflavin and pyridoxine or by a sudden change of climate (e.g, excessive cold). When associated with dysphagia in women, post-cricoid carcinoma should be suspected.

(c) *Angular stomatitis* (cheilitis) is the inflammation of the skin at the angle of the mouth and consists of superficial and reddish brown linear ulcers radiating from the angle of the mouth.

Causes : (i) Ill-fitted dentures. (ii) Vitamin deficiencies, especially that of riboflavine, (iii) Starvation. (iv) Iron deficiency anaemia and other debilitating diseases, (v) Occult candidiasis, (vi) Contact dermatitis etc.

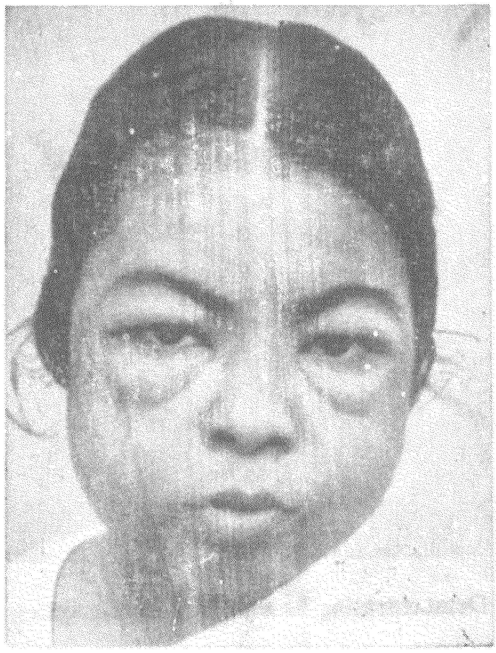


Fig. 4A : This was a case of membranous glomerulonephritis Note the puffiness of the face and especially the Swelling of the eyelids,

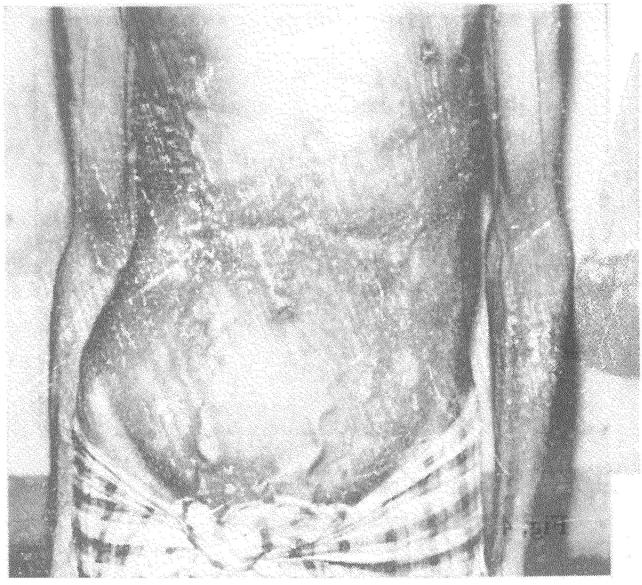


Fig. 4B : Inferior Vena Cava obstruction

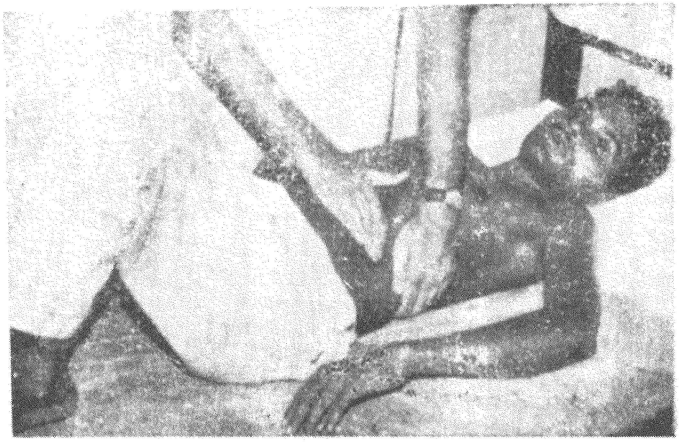


Fig. 4C : Demonstrating how to palpate the spleen

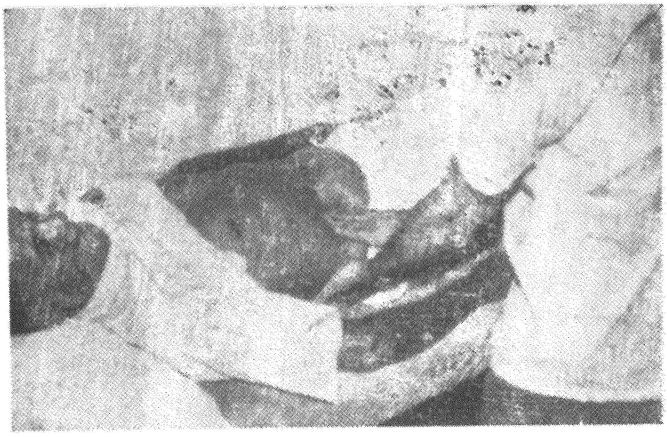


Fig. 4D : Demonstrating how to palpate the kidney bimanualy

(d) *Colour of lip*–The lips may be blue due to cyanosis or methor sulphhaemoglobinaemia.

(e) *Cancrum oris*–gangrenous e.g. in chronic kala-azar.

(f) The mucous surface of the lips should be examined for *aphthous ulcers* and *retention cysts*. The aphtchous ulcers are painful, small and superficial with a whitish base surrounded by a red area of hyperaemia.

Cheek–The cheeks should be inspected with the help of a torch and look for–(i) Pigmentation that may be found in Addison's disease, haemochromatosis, polyposis of the colon (Peutz-Jegher syndrome), chronic arsenical poisoning etc. (ii) Koplik's spot around the opening of the parotid duct, found in measles. (iii) Cancrum oris. (iv) Haemorrhagic spots which may be due to (a) Haemophilia. (b) Purpura. (c) Hereditary haemorrhagic telantgiectasia (Rendu Osler Weber's disease).

Gum–Normally the gum is pink coloured. The lips should be retracted before examining the gum. With gingivitis the gums are not pinkish, bleed easily on slight pressure and may be swollen.

A blue line in the gum is found in lead poisoning. A bluish black or grey line of the gum may be seen in tartar on teeth and may be produced by *bismuth*. A wedge shaped slip of white paper is inserted between the gum and the teeth. The stippled line of lead poisoning will be rendered more distinct but discolouration due to tartar on the teeth will disappear. *Mercury* poisoning may also show as a darkline along the gingival margins.

Accumulation of food debris between the unhealthy gum and the teeth helps in the growth of commensal bacteria as well as the pathogenic organisms leading to the development of *pyorrhoea alveolaris*. The danger of pyorrhoea alveolaris are subsequent developments of : (i) Infective

endocarditis. (ii) Rheumatic carditis, (iii) Aspiration pneumonia. (iv) Premature fall of teeth. Look for any *ulceration* of the gum. The ulcers of the gum are of the following types:

(i) Aphthous ulcers–These start as vesicles and ultimately rupture, are very painful and superficial with greyish edges and self-limiting.

(ii) Ulcers of Vincent's angina also known as acute necotizing gingivitis. Plaut Vincent's infection, trench mouth and fusospirochetosis–These are deep, clean cut ulcers with sharp edges associated with painful, bleeding gums and foulbreath. A swab from these ulcers will show fusiform bacilli (fusobacteria) and spirochaetes Borrelia vincenti) on dark ground illumination. Vincent's gingivostomatitis destroys interdental papillae, and produces halitosis.

(iii) Snail track ulcers of secondary syphilis.

(iv) Big ragged ulcers with or without sloughing may be due to acute leukaemias or agranulocytosis.

(v) Acute herpetic gingivostomatitis caused by herpes simplex virus is rather common in infants and children.

The gum may be *hypertrophied* in–

(a) Epileptics treated with sodium phenytoin, probably due to inhibition of collagen catabolism.

(b) Pregnancy.

(c) Leukaemias particularly acute monocytic leukaemia where there is also gum bleeding.

Lastly palpat for any tumour of the gum (epulis) and for any stone in the salivary gland.

Teeth–Count the number of teeth and note their colour. Normally there are 16 pairs of permanent teeth.

	M	P	C	I	Incisor	Canine	Premolar	Molar
Upper–	3	2	1	2	2	1	2	3
Lower –	3	2	1	2	2	1	2	3

Teeth may be less than normal in number due to–

(1) Traumatic loss, (2) Bad oral hygiene. (3) Extremes of age, (4) Delayed dentition as occurs in hypoparathyroidism and rickets. Teeth may become yellow and mottled with excessive use of tobacco and betels. *Fluorosis* also makes the teeth yellow and mottled. If expectant mothers after the first trimester of pregnancy or children up to 8 years of age are treated with *tetracyclines*, both the deciduous and permanent teeth of the child acquire permanent staining in the form of horizontal bands of yellow or grey colour.

Hutchinson's teeth–characteristic of *congenital syphilis,* the two central upper permanent incisors are broader near the gum than at the crown and there is a semilunar notch at the biting edge. The cross-sections of these incisors look rounded with inward sloping (inverted peg shaped). The lower teeth may close outside the upper ones causing an alteration of bite as is found in *acromegalic* patients with prognathism.

Breath–It is very important to smell the breath of a patient because some diseases give distinctive odour to the breath e.g. (1) Foul smelling (known as halitosis) present in gangrene of lung, lung abscess, bronchiectasis, pyorrhoea alveolaris, chronic pulmonary tuberculosis etc. (2) Sweetish or 'fruity odours' due to presence of acetone in expired air. This is encountered in diabetic ketoacidosis. (3) Garlic odour of breath in bismuth toxicity. (4) Pungent smell after administration of paraldehyde. (5) Mousy smell of hepatic failure. (6) Ammoniacal odour in uraemia.

Tongue–Ask the patient to protrude out the tongue. It may fail to protrude fully if there is a short frenum; known as tongue tie.

Note the following points–

(1) Hydration–(a) The tongue may exhibit minimum of secretion due to severe dehydration, after haemorrhage,

with administration of drugs like morphine or atropine, in mouth breathing, in uveoparotid fever (Heerfordt's syndrome, found in sarcoidosis) etc.

(b) Tongue may be excessively moist in heavy metal poisoning like arsenic, mercury, lead etc. in post-encephalitic Parkinsonism and also after administration of lozenges.

(2) Tremor–Fine tremors of the tongue is found in anxiety neurosis, thyrotoxicosis and chronic alcoholism. Coarse tremors of the tongue is characteristic of Parkinsonism. Coarse forward and backward movement of the tongue (also known as "trombone tongue") is characteristic of dementia paralytica.

(3) An apparent deviation of the tongue occurs in unilateral seventh nerve palsy. The tongue is deviated to the opposite side of the lesion in a case of upper motor neurone disease and to the same side of the lesion in lower motor neurone disease.

(4) Spasticity–characteristic of pseudobulbar palsy.

(5) Fasciculations, wasting and deep grooves or fissures in the tongue are observed in lesions of the hypoglossal nerve or nuclei e.g. progressive bulbar palsy or amyotrophic lateral sclerosis. This is lingual hemiatrophy; the tip deviates towards the side of lesion and the median raphe is concave towards the side of the lesion (see chapter 3 of part II).

(6) Colour–The tongue looks pale in anaemia. It may be blue in *central cyanosis*. The undersurface is examined to detect *jaundice*. An angry looking tongue with sores is characteristic of *niacin deficiency*. In chronic-superficial glossitis due to syphilis (syphilitic leucoplakia) the tongue surface is full of smooth and glazing patches. The tongue in typhoid fever is characterised by dryness of the surface with a central brown fur and red edges.

(7) Conditions of the papillae–A smooth of bald tongue due to generalised atrophy of the papillae is common in iron deficiency anaemia, sprue, pellagra, pernicious anaemia etc.

(8) Ulcers–A snail track ulcer over the dorsum of the tongue is highly characteristic of *secondary syphilis*. White patches with or without ulcerations are present in chronic superficial glossitis, Congenital fissures of the tongue and chronic superficial glossitis are differentiated clinically by the presence of normal papillae in the former.

The tongue may be ulcerated at the margins due to illfitted sharp dentures, short frenum etc. *Tubercular ulcers* are usually situated on the dorsum of the tongue whereas *malignant ulcers* can develop anywhere especially on the already existing leucoplakias. *Frenal ulcers* are frequently found in whooping cough and is occasionally due to persistent coughing.

(9) Size –The tongue may be large (macroglossia) or small (microglossia), Macroglossia is associated with acromegaly myxoedema, cretinism, primary amyloidosis, mongolism etc. Microglossia or glossal atrophy is seen in cerebral diplegia, pseudobulbar palsy and hypoglossal nuclear lesions as in amyotrophic lateral sclerosis, syringomyelia. etc.

(10) Taste sensation–Vide examination of the nervous system.

Palate –Ask the patient to open his mouth and see the colour, degree of the arch and presence of cleft, if any. Note also the movements by asking the patient to say 'Ah'. It does not move in palatal palsy as in diphtheritic polyneuritis or bulbar paralysis. The degree of anaemia and jaundice can be assessed by the colour of the soft palate. The high arched palate may be found in Marfan's syndrome, mon-

golism or in association with some congenital heart diseases (see Sick Children). Congenital cleft in the palate is an occasional finding. Pinhead petechial haemorrhagic spots are often found over the hard palate in glandular fever. In herpes zoster of the maxillary division of the trigeminal nerve, vesicles are found on one side of the hard palate which progress to painful oral ulcers.

Tonsils – These are usually slightly enlarged up to the age of 12 years. The septic tonsils are very big and red with liberation of pus on squeezing. The septic tonsils are the predisposing factors of rheumatic fever and acute glomerulonephritis.

Tonsils should be inspected after depressing the tongue with the help of a tongue spatula.

The patches *of faucial diptheria* are greyish white membranes confined to the tonsils and they cannot be easily removed. Oozing of blood occurs after removal of the membrane.

In thrush i.e. candidiasis–the plaques are curdy white and can be easily removed without any bleeding.

The white or yellowish grey patches of follicular tonsillitis are easily removed and on removal leave behind a sound surface which does not bleed.

Fauces –These are examined with the help of a torch after depressing the tongue with a tongue spatula. Note the colour, texture and any discharge from the mucosa. The posterior pharyngeal wall looks congested and granular in heavy smokers and orators. Look for cherry red spots on the pillars of the oropharynx, diagnostic or infectious mononucleosis.

Pharynx–Normally on its surface small adenoid swellings are seen–these are much increased and the mucous membrane of the pharynx become congested in chronic

pharyngitis while it may bulge inwards in retro-pharyngeal abscesses.

EXAMINATION OF THE OESOPHAGUS

Ask the patient to swallow water as well as solid food and note if there is any difficulty in swallowing.

The oesophagus is 10 inches long and extends from the level of the cricoid cartilage up to the 9th thoracic spine. There are two sphincters in the oesophagus–pharyngooesophageal and oesophagocardiac. A difficulty in swallowing is known as *dysphagia*. The dysphagia may be due to lesions in the bulb (e.g. bulbar palsy) or due to any local pathology, e.g. due to poor salivation. Paresis of the tongue or painful conditions of the mouth or pharynx. Besides, dysphagia may be due to–(1) Involvement of the *upper part* of the oesophagus–(a) Kelly-Patterson syndrome or Plummer-Vinson syndrome–This type is also known as sideropenic dysphagia. In oesophageal dysphagia the feeling is as if the food is lodged at the end of the throat or behind the sternum. *Mechanism*–Iron in the body is stored up in the epithelial cells. In severe iron deficiency states, the iron stored in the epithelial cells of the mucous membrane of oesophagus is used up to maintain haemoporesis as a last resort. The epithelium is thereby denuded and the Auerbach's plexus becomes exposed to irritants and food particles. Reflex spasm occurs during the passage of food bolus.

(b) Benign strictures e.g. due to ingestion of corrosives.

(c) Pressure from outside e.g. carcinoma of the thyroid. retropharyngeal abscess, caries spine etc.

(d) Pseudobulbar palsy.

(e) Carcinoma oesophagus, upper part.

(2) *The middle part* of the oesophagus may be narrowed by the pressure of bronchogenic carcinoma, aneurysm of

the aorta, congenital aortic ring, enlarged left atrium (in mitral stenosis), caries spine etc. The lumen is also narrowed by malignant strictures of the oesophagus, carcinoma oesophagus middle third etc.

(3) *The Lower part* of the oesophagus plays an important part in the etiology of dysphagia as it is the site for cardiospasm, carcinoma oesophagus, scleroderma, hiatus hernia etc.

Normally the food bolus takes 6 seconds to reach the stomach after deglutition.

N.B. *Dysphagia lusoria* is a very rare condition due to compression of the oesophagus by an aberrant right subclavian artery.

Besides the routine clinical examination of the relevant systems, a routine blood examination, a straight X-ray of chest, screening of the chest, Ba-swallow X-ray of the oesophagus and oesophagoscopy and biopsy must be done in order to find out the cause of dysphagia.

EXAMINATION OF THE ABDOMEN

Anatomical subdivisions and landmarks–For the purpose of localising the sings and symptoms, structures and organs within the abdomen in relation to the anterior abdominal wall; the abdomen is subdivided into *nine regions* by two horizontal and two vertical lines or planes.

The vertical line on either side is drawn vertically upwards from the midpoint of the line joining the anterior superior iliac spine and the symphisis pubis or the midinguinal point. The upper horizontal line lies at the level of the lowest points of the chest wall i.e. joining the tips of the tenth costal cartilages on either side. The lower horizontal plane or line lies at the level of the highest points of the iliac crests as seen from the front.

The upper section is subdivided into the *right* and the *left hypochondric* and *epigastric* regions; middle section into *umbilical* and *right* and *left* lumber regions and the lower section into *hypogastrium (or suprapubic region) and right* and *left iliac* regions.

The intraabdominal structures and organs in relation to these arbitrary subdivisions should be kept in mind and are as follows–

(1) *Right hypochondrium*–right lobe of the liver, gall bladder, hepatic flexure of the colon, upper part of the right kidney and the right suprarenal gland.

(2) *Epigastrium pylorus of the stomach*– a part of the liver; the pancreas, the aorta and the duodenum.

(3) *Left hypochondrium*–the spleen, the tail of the pancreas, the splenic flexure of the colon the upper part of the left kidney, the left suprarenal gland and a part of the stomach.

(4) *Right lumber region*–the lower part of the right kidney, the ascending colon, parts of the duodenum and the jejunum.

(5) *Umbilical region*–the omentum, the transverse colon, parts of the jejunum and the ileum.

(6) *Left lumbar*–the lower part of the left kidney, the descending colon, parts of the jejunum and the ileum.

(7) *Right iliac*–the lower end of the ileum, the caecum, the appendix, right ureter, the right ovary in the female and the right spermatic cord in the male.

(8) *Hypogastrium*–the urinary bladder, the enlarged or gravid uterus and coils of the ileum.

(9) *Left iliac*–the sigmoid colon, the left ureter, the left ovary in the female and the left spermatic cord in the male.

The above anatomical recapitulations will help the student of medicine to correlate symptoms and signs in rela-

tion to the regions of the abdominal wall, with the organs and structures underneath. In hiatus hernia a portion of' the stomach enters into the thoracic cavity due to a laxity of the oesophageal hiatus of the diaphragm. It occurs usually in obeses subjects aggravated by a raised intraabdominal pressure (e.g. pregnancy) and kyphoscoliosis. Symptoms are mainly due to acid reflux. Heart burn on stooping or lying down, lower sternal pain, dysphagia, haemorrhage and pallor are the main symptoms.

INSPECTION

The patient should be lie flat on his back, quite straight. The legs are extended. Extended, the arms should lie parallel to the body. A broad daylight is preferable and inspect the abdomen from the foot end of the bed. A systematic examination is to be done following these guidelines–

(i) The general contour of the abdomen should be first described--whether normal, scaphoid or distended. The *scaphoid* abdomen is commonly found in cases of undernutrition, tuberculous peritonitis, extreme degrees of dehydration etc.

The abdomen may be distended due to the presence of fluid in the peritoneal cavity, pregnancy, acute intestinal obstruction, intraabdominal tumours, obesity, full bladder, acute dilatation of the stomach etc. The flanks appear full if there is free fluid in the peritoneal cavity. The localised swellings should be inspected in relation to the conventional areas of the abdomen.

(ii) Inspect the general condition of the skin for any striae or excessive *shininess* or pigmentation at the creases. *White striae* are commonly met with when an obese person suddenly loses weight or in women following pregnancy, after paracentesis of the abdomen in a case of ascites, thyrotoxicosis, diabetes mellitus etc.

Purple striae are characteristic of Cushing's syndrome and prolonged corticosteroid therapy. After repeated pregnancies broad silvery lines called striae gravidarum are seen in the abdominal wall.

(iii) Observe for any alteration in the position of the umbilicus, whether it is inverted to everted, whether there is any bluish discolouration or any sinus over it.

A blue discolouration around the umbilicus is known as *Cullen's sign* and is due to extravasated blood coming forwards from the retroperitoneal space and the sign is seen in cases of ruptured kidney, leaking abdominal aneurysm, acute pancreatitis (which also causes a green discolouration in the loins–*Grey-Turner's sign*) and occasionally in ruptured ectopic gestation. An inverted umbilicus may be found in obesity and in bowel obstructions. An everted umbilicus is found in any conditions giving rise to increased intra-abdominal tension as occurs with an adynamic bowel e.g. Hirschprung's disease in which the abdomen becomes enormously tense and tympanitic with umbilical eversion and marked widening of the subcostal angle.

The umbilical sinuses are produced as a result of malignant or tuberculous peritonitis, Meckel's diverticulum and persistent urachus. The black rash of acanthosis nigricans may be seen around the umbilicus in addition to axillae, neck, groins and nipples and is associated with intraabdominal adenocarcinomas in nonobese adults in the majority of cases.

Distended veins around the umbilicus (caput medusae) are characteristic of portal hypertension syndrome. It may also be found if there is a persistent patency of recanalisation of the umbilical vein. A red, raw angry looking tissue may at times be seen at the umbilicus which might be due to,

(i) chronic infective granuloma of the umbilicus, (ii) umbilical adenoma or Raspberry tumour, or a (iii) secondary carcinoma (primary being situated in the stomach, colon, ovary or breast).

An umbilical abscess or nodule may be a clue to an obscure abdominal pain, though the lump itself is painless usually, e.g. that due to chronic abdominal infection like tuberculosis and pneumococcal peritonitis.

Endometriosis of the umbilicus also look raw and red but it bleeds at each menstrual cycle.

(iv) Note the movement of the abdomen with each respiration. Normally the abdomen bulges during inspiration and goes back during expiration, the order being reversed in a diaphragmatic palsy. The movement may be diminished as a whole in perforative peritonitis and tuberculous peritonitis.

(v) Look for visible peristalsis and pulsations, if any. A left to right visible peristalsis indicates *reflex pylorospasm* or *organic pyloric stenosis.* Pylorospasm is produced by inflammation of the 1st and 2nd parts of duodenum or of the gall bladder as they are supplied by the 9th thoracic segment. Organic pyloric stenosis may be produced by fibrosis in chronic duodenal or peptic ulcer diseases, carcinoma of the stomach affecting the pyloric antrum or may be due to congenital hypertrophic pyloric stenosis. If the stomach is grossly, dilated, a visible peristalsis will be seen passing down to the suprapubic region and then again ascending to the right side of epigastrium. When dilated to such an extent, the stomach may hold even two litres of fluid–and on shaking the abdomen a splashing noise is produced. This is the *succusion splash.*

Obstruction of the *transverse colon* is diagnosed by distension of the midpart of the abdomen and a right to left

visible peristalsis. A visible peristalsis in the middle part of the abdomen around the umbilicus in a zigzag fashion is diagnostic of *small intestinal obstruction.*

An *epigastric pulsation* may be visible in right ventricular hypertrophy, a tumour overlying the abdominal aorta, aneurysm of the abdominal aorta and may be present normally in thin built persons.

(vi) Superficial veins are engorged in portal hypertension and inferior vena caval obstruction. The engorgement can be unmasked by asking the patient to sit up and to hold the breath in deep inspiration or by vigorous coughing.

Portal vein obstruction	***Inferior vena cava obstruction***
1. Direction of flow is away from the umbilicus.	1. Direction of flow of the veins below the umbilicus is towards the superior vena cava i.e., towards the umbilicus.
2. Ascities precedes oedema.	2. Oedema precedes ascites.
3. Spleen is enlarged.	3. Spleen is not enlarged.
4. Varicosity is present in the paraumbilical vein (Cruveilheir-Baumgarten syndrome)	4. Varicosity affects the veins of the lower limbs especially those of the calfmuscles.
5. Haematemesis from rupture of the oesophageal varices and bleeding per anum from the haemorrhoids are common.	5. None of these occur.
6. Portal venous pressure is high.	6. Femoral venous pressure is high.

Next look for any hernia that may be–(a) inguinal on one or both sides (b) umbilical or paraumbilical (c) epigastric (small hernia due to extrusion of portions of the extraperitoneal fat) (d) incisional, protruding through an incision site of post-operation or (e) femoral hernia.

PALPATION

The patient lies on his back with the head and shoulder's supported by a pillow and the knees drawn up to relax the abdominal muscles. The clinician stands or sits on a tool to the right of the patient.

Warm your hands by rubbing each other if they are cold. Gently place the right hand first over the left iliac fossa and palpate it. Subsequently pulpate the left lumber. Left hypochondrium epigastrium, right hypochondrium, right lumbar, right iliac fossa hypogastrium and umbilical regions. Palpation should always be done by the flat of the hand and finger movements must be gentle and from the metacarpophalangeal joints. *Superficial palpation* should be done to get the following informations :

(1) *Consistency or feel of the abdomen*–The normal feel of the abdomen is elastic. Rigidity is a sign of peritonitis or any localised infection. Doughy feel is obtained in tuberculous peritonitis. Abdominal guarding is due to contraction of the abdominal muscles. It occurs reflexly as a part of a defense (protective) mechanism which may be localised (e.g. over an inflamed organ) or generalised (as in peritonitis).

(2) *Tenderness*–It indicates an inflammation of the peritoneum or a stretching of the capsule of intra-abdominal organs as a result of inflammation or enlargement. Ask the patient to show the site of pain by his index finger (*Pointing sign*).

In peptic ulcer, particularly in chronic duodenal ulcer, the

tenderness corresponds with the area shown by the pointing finger.

(3) *Any Localised lump*– If it is superficial, do the rising test;–ask the patient to raise the head from the bed and see whether the lump becomes more superficial (perietal (swelling) or disappears (intra-abdominal swelling). During superficial palpation try to locate the lump in relation to the area of the abdomen.

4) *Fluid thrill*–The patient lies flat on the bed with thighs flexed. The ulnar border of the right hand of the patient is placed vertically on the middle to prevent transmission of vibrations by the abdominal wall. Place your left palm on one flank and sharply tap the other flank with the right hand. If there is any enlargement of a local organ tapping should be done from that side for better conduction. To have a fluid thrill there must be at least 2 litres fluid in the peritoneal cavity under tension. Fallacy of the test :–It may be positive even in conditions where there is no free fluid in the peritoneal cavity such as obesity, ovarian cyst, encysted ascites and also in paralytic ileus. In ascites the bulging is mainly lateral whereas in an ovarian tumour it is an anteroposterior bulging of the abdominal wall. The dullness in the ovarian tumour is central and does not change with the position of the patient.

(5) *Divarication of the recti*– Sometimes even in healthy individuals separation of the rectus muscles are found to produce a wide gap, the abdominal viscera being palpable distinctly, only separated by a thin abdominal wall. This is called the divarication of the rectus muscles and is common in those with chronically distended abdomen e.g. in long standing huge ascites.

(6) *Direction of blood flow in prominent abdominal veins*– It is examined if the abdominal veins are prominent. A promi-

nent abdominal vein is selected and made more prominent by making the patient sit up and cough, if possible. Standing on the right side of the patient place the index fingers of both the right and the left hands on the vein side by side; the left index finger being placed above that of the right. Empty a portion of the vein by milking the two fingers in two directions i.e., the left index finger drawn upwards and the right downwards. Each end of the emptied vein is sealed with the pressure of the finger. One of the fingers is then removed and the rate and rapidity at which the vein fills up is noted. The same procedure is repeated, removing the finger at the other end. The rate and rapidity of filling of the vein indicates the direction of the blood flow. The direction of the flow of blood in prominent abdominal veins in portal venous obstruction and in inferior vena cava obstruction has already been discussed.

Deep Palpation

Now the abdomen is palpated more deeply with the flat of the hand as well as with the fingers. The patient should be asked to breath quietly with the head turned to the left side. During palpation of the liver, spleen and the kidneys, the patient should be asked to take deep breaths by mouth.

(i) Palpate the different areas for detection of any mass. The organs or the portions thereof that occupy the different anatomical regions have been discussed earlier.

If a mass is felt, the points to be described are – *site, size, shape, consistency, margin; surface, movement with respiration, pulsatile or not, ballotable or not, tenderness, condition of the overlying skin etc.*

(ii) During each expiratory phase of respiration, gradually apply pressure on the abdomen and then suddenly take off the hands and look for an expression of agony in the patient's face, if there be any. This is known as *rebound*

tenderness found in peritonitis from any cause viz, appendicitis.

Sometimes a sharp pressure over the left iliac fossa elicits a pain over the appendix due to pressure exerted by gas on the wall of the inflammed caecum. This is known as *Rovsing's sign* found in acute appendicitis.

(iii) *Palpation of the liver*

Sit by the side of the patient and place the two hands side by side in the right subcostal region (Fig 4-2). The hands should lie flat on the abdomen lateral to the rectus abdominis, fingers pointing towards the ribs. If any resistance is encountered, the hands should be moved down-

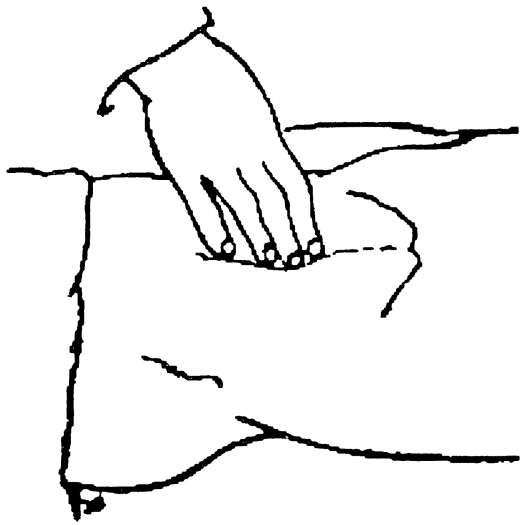


Fig. 4-1 · *Palpation of the liver–Conventional mathod*

wards till there is no resistance, The patient is then asked to inspirate deeply and at the height of inspiration, the fingers are pressed firmly upwards and inwards. A palpable liver is felt as a sharp regular border riding beneath the

fingers. The whole process is repeated from the lateral to the medial regions to find out the edge of the liver.

The liver can also be palpated by the conventional method (Fig 4-1). Ask the patient to turn his head to the left and breath regularly. Place the right hand firmly and flatly over the right iliac fossa and slightly press it inwards and upwards during the phase of inspiration. The radial border of the right hand should be kept parallel to the lower border of the liver and on the outer side of the rectus muscle to avoid its upper septum. Keep the hand steady when the patient takes a deep breath by mouth. In this way go on palpating upwards. At the height of deep inspiration, the tip and the radial margin of the index finger should slip over the lower border of the liver, if it is palpable. Trial is made at different levels before concluding that the edge cannot be felt.

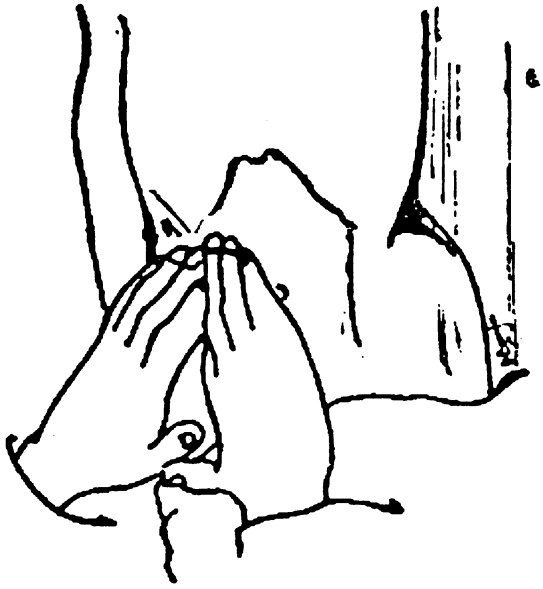


Fig. 4-2 : Palpation of the liver–Better method

The edge of the liver can be felt in normal children and *emphysema*. The enlargement of the liver in the epigastrium should be noted in the usual way as a routine. A simple anatomical variation of the liver, the Riedel's lobe, is a downward tongue-like projection from the right lobe and is more-frequent in women;–occasionally it gives rise to diagnostic difficulty. It is usually freely mobile and may be mistaken for a movable kidney or if situated closer to the midline, for a gall bladder lump, It is usually palpable in the right upper quadrant of the abdomen. If the liver is palpable, note–

(a) The degree of enlargement expressed by the number of fingers placed between the costal arch and the lower border of the liver or by the number

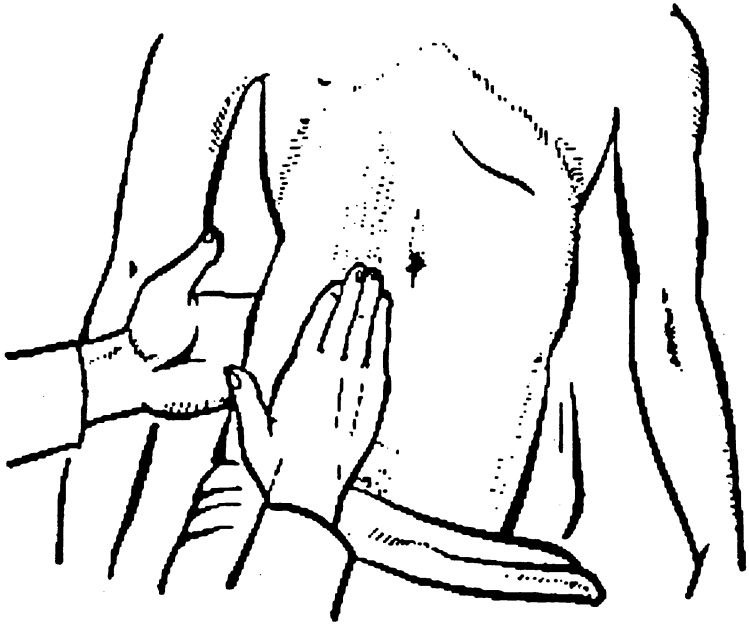


Fig. 4-3 : Bimanual palpation of the liver

of inches or centimeters below the costal margin up to the lower border of the liver. Note that a *palpable liver* is not necessarily an enlarged one; to confirm the latter the upper border of the liver dullness should be detected.

(b) The consistency–This may be soft, firm or hard. An enlarged liver of soft consistency is found in congestive cardiac failure, fatty liver, infective hepatitis, acute malaria and early stages of amyloid disease.–

A *firm* liver is due to chronic kala-azar, Hodgkin's disease, infective hepatitis, Weil's disease, amoebic hepatitis, early stage of cirrhosis, intrahepatic cholangitis etc.

A *hard* liver is characteristic of carcinoma and secondary deposits in the liver; but may also be seen in chronic malaria, chronic myeloid leukaemia, cirrhosis of the liver etc.

(c) The surface–A *smooth* surface of the enlarged liver is found in congestive cardiac failure, chronic malaria, kala-azar, infective hepatitis, amoebic hepatitis, Weils' disease, chronic myeloid leukaemia etc. *Finely irregular* surface is characteristic of portal cirrhosis in the early stage. A grossly *irregular* liver with nodules is a clinical finding in post-necrotic cirrhosis, carcinoma of the liver, secondary deposits in the liver and gumma liver.

(d) Any tenderness–This is due to stretching of the capsule because of enlargement of the liver or an inflammation causing perihepatitis. A tender liver is commonly seen in congestive cardiac failure, amoebic hepatitis, infective hepatitis, Weil's disease and occasionally in carcinoma of the liver.

(e) The margins–A hard or a firm liver usually shows a sharp margin whereas a soft liver has a rounded margin.

(f) Any pulsation–Sitting in a low chair beside the patient place the right hand on the anterior abdominal wall over the liver and the left hand over the costal arch in the back (Fig 4-3). Now ask the patient to hold breath after deep inspiration. Note if there is any *expansile pulsaion* and also decide whether it is systolic or presystolic. Systolic and presystolic pulsation of the liver are the characteristics of tricuspid incompetence and tricuspid stenosis. respectively. Haemangioma of the liver is also pulsatile, Hepatic pulsations may be transmitted from an enlarged right ventricle or from the aorta in thin built subjects, but then they are not expansile.

(g) The movements with respiration–Normally the liver moves 1 to 3 cm downwards with deep inspiration and may be particularly conspicuous in athletes and singers.

(h) The upper border of the liver dullness–This is mappd out by percussing the chest downwards along the right midclavicular line. Find out the lower edge by light percussion moving upwards from the umbilicus towards the costal margin. A *small liver is clinically* detected by this method only. The upper border of the liver dullness may be *obliterated* or *lowered* in (a) emphysema, (b) right sided pneumothorax, (c) perforated peptic ulcer, due to accumulation of gas under the diaphragm, (d) cirrhosis of the liver, due to shrinkage, (e) pneumoperitoneum, (f) Hirschprung's disease etc.

The upper border of the liver dullness may be raised by (a) amoebic liver abscess, (b) subdiaphragmatic abscess, (c) basal pneumonia and (d) right sided pleural effusion. On deep inspiration the upper border of the liver dullness goes down in the diseases mentioned above except in pleural effusion where it remains unchanged.

N.B.– In presence of huge ascites the liver is palpated by the method of *dipping* i.e., the tip of the fingers are sharply and quickly but gently thrust into the abdomen to displace the fluid and feel for the edge of the liver.

Enlargement of the liver (hepatomegaly), may be mild, moderate, or huge.

Causes of (*mild hepatomegaly*) (1-2 fingers) are : (a) infective hepatitis, (b) early congestive cardiac failure, (c) acute malaria and kala-azar, (d) serum hepatitis, (e) ascending cholangiohepatitis, (f) amyloidosis, (g) septicaemia or pyaemia. (h) haemolytic anaemias etc.

Moderate hepatomegaly (2-5 fingers)–Found in chronic malaria, chronic kala-azar post-necrotic cirrhosis, pre-cirrhotic liver, amoebic liver abscess, chronic myeloid leukaemia, Hodgkin's disease, hydatid cyst of the liver and acquired haemolytic anaemia.

Huge hepatomegaly (more than 5 fingers)–Found in chronic malaria, chronic kala-azar, post-necrotic cirrhosis, carcinoma of the liver, chronic myeloid leukaemia and myelosclerosis.

Jaundice associated with hepatomegaly occurs in infective hepatitis, ascending cholangic-hepatitis,

haemolytic anaemias, carcinoma of the liver, Weil's disease, chronic malaria and post-necrotic cirrhosis with hepatocellular failure.

The combination of *hepatomegaly* and *ascites* may be due to hepatic cirrhosis carcinoma of the liver, cirrhosis of the liver with tuberculous peritonitis, chronic myeloid leukaemia, abdominal Hodgkin etc. *Budd-Chiari syndrome* causes acute tender gross hepatomegaly with intractable severe ascites

Lymphadenopathy with hepatomegaly occurs in Hodgkin's disease, chronic lymphatic leukaemia, infective hepatitis and many other conditions where there is generalised lymphadenopathy (see chapter I). The liver is moderately enlarged and associated with *rise of temperature* in–(i) infective hepatitis, (ii) amoebic hepatitis and amoebic liver abscess, (iii) acute malaria, (iv) acute kala-azar. (v) Hodgkin's disease, (vi) Weil's disease, (vii) septicaemia and pyaemia, (viii) hydatid cyst of the liver when secondarily infected etc.

Predominant enlargement of the left lobe of the liver might be due to (i) liver abscess (amoebic), (ii) primary hepatoma, (iii) secondary deposits or (iv) hepatic syphilis (gumma).

The left lobe may be severely *atrophied* and is not uncommonly detected at postmortem, the usual cause being interference with the left branch of the portal vein when the degeneration occurs at birth and atrophy persists into the adult life. Rare causes in later life are–compressions of the left hepatic duct or the left branch of the hepatic artery or the left branch of the portal vein by malignant diseases.

It must be remembered that the palpable liver may occasionally be due simply to a downward displacement of the liver without any hepatomegaly and this might be caused by a right sided massive pleural effusion, empyema or pneumothorax; pulmonary emphysema and severe kyphoscoliosis.

N.B.–A liver palpable below the umbilicus is commonly caused by (i) malignant deposits, (ii) polycystic disease, (iii) Hodgkin's disease, (iv) amyloidosis, (v) congestive heart failure, (vi) gross fatty change etc. Rapid change of the liver size may occur with–(i) correction of congestive heart failure, (ii) relief of cholestatic jaundice, (iii) control of severe diabetes, (iv) control of fatty liver etc.

In hepatopsis or wandering liver the organ leaves its normal situation and can be moved laterally or rotated manually about a horizontal axis–patients usually present with a dragging pain and heaviness in the hepatic region.

(iv) *Gall bladder*

This pear shaped saccular organ is situated in a fossa on the visceral surface of the liver. It is about three inches long and weights about $1^1/_2$ ounces. Its fundus is rounded and projects beyond the inferior margin of the liver. At the level of the tip of the ninth costal cartilage and the outer border of the right rectus muscle the gall bladder comes in contact with the anterior abdominal wall.

The organ is felt as a pear shaped, smooth, tense swelling projecting beneath the right costal margin in the direction of the umbilicus only when it is distended. A pyriform swelling situated just outside the right rectus muscle, moving freely from

side to side around a point opposite the 9th costal cartilage and moving freely with respiration is the classical feature of a gall bladder swelling. In order to palpate the organ, the patient is asked to take a deep breath and at the same time the hand is moved upwards under the costal margin. During palpation note for the gall bladder tenderness and Murphy's sign.

Murphy's sign–If a continuous gentle pressure is exerted over the right hypochondrium while the patient takes a deep breath, there will be a catch in the breath at the height of inspiration. To elicit the Murphy's sign place your hand over the right costal margin with the thumb resting at the junction of the tip of the ninth costal cartilage and the lateral border of the right rectus muscle. The

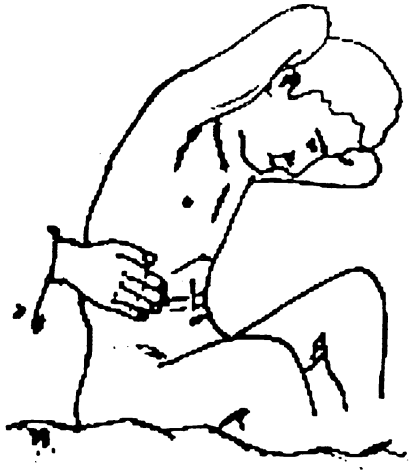


Fig. 4-4 : Elicitation of Murphy's sing.

patient is then asked to take a deep inspiration when the descent of the diaphragm causes the

gall bladder to strike against the examiner's thumb and just at that moment, the patient feels a sharp pain and there is a catch in the breath before the zenith of inspiration. This is *Moynihan's* method of eliciting the Murphy's sign.

In some cases of acute cholecystitis subcutaneous oedema may be demonstrated by carefully comparing the appearances of the skin and the subcutaneous tissues above the tip of the 8th and the 9th ribs on the right side with that of the left side. This right sided subcutaneous oedema is called *Leake's oedema test.*

Sometimes in acute inflammation of the gall bladder an area of hyperaesthesia may be demonstrated between the 9th and the 11th ribs posteriorly on the right side–this is the *Boas' sign.* A distended gall bladder in the presence of jaundice is probably not due to a gall stone impacted in the common bile duct; in cholelithiasis, previous episodes of cholecystitis have already made the gall bladder fibrotic and small which, therefore, cannot distend. This is *Courvoisier's law.* In such cases the cause of the palpable gall bladder along with obstructive jaundice is probably carcinoma of the head of the pancreas.

An enlarged gall bladder had to be distinguished from a visceroptotic right kidney. The latter can be displaced towards the pelvis and has to resonant colon anteriorly.

(v) *Spleen*

The spleen lies behind and below the 9th, 10th and 11th ribs with its long axis along the direction of the 10th rib. Its posterosuperior end lies $1^1/_2$ inches lateral to the 10th thoracic spine and its anteroinferior end extends anterioly up to the midaxillary line. The spleen moves with respiration

and very rarely with deep inspiration may be normally palpable. It beomes palpable when it enlarges to about thrice or more its normal size.

Palpation of the spleen : Preliminary preparation of the patient is the same as that during palpation of the liver. The examiner should stand on the right side of the patient. Ask the patient to take deep breaths by mouth. Place the left hand over the edge of the left costal margin (i.e., over the 9th, 10th and 11th ribs on the lateral side) and flank; firmly press medially and forward so that the ribs and the lateral abdominal wall are brought a little forward and medially. Now palpate the spleen with the right hand, starting from the right iliac fossa, going gradually more towards the left hypochondrium.

The spleen, as already stated is not normally palpable and needs to be enlarged to about 3 times its usual size to become palpable. A *just palpable spleen* offers a great difficulty in detection which may be overcome by turning the patient to the right side (i.e., in the right lateral position) and then palpating the spleen with the help of the finger tips of the right hand placed loosely under the costal arch. Here the hooked fingers of the examiner's right hand are placed under the left costal margin and the patient is asked to take deep breaths. A just palpable spleen touches the finger tips at the height of inspiration.

The following findings should be noted in case a palpable spleen is found–situation, margin, splenic notch size, shape, consistency (i.e., soft, firm or hard), surface, tenderness, movement with respiration, any palpable splenic rub or fremitus (found in cases of perisplenitis) and whether the fingers can be insinuated between the costal margin and the enlarged spleen.

A palpable spleen should be differentiated from a kidney swelling by the following points :

Splenic swelling	Kidney swelling
1. The spleen enlarges downwards, forwards and to wards the right iliac fossa. It has a tendency to buldge forward.	1. The kidney enlarges downwards towards the respective iliac fossa. It has a tendency to buldge into the loin.
2. The margin is sharp.	2. The margin is rounded.
3. The spleen has a notch on the anterior border.	3. There is no palpable notch.
4. The fingers cannot be insinuated between the costal margin and the spleen and an enlarged spleen cannot be pulsed back into the loin.	4. Fingers can be insinuated easily between the costal margin and the kidney and there is a fullness of the loin (at the back) due to the enlargement of the kidney.
5. The fingers can be insinuated between the sacrospinalis and the subcostal margin on the back.	5. In the back the fingers cannot be insinuated between the kidney lump and the erector spinae muscle as there is no space.
6. Percussion note is dull over the spleen.	6. A band of colonicresicres onance is found over the kidneys
7. It is not palpable bimanually and is not ballotable.	7. It is bimanually palpable and ballotable.
8. Renal angle is not tender.	8. Renal angle may be tender.

Other swellings or enlargements which should be differentiated from an enlarged spleen are—enlarged left lobe of the liver, rolled up carcinomatous or tuberculous omentum, carcinoma of the body of the stomach, malignant left suprarenal tumour and carcinomatous mass involving the splenic flexure of the colon.

Mild enlargemenf ot the spleen (I finger) may be found in enteric fever, infective endocarditis, acute malaria, acute kala-azar, haemolytic anaemias, miliary tuberculosis, acute leukaemia, infectious mononucleosis, in a few per cent cases of idiopathic thrombocytopenic purpura, infective hepatitis etc. *Moderate enlargement of the spleen* (2-5 fingers) found in chronic malaria, chronic kala-azar, portal hypertension, Hodgkin's disease, congenital haemolytic anaemias, chronic myeloid and lymphatic leukaemias, sarcoidosis, non-Hodgkin (lymphocytic) lymphomas, haemochromatosis, chronic active hepatitis, sarcoidosis etc. *Huge splenomegaly* (5 fingers or more) is characteristic of chronic myeloid leukaemia, chronic malaria, chronic kala-azar, myelofibrosis, myelosclerosis, tropical splenomegaly syndrome, primary polycythaemia, portal hypertension, thalassaemia major, in children Gaucher's and other lipid storage diseases etc. Enlargement of the *spleen and the lymph nodes* may occur in chronic lymphatic leukaemia, Hodgkin's disease, miliary tuberculosis sarcoidosis, non-Hodgkin's (lymphocytic) lymphoma, infectious mononucleosis etc. In fact any condition which causes generalised lymphadenopathy may cause splenomegaly.

Ascites may be present along with splenomegaly in portal hypertension syndrome, chronic myeloid leukaemia, lymphoma etc.

Rise of temperature may be associated with splenomegaly in enteric fever, infective endocarditis, kala-azar, acute malaria, infective hepatitis, miliary tuberculosis, acute leukaemias, crisis in haemolytic anaemias etc.

The combination of *hepatomegaly and splenomegaly* is a common clinical finding and usually occurs in chronic malaria, kala-azar, chronic myeloid leukaemia, heamolytic anaemias, myelofibrosis, myelosclerosis, infective hepatitis, post-hepatitic cirrhosis with portal hypertension.

Hypersplenism– This often accompanies splenomegaly and is characterised by an enlarged spleen, anaemia leucopenia and/or thrombocytopenia in addition to hyperactivity of the bone-marrow. Splenectomy often reverses the condition. Though hypersplenism may be present in most splenomegalic states, *splenomegaly does not always cause hypersplenism.*

Besides the bedside clinical methods of detecting splenomegaly, the best method is *scanning* of the spleen by introducing IV a colloid tagged with technetium 99m. The latter is taken up by the RE cells and helps in visualizing the size, shape and defects of spleen.

(vi) *Kidneys*

The kidneys lie on either side of the dorsolumbar vertebrae; their upper poles roughly corresponding to the upper border of the 12th thoracic vertebra; the left kidney being placed slightly

higher than the right. Kidneys are about 10 cm in length. The hila lie opposite the space between the transverse processes of the 1st and 2nd lumbar vertebra.

Method : Sit on a low chair on the right side of the patient who lies on his back with the knees flexed. To palpate the right kidney place the left hand over the renal angle and press the loin forward. The fingers of the right hand are placed over the lower hypochondrium and upper lumbar regions of the abdomen and pressed backward, upward and inward. Ask the patient to relax the abdominal muscles and take a deep breath when a firm mass with a rounded lower pole may be felt lying in the posterior abdominal wall. Now give a sharp tap by the left hand on the back and as the kidney is *ballotable*, the right hand should feel it following which the kidney falls back on the posterior abdominal wall which will be felt by the left hand. The left kidney is best palpated from the left side; the clinician's right hand being placed posteriorly.

Following points should be noted : situation, size, shape, (kidneys are ovoid in shape), margins (rounded), ccnsistency (normally resilient feel), surface (irregular in polycystic kidney), movement with respiration and *tenderness over the renal angles*. Tenderness of the renal angles may be found in acute pyelonephritis, stone, renal tuberculosis, hypernephroma and perinephric abscess. The kidneys may be palpable in cases of *dropped kidney*. In *normal thin built* persons the lower pole of the right kidney may be palpable. But the left kidney is rarely palpable unless enlarged or displaced.

The movable kidney can be easily restored to its original position by manipulation. A movable right kidney is often mistaken for a distended gall bladder. The distended gall bladder can only be temporarily pushed back and always comes forward rapidly.

Both kidneys are enlarged in polycystic disease and in bilateral hydronephrosis due to obstruction of the urinary tract at the level of the bladder or below it. *One kidney may be enlarged* in unilateral hydronephrosis hypernephroma. Wilm's tumour and in a large solitary cyst of the kidney.

Shape of the kidney may vary due to congenital defects in development, e.g. in horse-shoe shaped kidney.

The kidney mass should be differentiated from : enlarged spleen, perinephric abscess, retroperitoneal tumours, tumours of the bowel, pancreatic cyst and Riedel's lobe of the liver (if present).

(vii) Left iliac fossa, right iliac fossa and hypogastrium should be palpated deeply with both hands–the left being placed over the right.

Deep palpation in the left iliac fossa may reveal a freely movable, tender, rope like structure in a case of intestinal amoebiasis, irritable colon syndrome etc. A scyballous mass, tumour or any other lump may be palpated in this way.

A lump in the right iliac fossa may be palpated in cases of appendicular abscess, amoeboma of the colon, iliocaecal tuberculosis, Crohn's disease, carcinoma of the caecum or ascending colon carcinoid tumour etc.

Palpate the splenic and the hepatic flexures in the left and the right hypochondria respectively.

A lump due to faecal accumulation occurs more often in the left iliac fossa than elsewhere and it pits on pressure and may disappear after administration of a high enema.

Palpate the hypogastrium for any tumour of the bladder or uterus. Tenderness. over the hypogastrium may be found in bladder stone, cystitis and acute retention of urine.

(viii) *Miscellaneous swellings* within the abdomen should be looked for. They are often due to lymphoglandular enlargement found in tabes mesenterica, Hodgkin's and non-Hodgkin lymphomas, leukaemias etc.

The *testes* should be examined for any tumour e.g., seminoma which metastasises by lymph vessels and as such. mesenteric glands may become enlarged.

(ix) One must perform a digital examination of the rectum to note the condition of the sphincter, presence of scybala, stricture or growth. In the male the prostate, the seminal vesicles and the trigone of the bladder may be recognised by this technique; in the female, the uterus and the pouch of Douglas can be examined. The anus should be examined for haemorrhoids, fistulae, fissures or growths.

Lastly palpate the hernial sites, i.e., the inguinal, the umbilical and the epigastric regions. Hernias of the abdominal wall become prominent in the erect position or after coughing.

PERCUSSION

(1) General percussion–It should be done to detect any alteration in the tympanitic note of the abdomen.

(2) Shifting dullness–*First of all palpate the liver and the spleen.* If they are enlarged avoid percussion over them. Go on percussing on the midline from the epigastrium to about the midpoint between the umbilicus and the symphysis pubis. Now change the direction laterally to that side where there is no enlargement of organs. When you get a dull not turn the patient to the other side keeping the fingers in the same position; wait about a minute for the intestine to float up and then percuss again. The dull note obtained previously will have changed to a resonant one and the resonant area near the midline will be dull. *Shifting dullness and fluid thrill, together of singly make the diagnosis of ascites certain.* However, they can be elicited in only half of the cases of ascites and absence of either or both does not exclude ascites.

A fluid thrill can be elicited when a large amount of ascitic fluid has accumulated. One hand is placed flat on the patient's flank. An assistant (or the patient) is asked to put the side of his hand firmly on the midline of the abdomen. When the other flank is flicked or tapped, a shock wave is transmitted to the palpating hand. The patient's or assistant's hand prevents any ripple from passing through the fat of the abdominal wall.

Common causes of ascites are :

(1) Hepatic–Portal hypertension syndrome, carcinoma

of the liver, hepatic vein thrombosis (Budd-Chiari syndrome).

(2) Cardiac–Chronic constrictive pericarditis, congestive cardiac failure.
(3) Renal–Nephrotic syndrome.
(4) Nutritional–Malabsorption syndrome, famine oedema.
(5) Haematological–Severe anaemia, Hodgkin's disease, chronic myeloid leukaemia.
(6) Lymphatic–Filariasis.
(7) Infection–Tuberculous peritonitis, pyogenic peritonitis.
(8) Malignancy – Malignant peritonitis.

Percussion of the individual organs like liver, spleen, kidney, stomach bladder etc. should next be performed. *Heavy percussion* should be done starting about the second rib and gradually going downward until *impairment* is detected. The upper limit of the liver dullness forms an almost horizontal line around the chest. The upper border of the right lobe is at the level of the 5th rib, 2 cm medial to the right midclavicular line while the upper border of the left lobe corresponds to that of the 6th rib on the left midclavicular line. The lower border of the liver is defined by *light percussion* from below upward.

The percussion note over the left hypochondrium is tympanitic because of air in the stomach. The fundus of the stomach is normally situated in Traube's space. As such any growth in the fundus of the stomach will produce a dull note on percussion over the Traube's space (vide Respiratory system). Enlargement of the liver and the spleen should be confirmed by percussion. In a case of

ruptured spleen the area of splenic dullness increases.

Percussion note over the lumbar region i.e., over the kidney is resonant owing to the presence of gas in the colon.

Bladder in percussed from the umbilicus, downwards, on the midline.

AUSCULTATION

Auscultation of the abdomen is an invaluable clinical method of diagnosis in many abdominal and extra-abdominal disorders. The chest piece of the stethoscope should preferably be placed to the right of the umbilicus and the auscultation might reveal–

(1) *Increased* peristaltic sounds in acute intestinal obstruction and an otherwise *silent* abdomen with the sounds of pulsations of the abdominal aorta only is characteristic of paralytic ileus.

(2) Peristatic sounds may also be *increased* in nervous diarrhoea, Crohn's disease, carcinoid tumour etc.

(3) *Succussion splash* induced by vigorous shaking of the trunk in pyloric stenosis and paralytic ileus. It may also be detected by placing the stethoscope on the abdominal wall and quickly depressing the abdominal wall by the free hand. A succussion splash may be *normally* found one to two hours after a meal.

(4) *Friction rubs* over the liver and spleen with the movements of respiration are characteristic of perihepatitis and perisplenitis. The commonest cause of the former is a liver biopsy. It may also be audible over the surface of splenic infracts.

(5) *Foetal heart sounds and uterine souffle* (which is

a systolodiastolic murmur) are important clinical signs of pregnancy.

(6) *A soft systolic murmur over the lumbar region* on deep pressure with the chest piece is *diagnostic of renal artery stenosis*. In this case a systolic murmur may also be present in the renal angle. *A systolic murmur* over the abdomen is a common clinical finding in coarctation of the abdominal aorta.

(7) A *presystolic gallop* is often present over the epigastrium in corpulmonale.

(8) *Pansystolic murmur* over the epigastrium or the tricuspid area increasing with inspiration (*Carvallo's sign*) is diagnostic of tricuspid incompetence.

(9) In aortic regurgitation, a *splashing sound* is often heard over the stomach if the patient drinks a glass of water. This is due to the splashing of stomach (partially filled with water) by the booming aorta.

(10) *Auscultopercussion* helps to delineate the outline of the stomach in cases of acute dilatation of the stomach and pyloric stenosis. This is done by placing the chest piece over the left hypochondrium and simultaneous percussion of the stomach by another clinician in the direction away from the chest piece. Note the character of the sounds and put markings over the skin where there is a change of character. Another method may be employed where the percussion is replaced by scratching the skin with the help of match sticks. This should be done in straight lines directed away from the chest piece.

(11) A *venous hum* may be heard on or below the

xiphisternum or at the umbilicus in *congenital hypoplasia of the portal venous system with patent umbilical vein* or may be due to a *dilated paraumbilical vein in* cirrhosis of the liver. This is known as Cruveilhier-Baumgarten syndrome.

Clinical signs in diseases of the stomach and duodenum

(1) Pointing sign–Indicates the area of maximum tenderness in duodenal ulcer.

(2) Unilateral rectus rigidity–An important sign of inflammation of the stomach or the duodenum such as chronic duodenal ulcer.

(3) Visible peristalsis–Ask the patient to drink a glass of water before he or she lies down. Look for visible peristalsis which may be induced by sharp tapping over the abdominal wall. A left to right peristalsis is found in pyloric stenosis.

(4) Succussion splash–Characteristically found in pyloric stenosis and acute dilatation of the stomach.

(5) Percussion of Traube's space–Dull if there is a growth in the fundus.

(6) Auscultopercussion–*vide supra.*

ANO-RECTAL EXAMINATION

Students should remember that the examination of the gastrointestinal system remains incomplete without a rectal examination. The physician might miss a very prognostically grave disease e.g. a carcinoma of the rectum if he avoids this part of the exmination. One should do a per-rectal examination with the patient in any of the following positions :

(1) Left lateral–In this position the right leg should be flexed while the left leg is extended. The buttocks should be placed over the edge of the bed. (2) Knee elbow. (3) Dorsal. (4) Lithotomy.

EXAMINATION–Inspection

First inspect the region of the anus. Note the presence of discharges–blood or pus, condylomas, external haemorrhoids, protrusion of the internal haemorrhoids, anal fissure, prolapse of the rectal mucosa, external opening of anal fistula etc.

Digital palpation

The petient is asked to open his mouth and breathe quietly. Wearing a glove in the right hand, smear the right index finger with petroleum jelly. Now gently and slowly introduce the finger into the rectum starting with the pulp of the finger. Following are the conditions that can be diagnosed by digital palpation :

(i) In the rectum–Apex of an intussusception, distended rectum (i.e., ballooning) in intestinal obstruction, internal opening of anal fistula, rectal polyps, carcinoma and stricture of the rectum.

(ii) Outside the rectum–ischiorectal abscess, pelvic appendicitis, salpingo-oophoritis, aneurysm of internal iliac artery, enlarged prostate, seminal vesicles etc.

After withdrawal of the index finger inspect it for blood, pus or mucus.

Proctoscopy and sigmoidoscopy

The patient is placed in the left lateral position with the left leg extended and the right leg flexed. A lubricated proctoscope with an obturator is gently introduced to its full depth per rectum very carefully and slowly. The obturator is then removed and the interior of the rectum and the anus is inspected carefully as the instrument is withdrawn slowly. For better visualisation a torch may be focussed at the sites of the mucous membrane that is being inspected. By this process about 3 inches of the rectum and the anal canal

is visualised. It is particularly helpful in the diagnosis of *haemorrhoids.* If more of the lower part of the large gut needs to be visualised, sigmoidoscopy should be done. The lubricated sigmoidoscope is introduced into the colon per rectally. As the instrument is longer than a proctoscope its use demands experience and some skill. A lighting arrangement is attached with the sigmoidoscope. It is a safe procedure and is useful in the *differential diagnosis of diarrhoea caused by colonic pathology* and also in the diagnosis of *polyps* and *malignancies.* At times a biopsy is also taken through the sigmoidoscope. With its help about 8 to 9 inches of the colon can be inspected.

PARACENTESIS ABDOMINIS

In paracentesis abdominis free fluid is withdrawn from the peritoneal cavity usually by means of a trocar and canula.

Indications :

(1) Diagnostic–The colour and the character of the fluid is studied to determine the cause.

(2) Therapeutic–To reduce the intra-abdominal pressure when the patient feels distressed due to breathlessness, palpitation etc.

Contraindications :

During the procedure of paracentesis if the patient starts having cardiorespiratory distress the manoeuvre must be stopped immediately.

Method :

(a) Ask the patient to empty his bladder before the procedure to prevent an accidental puncture of the bladder by the trocar.

(b) Then make the patient sit at the edge of the bed, supported with a back rest. A rubber sheet is placed over the patient's lap.

(c) The site of puncture is just outside the midpoint of the line between the umbilicus and the anterior superior iliac spine to prevent any damage of the abdominal viscera. Before selection of the site the abdominal organs should be examined avoid an injury to the *spleen* or the *liver* in cases of huge splenomegaly or hepatomegaly respectively and the uterus in a case of pregnancy.

(d) The selected site is sterilised with iodine and spirit.

(e) Anaesthetise the site of puncture by 2% lignocaine.

(f) Put an abdominal binder above the umbilicus in order to maintain a steady external pressure over the abdomen.

(g) Puncture the abdominal wall by the trocar and canula. Initially a resistance is met with which is due to the subcutaneous tissue, fascia and muscles. The resistance suddenly disappears when the abdominal cavity is reached.

(h) The fluid comes out of the canula like a jet and collected in a tumbler.

(i) Samples of the fluid are stored in sterile test tubes for subsequent biochemical and pathological examinations.

(j) After removal of 2 to 3 litres of fluid the trocar is reintroduced into the canula and both are now removed together from the abdominal cavity.

(k) Seal the puncture site with Tinc. Benzoin and apply dressings.

N.B.–For small samples of fluid and also for a continuous slow drainage a wide bore needle may be used in place of a trocar and canula.

Complications :

(1) Sudden cardiorespiratory distress or shock caused by blood polling in the acutely decompressed systemic veins of the abdomen.

(2) Introduction of infection.

(3) Precipitation of hepatic encephalopathy because of the mechanisms as in (I) above.

Investigation of a sample of ascitic fluid :

The fluid withdrawn may be an exudate or a transudate.

A *transudate* is formed by the passive transudation of fluid into the peritoneal cavity and is commonly found in cirrhosis of the liver, nephrotic syndrome and severe anaemia. The fluid contains protein less then 2.5gm%, cells not more than 250/cmm in cirrhosis and less than 1000/ cmm in congestive cardiac failure which are endothelial cells and culture and animal inoculation tests are negative.

An *exudate* is formed by the inflammatory reaction of the peritoneum itself due to infection, chemical irritation or malignancy. Exudates are commonly found in Koch's peritonitis, pyogenic and malignant peritonitis and it may be serous, purulent or haemorrhagic. The *protein content* is more than 2.5gm% and *sugar* is less than normal. Sp. gr is more than 1016, spontaneous coagulation occurs on standing (due to a high protein content), cells are more than 1000/ cmm (mostly lymphocytes) in Koch's peritonitis and more than 1000/ cmm (mostly polymorphonuclear cells and pus cells) in pyogenic peritonitis. In 20% of neoplastic peritonitis more than 10000 cmm RBC's are present. Bacteriological examination of a smear, culture, and animal inoculation tests are to be carried out.

(A) *Physical character* :

(1) Appearance :

(a) In ascites due to cirrhosis of the liver, nephrotic syndrome, congestive cardiac failure and severe anaemia the fluid will be *clear and straw coloured.*

(b) *Opalescent* or even clear fluid may be obtained in Koch's peritonitis.

(c) *Haemorrhagic* fluid may be due to malignancy or Koch's infection.

(d) *Chylous* fluid may be turbid, milky or creamy due to presence of thoracic or intestinal lymph. It results from lymphatic blockage caused by trauma. tumour, filariasis, tuberculosis etc. The turbidity of such a fluid tends to disappear on addition of fat solvents like ether or chloroform. Rarely congenital abnormalities of lymphatic vessels or nephrotic syndrome may produce chylous ascites.

(e) *Turbid of frankly purulent fluid* is due to pyogenic infections.

(f) Rarely *mucinous* ascitic fluid may be due to pseudomyxoma peritonei or gastric or colonic colloid carcinoma with peritoneal implants.

(2) Specific gravity–If the specific gravity is below 1016 the fluid is a transudate and this is commonly found in ascites due to cirrhosis of the liver, nephrotic syndrome, congestive cardiac failure etc. In Koch's peritonitis, pyogenic infections and malignant peritonitis, the specific gravity is more than 1016 (exudate).

(3) Spontaneous coagulation–Fluid of high specific gravity may clot on standing due to its high protein content.

(B) *Chemical examination* :

(i) Protein more than 2.5gm% in exudates.

(ii) In transudates the protein content is less than 2.5 gm%

(iii) Sugar grossly reduced in ascitic fluid due to pyogenic and Koch's peritonitis. But in the ascitic fluid of cirrhosis or congestive cardiac failure the sugar content will not be altered.

(C) *Cytological examination* :

In transudates the cell count is not more than 250/ cmm and are mostly endothelial cells.

In Koch's peritonitis the cell count is more than 1000 cmm and are mostly lymphocytes.

In pyogenic peritonitis the cell count is more than 1000/ cmm they are mostly polymorphs.

In haemorrhagic ascities due to malignancy the centrifuged deposit is stained specially to detect malignant cells.

(D) *Bacteriological study* :

(i) A smear prepared from the centrifuged deposit of the ascitic fluid when stained with Gram's stain may show gram-positive cocci or gram-negative bacilli in cases of pyogenic peritonitis. The type of the organism may be confirmed by further culture, biochemical reactions and animal inoculation tests.

(ii) Centrifuged deposit smeared and stained by Ziehl-Neelsen's method may show acid fast bacilli. The diagnosis may be confirmed by culture and animal inoculation tests.

(iii) If the fluid is milky white in character, the centrifuged deposit should be examined by ordinary coverslip preparation or staining by Leishman's method. The slides may show Microfilaria.

INVESTIGATIONS FOR LIVER DISEASES

(A) LIVER FUNCTION TESTS

These are done to :

(i) confirm whether the liver cells are diseased or not;

(ii) detect whether there is parenchymal disease or obstruction of the biliary tree (cholestasis) or both;

(iii) assess the degree of severity or liver damage and,

(iv) indicate the prognosis of the patient.

Note all the tests are routinely done.

Commonly done tests are :

(1) *Serum bilirubin*–Normal value is 4–1.8 μmol/ litre (0.2–1.0 mg 100ml). Unconjugated and conju-

gated factors are estimated. *Unconjugated* hyperbilirubinaemia occurs in haemolytic anaemias early stage of infective hepatitis, Glibert's syndrome etc. *Conjugated* hyperbilirubinaemia occurs in later stages of infective hepatitis and in obstructive and cholestatic jaundice.

(2) *Plasma proteins,* particularly serum albumin and globulin. Serum albumin (normal 35.45 G/litre) is reduced in chronic liver disease e.g. cirrhosis, while serum globulin is increased (>30 G/litre) increase in gamma globulin peak can be detected by plasma protein electrophoresis.

(3) *Serum enzymes* :

(i) Alanine aminotransferase, ALT (GPT or SGPT). Normal value–3-30 units/L.

(ii) Aspartate aminotransferase AST (GOT or SGOT). Normal value–5-40 units/L.

Both are increased in hepatic cell damage. AST is more than ALT in secondary deposits or infiltrations; the reverse is seen in acute hepatitis, chronic active or persistent hepatitis and cirrhosis of the liver.

(iii) Serum alkaline phosphatase : Normal value is 20-100 U/L or 3-13 KA/100 ml. It is moderately raised (up to 250 u/litre) in hepatocellular damage but greatly raised in biliary obstruction, *Extrahepatic conditions* where alkaline phosphatase may be raised are metastatic tumours of bones, Paget's disease, hyperparathyroidism pregnancy and rickets.

(iv) Other enzymes : (a) 5' nucleotidase–rasied in biliary obstruction (b) γ-glutamyl transpeptidase (GGT), an enzyme of the liver cells rises in liver

diseases and also after taking cetain drugs and alcohol.

(4) *Prothrombin time :* Impairment of synthesis of coagulation factors like II, VII, IX and X (vitamin K dependent factors) results in prolongation of the prothrombin time. It indicates both the severity of damage and the prognosis of the patient, especially in infective hepatitis.

(5) *Urine urobilinogen* : Normal value is 1-2 mg/ day and increased excretion occurs in haemolytic diseases and parenchymal diseases of the liver e.g. viral hepatitis, cirrhosis, congestive cardiac failure, hepatic malignancy etc.
Decreased excretion occurs in cholestasis. Difficulty arises in interpretation of the results when both parenchymal disease and cholestasis are simultaneously present.

(6) *Urine bilirubin* : Responsible for the smoky appearance of the urine which should be differentiated from haemoglobinuria and myoglobinuria. It indicates excretion of conjugated bilirubin in the urine.

(7) *Bromsulphthalein* (BSP) *excretion test*–5 mg of BSP per kg body weight is injected IV and the blood concentrations are measured after 45 minutes and 2 hours. BSP is found almost completely (except about 2%) to albumin, carried to the liver and excreted in bile after conjugation with glutathione. Normal blood concentration after 45 minutes will be less than 5 per cent; this is a *sensitive but non-specific test.* False results may be obtained in old, febrile or hypoalbuminaemic patient and is valueless in presence of jaundice.

Hypersensitivity reaction to the dye may limit its use.

(8) *Serum lipids* : Serum total cholesterol (normal 130 to 230 mg/ 100ml) and cholesterol esters are measured. In cholestasis total cholesterol level is increased. There is a rise in low-density lipoprotein level of the plasma, whereas the high density fraction is reduced. An abnormal low-density lipoprotein, lipoprotein X is an important marker or cholestasis.

Other tests that are infrequently done :

(1) Lactate dehydrogenase (LDH)–Not a specific test but the serum level is moderately elevated in acute viral hepatitis and in cirrhosis of the liver.

(2) Alpha fetoprotein (AFP) : Marked increase or rising serum level occurs in hepatoma. Increased serum levels may be found in acute viral hepatitis, chronic hepatitis, gastric carcinoma, ovarian or testicular embryonal tumours, pregnancy, ataxia, telangiectasia etc.

(3) Detection of hepatitis B surface antigen (HBsAg) by haemaglutination and radioimmunoassay methods. It is present in type B hepatitis; sometimes in cirrhosis, chronic hepatitis, hepatic carcinoma and carriers of type B hepatitis virus. Carriers who are HB_3 Ag positive are much more infective than those who are not.

(4) IgM detection of anti-HAV for diagnose H hepatitis A.

(5) Igm_2 anti HCV to discover acute infection with hepatitis C virus.

(6) Igm_2 anti-HDV to find out if there is a superinfection or coinfection with hepatitis D virus upon a hepatitis B.

(7) IgM anti-HEV to detect acute hepatitis E.
(8) Serum ceruloplasmin level –Decreased in Wilson's disease (Normal 30-60/100 ml).
(9) Serum iron, iron binding capacity and serum ferritin concentrations– for detection of *haemochromatosis*–higher values are found.
(10) Leucine aminopeptidase and isocitrate dehydrogenase levels in, serum are increased in hepatic parenchymal damage.
(11) Plasma cholinesterase level *falls* in hepatocellular diseases.

(B) RADIOISOTOPE SCAN

Radioactive isotopes that emit gamma rays are injected IV; these are selectively extracted by the liver which is followed by external radiation scanning of the upper abdomen by a gamma camera. Liver scans are of 3 types– (i) Colloidal (ii) HIDA or PIPIDA and (iii) gallium.

(i) Colloidal scan employs colloidal gold 198 Au or 99m Tc sulfur colloid which are taken up by kupffer cells and demonstrates filling defects greater than 2-3 cm diameter, so is *falsely negative in smaller diffuse metastatic deposits while falsely positive in cirrhosis* because of distroted lobular architecture.

(ii) In HIDA or PIPIDA scans the iminodiacetic dye is taken up and excreted by hepatocytes and so a *liver abscess* or *hepatoma* produces a reduced uptake area known as a hole.

(iii) Gallium scan employs radioactive 67 Ga that is taken up by neoplastic and inflammatory cells selectively and so shows a 'hot spot' in *hepatoma* or *liver abscesses.* The HIDA or PIPIDA hepatic scans are of great value in the diagnosis of *acute cholecystitis* when the dye may fail to enter the gall bladder thus showing cystic or common bile duct obstruction.

(C) ULTRASOUND

This is done by moving a probe (that emits ultrasonic pulses) across the liver and its surrounding areas and echoes are received with a transducer, amplified and then displayed. Cysts abscesses, dilated intrahepatic bile ducts and metastases are suitably demonstrated.

(D) CAT SCAN

By computerized axial tomography cross-sectional images of the liver can be obtained and different lesions can be diagnosed.

(E) NEEDLE BIOPSY

This is an established, state and reliable method. The material obtained is subjected to histopathological study. It may be done with the Vim-Silverman or the Menghini needle :– inserted through an intercostal space using local analgesia and sedation. *Best results* are obtained in patients with *diffuse liver diseases.* Complications are bleeding, abdominal pain, shoulder pain and rarely biliary peritonitis. It is contra-indicated in patients with suspected hydatid disease, haemorrhagic diatheses, deep obstructive jaundice, ascites etc. It is also contra-indicated in uncooperative subjects, but in them it may be done by a *transvenacaval* approach, entering the liver through the hepatic vein.

(F) SELECTIVE ANGIOGRAPHY OF THE COELIAC AXIS AND HEPATIC ARTERY

This invasive procedure is not used frequently. Abnormal vascular patterns in isolated lesions e.g. in *hepatoma* can be demonstrated where other procedure have failed.

Some important laboratory tests in common hepatic disorders–

(i) *Acute hepatitis* – ALT AST ↑, ALT > AST; Alkaline phosphatase N or mild ↑; Albumin N, γ-globulins usually N, IgM slightly ↑ in viral hepatitis; bilirubin variable.

(ii) *Chronic hepatitis*–AST may be N, ALT > AST; Alk phosphatase ↑ or N γ-globulins all ↑ especially IgG; Bilirubin N or variable; Albumin N or ↑.

(iii) *Cirrhosis*–AST > ALT or ALT or both N : Alk phosphatase ↑ or N; Albumin ↓; γ-globulin all ↑ especially IgA; Bilirubin N; Prothrombin time may be pronlonged (Vit K does not help),

(iv) *Biliary cirrhosis*–ALT, AST variable; Alk phosphatase ↑↑; Albumin usually N; γ globulin IgM ↑; Conjugated bilirubin↑.

(v) *Total biliary obstruction*–ALT, AST variable; Alk phosphatase ↑↑; Albumin γ-globulin usually N; Conjugated Bilirubin ↑↑; PT pronlonged and responds to Vit K.

INVESTIGATIONS FOR DISEASES OF THE GALL-BLADDER AND BILE DUCTS

(A) *Straight X-Ray* : It may show–

(i) Stones in the gall-bladder or bile ducts (10% cases)

(ii) Soft tissue shadow of the inflammed gall-bladder.

(iii) Rarely gas in the biliary tree due to *fistulous* communications with intestine or in *emphysematous cholecystis* occurring as a complication of diabetes mellitus.

(iv) Pancreatic calcification, if present, will be demonstrated.

(B) *Oral Cholecystography :* An iodine containing substance (Telepaque tablet) is given orally the night before the investigation. After absorption from the intestine it is excreted by the liver and concentrated in the gall-bladder, thus rendering it opaque.

Normal gall-bladder is seen as an ovoid homogeneous opacity which is absent in case of a nonfunctioning gall-bladder.

Radiolucent stones and rarely a tumour may be seen a filling defects. Also anatomical variations and failure to contract in response to a fatty meal be demonstrated.

N.B.–In case of a nonfunctioning gall-bladder, the investigation is to be repeated with a double dose of the contrast media.

(C) *Intravenous Cholangiography* : This is performed by administering Iodipamide Methyglucamine (Biligrafin) intravenously. The bile ducts and any pathology thereof e.g. dilatation, stones etc. can be demonstrated. As in cholecystography it cannot be performed on a jaundiced patient.

(D) *Percutaneous Transhepatic Cholangiography* (PTC): It is nontraumatic procedure because a five gauge (FR 22, 25) needle is used and can be performed even in the presence of obstructive jaundice provided prothrombin time and platelet count are normal. As in intravenous cholangiography, pathology in the biliary tree can be demonstrated. Almost all patients other than young children tolerate this procedure well. The needle should be incrementally withdrawn through the liver whilst contrast injection is screened.

(E) *Endoscopic Retrograde Cholangiopancreatography* (ERCP) :

This is a very useful application of fibreoptic endoscope. A fibreoptic duodenoscope is introduced and the duodenal papilla is identified. Biopsy and brushing of the papilla are taken for cytological and histological examinations. Then the ampulla of Vater is cannulated by a fine bore catheter and a radio-opaque dye is injected into the pancreatic and biliary ducts. Any abnormalities of these ducts are therefore, well demonstrated. The procedure is very useful in suspected pancreatic diseases and obstruction of the main

bile ducts. Complication rate is extremely low in the hands of an experienced operator and pancreatitis and cholangitis remain the main problem.

(F) *Operative T-tube Cholangiography.*

(G) *Ultrasonography* : A non-invasive procedure, it can demonstrate dilated biliary tree caused by mechanical obstruction and can also detect gall stones. It is particularly useful in pregnancy.

INVESTIGATIONS FOR DISEASES OF STOMACH AND DUODENUM

Gastric intubation– Intubation is done both for *therapeutic* and *diagnostic* purposes. Either an original rubber Ryle's tube or a disposable plastic tube of about 16 gauze is used. These tubes have bulbous ends containing a solid metal olive to make it heavy as well as radio opaque. There are 4 marks on the tubing to show the position of the tip of the tube from the incisor teeth : mark 1 at 40 cm and the tip is at the *cardiac* end of the stomach; mark II at 50 cm and the tip inside the *stomach;* mark III at 57 cm and the tip is at the *pylorus* and mark IV 65 cm and the tip is inside the *duodenum.*

The different uses are :

A. *Diagnostic*–(i) Gastric acid studies and measurement of secretory functions (ii) Diagnosis of gastric diseases (by occult blood, mucus, exfoliated malignant cells and poisons in the analysis of gastric contents) (iii) Diagnosis of extragastric diseases e.g. Mycobacterium tuberculosis in the gastric lavage of children to diagnose pulmonary tuberculosis (as children cannot cough out sputum).

B. *Therapeutic*–(i) Gastric lavage (2 sodi bicarb etc.)

(ii) Gastric decompression e.g. in acute dilatation of the stomach.

(iii) Feeding and administering drugs to a comatose patient. Before the study of gastric acid the position of the tip should be confirmed by X-ray screening–it should be in the most dependent part of the stomach.

Fractional test meal–An overnight fast should follow a light evening meal. In the morning remove the resting juice by manual suction (first sample). Then give the patient a test meal of oat meal gruel, barley water of 7% alcohol. Next suck out manually 6.8 ml of the gastric juice every 15 minutes till 12 samples are drawn. *All the samples are studied for total acid. free acid, blood, bile or mucus.* This is the fractional test meal study. In addition, if malignancy is suspected malignant cells can be searched for by special staining and microscopy (exfoliative cytology).

Definite variations of gastric secretions occur among normal subjects and patients of gastric and duodenal ulcers. But the wide range of secretion to each group makes the differences between individuals of little value. As such, fractional test meal is an obsolete procedure, and only of historical value.

Gastric acidity is studied–

(i) To demonstrate *achlorhydria* in patients of pernicious anaemia, gastric carcinoma (18%) and chronic gastritis etc. Achlorhydria is defined as the failure of the stomach to produce a juice of pH less than 7 even after a maximal stimulus by parenteral *histamine* (0.04 mg/kg body weight) or *pentagastrin* (6µg/kg body weight) see MAO below).

(ii) To demonstrate gross elevation of the basal acid output (BAO) as occurs in *Zollinger-Ellison syndrome.*

(iii) In surgical practice to *decide the type of operation* to be done in a particular case of peptic ulcer.

(iv) *To verify the completeness of a vagotomy* by Hollander's insulin test.

(v) In the *diagnosis of atrophic gastritis* and *gastric carcinoma.*

Basal Acid Output (BAO) : After an evening meal the patient is not allowed to eat, drink or smoke any more. In the morning a tube is inserted into the stomach and the position of its tip in the most dependent part of the stomach is confirmed fluoroscopically. All the resting fluid is removed completely by manual syringe suction and this first aspirate is discarded. The patient is asked to expectorate out any saliva or sputum. During the next *one hour* the gastric juice is aspirated and a total of four samples are taken out by manual suction–this is the *basal secretion* in which basal acid output is measured. So basal secretion is the juice obtained in the morning after an overnight fast, from an unstimulated stomach. If reflects the *vegal plus the hormonal factors* acting on the gastric mucosa. This is usually measured for one hour. In normal individuals the basal secretion is only a few millilitres containing up to 10 mEq HCl. The volume of the basal secretion is *high in duodenal ulcer* and *much higher in Zollinger-Ellison syndrome* and BAO may be as high as 500 mEq/hour.

Maximal Acid Output (MAO) : After the basal secretion has been measured for 1 hour either subcutaneous histamine (0.04 mg per kg body weight) or subcutaneous or intramuscular pentagastrin 6 μg/kg body weight is given. This causes a maximum acid output which *cannot be increased* further by increasing the dose of the stimulant. The gastric juice is then collected for 1 hour and the *total acid secreted is the maximum acid output* (MAO). In normal individuals the maximum value reached is about 20 mEq/hour in females and 24m Eq/hour in males. In patients with Zollinger-Ellison syndrome much higher values are found and in cases of duodenal ulcer values from 20-110 mEq/hour may be found.

The maximun acid output is *proportional to the 'Parietal cell mass'.* If the stomach so stimulated fails to produce a juice of pH less than 7, *achlorhydria is said to be present.*

N.B.–Other diagnostic procedures that help in confirming the diagnosis of *gastrinoma* (i.e. Zollinger-Ellison syndrome) are–(i) Ba-meal radiography, (ii) Demonstration of increased serum gastrin levels by radioimmunoassay which even in fasting state is almost always greater than 200pg/ml, while in a normal subject is about 60 pg/ml and in one, with typical duodenal ulcer not more than 160 pg/ml. Other provocative tests include (iii) *Calcium infusion test* and (iv) *Secretin infusion test* and of these the last one is of greatest diagnostic value.

The Insulin (Hollander) test :

Insulin causes hypoglycaemia and stimulates the vagus nerve to secrete gastric acid. In complete vagotomy this fails to occur. So measurement of gastric acidity following hypoglycaemia after insulin injection can test whether vagotomy had been complete and is particularly useful in recurrence of duodenal ulcer following vagotomy. The procedure is as follows : The blood sugar level should fall to below 45 mg/dL. It is measured both before and after administration of 15 units of soluble insulin slow IV, (or 2 units/10kg) following which gastric aspiration is done for 120 mins and the patient is watched to avoid hypoglycaemic coma. If the acid secretion rises to 20 mEq/L or more above the basal level, vagotomy had been incomplete.

II. *Plain X-ray* : Plain X-ray of the abdomen is taken both in the supine, and erect postures to detect gas under the diaphragm (commonly on the right side) which confirms perforation of a gastric or duodenal ulcer.

III. *Barium Meal* : Passage of radio-opaque barium sulfate is observed by fluorescent screening and films are taken. The followings are looked for–

Gastric Ulcer : Barium filled crater as a rounded deposit or as a projection from the wall.

Duodenal Ulcer (i) Stellate appearance of mucosal folds.
(ii) Deformity of the duodenal cap.
(iii) No definite crater may be seen.

Pyloric Stenosis : Grossly enlarged stomach emptying slowly

Polypoid Carcinoma : Filling defects.

Infiltrating Carcinoma : (i) Rigid conical shape of the stomach.
(ii) Absence of peristalsis.
(iii) No ulceration.

IV. *Endoscopy and Biopsy* : Flexible fibreoptic gastroscope and duodenoscope are used for visualization of oesophagus, stomach and duodenum for taking biopsy specimens for cystological and histological examinations.

INVESTIGATION FOR DISEASES OF SMALL AND LARGE INTESTINES & PANCREATIC FUNCTION TESTS

(1) *SMALL INTESTINE*

(a) **Estimation of Faecal Fat**–This is useful for demonstrating steatorrhoea (see Pancreatic Function Test).

(b) **Barium Meal Follow-through**– *In malabsorption*– Abnormal transit time to colon, dilatation, narrowing, flocculation which is known as *moulage sign* etc.

In Crohn's disease–Narrowing with proximal dilatation, mucosal abnormalities e.g. skip lesions, cobblestone appearance, small ulcerations occurring on small irregular nodules which may extend to produce longitudinal ulcers and transverse fissures; irregular thickening and fibrosis leading to stricture formation that may be multiple : and loss of mucosal detail and rigidity of involved segments due to submucosal oedema and stenosis : *Kintor's string sign* caused by gross narrowing of terminal ileum lumen due to thickened walls–a long continuous thin column of barium

with irregular edges resembling a frayed piece of string. Beside these, fistulae and sinuses may be demonstrated.

Neoplasms–Filling defects

Small bowel diverticula.

(c) **Small intestinal Biopsy**–A spring loaded capsule (the Crosby capsule, which is 7 mm in width and 1.5 cm in length and contains a cutting blade to cut out small pieces of intestinal mucosa when negative suction is applied via the attached tube) is used. The patient swallows this capsule and when it has reached the desired site of the small intestine, its position is checked by X-ray screening. After the biopsy tissue has been cut out, the capsule is removed and the biopsy specimen is examined microscopically without delay. It is particularly important in the investigation of *malabsorption syndrome*. One of the important findings is flattenning of the vill which may be virtually absent with elongation of crypts.

(d) **Endoscopy, Endoscopic Biopsy and Gastrocamera technique** : With the help of a conventional gastroscope or a flexible fibreoptic gastroscope it is possible to visualise the whole of the oesophagus, stomach and duodenum. It is also possible to see the orifices of the bile and pancreatic ducts. The procedure can be carried out rapidly and safely. Through a channel in the same instrument it is possible to introduce a biopsy forceps or brush to obtain the specimen for the cytological examination. In some instruments a proximal camera is attached with which photographs of the inner mucosal walls can also be taken.

(e) **Ultrasonography**–Noninvasive test to find pancreatic tumors or defects due to necrotic lesions.

(2) LARGE INTESTINE, RECTUM AND ANAL CANAL

(a) **Proctoscopy**– Piles, opening of an anal fistula, polyps and anal fissures can be seen.

(b) **Sigmoidoscopy**–Ulcers, polyps, carcinomas, proctitis are visualized and biopsy can be taken.

(c) **Barium enema**– Carcinomas, polyps diverticular disease, fistulae and colonic obstructive lesions can be demonstrated. In ulcerative colitis the earliest features are *irritability and incomplete filling* because of concomitant inflammation : fine ulcers of early stages give way to *deeper ulcers* with progression of disease and *polypoid defects* may appear. In chronic ulcerative colitis the features are shortening of the gut, narrowing of lumen, *rigidity, loss of haustration* and *tubular appearance*–the pipe stem colon.

N.B.– If the lumen becomes eccentric, a carcinoma should be suspected.

(d) **Coloscopy**–With the help of a *fibreoptic instrument* it is possible to inspect the whole of the colonic mucosa and take biopsy specimens. At times pedunculated polyps can be removed via a coloscope without going for a laparotomy.

TESTS FOR PANCREATIC DISORDERS

(a) STOOL should be examined for :

(i) Estimation of faceal fat in 24 hours. If it exceeds 7 gms in 24 hours with a diet containing 100 gms fat it is suggestive of steatorrhoea of pancreatic origin.

(ii) Presence of undigested meat fibres *(creatorrhoea)* indicates deficiency of proteolytic enzymes of the pancreas.

(iii) In chronic pancreatic disorders excretion of nitrogen will be more than 2.4 gm in 24 hours.

(b) Blood should be sent for estimation of *serum*

amylase which exceeds 1000 Somogyl units per 100 ml serum within the first few hours of acute pancreatitis (Normal : 150 to 340 units/L or 80 to 180 Somogyl/100 ml) Serum amylase estimations are of *little value* in the diagnosis of chronic pancreatitis. Provocative tests with *'secretin'* or *'pancreozymin'* demonstrate low volume of secretion and reduced concentration of amylase and bicarbonate in chronic pancreatitis.

(c) LUNDH TEST : Duodenum is intubated, pancreas is stimulated by prior administration of a meal, pancreatic juice is aspirated and *tryptic activity* assessed. Pancreatic insufficiency (of the exocrine pancreas) is said to be present if the mean tryptic activity is below 6 IUL.

(d) TRIPLE TEST : It consists of 3 parts. A special double lumen tube is made to swallow and positioned into the loop of the duodenum under screening. After that–

(i) Duodenal aspiration is done after secretin injection and again after pancreozymin injection; the aspirate is then subjected to physical and chemical examinations for bicarbonate, lipase, amylase and trypsin.

(ii) Fresh aspirate is subjected to Pap's stain and cytological examination.

(iii) Hypotonic duodenography after injecting an antispasmodic drug e.g. *hyoscine butylbromide* (BUSCOPAN) is performed.

(e) ERCP : This has been described earlier and is contraindicated in *acute pancreatitis* and *suspected pneudopancreatic cyst.*

OTHER TESTS FOR INTESTINAL DISORDERS

(i) The nitrogen content of the stool should be estimated as mentioned above.

(ii) Glucose tolerance test will show a flat curve.

(iii) Xylose excretion test in the urine will reveal diminished excretion as the intestine cannot absorb them properly.

(iv) Examination of stool–

(a) Passage of bulky, offensive and pale stool is due to defective absorption of fat in case of steatorrhoea.

(b) Liquid stool with pus and some amount of mucus and blood may indicate intestinal malignancy.

(c) Semiliquid and small amounts of stool mixed with mucus blood and pus may be due to bacillary dysentery.

(d) When the colour of the stool is tarry it indicates gastroduodenal haemorrhage, common blood dyscrasias or intussusception.

(e) The stool may be greenish and semiliquid in case of diarrhoea in children and sometimes in typhoid fever.

(f) The stool may be blackish if the patient has been taking iron or too much of *green vegetable* and also sometimes after taking bismuth *meat.*

(g) The stool may show cysts or vegetative forms of Entammoeba histolytica, ova or tapeworms, round worms or hookworms, as well as round worms or segments of tapeworms etc.

(h) Benzidine test reveals occult blood in gastrointestinal haemorrhage.

VOMITUS

Vomiting may occur in (i) acute gastric ulcer; (ii) peritonitis; (iii) liver diseases; (iv) acute cholecystitis; (v), acute

haemorrhagic pancreatitis; (vi) fulminating pneumonias; (vii) whooping cough; (viii) increased intracranial tension; (ix) pregnancy; (x) diabetic coma; (xi) Addison's disease : (xii) following anaesthesia; (xiii) acute intestinal obstruction; (xiv) Meniere's syndrome; (xv) after consuming alcohol, emetics etc.; (xvi) digitalis toxicity; (xvii) terminal stages of uraemia; (xviii) acute myocardial infraction especially of the inferior wall. In most of these cases nausea is associated with vomiting. Huge quantity of vomitus may be seen in congenital and acquired pyloric stenosis, carcinoma of the stomach etc.

In intracranial diseases especially with raised intracranial tension *nausea is usually absent* or less conspicuous, vomiting occurs suddenly and without warning; there is no relation with food intake. This is generally known as 'cerebral' vomiting.

Haematemesis means blood vomiting: it occurs in; (1) acute gastric erosion; (2) chronic duodenal ulcer; (3) gastric ulcer; (4) carcinoma stomach; (5) cirrhosis of the liver with or without portal hypertension; (6) blood dyscrasias like haemophilia; (7) Mallory-Weiss syndrome (mucosal rupture of the lower end of the oesophagus); (8) portal hypertension syndrome resulting from various causes; (9) Rendu Osler Weber's syndrome (hereditary haemorrhagic telangiectasia). In haematemesis the blood is usually dark red in colour due to conversion of Hb into acid haematin by HCl of gastric juice.

N.B.–Remember that the *Munchausen syndrome* may present with malingering of haematemesis which is a not uncommon condition.

EXAMINATION OF URINE

(1) *Amount*

Normal excretion of urine in 24 hours is about 1500 ml.

It may be persistently increased (polyuria) in : (a) diabetes mellitus, (b) diabetes insipidus, (c) kidney diseases associated with uraemia, (d) hypercalcaemia, (e) hypokalaemia, (f) administration of diuretics, (g) after successful treatment of heart failure, (h) recovery from nephrotic syndrome etc.

The urinary output may be so small that it may fail to maintain life processes in a steady state (oliguria) in– (a) acute glomerulonephritis, (b) congestive cardiac failure, (c) acute renal failure, (d) cirrhosis with huge ascites etc. Less than 400 ml urine in 24 hours in an average adult subject is conventionally known as oliguria. Less than 100ml/24h is known as anuria.

(II) ***Colour***

Normal urine has an amber or straw colour when freshly passed, and is quite transparent. Urine may be *opalescent* due to the presence of various substances in suspension e.g. pus, bacteria or phosphates, Add a few drops of 10% acetic acid;–if the opalescence disappears, it is due to *phosphates*. Filter the urine, if the opalescence persists after filtration it may be due to the presence of bacteria.

Urine may be high coloured in conditions like (a) excessive heat as in the tropics; (b) high rise of temperature in any infectious disease; (c) jaundice; (d) acute glomerulonephritis (smoky urine due to microscopic haematuria): (e) haemoglobinurias (dark red to brownish black); (f) prophyria (port wine colour due to prophobilinogen which condenses to prophyrins); (g) drug induced e.g. phenylindanediones (pink), anthracene purgative (orange), rifampicin and phenazopyridium (red), methyldopa and iron sorbitol (grey), desferrioxamine (reddish brown) and furazolidone (brown).

There may be frank blood in the urine (*haematuria*).

(i) If the haematuria is due to diseases of the bladder,

the first part of the urine is clear and the last part contains blood.

(ii) If the haematuria is due to urethral haemorrhage, the first part of the urine is mixed with blood and the last part is clear.

(iii) In case of haematuria of renal origin in the urine is uniformly mixed with blood.

Chemical examination with benzidine and the guaiac test are positive in microscopic or frank haematuria.

Common causes of haematuria

(i) Prerenal – (a) haemorrhagic diseases, (b) excessive anticoagulant therapy.

(ii) Renal–(a) acute glomerulonephritis, (b) renal tuberculosis, (c) neoplasm, (d) traumatic, (e) malignant hypertension, (f) idiopathic (essential haematuria), (g) renal stone, (h) collagen diseases etc.

(iii) Ureteric–(a) ureteric stone, (b) pyelitis.

(iv) Bladder–(a) calculus, (b) cystitis, (c) carcinoma and papilloma.

(v) Urethral–(a) calculus, (b) enlarged prostate, (c) rupture of the urethra.

(III) ***Specific gravity***

The concentration of urine is expressed as its specific gravity for all practical purposes. The specific gravity depends on the type and the number of solute particles. Normal specific gravity varies from 1010 to 1035.

The specific gravity is determined by the *urinometer*. Urine is taken in a clean long cylindrical glass jar and the urinometer is made to float freely in it when it remains partially submerged. The urinometer reading at the upper level of the urine (at the bottom of the concave meniscus) is recorded and this indicates the specific gravity of the urine. Falsely low specific gravity may be recorded if the

urine is tested while it is warm (shortly after it has been passed). So it should be first cooled to the room temperature. In the normal urine the specific gravity is proportional to the concentration of the sodium and the urea content of the urine. Also presence of glucose, protein, radio-opaque media etc. in the urine increases its specific gravity. Presence of 1% protein raised the specific gravity by 3 points. In a normal individual who has not taken any water for 10-12 hours and who is on a normal diet; the specific gravity of the urine should be about 1020. In chronic renal failure due to chronic nephritis, malignant hypertension or chronic pyelonephritis the specific gravity becomes *low* and *fixed* (1010) when it is known as *isosthenuria*; whereas in diabetes mellitus, acute glomerulonephritis, nephrotic syndrome, water deprivation and in any condition causing proteinuria the specific gravity of the urine becomes high.

(IV) Character of any visible deposit

Normal urine is clear and transparent. Urinary ingredients like phosphate, urates, uric acid etc. may separate out as a deposit on standing. A white deposit is formed by phosphate whereas a light yellow, brown or at times red deposits are formed by uric acid and urates. A wooly appearing cloudy deposit at the bottom of the tube may be formed by calcium and mucus only. Magnesium phosphate deposits are found in an alkaline urine whereas ammonium, sodium and potassium urates are found in acidic urine.

(V) Reaction (pH) of the urine

Normal urine is acid and the reaction is examined by dipping in it a litmus paper. An acid urine turns blue litmus red. An alkaline or neutral urine indicates either impairment of renal tubular capacity to excrete acid or ingestion of a systemic or urinary alkaliser.

(VI) ***Chemical constituents***

(1) **Protein**–Proteinuria is the excretion of a sufficient amount of protein in the urine which can be detected by- (a) heat coagulation or (b) sulphosalicylic acid test or (c) commercial strips (e.g. Albustix). Quantitative estimation of protein can be done by Esbach's albuminometer. Normal daily protein excretion is up to 150 mg.

Heat coagulation test– Urine is taken in a test tube and the upper part of the column is heated to boiling;–a white cloudy precipitate appears which may be due to phosphate or protein. On addition of 10% acetic acid drop by drop, in *exces phosphate precipitate disappears but the protein precipitate persists.* A false positive result may be due to the presence of radio-opaque media in the urine or if the patient is being treated with *tolbutamide* or large doses of *penicillin.* Bradshaw's test–This indicates the presence of globulins including light chains and is negative in presence of *albumin only.* Urine is genlty layered on to few millilitres of conc. HCl when a heavy white precipitate indicates the presence of globulins.

Albustix – It is a commercial strip whose test area is impregnated with *buffered tetrabromophenol* blue. When dipped in urine the test area shows a change of colour from yellow to various shades of green depending on the amount of protein in the urine.

Causes of proteinuria :

(A) *Tubular proteinuria*–This occurs in tubulointerstitial disease like–

(i) Analgesic and lead nephropathy.
(ii) Gouty and acute uric acid nephropathy.
(iii) Hypercalcaemic and hypokalaemic nephropathy.
(iv) Acute and chronic pyelonephritis.
(v) Hereditary nephritis or Alport syndrome.
(vi) Multiple myeloma.

(B) *Overflow proteinuria*–This is essentially a form of tubular proteinuria and occurs when the filtered load of protein overwhelmes the tubular reabsorptive capacity. This is exemplified by–
(i) Bence Jones proteinuria.
(ii) Myoglobinuria.

(C) *Glomerular proteinuria*–This is caused by the loss of normal selective filtration mechanism and is exemplified by–
(i) Acute glomerulonephritis.
(ii) Rapidly progressive glomerulonephritis.
(iii) Chronic glomerulonephritis.
(iv) Nephrotic syndrome.
(v) A symptomatic urinary abnormalities due to glomerular causes including *orthostatic proteinuria*,–a primary glomerular disease.

An abnormal protein, the Bence Jones protein consists of the light chains of immunoglobulin molecules and is diagnostic of multiple myeloma and it coagulates at 55°C and again disappears at 85°C.

(2) **Glucose**–It can be detected by Fehling's or Benedict's reagent. *Benedict's test*–To 5 ml of Benedict's reagent 8 drops of urine is added and boiled for 2 minutes. On cooling, a coloured precipitate will appear, and this colour indicates the approximate concentration of sugar, as follows–

(i) Light green turbidity (0.1 t0 0.5% sugar).
(ii) Green precipitate (0.5 to 1.0% sugar).
(iii) Yellow precipitate (1.0 to 2.0% sugar).
(iv) Brick red precipitate (2.0% or more sugar).

More simple methods of testing urine for sugar by clinitest tablet, clinistix test strip or Diastix test strip are available now-a-days.

Sugar in the urine is found in (a) diabets mellitus (b) thyrotoxicosis, (c) acromegaly, (d) renal glycosuria, (e) lactosuria in cases of pregnant and lactating women etc.

N.B.–Benedict's test with urine may be positive, other than glycosuria in–(i) lactosuria, pentosuria, fructosuria and galactosuria; (ii) conditions where drugs are excreted as glucuronides e.g. salicylates, chloral etc. and (iii) excretion of some metabolites or homogentisic acid. However, *90 per cent of all cases of positive Benedict's test is due excretion of glucose in urine.*

(3) **Calcium**–Excess of calcium in the urine can be detected by Sulkowitch's test. This may be due to (a) hyperparathyroidism, (b) prolonged immobilisation; (c) hypervitaminosis D. (d) renal rickets, (e) malignant disease of the bone, (f) sarcoidosis and (g) idiopathic causes.

(4) **Ketone bodies**–Acetone, aceto acetic acid and belahydroxybutyric acid are ketone bodies. Their presence in the urine can be detected by (i) Rothera's test or (ii) Gerhardt's (ferric chloride) test.

Rothera's test–10 ml urine in a test tube is supersaturated with ammonium chloride. To this is added 3 drops of freshly prepared sodium

nitroprusside solution. Then liquor ammon forte is added to it very gently; pouring by the side of the test tube (about 2 ml). At the junction of the two luquids a *deep purple ring* appears. Simple methods of detecting ketone bodies in the urine are by (i) Acetest tablet or by (ii) Ketostix reagent strip Ketone bodies are found in the urine in severe diabetes mellitus and often in starvation.

(5) **Bilirubin**–Unconjugated bilirubin or haemobilirubin cannot be excreted by the kidney. If only the circulating level of conjugated bilirubin or bilirubin glucuronide (cholebilirubin) is high it is excreted in the urine. Absence of bilirubin in the urine of a jaundiced patient suggests either *haemolytic disease of congenital hyperbilirubinaemia,* while its presence suggests *obstructive or hepatocellular jaundice.*

(6) **Urobilinogen**–Bilirubin excreted by the liver into the intestine gets converted into urobilinogen and urobilin. Some of this enters the circulation from the intestine by reabsorption. Majority of this circulating urobilinogen is reexcreted into the bile but a small amount is excreted in the urine. Presence of urobilinogen in the urine can be tested by using Erhlich's aldehyde reagent or urobilistix.

If in a jaundiced patient no urobilinogen of urobilin is found in the urine it indicates complete obstruction of the bile ducts (*obstructive jaundice*). If in patients with jaundice an excess of urobilinogen is found in the urine it indicates either *hepatocellular* or *haemolytic jaundice.* If in patients without jaundice an excess of urobilinogen and urobilin is

found in the urine it indicates *hepatic cell dysfunction* and inability of the liver to excrete these substances and may be due to (i) preicteric stage of infective hepatitis; (ii) congestion of the liver in congestive heart failure (iii) cirrhosis of the liver provided haemolytic disorder can be excluded.

(VII) ***Microscopical constituents***

Microscopical examination of the urine may show RBC, WBC, pus cells, epithelial cells, casts, crystals, microorganism, occasional parasites (bilharzia e.g. Schistosoma haematobium), spermatozoa and elongated prostatic threads (in chronic inflammation of the prostate).

(a) ***Casts :***

These are cylindrical structures produced by the precipitation of Tamm-Horsfall mucoproteins (THM) in the renal tubules and hence shaped by the tubules. They are of microscopic size.

(i) *Hyaline casts* are pale, hamogeneous, transparent casts produced by the precipitation and coagulation of protein (Tamm-Horsfall protein). This is the basic cast found at times in normal individuals and in cases of proteinuria.

(ii) *Epithelial cast* is a hyaline cast covered with renal epithelial cells, found in acute glomerulonephritis.

(iii) *Granular cast* is a hyaline cast covered with degenerated renal epithelial cells and the cells show granular degeneration.

(iv) *Fatty cast* is a hyaline cast covered with epithelial cells showing fatty degeneration. Found in lipoid and diabetic nephrosis.

(v) *Blood cast* is a hyaline cast covered with RBC; found in acute glomerulonephritis.

(vi) *Waxy cast* is a larger hyaline cast found in any case of proteinuria.

(vii) *Broad casts* are unusually wide and possibly arise in the dilated tubules which have undergone compensatory hypertrophy due to reduced functioning renal tissue. Found in chronic glomerulonephritis.

Casts are easily missed if the urine is centrifuged too long with very rapid rotation;–too long and too rapid centrifugation of the urine leads to a disruption of the fragile casts. Prolonged standing of the urine also disrupts the cells and casts–so it is best to examine a fresh sample.

In bright illumination it is difficult to see the casts; so the microscope diaphragm should be kept partially closed and the condenser should be racked down. Casts must be differentiated from the rolled up epithelial cells (so-called cylindroids) and prostatic threads.

(b) ***Cells*** :

(i) Pus cells in the urine are commonly found in acute and chronic, pyelonephritis cystitis and gonococcal urethritis. Pus cells or leucocytes are easily seen if the red cells are disintegrated by the addition of a few drops of glacial acetic acid. Pus cells disintegrate rapidly on standing; so they should be examined in fresh urine.

(ii) RBC–Normal uncentrifuged urine do not contain more than 3 to 5 RBC/cmm. Presence of RBC in the urine is known as haematuria. *Microscopic haematuria* is found in *infective endocarditis.*

Intact RBC in the urine indicates that there is whole blood in the urine, this is *haematuria.* But when only haemoglobin is present in the urine and no blood corpuscles, it is called *haemoglobinuria.*

If by microscopic examination no RBC is found then the suspected haemoglobinuria can be confirmed by benzidine or orthotoluidine test. A positive benzidine or orthotoluidine test indicates the presence of blood or haemoglobin in the urine. These can also be tested by dipping a Haemostix reagent strip in the urine.

Haemoglobinuria may occur in incompatible blood transfusion, (iii) autoimmune haemolytic anaemias, (iv) DIC, (v) march haemoglobinuria (exertional), (vi) haemolytic uraemic syndrome (vii) blackwater fever, (viii) snake and spider bites, (ix) Oxidant drugs in G6PD deficiency, (x) septicaemia e.g. due to Clostridium welchii, (xi) Thrombotic thrombocyto-penic purpura etc.

(c) ***Bacteria and Parasites*** :

(i) Bacteria may be seen by staining a smear of the centrifuged deposit of urine by Gram's method. Gram-negative bacilli are found in E. coli pyelitis.

(ii) In urine of women most often you find E. coli (escherichia coli) or S. saprophyticus (Staphylococcus). Trichomonas may be found.

(iii) Male urethritis might be caused by E. coli (most common) or N. gonorrhoeae (Neisseria) or Chlamydia etc.

(d) ***Deposit*** :

(i) Ca phosphate and Mg phosphate are found in alkaline urine. They are colourless deposits and are usually mistaken for pus. Phosphates dissolve on addition of dilute acetic acid but not the pus.

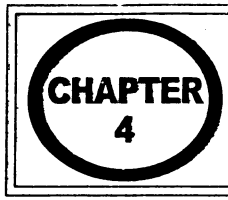
(ii) Ammonium, sodium and potassium urates are found in acidic urine. They are found in normal

individuals. They disappear on heating and re-appear on cooling.

(iii) Ca-oxalates are found in acid urine; these are envelop-like crystals.

Other investigations for the confirmation of disorders of the genitourinary system include :–

(i) Urine culture and antibiotic sensitivity.
(ii) Ultrasonography of kidney and urogenital system, down to bladder.
(iii) Cystoscopy and urethroscopy.
(iv) Intravenous pyelography or excretory urography.
(v) Micturating cystourethrography.
(vi) Retrograde pyelography.
(vii) Renal biopsy.



RESPIRATORY SYSTEMS

The respiratory system is concerned mainly with the oxygenation of blood by the lungs which are contained within a bony cage, the thorax or chest. The thorax is the region between the abdomen and the neck. It contains the heart and the great vessels, the lungs, the terminal part of the trachea and the bronchi; and is traversed by structures that pass from the neck to the abdomen. The bony thorax is bounded by 12 thoracic vertebrae behind, the sternum in front and 12 pairs of costae (or ribs and costal cartilages) which extend from the vertebrae behind to the sternum in front.

The respiratory tract is composed of –

(A) Upper respiratory tract which is up to the lower border of the cricoid cartilage and consists of (1) nose and nasal septum, (2) air sinuses, (3) nasopharynx, and (4) larynx.

(B) Lower respiratory tract which includes the trachea, the bronchi, the bronchioles and the alveoli.

(A) Upper Respiratory Tract :

(1) Nose and nasal septum–Examine with the help of a torch and a nasal speculum and–

(a) Look for any deviation of the nasal septum. This may cause constant nasal obstruction and mouth breathing leading to chronic upper respiratory tract infection.

(b) Inspect the nasal mucosa for congestion polyps or bleeding areas. If it is red-boggy and oedematous,it indicates nasal allergy or acute rhinitis. Look for any obvious swelling of the nose, or any obvious nasal discharge or bleeding through the nose.

In case of bleeding from the nose (epistaxis) an area in the anterior part of the nasal septum just beyond the mucocutaneous junction of the nasal vestibule, called the Littles area or Kiesselback's area should be carefully examined because in the majority of epistaxis bleeding occurs from this place.

(c) Note if there is excessive nasal discharge (rhinorrhoea) Paroxysmal rhinorrhoea(hay fever) is characterised by sneezing and excessive nasal discharge. It is the manifestation of an antigen antibody reaction; the antigen being dust, pollen grain, smoke etc. Inflammation of the nasal mucosa by Rhinoviruses is known as common cold which is characterised by stuffiness of and watering from the nose and sneezing but no fever. Vasomotor rhinitis is due to vasomotor imbalance in the blood vessels of the nasal mucosa. The triad of symptoms include nasal obstruction, bouts of sneezing, and excessive nasal discharge which is usually watery but occasionally thick and mucoid. On examination, the nasal mucosa is found to be slightly oedematous, often slightly bluish or pallid in colour and covered with excess secretions and at times a mucous polyp may be seen in the middle meatus if the vasomotor rhinitis is associated with allergic rhinitis. If sinus infection complicates the picture, mucopus will be visible in the middle meatus and postnasal space.

Search for the causes of epistaxis which are–

(I) Physiological–vicarious menstruation, change of climate (extreme hot) etc.

(2) Pathological–

(i) *Local causes :* Acute rhinitis, ulcers and polyps of the nose, foreign body in the nose, head injury, local trauma, neoplasms, separation of crusts as in rhinitis sicca or atrophic rhinitis.

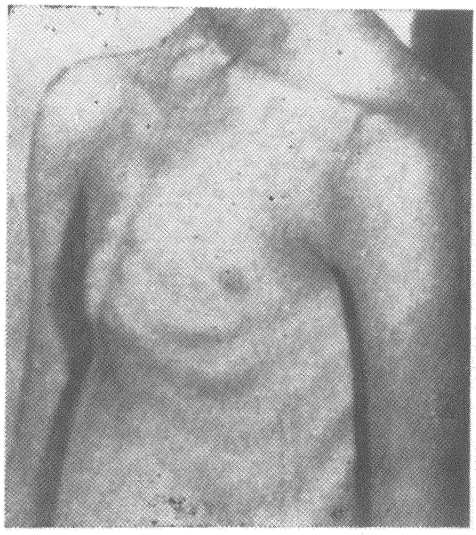


Fig. 2A : Funnel shaped chest.

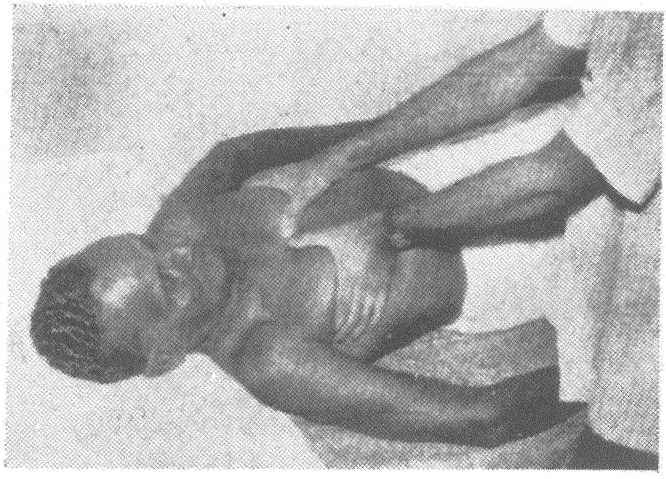


Fig. 2B ; Showing how to examine respiratory movements of the two sides of the chest.

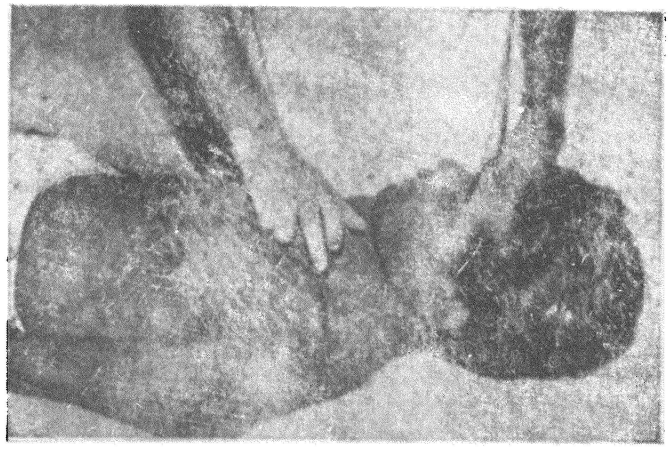


Fig. 2C : This is how clavicular percussion is done.

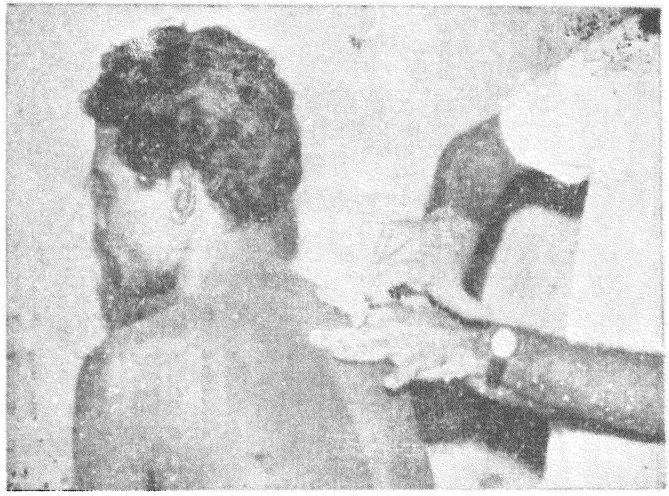


Fig. 2D : Demonstrating the percussion
of the back of the chest.

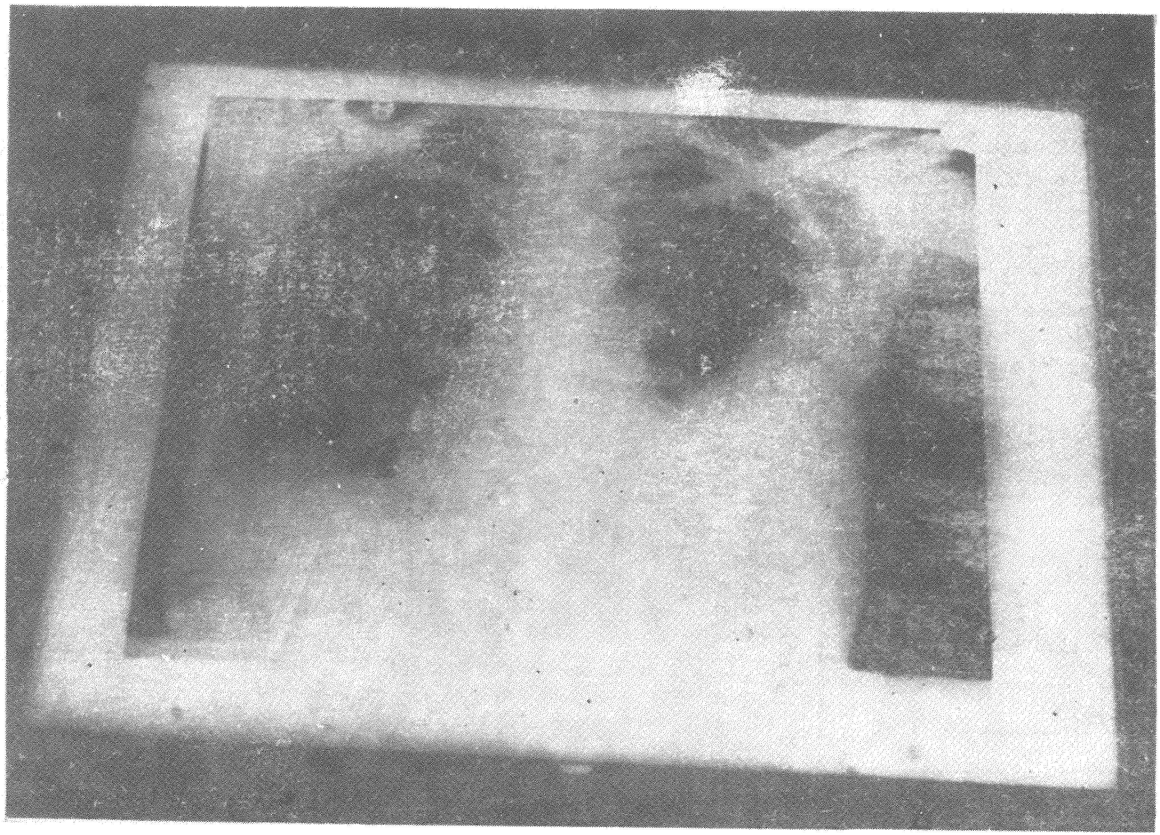


Fig. 2E : Left sided Pleural Effusion.

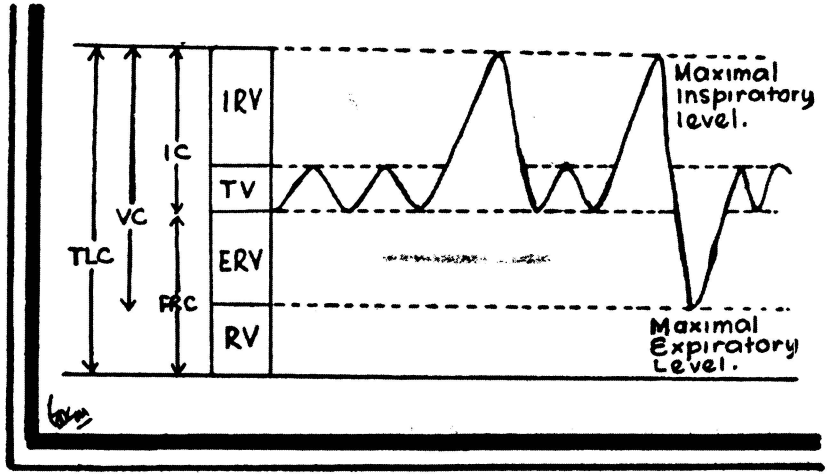


Fig. 2F : TLC : Total lung Capacity ; VC : Vital Capacity ; IC ; Inspiratory Capacity ; FRC : Forced Residual Capacity : IRV : Inspiratory Releive Volume ; TV : I : dal Volume ; ERV : Expiratory Reserve Volume ; RV : Residual Volume.

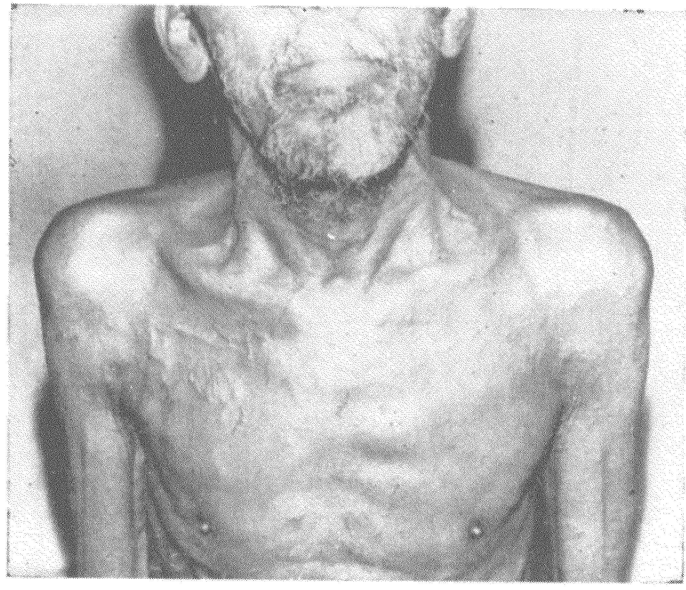


Fig. 2G : A case of superior Vena Cava obstruction by Bronchogenic carcinoma.

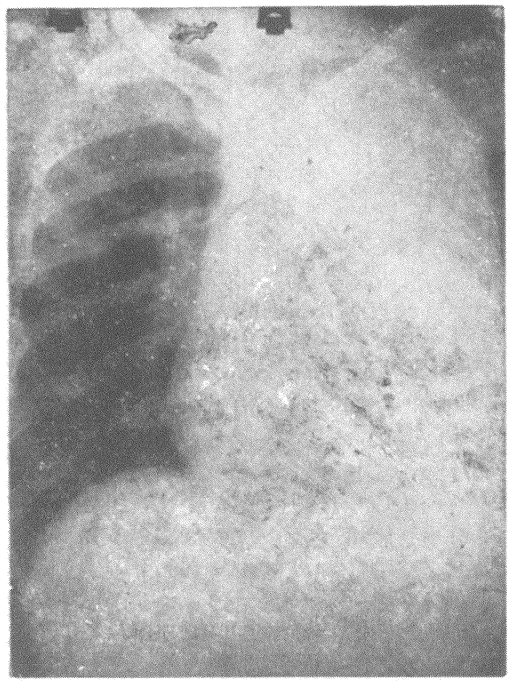


Fig. 2H : Massive pleural effusion of the left side.

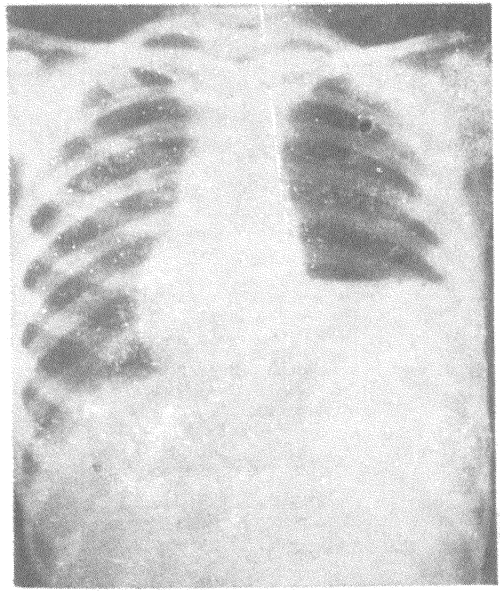


Fig. 2 I : Hydropneumothorax of the left side

(ii) *Systemic causes* : Hypertension, blood dyscrasias, lymphomas. hereditary haemorrhagic telangiectasia, prolonged anticoagulant therapy and rarely over-dose of drugs such as quinine and salicylates.

(d) Alae nasac–These usually work with the other accessory muscles of respiration when the latter are brought into play.

(II) Sinuses (frontal and maxillary air sinuses)–Gently press over the sinuses for tenderness and ask the patient to sit up and lean forward and note if he complains of any heaviness of head. Look for nasal regurgitation and palatal palsy by asking the patient to swallow water. These are found in bulbar palsy which may be due to bulbar poliomyelitis, diphtheria, brain tumours, disseminated sclerosis etc.

(III) Inspection of the tonsils and faucies should be done for diphtheritic patch, follicular tonsillitis etc.

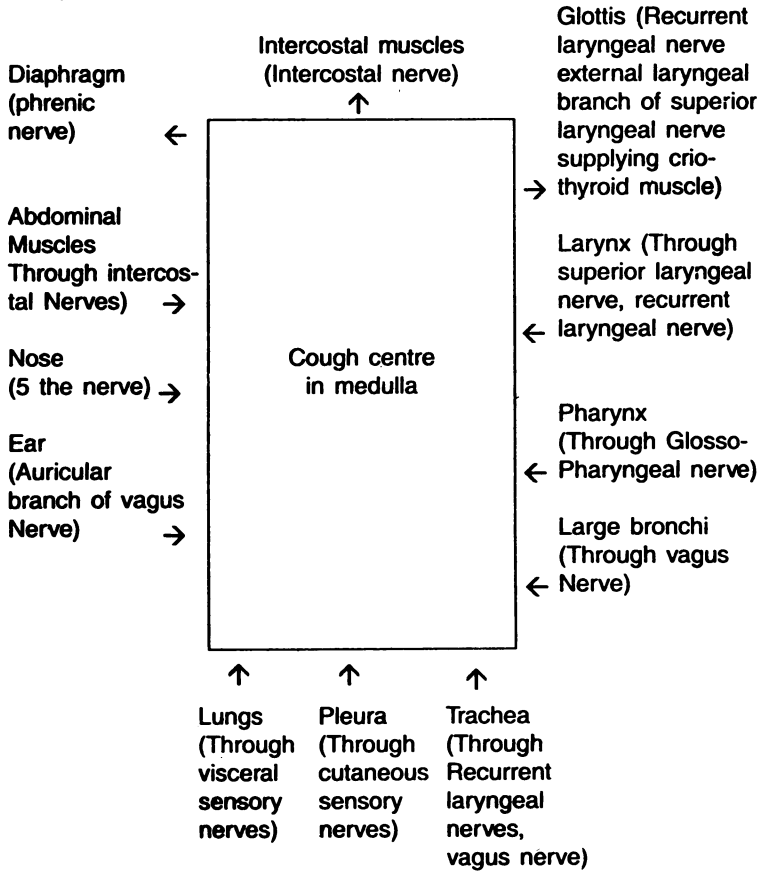
Note whether there is any stridor or hoarseness of voice. Stridor is defined as inspiratory difficulty in breathing which may be due to foreign body in the larynx, laryngeal oedema (in angioneurotic oedema) or involvement of larynx in diphtheria and in vocal cord palsy due to involvement of the recurrent laryngeal nerve. Constant stridor since birth may be due to laryngeal webs. Stridor in infants which becomes more pronounced during crying or becomes worse during respiratory infection is due to persistence of infantile appearance of epiglottis and aryepiglottic fold in exaggerated form. About the age of three these stridors disappear. Infantile larynx is relatively smaller than that of the adult and readily develops oedema and spasm during acute inflammation. Consequently a stridor (often termed as croup) is not a rare finding in infants during any respiratory infection or exanthems. Two very important causes of stridor

are acute laryngotracheo bronchitis and laryngeal diphtheria.

(IV) *Nasopharynx* should be carefully examined for any diphtheritic patch.

Cough : Cough is a forceful reflex or voluntary expulsion of the inspired air from the respiratory tract and one of the most frequent cardiorespiratory symptoms. It is an explosive expiration that provides a means of clearing the tracheobronchial tree of secretions and foreign bodies.

Afferent and efferent paths of cough and control of cough centre–



Cough may be :

(1) Dry–Acute tracheobronchitis, acute dry pleurisy.
(2) Wet–Resolution stage of pneumonia. Bronchopneumonia, bronchiectasis.
(3) Bovine–Recurrent laryngeal nerve palsy (e.g. due to bronchogenic carcinoma.)
(4) Hacking–Heavy smokers.
(5) Whooping–It is characterised by paroxysms of dry cough interrupted by a whoop.

The most sensitive areas stimulation of which initiates the cough mechanism are (i) bifurcation of the trachea (known as carina); (ii) upper part of the larynx, and (iii) the pharynx.

The cough centre is *depressed* (i) in deep unconsciousness, and (ii) by morphine and codeine administration.

Effects of cough : A paroxysm of violent cough may lead to–

(1) Severe vomiting.
(2) Sudden unconsciousness due to cerebral anoxia.
(3) Rupture of an emphysematous bulla resulting in spontaneous pneumothorax.
(4) Subconjunctival, retinal or bronchial haemorrhage. If cough is severe, cerebral haemorrhage may occur in susceptible individuals.
(5) Fracture rib in old age.
(6) Hernias and prolapse of the rectum or uterus.

Recurrent laryngeal nerve palsy :

Signs : (1) Bovine cough. (2) Stridor. (3) Hoarseness of voice.

Causes :

(1) Neurological–Bulbar poliomyelitis, motor neurone disease, vascular lesions of the brainstem, dephtheritic neuritis etc.

(2) Others–Carcinoma oesophagus, carcinoma thyroid after thyroidectomy, enlarged hilar lymph node secondary to bronchogenic carcinoma, aneurysm of the arch of the aorta, right subclavian aneurysm, after operation on lungs or heart etc.

Hoarseness of voice may be the presenting symptom of–

(1) Acute laryngitis–usually found in measles.
(2) Inhalation of irritant gas or smoke.
(3) Chronic laryngitis due to repeated attacks of acute laryngitis due to heavy smoking and excessive use of voice as in case of pleaders and actors.
(4) Obstruction at the vocal cord due to foreign bodies, diphtheria tetany, angioneurotic oedema and recurrent laryngeal nerve palsy etc.

(B) *Lower Respiratory Tract*

Anatomy

Surface making of the lungs (Fig. 2-1)

(1) Oblique fissure–It starts from the 2nd dorsal spine posteriorly and goes downwards, forwards and medially to the 6th costochondral junction.
(2) Transverse fissure–It start from the right 4th costochondral junction and proceeds transversely up to the midaxillary line and joins the oblique fissure.
(3) Upper border–2.5 cm above the medial third of the clavicle.
(4) Lower border–
On midclavicular line–6th rib.
On midaxillary line–8th rib.
On scapular line–10th rib.
(5) Lower limit of the pleura–
On midclavicular line–8th rib.
On midaxillary line–10th rib.
On scapular line–12th rib.

(6) The hilum of the lung lies opposite the spines of the 4th, 5th and 6th thoracic vertebrae at a site between the midline and the vertebral (i.e. medial) borders of the scapulae.

When seen from behind, greater part of each lung is composed of the lower lobes, only a minor area near the apex belongs to the upper lobes. But seen from the front, the middle and the upper lobes on the right side and the upper lobe on the left side occupy most of the area.

(7) Angle of Louis–At the junction of the manubrium stemi with the body of the sternum there is a well defined ridge called the angle of Louis. It is a very important anatomical landmark because–

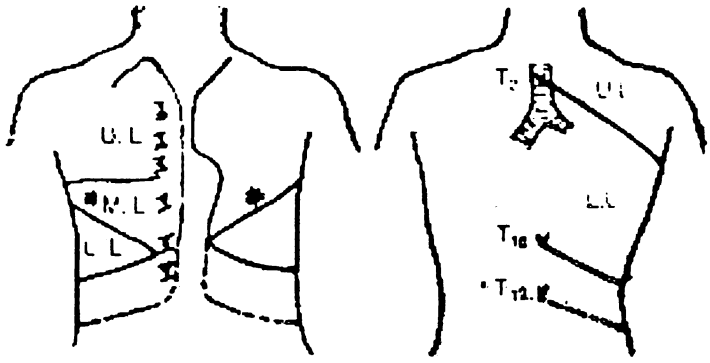


Fig. 2-1 : Surface markings of the lungs on the anterior and postenior chest walls UL–upper Lobe, KL-Middle Lobe. LL-Lower Lobe.

(a) it is the anterior level at which the trachea divides into its two main bronchi;

(b) behind, it corresponds with the disc between the fourth and fifth thoracic vertebrae;

(c) traced laterally, the transverse ridge that indicates the angle conducts the palpating fingers to the second costal cartilage and second rib.

The 12th rib is not always palpable and even may be absent. So the ribs and intercostal spaces should be counted from above downwards. First the sternal angle or the angle of Louis should be found out–and the rib that lies at its level laterally is the 2nd rib and the intercostal space just below this is the 2nd intercostal space. Count from this space or rib downwards.

Transverse and anteroposterior diameters of the chest are increased by the movements of inspiration. The vertebrosternal ribs move forward and upward during inspiration, thereby increasing the anteroposterior dimensions. They are also everted, around the anteroposterior axis through their ends, thereby increasing the transverse diameter. Elevation of the vertebrochondral ribs result in outward and backward movement to produce and increase in the transverse diameter only.

Muscles of respiration :

(1) Normal–External and internal intercostals, diaphragm.

(2) Accessory–Alae nasi sternomastoid, pectoralis, serratus anterior, trapezium scalenii.

Normal respiration–The rate is 18 to 20 per minute.

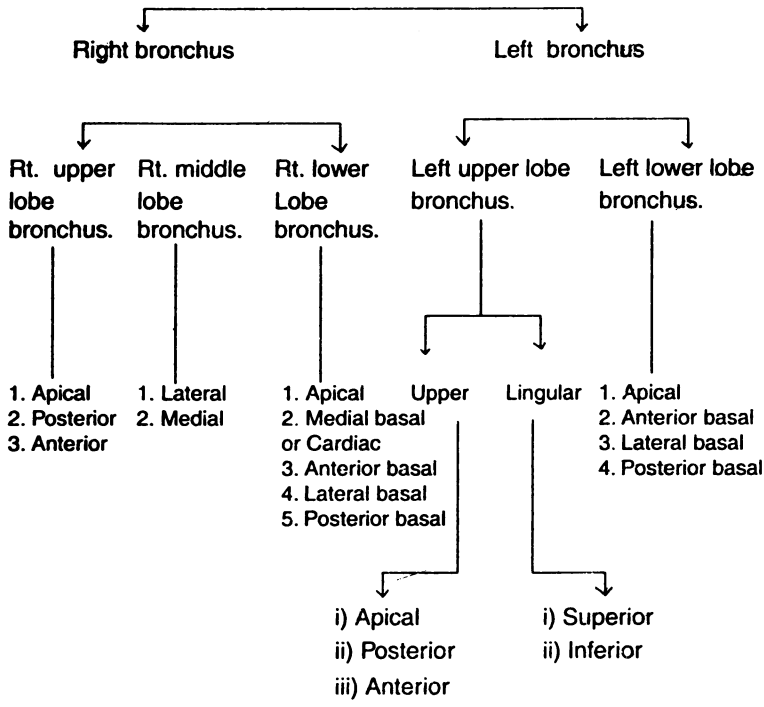
(1) Tidal air – 500 ml.

(2) Supplemented air during inspiration – 1500 ml.

(3) Supplemented air during expiration – 1500 ml.

(4) Vital capacity – Approximately 4,800 ml in males and 3,100 ml in females.

Divisions of bronchi



Physiology of the Lungs

(a) Primary function of the lungs is gas exchange–a process by which transfer of oxygen and carbon dioxide between environment and blood occurs. (b) Another function is regulation of hydrogenion concentration (which is also influenced by functions of the kidneys). The gas exchanges process can be considered to consist of four steps; (i) Ventilation, (ii) Gas transfer, (iii) Pulmonary blood flow, (iv) Blood gas transport.

(i) *Ventilation* : It is the mass movement of air into and out of the lungs and distribution of air within them. Rhythmic contractions of the respiratory muscles produce pulmonary ventilation by expansion of the thorax and the lungs.

By contracting, these muscles overcome both the elastic forces of the tissue as well as the resistance of the airways to the flow of air through them. The physiological dead space is not much greater than the anatomical dead space in the recumbent position, but in the erect position, physiological dead space is increased because of little blood flow to and gas exchange in the apical regions. Of the subdivisions of total volume of air in the lungs, those that need remembering are :– (a) Tidal volume – volume of air inspired or expired during breathing. (b) Vital capacity –The volume of air expelled by maximum voluntary expiration after maximum inspiration. (c) Functional residual capacity–The volume of air remaining in the lungs at the end of normal expiration. (d) Residual volume–Volume of air remaining in the lungs after maximum expiration.

The *vital capacity* is related to the size and development of the individual. Normal values have been mentioned above. It is *increased* in swimmers and divers and *decreased* in old age and diseases of respiratory apparatus e.g. poliomyelitis, pleural effusion, respiratory obstruction, pneumothorax, pulmonary fibrosis, emphysema and pulmonary oedema. It is also diminished in pregnancy and ascites.

(ii) *Gas transfer*: Oxygen and carbon dioxide exchange between alveolar air and capillary blood depends on :

(a) Distribution of ventilation and blood flow–of major functional importance in the distribution of ventilation in relation to blood flow. If the proportion of ventilation to blood flow in all the alveoli is neither uniform nor appropriate to the mixed venous and inspired gas composition, the arterial blood gas composition will not be normal.

(b) Diffusion–Total surface area of functional respiratory membranes is approximately 70 sq. metres and at any given moment the amount of blood present in the capillar-

ies of the lung is about 100 ml. This 100 ml of blood is effectively spread over the 70 sq. metres surface area. Diffusion of gases into pulmonary capillary blood depends upon the functional alveolar surface area and the distance through which the gases diffuse. Diffusion of carbon dioxide occurs almost instantaneously and more readily than that of oxygen because of its greater solubility. If the surface area for gas exchange decrease (e.g. emphysema) or the thickness increase (e.g. increased interstitial fluid), gas exchange is impaired.

(iii) *Pulmonary blood flow* : This is primarily determined by gravity. At the apex the arterial pressure is less than the alveolar pressure and no blood flow occurs. In the middle of the lung arterial pressure is more than the alveolar pressure which is again more than the venous pressure,– the blood flow is determined by the arterial alveolar pressure difference. At the base, the arterial and venous pressures are higher than the alveolar pressure–blood flow is determined by the arterial-venous pressure difference.

Under normal conditions, distribution of blood flow is matched to distribution of ventilation. Homeostatic mechanisms exist to maintain this relation, even in the presence of considerable maldistribution of air or blood. But they may be overwhelmed by severe widespread diseases when enough functioning lung tissue does not remain.

(iv) *Blood gas transport* : A detailed account of blood gas transport is beyond the scope of this book and students should consult text-books of respiratory medicine or physiology for the same.

Examination of lower respiratory tract :

INSPECTION

Inspection of the chest should be done after proper exposure in good light and warm atmos-

phere. Inspection of the chest may be carried out in the sitting, standing or lying down position. It is important that the patient be sitting or lying down absolutely straight. A slant will cause a curvature of the spine and in turn lead to apparent asymmetry of the thorax. The front of the chest is inspected systematically for size, shape, symmetry, respiratory movements and obvious swelling etc. and the back is also inspected similarly. Then the chest is inspected from the sides in profile; patient being seated on a tool; particularly for evidence of any kyphosis or lordosis or for any increase in the anteroposterior diameter of the chest as in emphysema. Finally the upper part of the chest is inspected standing behind the seated patient and looking from above downwards over the shoulders of the patient.

When the patient is recumbent, the chest should be inspected first from the foot end of the bed and later from its sides in profile, keeping the eyes of the examiner at the level of the anterior wall of the chest of the patient either by sitting on a tool or by kneeling beside the patient.

Following features must be noted in all cases :

(a) Size and shape of the chest.

(b) Presence of any asymmetry e.g. any prominence of any side of chest, any localised bulging any depression of the chest wall or any localised retraction.

(c) Respiration–its rhythm, rate, type, movement of both sides of the chest along with respiration, presence of any intercostal suction or retraction along with inspiration, and whether the accessory muscles of respiration are working.

(d) Additional features e.g. sternomastoid sign, and dilated veins over the chest wall etc. should be looked for.

I. *Shape of the chest*–The normal healthy adult chest is elliptical in cross-section, the transverse diameter being greater than the anteroposterior diameter (in a ratio of 7 : 5), more or less bilaterally symmetrical. In a normal chest the subcostal or epigastric angle is about 90. In males this angle is more acute than in females.

In childhood the chest is more or less circular in cross-section.

(i) *Pigeon chest*–It is characterised by an unduly prominent sternum; triangular shape of the chest; rickety rosary i.e., bead-like enlargements of the costochondral articulations particularly of the 4th, 5th and 6th ribs; and Harrison's sulci i.e., sulci or grooves extending transversely from the sides of the xiphoid process on either sides of the chest wall producing a transverse constriction on the thorax. Harrison's sulcus is caused by the depression of the ribs at the costal attachment of the diaphragm due to pulling in of the softened ribs by diaphragmatic contractions during respiration. Pigeon chest is found in rickets and recurrent lung infections in infants.

(ii) *Funnel chest* (pectus excavatum)–This comprises of depressed lower part of the sternum, prominent costochondral junction and diminished anteroposterior diameter of the chest.

Pectus excavatum is usually asymptomatic.

Dangers of severe funnel shaped chest are–

(a) Prevents proper ventilation leading to the development of corpulmonale in later life.

(b) Prone to develop repeated lung infections.

(c) Obstruction of right ventricular outflow tract by depressed sternum may lead to functional pulmonary stenosis and congestive cardiac failure.

Children who suffer from repeated lung infections can develop funnel shaped chest.

(iii) *Barrel shaped chest*–It has the following features.

(a) Chest cavity is held in the position of deep inspiration. It is circular in cross-section.

(b) Anteroposterior diameter is increased with marked kyphosis.

(c) Supraclavicular fossae are full.

(d) Intercostal spaces are prominent and ribs are wide apart and more horizontal.

(e) Subcostal angle is wider than normal.

(f) Venous girdle is present in the lower part of the chest.

Bilateral emphysema (centriacinar or panacinar) is the cause of barrel shaped chest.

(iv) *Alar chest*–This is characterised by drooping of the shoulder and the ribs are more obliquely placed with prominent shoulder blades and the chest looks tubular. It is found normally in some thin built persons.

(v) *Deformity due to kyphosis and scoliosis*– Kyphosis means backward bending of vertebral column with convexity posteriorly and concavity anteriorly. Causes of kyphosis are–

(a) Carries spine–There may be gibbus formation in late stage.

(b) Osteoarthritis.

(c) Occupational.

(d) Secondary to osteoporosis – e.g. after prolonged steroid therapy.

(e) Bilateral emphysema, ankylosing, spondylitis.

Scoliosis means the lateral bending of the vertebral column which may give rise to unilateral depression of the chest and is usually congenital in origin. Kyphosis and scoliosis may lead to an asymmetry of the chest wall and may diminish the size of the thoracic cavity and restrict pulmonary movements. Clinical and radiological displacements of the trachea and the apex beat may be found in cases of simple scoliosis. *Extensive unilateral fibrosis* or collapse of the lung in childhood can lead to scoliosis. Kyphos coliosis may be associated with *neurofibromatosis, poliomyelitis, Friedreich's ataxia, cerebral palsy* etc.

II. *Unilateral fullness or bulging of the chest*

Look whether there is any unilateral fullness of the chest. If present, it may be due to massive pleural effusion, empyema thoracis, pneumothorax, bronchogenic carcinoma etc.

III. *Localised fullness or bulging of the chest wall* can be detected in cases of empyema necessitates (due to accumulation of pus under the skin of the chest wall due to rib infection or empyema communicating with the chest wall), encysted pleural effusion, lung abscess, bronchogenic carcinoma, enlargement of one of the cardiac chambers, pericardial effusion, fibroma, lipoma and metastatic nodules of the chest wall, aortic aneurysm, surgical emphysema etc.

IV. *Unilateral depression of the chest* is a presenting feature of unilateral fibrosis and collapse of the lung.

V. Localised depression of the chest is encountered with segmental collapse of the lung, localised pul-

monary fibrosis and after rib resection in thoracotomy. Localised flattening of the upper part of the chest wall may be due to apical tuberculosis.

VI. *Visible veins*–Superficial veins in the chest wall are visible in superior vena caval obstruction and in azygos vein obstruction. In superior vena caval obstruction there is venous engorgement with oedema of the face and upper limbs. Superficial veins are engorged in the chest with flow of blood from above downwards, towards the inferior vena cava. This is known as the superior mediastinal syndrome or reversed congestive cardiac failure and is almost always due to malignant conditions e.g. bronchogenic carcinoma, lymphomas or leukaemias.

VII. *Accessory nipples* (Polythelia) Accessory nipples may be associated with the development of systemic and pulmonary hypertension, cardiomyopathy or congenital heart diseases.

VIII. *Sinuses over the skin of the chest*–These are common in tuberculous osteomyelitis of the rib and sometimes in cases of empyema after drainage, and also in actinomycosis of lung.

IX. *Herpes zoster*–It is a cluster of vesicles on an erythematous base of viral origin, distributed over an area of skin supplied by one or more spinal nerve segment. It manifests as severe chest pain and is most commonly unilateral.

X. *Intercostal suction*–It is produced either by positive atmospheric pressure pressing the lower intercostal spaces when the intrathoracic pressure in negative due to failure of air entry through upper respiratory tract as in vocal cord palsy (always

associated with stridor) or due to withdrawing of the lower posterolateral aspect of chest with each ventricular systole. The latter is due to adhesions of the parietal pericardium with the diaphragm in case of adherent pericarditis (Broadbent's sign).

XI. *Cardiac pulsation*–Apical impulse should be located carefully because displacement of the cardiac apex may reveal mediastinal shift due to underlying lung diseases as occurs in massive pleural effusion, empyema thoracis, pneumothorax and hydropneumothorax.

The apex beat is shifted to the opposite side of the lesion in the above mentioned cases whereas pulmonary fibrosis and collapse draw the apex beat to the side of lesion.

Epigastric pulsations should be looked for in a case of chronic corpulmonale as there is right ventricular hypertrophy with a low diaphragm due to emphysema.

XII. *Movements of the chest*– Look for the movements of the two sides and both upper and lower parts of the chest on quiet respiration. The movement may be restricted on both sides or on one side only.

Causes of bilateral diminution of chest movement:

(1) Bilateral emphysema.

(2) Bilateral hydrothorax.

The unilateral restriction of movement of the chest may be observed in pleural effusion pneumothorax, hydropneumothorax, fibrosed lung, collapse of the lung and bronchogenic carcinoma.

Respiratory movement–Type of respiration is next noted. In men the diaphragm moves more freely than the inter-

costal muscles and its downward excursion with inspiration causes free movements of the abdominal wall. Thus it is a predominantly abdominal type of respiration. This type of breathing is also seen in children. On the other hand, in women movements of the intercostal muscles are greater than the movements of the diaphragm. So the respiration is predominantly of thoracic type. Various combinations of thoracic and abdominal breathing may be seen in health. Normally during respiration the two sides of the chest should move symmetrically and simultaneously.

The rate of respiration for a normal adult is 18-20 per minute. It is higher in childhood, and at birth it is about 40 per minute. Increased rate of respiration is called *tachypnoea.* It is an important sign of pulmonary disease. The rate and depth of respiration should be observed without the patient's knowledge as consciousness of this act tends to make it irregular. Depth of respiration should also be observed. An increase in rate or *tachypnoea* and increase in depth or *hyperpnoea* is found when there is an increased demand for ventilation.

The ratio between the respiration and pulse is 1 is to 4 (18 respirations to about 72 pulse beats per minute). In pneumonia the rate of respiration is very much increased. In narcotic poisoning the respiration rate is much depressed changing the respiration to pulse ratio to about 1 is to 7. The depth of breathing or tidal volume is about 500 ml at rest. This can be measured with the help of a spirometer.

Types of abnormal breathing

(a) Cheyne-Stokes breathing : The amplitude progressively depends to a maximum and then gradually decreases to a period of apnoea and after this short apnoea the whole cycle is repeated. It is due

to diminished sensitivity of the respiratory centre to CO_2, and is most conspicuous when the patient is unconscious or is in sleep. It is rarely found except in gravely ill patients. It is found in severe cardiac failure renal failure. pneumonia, narcotic poisoning and increased intracranial pressure.

(b) Stertorous breathing : It is a noisy breathing found in comatose and dying patients. The noise is produced by the vibrations of the soft tissues of the nasopharynx, larynx, and cheeks. It is produced by the laxity of the soft palate and is found in patients who are unconscious as a result of severe brain damage, caused by trauma, haemorrhage or infraction.

(c) Kussmaul breathing (air hunger) : In this, there is an increase in both depth and rate of breathing; the hyperventilation being a conspicuous feature of metabolic acidosis. It is found in diabetic coma, uraemia cerebral tumour, and in some cases of acute alcoholism. The hyperventilation is often without dyspnoea or consciousness of laboured respiration.

(d) Asthmatic breathing or wheezy respiration : Here the expiratory phase is unduly prolonged associated with an expiratory wheeze. It is typically found in bronchial asthma.

Dyspnoea : It is a subjective state in which the effort of breathing reaches consciousness. It is defined as the undue awareness of every respiratory effort. Normally breathing is an involuntary act carried out by the voluntary muscles. Hyperpnoea is a state where the volume of ventilation or depth of respiration is increased and

tachypnoea is a state where the rate of respiration is increased.

Orthopnoea or upright breathing : In this, the patient remains in a sitting posture because the dyspnoea increases greatly in recumbency.

Causes of dyspnoea : **Respiratory** : Lobar and bronchopneumonia.
Pneumothorax.
Pleural effusion.
Bronchial asthma.
Chronic bronchitis with emphysema.
Bronchogenic carcinoma.
Advanced pleural endothelioma.
Fibrosing alveolitis.
Interstitial pulmonary fibrosis.
Pulmonary embolism.
Congenital cystic lung diseases, obstructed air passage anywhere from nostrils up to the bronchioles by a foreign body or pressure by enlarged mediastinal lymph gland over the lower respiratory tract, bilateral extensive pulmonary tuberculosis and miliary tuberculosis.

Cardiovascular : Left ventricular failure.
Mitral stenosis ("critical" uncompensated).
Pulmonary hypertension.
Constrictive pericarditis.
Pericardial effusion.
Superior mediastinal syndrome.
Rapid cardiac arrhythmias.

Blood disorder : Severe anaemia.

Metabolic : Diabetic acidosis, thyrotoxicosis, high fever etc.

Neurological : Poliomyelitis.
Increased intracranial tension.
Acute ascending myelitis.
Anoxia : High altitude (pulmonary oedema).
Severe exercise or exertion.
Mechanical : Huge ascites
Psychogenic : Hysteria.

PALPATION

During palpation the patient's head must be placed in a midline and neutral position. The patient must be lying down straight. The palpation should be very gentle and systematic. At the outset it would be wise to put the hand on that part of the chest where there is any obvious swelling or where the patient is complaining of pain.

Assess the severity of the pain or tenderness at that particular site by looking at the face of the patient. This helps the examiner to avoid causing unnecessary pain to the patient.

This pain may be due to

(i) Inflammatory conditions e.g. abscesses or boils on the chest wall.
(ii) Injury to the chest wall e.g. fractured ribs.
(iii) Costochondritis.
(iv) Pre-eruption stage of herpes zoster.
(v) Secondary malignant deposit (s) on the ribs.
(vi) Acute dry pleurisy.
(vii) Pericarditis.
(viii) Intercostal myalgia.

After this the following points should be noted carefully:

I. *Movement :* Normally the two sides of the chest move equally. This is assessed by placing the palms laterally on the chest in such a manner that

the thumbs touch each other at the midline in front of the chest with a fold of skin between the thumbs.

Note the separation of the thumbs with each inspiration. The procedure should be repeated from above downwards and also similarly in the back.

The movement of the apex of the lungs are determined by placing the palms on the shoulders in a way such that the thumbs are touching each other on their radial borders over the nape of the neck and the fingers are lying flat on the supraclavicular fossa. The patient should be in a sitting posture while the procedure performed from the back. If the patient is lying down the hands of the examiner should be placed over the two clavicles from the front. But noting the extent of lifting of the hands during deep inspiration the expansion of the apical region of the chest is assessed.

The clinicial should now measure the expansion of the chest with a tape. The expansion of the chest during inspiration is at least 2 inches in normal adult individual.

II. *Trachea* : Extend the neck and lightly grip the trachea with the help of index and middle fingers in the suprasternal notch and decide whether it is placed centrally or deviated to one side in relation to the suprasternal notch and sternomastoid muscles. Deviation of the trachea to one or the other side of the midline is an important sign of *shifting of the superior mediastinum*. Another way of examining the position of the trachea is to push

gently and carefully the index finger of the examiner's right hand directly backwards in the suprasternal notch;–normally the trachea is felt in the midline. If a tracheal deviation to any side is present the finger will slide down along the other side of the trachea.

The sternomastoid muscle becomes prominent (*sternomastoid sign*) on the side of deviation of trachea as both of these are covered with the same endotracheal fascia.

Normally the trachea remains slightly diviated to the right. In upper lobe lesions a mediastinal shift is judged by the deviation of the trachea and not by the apex beat.

In dextrocardia the trachea is central in position but the apex is on the right side; in dextroversion due to extracardiac causes, the trachea and apex beat will be shifted to the right. But the term dextrocardia is now used for any acquired or congenital causes where the heart is in the right hemithorax and the apex palpable on the right.

The trachea may be shifted in parenchymal lesions of the lungs or pleura and also in some local conditions such as enlargement of a lobe of the thyroid, mediastinal growth, aneurysm of the arch of the aorta etc.

Shifting of the trachea to the *opposite side* of the lesion occurs in pleural effusion, (massive) pneumothorax, hydropneumothorax, empyema, bronchogenic carcinoma with pleural effusion etc.

Tracheal shift may be to the *same side* of the lesion in fibrosis and collapse of the lungs. In combined pleural effusion and collapse of the lung

of the same side the upper part of the trachea is pulled to the same side due to collapse while the lower part of the trachea may be pushed to the opposite side due to pleural effusion. This is known as 'S' shaped trachea, found in bronchogenic carcinoma.

Tracheal tug (Oliver's sign) is the up and down movement of the trachea with each cardiac cycle found in aneurysms of the arch of the aorta as the left bronchus is then pulled down by the arch of the aorta during each cardiac systole. This sign can be elicited by raising the chin of the patient and applying a firm upward traction on the trachea with two fingers placed on the borders of the cricoid cartilage. A downward pull or tug on the trachea is felt with each heart beat.

III. *Apex beat* : Position of the apex beat offers information about the shifting of the lower mediastinum in the diseases of respiratory system.

Palpate the apex to find out the exact site. Normally it is in the left 5th intercostal space half an inch inside the midclavicular line in adults and in the left 4th space in children.

The apex may be shifted to the left in cardiac diseases with left ventricular hypertrophy or dilatation e.g. mitral regurgitation aortic stenosis, hypertension, aortic regurgitation, cardiomyopathy etc.

Pulmonary diseases are reflected in the form of shifting of the apex to the same side of the lesion in fibrosis or collapse of a lung and scoliosis; and to the opposite side of lesion in pleural effusion

pneumothorax, hydropneumothorax and empyema thoracis.

Scoliosis alone may displace the cardiac impulse or apex beat. The commoner form of scoliosis with its convexity to the right side displaces the apex beat to the left side and vice versa.

The apex beat may be palpable on the right side in dextrocardia.

IV. *Vocal fremitus* :

Definition : These are the vibrations of the sounds produced at the vocal cord, transmitted by the larynx, trachea, bronchi, bronchioles and alveoli and picked up by the palm of the clinician from the chest wall. In other words, these are palpable vibrations of the chest wall which are produced during the act of phonation.

Method : The palm of the hand is placed flat on the chest. The patient is asked to utter the word 'ninetynine' repeatedly and clearly keeping the intensity and pitch of the voice constant, at the same time the conduction of the vibrations of vocal cords, are felt over the two sides of the chest. Vocal fremitus over equivalent areas of the two sides of the chest are compared for relative intensities. Only one palm is used on both sides of the chest in order to exclude the possibility of an unequal sensitivity of the two palms.

Remember that the vocal fremitus is slightly more on the right infraclavicular regions as the right bronchus is close to the chest wall and is slightly less over the bare area of the heart which remains close to the left middle part of the chest wall.

The palm should be placed anteriorly over the

upper, middle and lower zones of the chest, laterally below the axilla and posteriorly on the interscapular and infrascapular regions.

The bare area of cardiac dullness should be skipped during palpation.

The causes of *increased* vocal fremitus are (i) consolidation of the lung, and (ii) a big superficial cavity with patent bronchus : due to the surrounding consolidation.

Diminished or *absent* vocal fremitus can be elicited on one side in (a) pleural effusion, (b) pneumothorax, (c) hydropneumothorax, (d) fibrosis of lung, (e) thickened pleura; and *bilaterally* in (a) chronic asthmatic bronchitis, (b) bronchopneumonia, (c) emphysema (d) bronchial asthma etc.

In pleural effusion the vocal fremitus is diminished or absent, though fluid is not a bad conductor of sound or vibration; and is due to a failure of conduction of the vocal fremitus by the collapsed lung itself. In fact fluid is a good conductor of sound or vibrations.

V. *Rhonchial fremitus* : *Rhonchi* are the sounds produced by passage of air through the narrowed lumen of the bronchi or bronchioles in chronic bronchitis or bronchial asthma. The vibrations produced by these sounds on the chest wall may also be palpated by the palm of the hand. These are called Rhonchial fremitus–found in bronchial asthma and severe chronic bronchitis.

VI. *Friction fremitus* : Whenever the opposing surfaces of the visceral and parietal pleura are roughened by inflammation as in dry pleurisy, a friction sound

or rub is produced during the phases of respiration. This is due to a friction of the two roughened surfaces of the inflammed pleura over one another. When present this sound or rub produced a vibration or sensation which can be felt by a palm placed on the chest wall. This is called the Friction fremitus or palpable pleural rub.

Palpable coarse crepitation :

Crepitations are discontinuous, bubbling or crackling sounds produced by air traversing the alveoli, bronchi, or cavities filled with liquid secretions. When the crepitations are coarse enough they may be palpable by the palm.

PERCUSSION

Method of percussion :

(1) The middle finger of the left hand is placed firmly and flatly on the part percussed. This is the pleximeter finger.

(2) No space should be left between the pleximeter finger and the skin.

(3) Back of the middle phalanx of the pleximeter finger is struck by the tip of the middle finger of the right hand; the percussing finger.

(4) Strokes should be delivered from the wrist and finger joints, not from the elbow.

(5) The percussing finger should be bent in such a way that when the stroke is made its terminal phalanx strikes the middle phalanx of the pleximeter finger perpendicularly.

(6) As soon as the stroke is made, the striking finger should be taken off.

(7) The wrist joint must move loosely and the stroke should not be too heavy unless the percussion is done over a solid viscus.

(8) The tip of the percussion finger should be away from the examiner.

Three vital principles of percussion :

(1) Percuss from a resonant to a dull area.

(2) The long axis of the pleximeter finger should be parallel to the edge of the organ which is to be delineated.

(3) The pleximeter finger should be in close contact with the chest wall. Strokes should be made not more than 2 or 3 times and must be light except over viscera.

Types of percussion :

(1) Diagnostic percussion–The main purpose of percussion is to determine the state of the underlying tissue such as the lungs or the pleura etc.

(2) Topographical percussion–Percussion to delineate the boundaries of a particular organ.

(3) Tidal percussion–Percussion of the lower border of the lung at the heights of deep inspiration and expiration.

Areas to be percussed :

(1) Directly over the most prominent part of the clavicle without using a pleximeter finger and only with the percussing finger.

(2) Along the midclavicular line, from above downwards. On the right side find out the upper border of the liver dullness and on the left side up to the upper limit of the area of cardiac dullness. Map out

the area of the cardiac dullness and then find out the upper border of the splenic dullness.

(3) Along the midaxillary line, go on percussing up to the 8th space from the axilia. During this technique the patient's hands should be kept above his head.

(4) Percuss over the back of the chest between the two scapulae in a bat-wing fashion till the infrascapular region is reached; then percuss from above downwards up to the 10th space. The patient should sit up and lean forward with his hands crossed on the knees.

(5) Traube's space. It is a triangular space bounded laterally by the spleen medially by the left lobe of the liver and above by the inferior surface of the diaphragm. It is resonant because of the fundus of the stomach and is obliterated by a left sided pleural effusion, an enlarged left lobe of the liver, an enlarged spleen and a carcinoma of the fundus of the stomach.

(6) Kronig's isthmus is the area of resonance over the shoulder bounded medially by the neck muscles and laterally by the shoulder joint. This represents the apex of the lung. Dullness of this area indicates an apical tuberculosis or a bronchogenic carcinoma of the apex or Freidlander's or staphylococcal pneumonia.

Characters of the percussion note :

(1) *Tympanitic* : It is the variant of hyperresonant note found over a hollow viscus containing air e.g. stomach and is found in–

(i) an open pneumothorax.

(ii) over a superficial cavity with a patent bronchus.
(iii) over an emphysematous bulla.

(2) *Hyperresonant* : It is an increased resonate note where there is obliteration of the hepatic, cardiac or splentic dullness and mimics the note elicited over the lungs after a deep inspiration in normal individuals. Pathologically it is found bilaterally in emphysema and lung cysts. In the diagnosis of emphysema the increased limits or resonance with loss of hepatic and cardiac dullnesses are more important than the quality of the resonance.

(3) *Resonant* : It is the usual note produced by the air present in multiple alveoli of the normal lung parenchyma separated by numerous septa.

(4) *Impaired resonance* : The normal resonance is slightly diminished by pulmonary tuberculosis especially of the apical area, patchy fibrosis of the lung and thickened pleura. It is normally present at the upper limits of the liver and splenic dullnesses. It may be found at the bases of the lungs in pulmonary oedema or in small bilateral effusions.

(5) *Dull note* is found over consolidations, pulmonary tuberculosis, atelectasis, thickened pleura, lung abscess, bronchogenic carcinoma, pleural endothelioma etc.

(6) *Stony dullness denotes* dull percussion note with a peculiar feeling of resistance in the percussion finger and can be elicited in pleural effusion over the fluid in hydropneumothorax and empyema.This change of resonant note to dullness is due to a

superimposition of fluid or solid media between the lung tissue and the percussing finger.

Skodaic resonance. It resembles the resonance heard by percussing a wooden box. Found just above the upper level of a moderate pleural effusion.

Tidal percussion : Both lungs are percussed posteriorly along the infrascapular line downwards in full inspiration and also after full expiration. Normally there is an increase in the area of resonance by one space during full inspiration due to movement of the diaphragm and downward excursion of the normal lungs. The findings may be–

(1) A little change in resonance on inspiration and expiration in bilateral emphysema as the hypertrophied lung has restricted the diaphragmatic movement.

(2) A dullness both on inspiration and expiration in cases of basal pleurisy and basal pneumonia.

(3) The note may be dull on inspiration and resonant on expiration (paradoxical resonance,–seen in diaphragmatic palsy.)

AUSCULTATION

The following should be noted during an auscultation of the chest :

(1) Character of the breath sounds on both sides of the chest.

(2) Character of vocal resonance with comparison between the two sides.

(3) Added sounds, if present.

The diaphragm should be placed firmly over the chest.

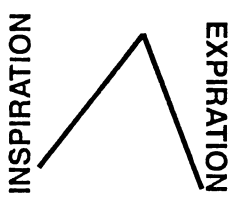
Ask the patient to breathe regularly and deeply without making any noise. Shivering of the patient may jeopardise the utility of auscultation.

Breath sounds

These are the sounds produced by the passage of air through the respiratory tract up to the alveoli and picked up by the stethoscope placed over the chest.

Types of breath sounds

(A) **Vesicular breath sound**



Features. (1) It is heard over the normal lung tissues (typically in infraaxillary inframammary and infrascapular areas).

(2) Intensity of the inspiration is greater than that of the expiration.

(3) Duration of the inspiration is more than that of the expiration because–

(i) Air is moving away from the chestpiece.

(ii) Intensity of the sound becoming low pitched from a high pitch as the air is moving from smaller bronchioles to bigger bronchi.

(iii) Expiration is a passive process.

(4) There is no gap between the inspiration and the expiration.

(5) It is rustling in character and low pitched.

(6) Expiration is heard only in its earlier part.

Variations :

(1) Puerile : Intensities of both inspiration and expiration are more than normal though the ratio is maintained. It is found normally in thin built children where the lung is very close to the chest wall.

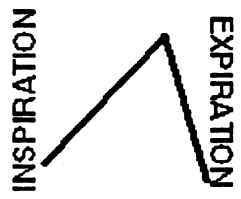
(2) Jerky : Interrupted or cog wheel breathing, found in nervous or hysterical individuals. It may also be observed in early pulmonary tuberculosis.

(3) Vesicular breathing with prolonged expiration. In this type the duration of expiration is more or less equal to that of inspiration. There is no gap. The cause of the prolongation of expiration is due to a partial obstruction of the bronchial tree as in bronchial asthma acute and chronic bronchitis, emphysema and early pulmonary tuberculosis. The increased duration of expiration is explained by the fact that the whole of the expiratory phase of respiration becomes high pitched as a result of narrowing of the lumen of the bronchioles and bronchi either by spasm or inflammatory exudates.

The pitch and intensity of the breath sounds diminish if two media prevail between the patent bronchus and the chest piece of the stethoscope e.g. air and fluid in moderate pleural effusion of solid and fluid in consolidation with pleural effusion. If the medium is single, be it fluid, solid or air, the bronchial breath sound can be transmitted to the chest piece in massive pleural effusion, consolidation or massive pneumothorax.

Breath sounds may be *diminished* or *absent* in thickened pleura pleural effusion, empyema, lung abscess pneumothorax, hydropneumothorax, pulmonary tuberculosis at the apex, bronchogenic carcinoma, fibrosis or collapse of lung, over the lung bases in left ventricular failure, huge ascites and liver abscess etc. The last two disorders produce collapse of the lung bases by pushing up the diaphragm.

(B) Bronchial breath sound



This is produced normally by the passage of air through the trachea and bronchial tree.

Features

(1) Typically heated over the trachea and infraclavicular and interscapular regions.

(2) Intensity of the expiratory sound is more than that of inspiration. The expiratory sound is also of high pitch.

(3) The duration of inspiration is equal to that of expiration.

(4) There is a gap between the inspiration and the expiration.

(5) It is blowing or aspirate in character and harsh in quality.

Varieties

(1) Tubular : It is a *high piched* bronchial breathing due to passage of air through the small bronchioles and conduction of the same by a solid media e.g. consolidation and is usually associated with aegophony.

(2) Cavernous : It is a *low pitched* bronchial breath sound heard over fibrotic lung tissues or a superficial moderately big cavity with a patent bronchus and is not associated with aegophony.

(3) Amphoric : It is a special variety of the high pitched bronchial breathing which resembles the sound produced by blowing air across the mouth of a jar. There is a tinge of metallic character e.g. tension pneumothorax and very large superficial cavities with patent bronchus.

(C) Bronchovesicular breath sound

It is a breath sound combining the characters of the vesicular and the bronchial breath sounds to some extent. It is heard when a breath sound from a superficial bronchus

is transmitted through the normal lung tissue. In a healthy individual it is typically heard near the roots of the lungs at the back, at the upper part of the right lung about an inch below the clavicle and the expiratory sounds have a more bronchial character than the inspiratory sounds.

Added sounds (adventitious sounds)

These may be dry, moist, or pleural origin.

(A) Dry sounds (rhonchi)

These are prolonged, uninterrupted high pitched sounds arising in the bronchi or bronchioles due to partial obstruction of the lumen by mucosal swelling in acute and chronic bronchitis, viscid secretions in bronchiectasis, spasms in bronchial asthma or a partial infiltration of the bronchial lumen by bronchogenic carcinoma.

Rhonchi may be high pitched, produced in smaller bronchi and of squeaky quality–known as *sibilant rhonchi.* It may be low pitched, produced in larger bronchi and of snoring quality–known as *sonorous rhonchi.*

Localised rhonchi are found in bronchogenic carcinoma and generalised rhonchi are heard in chronic bronchitis and bronchial asthma.

(B) Moist sounds (crepitations)

These are discontinuous, crackling or bubbling sounds produced either in alveoli, terminal bronchioles or cavities. They are produced only in the presence of liquid secretions.

Classification–

(1) Fine crepitations–These are produced when the stickly walls of the alveoli separate by the passage of air during the last phase of inspiration and are heard in early stages of consolidation or lobar pneumonia, acute pulmonary oedema, pulmonary tuberculosis and bronchopneumonia. Fine crepitations occur only near last part of inspiration and indicate the presence of exudates in the

alveoli of a particular region. It is also heard at the bases of the lungs in left heart failure.

(2) Coarse crepitations–These are produced in the bronchi, bronchioles, alveoli or in the big superficial cavities due to the presence of exudates and are heard in both phases of respiration in bronchiectasis, bronchopneumonia, chronic bronchitis, resolution stage of lobar pneumonia, lung abscess and cystic diseases of the lungs.

Localised crepitations are heard in pneumonia, lung abscess and/or cavity, pulmonary tuberculosis and *generalised* crepitations are characteristic of pulmonary oedema, bronchiectasis and cystic diseases of the lungs. When coarse crepitations are restricted to the lung bases the possibility of bronchiectasis or fibrosing alveolitis should be kept in mind.

Post-tussive crepitations–These are the crepitations brought out by coughing and are diagnostic of superficial tubercular cavity.

Normally the crepitations are not easily heard as the secretions remain deep in the cavities and are only brought out by coughing when the secretions become superficial.

(C) Added sounds of pleural origin.

Pleural rub–It is a leathery or creaking sound produced by movements of an inflammed and roughened visceral pleural over the parietal pleura.

Features : (1) It can be heard in any phase of respiration.
(2) Rough, grating in quality.
(3) More or less localised.
(4) Disappears when the breath is held.
(5) No change in character after coughing.
(6) On pressing the chest-piece over the chest wall, the intensity increases.
(7) May have relation with the cardiac cycle in case of pleuropericardial rub.
(8) In some cases this pleural rub may be

palpable when it is called friction fremitus.

Causes : (1) Acute dry pleurisy
(2) Over an area of consolidation due to adjacent pleuritis.
(3) Over an area of pulmonary infraction due to adjacent pleuritis.
(4) Over an area of bronchiectasis or lung abscess or bronchogenic carcinoma due to adjacent pleuritis.
(5) Maliganancy of pleura e.g. pleural endothelioma.

Causes of dry pleurisy.

(1) Pulmonary.
  (a) Pulmonary tuberculosis.
  (b) Bronchogenic carcinoma.
  (c) Pulmonary infraction.
  (d) Pneumonia.
  (e) Bornholm disease (due to coxsackie B virus.)
(2) Inter lobar.
  (a) Pulmonary tuberculosis.
  (b) Lobar pneumonia.
  (c) Pulmonary infraction.
(3) Diaphragmatic.
  (a) Subdiaphragmatic abscess.
  (b) Liver abscess.
  (c) Perihepatitis.
  (d) Perisplenitis.
  (e) Perinephric abscess.
  (f) Pyogenic peritonitis.

Signs of diaphragmatic pleurisy

(a) Pain radiates to the tip of the shoulder (4th cervical segment irritation)
(b) Abdominal pain and tenderness over the
  (i) epigastric and hypochondriac regions.
  (ii) area or a point 2" away from the midline on the right side in the subcostal

plane. This is known as "Bouton diaphragmatique of Gueneau de Muss."

(c) Hiccough.

(d) Restricted movements of the diaphragm on screening.

Hippocratic succussion or *succussion splash* : It is the splashing sound heard when the chest of the patient is shaken vigorously by the examiner. It can be heard even with the unaided ear. Normally it can be heard over an empty stomach just after taking any liquid e.g. a glass of water. When heard over the chest it is always pathological which may be due to hydropneumothorax or herniation of the stomach into the thoracic cavity through the diaphragm. It is also heard in pyloric stenosis.

VOCAL RESONANCE

The procedure is the same as that of vocal fremitus but here we hear the resonance with the help of the chest-piece.

So this is nothing; but auscultation of the laryngeal vibrations on the chest wall.

Vocal resonance being the auscultatory equivalent of vocal fremitus the same principle is involved in the mode of production, transmission, elicitation and abnormalities of both phenomena.

How to elicit : The patient is asked to repeat the word 'ninety nine' in a constant tone and voice and symmetrical areas on both sides of the chest are auscultated alternately, starting from the upper zone, up to the lower zone, on the front, sides and back.

Types :

Normal. It generally conveys the impression of the sound being produced at the chest-piece. It is heard as an indistinct rumbling sound where the individual syllables are blurred and indistinguishable.

Bronchophony : The sound is increased and it seems to arise from the ear-piece. It is found in lobar pneumonia tubercular consolidation and above the level of the fluid in some cases of pleural effusion. Bronchophony and bronchial breathing are usually present in similar cases.

Whispering pectoriloquy : If the bronchophony becomes so intense that the acute syllables are heard distinctly the term pectoriloquy is used. Here the sound is so much increased that it seems to be spoken right into the ear even with a whisper. It is detected in consolidation, a superficial big cavity with a patent bronchus, collapse of the lung with a patent bronchus and above the level of the fluid in pleural effusion.

Aegophony : This is nothing but the high pitched nasal intonation or bleating character of the voice characteristically found just above the level of a pleural effusion and in some cases over an area of consolidation. It resembles the bleating of a goat and is due to the interceptions of the low pitched elements of the sound and the fluid level.

Pneumothorax click

This is a sharp sound synchronous with the cardiac systole if there is air under tension inside the mediastinal pleura below the hilum.

Coin sounds (bruit d' airain : airain means brass) : When a metallic coin is placed over the affected side of the chest on the posterior wall and is percussed with a second coin a high pitched tympanitic or metallic sound can be heard by placing the chest piece of the stethoscope on the anterior wall of the chest of the affected side. It is also called brass sound, bell sound, or bell tympany. It is frequently found in pneumothorax. *Causes of pneumothorax* include rupture of subpleural emphysematous bulla or the pulmonary end of a pleural adhesion;

rupture of a subpleural tuberculous focus into the pleural space; benign spontaneous pneumothorax, following staphylococcal lung abscess, pulmonary infraction, bronchial carcinoma etc. Pneumothorax can also occur following a stab injury, fracture rib or any chest injury or may be *iatrogenic* following a lung or pleural biopsy, thoracocentesis and surgery in the lower neck.

Spontaneous pneumothorax is also associated with congenital cysts, pneumoconiosis particularly that associated with aluminium (bauxite) and cystic fibrosis. A special form of pneumothorax may be seen at the time of menstruation (*catamenial pneumothorax.)*

Pericardial and pleuropericardial rub

The former is related with systole and diastole and seems to be increased if the chest-piece is pressed against the precordium. If the patient is asked to hold respiration, the pleuropericardial rub usually disappears but the pericardial rub remains unchanged.

Causes of pericardial rub :

(1) Viral pericarditis
(2) Pyogenic pericarditis
(3) Koch's pericarditis
(4) Mycotic pericarditis
(5) Acute myocardial infraction
(6) Uraemia
(7) SLE
(8) Rheumatoid arthritis
(9) Scleroderma
(10) Dressler's syndrome
(11) Traumatic pericarditis etc.

Physically signs in some common diseases of the respiratory system :

	Shifting of Mediastinum	Vocal fremitus	Percussion note	Breath sound	Vocal Resonance	Any other adventitious sound on auscultation
Emphysema Lung	No mediastinal shift	Diminished	Hyper resonant; obliteration of area of Hepatic and Cardiac dullness	Diminished in Intensity, expiration prolonged	Diminished	No adventitious sounds. At times an expiratory wheeze may be heard
Pulmonary Fibrosis	Mediastinum shifts towards the side of fibrosis	Diministed	Impaired resonance or dull	Diminished in intensity or may be bronchial	Diminished in Intensity or may be bronchophony	No other adventitious sounds heard
Pneumothorax	Mediastinum shifts to the opposite of Pneumothorax	Diminished or Absent	Hyper resonant or Tympanitic	Diminished or absent. At times amphoric. Coin bell sound	Diminished in Intensity. or absent.	No adventitious sounds heard
Pleural Effusion	Mediastinum shifts to the opposite side of effusion	Diminished or absent	Dull or Stony Dull	Diminished or absent. At times may be bronchial over collapsed lung in massive pleural effusion.	Diminished in Intensity Aegophony heard just above the fluid level	No other adventitious sounds heard
Collapse of lung	Mediastinum shifts towards the side of collapse	Diminished	Dull on percussion	Absent or Bronchial breath sounds.	Diminished in Intensity or bronchophony	No other adventitious sounds heard
Cavitation	No Mediastinal shift.	Increased usually.	Hyper resonant (when filled with air); Dull (when filled with fluid)	Amphoric or Cavernous breath sound (if communicating with a patent bronchus	Bronchophony but a times Whispering pectoriloquy	Crepitations after cough Creptiations are better heard at times.
Consolidation of Lung.	No mediastinal shift	Increased	Dull on Percussion	Bronchial breath sounds	Bronchophony at times whispering pectoriloquy	In the early stages and at the time of resolution. Fine crepitations at the end of inspirations

METHOD OF ASPIRATION :

(1) Position of the patient--Prop up the patient near the edge of the bed and ask him to keep the hands over his head.

(2) The area is sterilised with iodine and spirit.

(3) Site of puncture–The 5th or 6th intercostal space in the mid-axillary line or the 8th intercostal space below the inferior angle of the scapula is the usual site if there is a huge amount of fluid and in case the amount of fluid in the pleural cavity is small or in an encysted pleural effusion the site selected should be the area of maximum dullness. In these cases, to localise the exact position of the fluid it is better to have an X-ray of the chest (both P A and lateral views).

(4) For local anaesthesia at the site of puncture infiltrate 2% novocaine.

(5) The aspiration needle connected with a three-way tube is then introduced into the pleural cavity. At first there will be a resistance due to the skin and muscles but it falls as soon as the needle enters the pleural cavity. The puncture should be done through an intercostal space, just above a rib margin.

(6) Now connect the three-way tube with a 50 cc syringe and aspirate the fluid.

(7) There are two opinions regarding the volume of the fluid to be aspirated. Some advocate that pleural fluid should be aspirated in several sittings, not more than 300–500 ml at a time. The other group observes that the fluid should be aspirated as much as possible and only to be stopped when the patient shows some complications like severe bouts of cough.

For diagnostic purposes at least 40 ml fluid must be aspirated for cytological, biochemical and physical studies.

(8) Withdraw the aspiration needle and seal the wound with tincture benzoin when the required amount of fluid is obtained. If the patient is restless or anxious it is better to give a sedative half an hour before the aspiration.

INDICATIONS OF ASPIRATION :

(1) Diagnostic : To know the physical, cytological and biochemical characters of the fluid for the detection of the cause of the pleural effusion.

(2) Therapeutic : To relieve the patient from respiratory distress in the following conditions :

(i) If fluid is up to the clavicular level;

(ii) If fluid persists even 15 days after the previous aspiration;

(iii) When there is rapid collection of fluid in the pleural cavity.

DANGERS OF ASPIRATION :

During aspiration of pleural fluid there is a chance of introducing infection giving rise to empyema thoracis and also air giving rise to hydropneumothrorax. If the fluid is rapidly aspirated there may be cardiorespiratory embarrassment or noncardiogenic acute pulmonary oedema.

INVESTIGATION OF THE PLEURAL FLUID :

The fluid is collected in separate sterile test tubes and is to be examined for physical, cytological and biochemical characters.

(A) **PHYSICAL CHARACTER :**

(1) *Appearance*

(a) The fluid may be turbid or frankly purulent in

empyema thoracis. The pus is due to streptococcal or staphylococcal infections. Greenish pus may be obtained in infections due to pneumococcus or Pseudomona pyocyaneous.

(b) In tuberculous effusion the fluid is amber or straw coloured.

(c) Clear serous fluid in hydrothorax is due to congestive cardiac failure, nephrotic syndrome, severe anaemia etc.

(d) Haemorrhagic pleural fluid is commonly found in malignant pleural effusion. Haemorrhagic fluid may also be due to tuberculous effusion, following pulmonary infraction, haemorrhagic diseases, in viral infections or trauma and in congestive cardiac failure.

(e) Milky white turbid chylous fluid may be due to filarial or malignant obstruction of the thoracic duct, eosinophilic lung, lymphomas, lymphangiomyomatosis etc.

(2) *Specific gravity*

In empyema or tuberculous effusion the specific gravity is more than 10, 6. In hydrothorax it is less than 1012.

(3) *Spontaneous coagulation*

In empyema thoracis and rarely in tuberculous effusion the fluid may clot if left in a test tube, due to its high protien content.

(4) Odour

In empyema caused by E. coli the fluid may be offensive fishy odour.

(B) **CHEMICAL EXAMINATION :**

In empyema and tuberculous effusion the total protein content is more than 3 gm%. In hydrothorax the protein

content is less than 3 gm%. Sugar is grossly reduced in empyema and in tuberculous effusion. In hydrothorax there will be no alteration of the sugar content of the fluid.

(C) **CYTOLOGICAL EXAMINATION**

In transudates due to hydrothorax of any cause only a few endothelia! cells are present per cmm of fluid. In empyema the cell count is more than 1000/ cmm and the majority of the cells are polymorphs. In tuberculous effusion the cell count is more than 1000/cmm and there is preponderance of lymphocytes. Plenty of RBC in the fluid indicates haemorrhagic effusion. Eosinophils may be found in the pleural fluid of those cases where repeated aspiration has been done or in chylous pleural effusion. In malignant effusion the malignant cells may be demonstrated by special staining.

(D) **BACTERIOLOGICAL STUDY :**

(1) In empyema a smear prepared from the centrifuged deposit should be stained by Gram's method for microscopical examination to identify the type of organism. This should be confirmed by culture and animal inoculation.

(2) In suspected tuberculous effusion the prepared smear from the centrifuged deposit is to be stained with Ziehl Neelsen's method for demonstration of acid fast bacilli. Culture and guineapig inoculation are to be done for confirmation of the diagnosis.

(3) Milky white fluid is to be examined for microfilaria by staining a smeared slide by Leishman's technique or by a cover slip preparation of the centrifuged deposit. The colour of this may become clear on adding ether.

Pleural effusion may be post pneumonic; tuberculous; malignant (following bronchogenic carcinoma

or pleural mesothelioma); or following lymphomas or penetrating wounds of the chest and myxoedema. In these cases the pleural fluid is exudative in nature. *Transudative pleural effusion* (hydrothorax) may occur following cardiac failure, nephrotic syndrome, hepatic failure (cirrhosis of liver, gross hypoproteinaemia. Meigs syndrome etc. *Haemorrhagic pleural effusion* is caused by penetrating wounds of the chest, neoplastic implant on pleura, primary mesothelioma of pleura and occasionally due to tuberculosis. Amoebic liver abscess may burst into the pleural cavity producing an anchovy sauce coloured fluid. Rarely coxsackie virus, psittacosis or infectious mononucleosis and very rarely a fungal infection such as coccidioidomycosis or blastomycosis also causes pleural effusion.

Differences between transudates and exudates

TESTS	TRANSUDATE	EXUDATE
colour	Usually colourless but may vary from, slight yellowish tint to milky or reddish tinge.	Usually variously coloured according to the cause
Appearance	Usually clear	Usually turbid
Coagulation	Does not coagulate	Coagulates often on standing.
Specific gravity	< 1015	>1020
pH	<7.8	>7.8
Protein	< 3gm%	>3gm%
Fluid to serum protein ratio	<0.5	>0.5
LDH	Low (>200 I.U.)	High (>200 I.U.)
Fluid to serum LDH ratio	< 0.6	> 0.6
RBC	Usually<10000/mm^3	May be>10000/mm^3
WBC	Usually<1000/mm^3	Usually>1000 mm^3

TESTS	TRANSUDATE	EXUDATE
Differential leucocyte count	Usually>50% lymphocytes Polymorphs usually absent	> 50% in Koch's and malignancy Polymorphs>50% in acute inflammation
Sugar	Same as in blood	Slightly or markably diminished
Amylase	–	>500 units/cc usually in pancreatitis, rarely in infection or neoplasm
Complements	–	C^3 and C^4 components are diminished in rheumatoid arthritis and SLE

SPUTUM AND ITS EXAMINATION

The term sputum is used in a broad sense and includes any material that is spitted out. The material may come from any portion of the respiratory tract. However, here we are concerned with the expectoration that comes from the larynx and below that.

(A) *MACROSCOPICAL*

In the process of examination, it is easy to pour the sputum into a petridish and place this against a black back ground and inspect through a hand lens. Some amount of sputum should also be taken in a test tube and allowed to settle and the following points are to be noted.

(i) *Amount*

To obtain precise information about the amount, it is better to collect the sputum in a graduated container for 24 hours.

Small amounts of sputum are expectorated in many lung diseases but large quantities (10-20

oz) are found only in bronchiectasis, pulmonary abscess or when an empyema ruptures into a bronchus. An increase in amount following a change of posture is characteristic of bronchiectasis.

(ii) *Character*

(a) Serous : It is described by the patient as thin and watery and sometimes forthy. Usually it is pinkish in colour due to the presence of RBC. The commonest cause is acute pulmonary oedema and rarely alveolar cell carcinoma. A ruptured hydatid cyst will cause expectoration of a clear salty fluid. It may be pinkish in colour due to the presence of RBC.

(b) Mucoid : It is clear, tough jelly-like and sticky and the amount is small. It is characteristic of early bronchitis. The amount becomes copious in a later stage of bronchitis when it becomes mucopurulent (indicating active bacterial infection of the respiratory tract) and in bronchial asthma.

(c) Mucopurulent : This is one of the commonest types of sputum. At the time of collection of such sputum, a lump of dense mucopus will be seen sinking down in the clearer media to the bottom of the test tube and becoming flat. This is usually seen in cases of pulmonary tuberculosis with cavity formation. If watery fluid is present in addition on standing the sputum will separate in three layers. The uppermost layer is thin forthy mucus. The middle layer is serous and may look greenish. The lowermost layer is brownish in

colour consisting of mucopus, anaerobic bacteria, Charcot-Leyden crystals, putrefactive products and foul smelling organic acids, RBC, tissue debris and yellow bodies called Dittrich's plug and sometimes elastic tissues if erosion has developed. This is characteristic of bronchiectasis.

(d) Purulent : In a pure form it is present only in cases of lung abscess, empyema and an extrinsic abscess rupturing into a bronchus.

(e) Expectoration resembling anchovy sauce. This is seen when an amoebic liver abscess bursts into the lung and the material is expectorated through a communicating bronchus.

(f) Haemorrhagic : Small amounts of blood mixed with sputum giving rise to a rusty appearance is seen in pneumonia. The pink, frothy sputum is the result of acute pulmonary congestion while red streaked sputum is frequently seen in pulmonary tuberculosis. Blood mixed with mucus, giving rise to prune-juice appearance is sometimes found in pulmonary neoplasms.

(g) In laryngeal diptheria pieces of membrane may be coughed out.

(h) Food particles may be found in the sputum in cases of tracheo-oesophageal fistula.

(iii) *Colour*

A serous sputum is colourless while a mucoid sputum looks grey. A purulent sputum is usually yellowish or greenish but may be only white. Black

discolouration due to presence of soot particles is found in coal miners, cigarette smokers etc. Rusty to golden yellow discolouration is characteristic of pneumococcal pneumonia. Blood streaked sputum may be seen in chronic bronchitis, pulmonary tuberculosis and sometimes in bronchial carcinoma while blood stained sputum is common in bronchial carcinoma, lung abscess, bronchiectasis etc. Pinkish colour occurs in acute pulmonary congestion. Anchovy sauce expectoration is the result of bursting of an amoebic liver abscess into the lungs.

(iv) *Odour*

'Nasty' odour is usually referred to that of purulent sputum. When the odour is extremely unpleasant and described by the patient as that of rotten egg or of sewer, it suggests the presence of bronchiectasis, more rarely of pulmonary suppuration following spirillum infection or of gangrene of the lung. Because of effective antibiotics this form of sputum is now rarely seen.

(B) *MICROSCOPICAL*

Microscopical elements in the sputum may have different origins. It may come from any portion of (i) respiratory tract and lungs, (ii) from blood, or (iii) It may be foreign bodies.

(i) *From the respiratory tract and lungs :*

(a) Epithelial cells are commonly seen in chronic bronchitis which may contain iron pigment following corpulmonale.

(b) Casts of smaller bronchi and their ramifications are found in fibrinous bronchitis. These casts may be mistaken for diphtheritic membranes, when culture of the latter will be diagnostic.

(c) Elastic fibres due to destruction of lung tissue may be obtained in bronchiectasis, gangrene of the lung, lung abscess, tuberculosis etc.

(d) Calcareous materials are sometimes obtained in pulmonary tuberculosis of long duration.

(e) Groups of malignant cells in the form of cell nests or sarcomatous tissue may rarely be seen in alveolar carcinoma.

(ii) *From blood* : RBC are found in the stage of red hepatisation while WBC are found in the stage of grey hepatisation in case of lobar pneumonia. Eosinophils are found frequently in asthma. Pus cells may be found in any acute infection of lung. Charcot-Leyden crystals and crystals of uric acid are occasionally found in asthma.

(iii) *Foreign bodies :* Asbestos bodies which are golden yellow and dumb-bell shaped are met with in cases of asbestos pneumoconiosis. Hooklets and scolices are found in hydatid cysts of lung. Fungus of actinomycosis are rarely seen. Structure of liver cells, crystals of lecithin and tyrosin are very rarely seen in amoebic liver abscess bursting into the lungs.

(C) BACTERIOLOGICAL

For a proper bacteriological examination the spu-

tum should be collected in the morning in a sterile container after mouth wash with only warm water and then it should be examined immediately by making a smear and staining. Culture followed by animal inoculation should be done if necessary. Smear should be stained by both Gram's and Zeihl-Neelsen's methods and examined under the microscope with the oil immersion lens. In suspected cases of Koch's infection, repeated examination is necessary if the first examination proves negative.

However, it is essential to remember that the sputum may normally contain many nonpathogenic bacteria. Certain variety of streptococcus, pneumococcus, diphtheroid bacilli and H. Influenzae may be discovered in the sputum even though there had been no respiratory disease.

SPECIAL DIAGNOSTIC PROCEDURES

I. RADIOLOGY

It is needless to mention the importance of X-ray examination of the chest which includes the following :

(A) Posteroanterior and lateral views of skiagram of chest

The posteroanterior film is taken with the film against the front of the chest and the X-ray tube placed six and a half feet behind the patient. A lateral view is essential for localisation of the lesion in terms of lobe, zone, particularly the midzone, and proximity of lesion to the anterior or posterior wall of the chest. A systematic plan of examina-

tion of the film is advised so that all the possible information that can be had from the film are ensured. The following points should be examined :

(i) Bony skeleton–scoliosis bony erosions, tumours and fractures.

(ii) Position of the trachea–This is seen as a dark line (due to air inside) in front of the upper dorsal vertebrae.

(iii) Position of the diaphragam–This is important because it may be pulled up by a collapse or fibrosis of the lower lobe of lung. The angles between the ribs and the diaphragm on both sides laterally (costophrenic anlges) and the angles beetwen the heart and the diaphragm (cardiophrenic angles) should be looked carefully to note whether they are clear, opaque or obliterated. Normally the costophrenic and cardiophrenic angles should be clear.

(iv) Heart shadow and other mediastinal contens–This will help to detect right and left ventricular hypertrophics (in the lateral view) and also other cardiovascular disorders.

(v) Lung fields–Excessive translucency indicates emphysematous change. More than 60% of the parenchymatous lesions can be diagnose by means of P A and lateral views of the film.

For radiological diagnosis, the lung fields are examined in reference to three zones :

(i) Upper zone–It extends from the apex down to the

level of the 2nd costochondral junction.

(ii) Mid-zone–It extends from the lower limit of the upper zone to the lower borders of the 4th costal cartilages.

(iii) Lower zone–It corresponds with the part of lungs below the lower limit of mid-zone. In posteroanterior view of the skiagram of chest sometimes a transverse line may be seen in the 3rd and 4th intercostal spaces on the right side which is the transverse fissure separating the right upper and middle lobes of the lung. The oblique fissure which separates the upper and lower lobes on both sides is not visualised in the P A skiagram of the chest.

(B) ***Fluoroscopy***

It is not much helpful in the diagnosis of lung diseases as compared to its diagnostic importance in cardiovascular diseases and diaphragmatic paralysis. The latter will show paradoxical movements of the diaphragm, that is the diaphragm descending when it should ascend (during expiration) and ascending when it should descend (during inspiration).

(C) ***Bronchography***

In this procedure a radio-opaque iodised oil is introduced via the trachea into the bronchial tree and then X-ray pictures are taken in PA and lateral views to visualise the particular bronchi or the whole of the bronchial tree. The oil may be introduced either,

(a) by a needle through the crico-thyroid membrane into the trachea, or,

(b) by a nasal catheter in the trachea through one of the nostrils, or,

(c) through a bronchoscope.

This is helpful in the diagnosis of bronchiectasis and to select preoperatively the particular lobe to be operated.

(D) ***Tomography***

Tomography is a particular method of X-ray in which structures at a particular plane and depth are kept in focus and anything out of the plane is kept out of focus so that they do not superimpose on the X-ray picture. In fact, it is nothing but an X-ray picture at a particular plane and depth. Its main use is to detect narrowing of the bronchus in cases of bronchogenic carcinoma and to detect a cavitation of the lung in an apparently opaque shadow.

II. **BRONCHOSCOPY**

This is done for direct inspection of the main bronchi and their branches and taking a biopsy especially to confirm the diagnosis of a bronchogenic carcinoma. It is also helpful in visualising and removing any foreign body from inside the bronchial lumen.

III. **THORACOSCOPY**

By this procedure the pleura is inspected after induction of an artificial pneumothorax.

IV. **MEDIASTINOSCOPY**

This is a recent procedure by which the structures in the mediastinum are directly inspected. The instrument is inserted behind the sternum and biopsy of the mediastinal structures e.g. lymph glands can be obtained.

V. **LUNG BIOPSY**

Drill biopsy of the lung is performed for histopathological

confirmation of the diagnosis of diffuse lung diseases e.g. diffuse interstitial fibrosis.

VI. LUNG FUNCTION TESTS

Detailed discussion of the different lung function tests is beyond the scope of this book; but a few simple clinical tests are briefly described. The lung function tests enable us to make a physiological and functional rather than a pathological diagnosis. They are useful *diagnostically* for objective assessment of the patients and their disabilities and *prognostically* to assess the progress of the disease and the effect of the treatment.

Spirometry– It is one of the most commonly used laboratory methods for evaluation of the patient. This includes measurement of vital capacity.

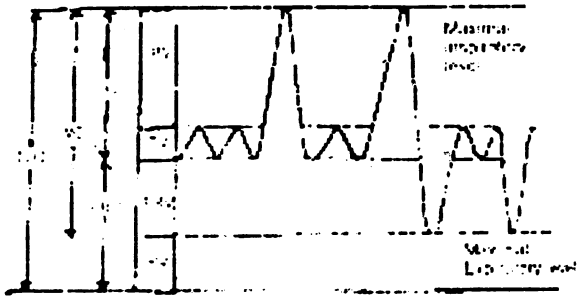


Fig. 2-2 The lung volumes TLC–Total ling capacity. VC–Vital capacity TV–Tidal volume IRV–Inspiratory reserve volume ERV –Expiratory reserve volume. RV–Residual volume.

Vital capacity is defined as the largest volume of air which the patient can exhale voluntarily beginning with the fully inflated lungs. The result is expressed in litres. Weakness, pain on deep breathing, lack of co-operation on the

part of the patient etc. may result in an underestimation of the vital capacity (normal 3 to 5 litres).

Reduction of vital capacity in the absence of obstruction is indicative of a *restrictive* ventilatory defect e.g. deformities of thoracic cage, pleural thickening, pleural effusion and lung fibrosis.

Spirometer tracings recorded during forced expirations (expiratory spirogram) are the most convenient indirect method of confirming the presence and assessing the severity of an obstructive ventilatory defect.

In normal subjects about 75 per cent of the vital capacity is expelled in the first second of forced expiration and the rest in about three seconds. So a normal forced expirogram is very steep in its initial portion, then smoothly curved and finally reaches a plateau in 3 to 6 seconds.

Forced expiratory volume in the first second (FEV) is 70 to 75 per cent of the vital capacity in normal individuals under sixty. When there is an obstruction to the outflow of the air from the lungs as in asthma, bronchitis, emphysema or intraluminal obstruction owing to mucus or oedema, this curve is flattened. By recording the FEV_1 a numerical value can be given to the degree of flattening of this curve. It is expressed as the ratio of FEV_1 FVC. In patients suffering from *obstructive* airway disease the percentage is below 70. In the presence of *restrictive* ventilatory defects the FEV_1 will remain 75 to 80 percent of the vital capacity or more depending on the age of the patient; provided the airways are not obstructed.

The test should be repeated after the use of aerosol bronchodilators for prognostic evaluation. Airway obstruc-

tion is said to be reversible if the FEV_1 improves. In cases of bronchial spasm due to bronchitis and bronchial asthma the obstruction is partial; it is not reversible in emphysema.

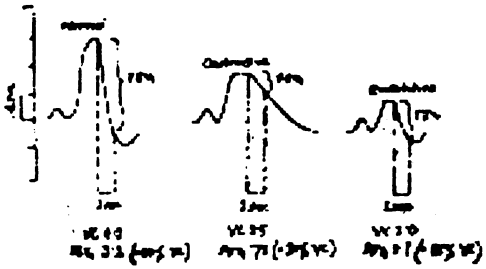


Fig. 2-3 : *Graphical representation of obstructive and restrictive defects of ventilation*

Alternatively, with the help of Wright's peak flowmeter, peak expiratory flow rate (PEFR) can be measured. The peak expiratory flow rate is reduced in airways obstruction. It is to be remembered that 150 ml of air fills the conducting airway which constitute the 'anatomical dead space' and take no part in gas exchange.

Defects of ventilatory capacity can be obstructive and or *non obstructive.* Obstructive defect of ventilatory capacity may be due to chronic bronchitis and bronchial asthma where variable airways obstruction occur due to bronchial secretion, oedema of bronchial mucosa and bronchospasm. Nonobstructive defect of ventilatory capacity may be due to (i) restrictive lung disorders and (ii) hypodynamic ventilatory defects.

A restrictive lung disorder is due to reduced lung compliance as in (a) diffuse infiltration of lung, (b) fibrosis of lung, (c) pulmonary oedema or stiffness or pleura of chest wall as in, (d) pleural thickening or, (e) kyphoscoliosis respectively or may be due to diminished ventilable pulmo-

nary volume as in, (f) pulmonary resection, (g) malignant tumour, (h) pneumothorax and, (i) pleural effusion.

Hypodynamic ventilatory defect may be due to (a) failure of central respiratory drive as occurs after depressant drugs, anaesthetics, cerebrovascular accidents or, (b) due to neuromuscular failure as in poliomyelitis, polyneuritis or after muscle relaxant drugs.

Lung function tests, thus include

(i) Tests for ventilatory capacity.
(ii) Tests for regional lung function.
(iii) Arterial blood analysis.

(i) *Tests for ventilatory capacity* : This includes the followings :

(a) *Forced expiratory time* (FET) : It is the best physical sign for diffuse intrathoracic airways obstruction – requiring no special instruments and can be readily performed at the bedside. FET is the time taken to deliver the vital capacity. In normal subjects this is accomplished in 3.4 seconds. Prolongation of FET beyond 6 second indicates airways obstruction and that FEV_1 FVC is less than 60%.

(b) *Forced expiratory volume* (FEV) *and forced vital capacity* (FVC) *:* In diseases causing diffuse airways obstruction e.g. asthma, bronchitis, emphysema etc. the inequality of the disease process in different airways causes them to close irregularly and progressively as expiration is continued. Result is that in severe cases less than 40% of the FVC is expelled in the first second and the rest of the air takes still much longer, or in other words FEV/FVC $\leq$ 40%. This is called

obstructive type of ventilatory defect. In interstitial lung diseases, ankylosing spondylitis etc. both FEV and FVC are nearly equally reduced. So FEV/FVC% will either remain normal or may even increase because of increase elastic recoil. This is restrictive type of ventilatory defect.

(c) *Peak expiratory flow rate* (PEFR) : It can be measured by the Wright peak flowmeter and the reading is obtained in litres/min. PEFR is diminished in obstructive airways diseases and as such, it is a good indicator of the severity of obstruction.

(d) *Maximum breathing capacity* (MBC) or *maximum voluntary ventilation* (MVV) : It is measured by instructing the patient to breathe as deeply and as repidly as he can for about 15 secs. The volume breathed may be recorded by a spirometer or collected in a bag. The result is expressed in litres min. It is rarely used now-a-days.

(ii) *Tests for regional lung function :* These include fluoroscopy bronchospirometry and radioisotope techniques. By fluoroscopy the proportion of ventilation received by each individual lobe and regional differences of pressure in the lungs can be ascertained from the intensity of 'lighting up' of the lungs and also from the movements of the ribs, mediastinum and diaphragm.

Bronchospirometry involves differential bronchial catheterization and collection of air separately from each lung. It is rarely used.

In radioisotope technique and isotopically labelled gas is administered and its concentration in differ-

ent regions or 'cores' of lung is determined by external counting. Xenon^{33} is an insoluble gas which is very suitable. Scanning of the radioactivity of the lungs after-IV administration of macroaggregated 1^{31} or, Tc^{99} labelled albumin is also widely used.

(iii) *Arterial blood analysis* : The pH, PaCo and PaO_2 are determined by special apparatus for arterial blood gas analysis and with these data, the type of respiratory failure (vide infra), the state of acid base balance, the therapy to be instituted and the response to therapy can all be ascertained.

Overall function of the lung is to maintain the physiological limits of blood gases needed for normal tissue metabolism by oxygenation of deoxygenated blood and elimination of CO_2. In respiratory failure carbondioxide is inadequately eliminated and so its level in the blood rises. Normal arterial oxygen saturation is above 97% and arterial carbon dioxide tension (PCO) is normally 37-43 mm of Hg and the pH of blood is 7-38 to 7-42. The partial pressure of oxygen in arterial blood (PO_2) is normally about 100 mm of Hg.

When PCO rises above 49 mm of Hg it indicates *respiratory failure* due to alveolar hypoventilation. Its level falls in hyperventilation and is less affected by diffusion impairment or right to left shunting of blood because of the relatively high diffusibility of Co. Routine PCO_2 estimation in arterial blood is not always possible. An easier method has been introduced by Campbell and Howell, where

the patient breathes and rebreathes into a bag until the gas mixture in the bag is in almost same gaseous equilibrium with the alveolar air and hence indirectly with the arterial blood. Final analysis of the gas mixture in the bag is then carried out using simple instruments and apparatus.

VII. **PLEURAL BIOPSY**

This is one of the indispensible diagnostic techniques for confirmation of tuberculosis and malignancy of pleura.

VIII. **SKIN TESTS**

In the diagnosis of the chest diseases skin tests can prove valuable Allergic asthma is associated with immediate skin reaction to allergen e.g. pollens etc. This is type I immune reaction or immediate hypersensitivity reaction. Cell mediated (delayed) hypersensitivity or type IV immune reaction is seen in Mantoux test used to detect the tuberculous infection (either past or recent). For sarcoidosis, an intradermal Kveim test is done which is relatively specific for this disease.

COR PULMONALE

Cor pulmonale is defined as enlargement of right heart secondary to malfunctioning of the lungs. The enlargement may be due to both hypertrophy and dilatation and malfunctioning of the lungs may be due to causes other than intrinsic lung diseases. The most obvious mechanism is obstruction of the pulmonary vessels. It may be due either to vasoconstriction from hypoxia or to distortion, compression or obliteration of the vessels by the

underlying disease process. Hypoxaemia may impair myocardial contractility and reduced left ventricular performance may contribute to the consequent cardiopulmonary derangements. Cor pulmonale may be either *acute* or *chronic*. In acute cor pulmonale the right heart dilates because of acute pulmonary embolism. Chronic cor pulmonale may be caused by–(i) intrinsic diseases of the lungs of airways, (ii) malfunctioning of the chest belows, or (iii) insufficient drive from the respiratory centre. Of all these causes chronic obstructive lung diseases (chronic bronchitis and emphysema) are the commonest. But cor pulmonale resulting from chronic obstructive lung diseases may remain undiagnosed till there is an episode of overt right heart failure. Diagnosis may be confirmed by arterial blood gas analysis (showing arterial hypoxaemia, hypercapnia and acidosis); roentgenographic and electrocardiographic evidences of enlargement of right ventricle; and if necessary, by catheterization of right side of the heart. Recently echocardiography has proved valuable in detecting pulmonary hypertension based on the movements of the pulmonary valve.

RESPIRATORY FAILURE

Respiratory failure is a clinical condition characterized by abnormality of blood gases present at rest and caused by a disorder of respiration or of its control mechanisms.

There are two types of respiratory failures in both of which the arterial O_2 tension is diminished, but may be distinguished by arterial CO tension.

In type I the $PaCO_2$ is normal or low associated with a low PaO_2

In type II there is elevated $PaCO_2$ associated with a low PaO_2

Type I respiratory failure is due to a defect in gas transfer caused by uneven distribution of inspired air and pulmonary blood flow. It is found in (i) Pulmonary oedema, (ii) Pulmonary fibrosis or infiltration, (iii) Pulmonary thrombo embolism and (iv) transiently in severe asthma and acute bronchial infection.

Type II respiratory failure is caused by deficient total alveolar ventilation. It is found in (i) chronic obstructive lung diseases like chronic bronchitis, emphysema and asthma, (ii) depression of respiratory centre caused by narcotic group of drugs or anaesthesia or vascular disorders, (iii) neuromuscular failure in conditions like poliomyelitis, polyneuritis, myasthenia gravis, chest injury etc. and (iv) end stage of type I respiratory failure.