PRINCIPLES OF HISTORY TAKING AND PHYSICAL EXAMINATION

- General Principles
- The Cardiovascular System
- The Respiratory System
- The Abdomen

Dr. Osama Mahmoud Mohamed
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PRINCIPLES OF HISTORY-TAKING
AND
PHYSICAL EXAMINATION

• General principles
• The cardiovascular system
• The respiratory system
• The abdomen

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GENERAL PRINCIPLES

Surface Anatomy Of The Neck

(A) Feel the following structures in the middle line as you pass your finger from the chin to the sternum:
1. The chin.
2. The body of hyoid bone at C3.
3. The notch of thyroid cartilage at C4.
4. The arch of cricoid cartilage at C6.
5. The rings of trachea from C6 downward.
6. The suprasternal notch.

Isthmus of thyroid gland lies over tracheal rings 2, 3, 4.

(B) The lower border of the cricoid cartilage is a very important Landmark, it marks the following features:
1. The level of the 6th cervical vertebrae.
2. The level at which you can compress the common carotid artery against the transverse process of C6.
3. The level at which larynx ends and trachea begins.
4. The level at which pharynx ends and esophagus begins.

(C) SURFACE MARKINGS

(1) The sternomastoid muscle:
Turn your face to the left side and notice that the right sternomastoid muscle becomes prominent.

(2) The jugular veins:
The external jugular vein can be seen on the surface of the sternomastoid, it lies in the superficial fascia and descends almost vertically from the angle of the mandible towards the middle of the clavicle. The internal jugular vein lies below the sternomastoid muscle.

(3) The Carotid arteries:
a- The common carotid artery enters the neck by passing behind the sternoclavicular joint, at the level of the upper border of the thyroid cartilage.
   You can feel the pulsation of the common carotid artery by pressing it backwards against the carotid tubercle. (Which is the anterior tubercle of the transverse process of C6) medial to the sternomastoid muscle.
b- The common carotid artery are represented on the surface by a line which joins two points:
   1- The sternoclavicular joint.
   2- A point midway between the tip of the mastoid process and the angle of the mandible.
SURFACE ANATOMY OF THE UPPER LIMB

(1) Shoulder region:
☆ The acromion process lies immediately above the smooth bulge of the deltoid muscle.

(2) Elbow region:
☆ Three bony landmarks which form a triangle:
  - Olecranon process of the ulna.
  - Medial and lateral epicondyles of the humerus.
☆ Brachial artery felt medial to the tendon of biceps.

(3) At the wrist:
☆ Feel the scaphoid bone in the anatomical snuff box.
☆ Feel pulsations of radial artery, lateral to tendon of flexor carpiradials.
☆ Feel pulsations of ulnar artery lateral to tendon of flexor carpiulnaris.

(4) Muscles:
☆ Pectoralis major forms the anterior fold of axilla while teres major and Latissimus dorsi form the posterior fold.
☆ Deltoid forms the smooth contour of the shoulder.

(5) Arteries: (Sites of palpation)
☆ Subclavian artery → against 1st rib.
☆ Brachial artery → against the humerus.
☆ Radial and ulnar artery → at wrist (as above).

(6) Nerves:
☆ We can feel the ulnar nerve near by the medial epicondyle.

SURFACE ANATOMY OF THE LOWER LIMB

(1) The femoral artery:
☆ You can feel its pulsations at the mid inguinal point.
☆ Surface markings, flex your hip and externally rotate it, then draw a line joining the mid inguinal point with adductor tubercle (run your fingers down the medial side of your thigh till they are stopped by the adductor tubercle). The upper 2/3 of this line is a mark of femoral artery.

(2) Popliteal artery:
☆ Patient in the prone position, flex knee and use firm pressure against the popliteal surface of his femur (see later).

(3) The Dorsalis pedis artery:
☆ Felt on the dorsum of the foot lateral to the tendon of the extensor hallucis longus, against navicular bone. In 10% of people it can not be felt.
(4) Post tibial artery:
- Felt below and behind the medial malleolus. It is not felt in 5% of population.

(5) Nerves:
- Only one nerve can be felt in LL. (lateral popliteal nerve), it can be rolled against
  the neck of the fibula.

**HISTORY TAKING**

(A) Personal history:

(1) Name:
- Insist upon recording the complete name including the family name (filing system).
- Fatal errors may occur when two patients with the same name have been under
  treatment in the hospital simultaneously.
- This gives sense of familiarity, sex identification.

(2) Sex:
- Diseases which are common in females as:
  2. Thyrotoxicosis - Myxoedema.
  3. Gall bladder diseases e.g. gall stones.
  4. Bronchial adenoma.
  5. Primary biliary cirrhosis.
- Diseases which are common in males:
  1. Coronary heart disease.
  2. Bronchogenic carcinoma.
  3. Hemophilia, Duchenne. (X - Linked)
  4. Peptic ulcer, Cancer stomach.

(3) Age:
- We ask about the age because some diseases are common in children and
  young adults e.g.:
  1. Acute rheumatic fever and rheumatic heart diseases.
  2. T.B.
  3. Viral hepatitis.
  5. Poliomyelitis - Duchenne myopathy - Friedrich's ataxia -

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Diseases which are common in old age:
1. Carcinoma.
2. Atherosclerosis and coronary artery disease.
3. Cor pulmonale.
5. Multiple myloma

(4) Occupation:
Certain occupations may expose the patient to certain diseases:
2. Glass workers → Silicosis → Interstitial pulmonary fibrosis
5. Manganese → Parkinsonism.
6. Aniline dyes → Cancer urinary bladder.
7. Sewers → Infections e.g.: leptospirosis.
8. Farmers → Bilharziasis.

(5) Residence & Address:
This may reflect socioeconomic condition and may occasionally point to a certain disease e.g.:
- Sharkia → Filariasis!
- Country → Bilharziasis, exposure to animals (Brucellosis) or insecticides.
- Towns → Hypertension, Anxiety and IHD.

(6) Marital State:
- Duration of marriage.
- Number of children.
- The age of the youngest child.

- High social class liable to Hypertension - I.H.D –
- irritable bowel disease

Social class
- Low social class liable to malnutrition, infections & parasites.

(7) Habits:
Special habit is a habit that makes the patient more susceptible than others to a certain disease.

- Smoking:
Ask about: Number of cigarettes /day, duration & type of smoking (pipe, cigarette).

Smoking predispose to:

1. Chest:
   (a) Chronic bronchitis, emphysema.
   (b) Bronchial carcinoma:
   The risk is directly proportional to the amount smoked and to the tar content of cigarettes. Staining on the fingers or teeth should raise strong suspicion that the patient is or until recently was a heavy smoker. Smoking habits which increases the risk of bronchial carcinoma are:
Starting to smoke at early age.

Increased number of puffs/cigarette.

Keeping the cigarette in the mouth between puffs.

Smoking down to the button-end.

(c) Cancer lips and tongue due to pipe smoking.

(d) Post operative pneumonia.

2. CVS: Arrhythmia - I.H.D - peripheral vascular diseases.


**Alcohol:**

Ask about the amount / day. The alcohol equivalents are: 30 ml of whisky, 100 ml wine or 250 ml bear contain 10 gm alcohol.

1. GIT:
   - Mallory-weiss syndrome: It is haematemesis characterized by laceration of mucosa at gastro-oesophageal junction.
   - Alcoholic fatty liver, hepatitis, cirrhosis.
   - Acute hemorrhagic pancreatitis.
   - Alcohol may aggravate peptic ulcer.

2. CNS:
   - Wernicke's - Korsakoff syndrome.
   - Polyneuropathy, proximal myopathy.
   - Optic atrophy.
   - Hallucination - delirium and coma.

3. CVS:
   - Cardiomyopathy (Potentially reversible).

**Opium:**

1. Constipation, its withdrawal symptoms are diarrhea, rhinorrhea
2. I.V addiction → Hepatitis - AIDS - Infective endocarditis.

**Diet habits:**

e.g. excessive intake of coffee, salt or spices.

(8) Menstrual and obstetric history:

- Frequency of the periods, regularity, duration, amount of blood.
- Date of Menarche and menopause.
- History of intake of contraceptive pills.
- Ask about child birth including miscarriages or therapeutic abortions.

**Some side effects of contraceptive pills:**

1. Nausea and vomiting
2. Headache.
4. Thromboembolic manifestations.
5. Hepatic cholestasis, hepatic adenoma and Budd-Chiari syndrome.
6 Increase the incidence of cancer breast.
7 Carpal tunnel syndrome.
8 Hypertension.
9 Impaired glucose tolerance, plasma lipoprotein may adversely affected.
10 Pseudo tumour cerebri.

(B) Presenting complaint: C/O

This should be:
1- The patient's complaint in his own words (try to avoid medical terms)
    For example:
    ➔ Shortness of breath/Dyspnea.
    ➔ Consciousness of heart beat / Palpitation.
    ➔ Swelling of both lower limbs / Oedema.
    ➔ Coughing blood / Haemoptysis.
2- It should be the presenting C/O (e.g.: the cause of admission to the hospital).
3- Avoid multiple C/O related to the same system.
4- We mention in the C/O its duration, also it is possible to write the onset and course of the C/O, or it is better to be mentioned in the history of present illness.

(c) History of present illness or present history:

1- We start by asking the patient about the last time at which he was symptom free?
2- You must mention the symptoms in chronological manner, with analysis of each one. (In the form of story)
3- Comment on the onset, the course and the duration of the main complain (some prefer to start the present history with analysis of the complaint)

ONSET: The time required to complete the full picture of the disease.

a- Acute onset:
   ➔ Dramatic i.e. within seconds & minutes e.g. embolism.
   ➔ Sudden i.e. within hours e.g. thrombosis.
   ➔ Rapid i.e. within days e.g. inflammation.

b- Gradual onset:
   i.e. Within weeks, months and years e.g. degenerative diseases and tumors.

c- Accidental onset:
   i.e. The patient discovers his C/O by chance, so you can't mention the duration e.g.:
   ➔ Lymph node enlargement.
   ➔ Thyroid swelling.
   ➔ Breast mass.
   ➔ Jaundice.

COURSE:

a- Regressive: as inflammation and vascular accidents.

b- Progressive: as malignancy and degenerative disease.
c- Stationary: It may be the end of progressive diseases?

d- Remission & Exacerbation: e.g.
  . Nephrotic syndrome.
  . Peptic ulcer. (Periodicity)
  . Disseminated Sclerosis.
  . Ulcerative colitis.
  . Rheumatoid arthritis.

DURATION: e.g. short → inflammatory, long → degenerative.

4- After obtaining much information about the symptoms related to the diseased system, other systems should be reviewed for disturbances related to the present illness. E.g.: if the case is cardiac you must ask about chest symptoms searching for a relations.

5- Enumerate any negative relevant data either related to the diseased system or to other systems.

(D) Past history:

Mention the relevant items in relation to the complain and present illness.

1- Similar attacks or previous illness as below.
   ☆ Diabetes – Hypertension!
      Diabetes can be considered as a past history e.g. gestational and stress diabetes. Also curable or stress induced hypertension can be put in the past history.
   ☆ Infectious diseases as hepatitis, T.B.
   ☆ Rheumatic fever, recurrent sore throat, or Bilharziasis.
   ☆ Venereal diseases e.g. syphilis.

2- Trauma, surgical operation, blood transfusion.

3- Drug treatment or radiotherapy.

4- Travelling abroad e.g suspecting case of HIV.

(E) Family history:

Ask about:

- Similar conditions.
- History of consanguinity.

Hereditary diseases are:

I. Multifactorial inherited diseases:
   1- Diabetes mellitus.
   2- Hypertension.
   3- Bronchial Asthma.
   4- Epilepsy.
   5- Gout.

II. Autosomal Dominant Diseases (appears in every generation)
   - Congenital Polycystic kidney. (The most common)
   - Myotonia.
   - Huntington's chorea
   - Fascioscapulo humeral myopathy.
III. Autosomal Recessive Diseases: (not appear in every generation)
- Scapulo humeral myopathy.
- Dubin Johnson $, haemochromatosis & Wilson disease.

VI. X-Linked recessive diseases: (Affecting males, females are carriers)
- Hemophilia.
- Duchenne myopathy.

GENERAL EXAMINATION

I- General Condition and General Appearance:
- Good.
- Bad
- Fair.
- Cachetic appearance in advanced malignancies.
- Infantile appearance as infantilism.

II- Mental state: (see neurology sheet)
- Consciousness
- Attention
- Memory
- Mood
- Intelligence.

III- Built:
- Over built.
- Under built
- Average.

The built is determined by noting the weight, height in relation to age and sex. It can be determined by body mass index (BMI) which is derived from the formula Wt/Ht$^2$ in kg/m$^2$. Normally it is “18-25” (average body built).

Abnormalities:
- Under built: < 18.
- Over built: 25 - 29.
- Obesity: 30 - 39.
- Morbid obesity: 40 or more.

Important notes:
- The height and span are almost equal. The height is the distance from the occiput to the heels in the up right position. The span is the distance between the tip of the third fingers with outstretched hands.
- The distance between symphysis pubis and floor is equal to that between occiput and symphysis pubis (lower and upper segments).
The upper segment also = the height – the lower segment.

Obesity means increase in body weight due to accumulation of fat in subcutaneous and deep tissues with BMI ≥ 30.

Obesity can be assessed by the thickness of skin folds e.g.:
- Lateral aspect of the arm 0.9 -1.1 cm.
- Abdomen = 1.5 cm.
- Buttocks = 1.5 - 2.5 cm.

Waist-hip ratio provides a simple assessment of visceral adipose fat. Persons with a pear shaped configuration and a waist – hip ratio of 0.8 or less in females or <0.9 in males have good prognosis. Apple shaped persons with a greater waist-hip ratio have an increased risk of developing cardiovascular disease.

Causes of obesity (for details, see endocrinology)
- Excessive intake.
- Deficient physical activity.
- Hypothyroidism.
- Froehlich's syndrome and Laurence Moon Biedl $.
- Cushing syndrome.
- Insulin resistance.
- Drugs e.g steroids.

Causes of underweight:
- Chronic infection
- D.M.
- Anorexia nervosa.
- Malignancy.
- Thyrotoxicosis.
- Depression.
- Malabsorption.
- Addison's disease.

Causes of Dwarfism: (Stunted growth or short stature)

1. Endocrine:
- Hypopituitarism in children.
- Juvenile hypothyroidism.
- Precocious puberty.
- Juvenile D.M.
- Pseudohypo-parathyroidism.

2. Racial and Familial:

3. Skeletal:
- Osteogenesis imperfecta.

4. Chronic diseases during childhood:
- Cyanotic heart disease.
- Polycystic lung.
- Malabsorption syndrome.
- Steroid therapy for long time.

5. Genetic:
- Down syndrome.
- Turner syndrome.
Causes of gigantism:
- Familial, racial
- Pituitary hyperfunction.
- Cerebral gigantism
- Eunuchiodism.
- Marfan’s syndrome
- Klinefelter’s syndrome.

IV- Decubitus
(Position of patient in bed in relation to certain disease)

1. Orthopnoea:
- Left sided heart lesions (Left sided heart failure, M.S.)
- Status asthmatics.
- Tense ascites (mechanical).

2. Squatting position: In Fallot’s tetralogy.

3. The praying Muslim position:
Patient prefer to lean forward e.g. pericardial effusion and mediastinal syndrome.

4. Lateral position in chest disease: (Treponea)
Some patients unable to lie supine or prone but prefer the lateral position e.g. (down with the good lung) to increase perfusion of the dependent normal lung as in cases of lung collapse as this → better ventilation/perfusion.
Other patients prefer to lie on the affected side e.g. lung abscess, pneumonia or haemotysis from one side (pus or blood may spill from the bad into the good lung). Patients with unilateral pulmonary fibrosis or effusion prefer to lie on the affected side for more comfortable breathing.

5. Position in meningitis:
There is hyperextension of the neck and spine together with flexion of the knee.

6. Position in peritonitis:
Patient lies quiet flat in bed supporting the abdomen with both hands.

7. Platypnea in hepato pulmonary syndrome:
Platypnea means dyspnea in the erect position relieved by recumbency.

V- (facies):
It is a peculiar and unusual facial features that often are pathognomonic of a particular disease.

1- Parkinsonism → Mask like face.
2- Myxoedema → Apathetic look.
3- Hyperthyroidism → Restless, staring look.
4- Acromegaly → Ape like appearance.
5- Congenital $ → Square like bulldog.
6- Uremia → Earthy look.
7- Myasthenia gravis → Weak smile, bilateral ptosis.
8- Facial palsy → C.N.S
9- Horner’s syndrome → C.N.S
10- Myopathic face → Expressionless, protrusion of the lower lip.
11- Toxic look → Infective endocarditis.
12- Elfin facies in congenital supravalvular A.S (William $).
VI- Complexion: (cyanosis – pallor – jaundice)

Abnormalities of complexion may be first noticed by patients or by their relatives or friends. The colour of the skin depends upon variations in oxyhaemoglobin reduced haemoglobin, melanin and carotene.

a) Cyanosis:

- Means bluish coloration of the skin and/or mucous membranes due to increase percentage of reduced Hb or abnormal Hb in the arterial blood.
- For cyanosis to occur there must be at least 5 gm reduced Hb/dl in the arterial blood perfusing the skin or mucous membranes (capillaries), so cyanosis may not be detected in cases with severe anaemia.

Types of cyanosis:

I- Central cyanosis:

Reduction in the oxygen saturation of arterial blood below 80-85%.

Causes:

a. Heart diseases: Congenital cyanotic heart disease e.g. Fallot's tetralogy, Eisenmenger's syndrome, Ebstein's anomaly and transposition of great vessels.

b. Chest diseases: (Pulmonary advanced chest disease):
   1- Chronic obstructive pulmonary disease.
   2- Interstitial pulmonary diseases or fibrosis.
   3- Pulmonary oedema.
   4- Bronchiectasis if advanced and extensive.

II- Peripheral Cyanosis:

Due to stagnant circulation or vasoconstriction through the peripheral vascular bed with excessive O₂ extraction from capillary blood. the arterial O₂ saturation is normal unless cardiopulmonary disease is also present.

Causes:

1- Reduced cardiac output.
2- Peripheral vascular diseases.
3- Cold weather.
4- Polycythaemia (↑ Hb content → cyanosis at higher levels of arterial O₂ saturation).
5- Venous obstruction e.g. Superior vena caval obstruction leading to cyanosis of the face. Also arterial obstruction → peripheral cyanosis.

III: Differential cyanosis:

i.e.: Cyanosis usually with clubbing limited to the lower limbs only, as in case of P.D.A. with reversed shunt (it is a central cyanosis).

Difference between central & peripheral cyanosis

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<th>Central</th>
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<td>1- It affects the skin only i.e. nails, tip of nose or ears.</td>
<td>1- It affects skin, nail, lips, tongue and mucus membranes.</td>
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<td>2- Hands are cold.</td>
<td>2- Warm.</td>
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<tr>
<td>3- Improvement with massage or warming of the hands.</td>
<td>3- No improvement.</td>
</tr>
<tr>
<td>4- No clubbing.</td>
<td>4- Usually there is clubbing.</td>
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<td>5- O₂ therapy → no improvement.</td>
<td>5- Improvement in case of chest disease but no improvement in cases of congenital cyanotic heart diseases.</td>
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Q: False Cyanosis or Chemical Cyanosis:
Blue discoloration occurs due to the presence of abnormal non-functioning Hb and not due to reduced Hb. In these cases, arterial oxygen saturation is normal?

➢ Clinically:
Picture of central cyanosis. It can be suspected when there is no apparent cardiac, chest or circulatory disturbance.

➢ Causes:
1. Met-Haemoglobinemia, due to nitrites.
2. Sulph-Haemoglobinemia, due to Sulphonamides.

➢ Diagnosis: by spectroscopy.

N.B.:

Q: When Central cyanosis does not appear in tongue? In cases of differential cyanosis.
Q: Why Peripheral cyanosis does not appear in tongue? As it is well perfused but this can occur in advanced circulatory failure.
Q: When peripheral cyanosis appears in tongue? In cases of SVC obstruction.

Partly central and partly peripheral cyanosis may occur with polycythaemia and massive pulmonary embolism?

b) Pallor:

➢ We examine in the following sites:
  ✓ Mucous membranes in the lips & conjunctiva.
  ✓ Palmar crease, Hb < 6-7, gm → Pale palmer crease.
  ✓ Skin. ✓ Nail. ✓ Tongue

➢ The degree of pallor depend on the state of capillaries, amount of blood within the capillaries, Hb, pigmentation & thickness of the skin.

➢ Examination of the mucus membranes may help to distinguish pallor of anaemia from that of other causes.

➢ Causes of Pallor:
  ✓ Anemia
  ✓ Shock or ↓ cop
  ✓ Malignant hypertension.
  ✓ Toxaemia e.g. infective endocarditis
  ✓ Edema of the face e.g.: Nephrotic syndrome
  ✓ Racial pallor (Far East).

c) Jaundice:

. It is a yellow discoloration of the sclera, mucous membranes and skin due to hyperbilirubinaemia (> 2.5 – 3 mg/dL).
. Jaundice is best seen in day light and may be undetected in artificial light.

➢ Ch. Ch. Of hemolytic jaundice:
  - Lemon yellow jaundice.
  - Normal urine (Acholuric Jaundice).
  - Dark stool.
  - Pallor + other signs of haemolytic anaemia

➢ Ch. Ch. of hepatocellular jaundice:
  - Orange yellow jaundice.
  - Dark urine.
  - Pale stool.
  - Signs of liver cell failure.
Ch. Ch. of obstructive jaundice:
- Olive green jaundice.
- Dark urine.
- Pale stool.
- Other signs of obstructive jaundice. e.g.: scratch marks due to pruritis.

Examinations:
1) Sclera and lower fornix. 2) Soft palate
3) Lunula of the tongue. 4) Skin.

D.D. of jaundice: Carotenamia.

Other abnormalities of complexion
- Hyperpigmentation and depigmentation. (See skin examination)
- Carotenamia: common in subjects who eat a lot of raw carrots and tomatoes, it can also occur in hypothyroidism it appears in skin (face, palms and soles) and not in the sclera or mucous membranes.

VII- Vital Signs:

A - Temperature:
- Sterilize the thermometer in 70% alcohol for at least 20 minutes.
- We put the thermometer in the mouth under the tongue - axilla - groin - rectum (for 3 minutes in old types of thermometers and 1 minute with the new models) or until we get two successive fixed readings.
  * In axilla (add 1/2 a degree), it is highly inaccurate
  * In rectum (subtract 1/2 a degree).
- Normal temperature is 36.8 ± 0.4°C.
- Fever means temperature > 37.2°C AM or > 37.7 PM.
- Hypothermia means temperature ≤ 35°C (rectal), it is missed by routine thermometers, it is detected by thermistor.
- Hyperpyrexia means temperature ≥ 41°C

Low reading clinical thermometers are available and should be used when hypothermia is suspected, temperatures < 27°C are not uncommon.

Types of Fever:

a- Sustained Fever:
Daily fluctuation does not exceed 1 C. it can be defined also as persistent elevation of body temperature with minimal variation. It is common with gm negative sepsis or CNS damage.

b- Remittent Fever:
Daily fluctuation exceeds 1 C e.g. viral disease & T.B..

c- Intermittent Fever:
Temp. falls to normal at least once during the day e.g. deep seated or systemic infections or malignancy. It can be defined as an exaggeration of the normal circadian rhythm, if this variation is extremely large the fever is termed (hectic)
**d- Cyclic (periodic or relapsing) Fever:**

Occurs in bouts of several days alternating with afebrile periods e.g.: Malaria - collagen disease - lymphoma - infectious mononucleosis - familial mediterranean fever.

- Hypothermia is defined as a central or core temperature ≤ 35°C.
- Hyperthermia means: core temperature ≥ 41°C (without elevation of the hypothalamic set point), is due to inadequate heat dissipation.
- Lower esophageal temperature closely reflect core temperature.
- Factitious fever is a self induced artificial fever, it can be induced by ingestion and hold hot liquids in the mouth immediately before a temperature check.

**Causes of hypothermia:**
- Cold weather
- Hypothyroidism
- Hypoglycemia
- Panhypopituitarism
- Adrenal insufficiency
- Alcohol toxicity

**Causes of hyperthermia:**
- Malignant hyperthermia (Halothane)
- Neuroleptic malignant $ (with phenothiazines)
- Serotonin syndrome due to serotonin reuptake inhibitors (SSRI)
- Pontine haemorrhage
- Heat stroke
- Status epilepticus.
- Thyrotoxic crises.

- Temperature-pulse dissociation (relative bradycardia) is seen with typhoid fever, brucellosis, leptospirosis, increase of intracranial tension and factitious fevers.

- For details (See tropical diseases)

Fever is an elevated body temperature that is mediated by an increase in the hypothalamic heat – regulating set point. In contrast, hyperthermia is an increase in body temperature above the hypothalamic set point i.e bypasses the normal homeostatic mechanisms.

**B - Pulse & Blood pressure:**

**BLOOD PRESSURE AND HYPERTENSION**

Arterial hypertension in adult is defined as persistent elevation of diastolic blood pressure > 90 mmHg or systolic ≥ 140 mmHg on at least two subsequent visits.

In healthy children and pregnant women, the blood pressure is typically lower so reading > 120/80 = hypertension

**Types of Hypertension:**

**Systolic hypertension:** (isolated systolic hypertension)

Elevation of systolic blood pressure ≥ 140 with diastolic blood pressure ≤ 90.

**N.B.:**
- Systolic blood pressure depends on COP (stroke volume x heart rate).
- Diastolic component depends on P.R and blood viscosity.
Causes of systolic hypertension:
- Atherosclerosis due to diminished compliance of arteries.
- Thyrotoxicosis.
- Complete heart block. (↓↓ HR → ↑↑ stroke volume), i.e.: bradycardia prolongs the filling time of the heart → (↑↑ stroke volume → ↑ systolic blood pressure).
- A.I (see C.V.S).

Diastolic Hypertension:
Elevation of diastolic Blood pressure > 90 mmHg.

Causes:

<table>
<thead>
<tr>
<th></th>
<th>Primary (idiopathic)</th>
<th>Secondary</th>
</tr>
</thead>
<tbody>
<tr>
<td>* Age:</td>
<td>35 - 55 years</td>
<td>&lt; 35 or &gt; 55 years</td>
</tr>
<tr>
<td>* Family history:</td>
<td>+ Ve F.H</td>
<td>- Ve F.H</td>
</tr>
<tr>
<td>* Cause:</td>
<td>No apparent cause</td>
<td>There is a cause e.g.: renal or endocrinal disease.</td>
</tr>
</tbody>
</table>

Classification & grades of Hypertension:

<table>
<thead>
<tr>
<th>Range (mmHg)</th>
<th>Classification</th>
</tr>
</thead>
<tbody>
<tr>
<td>(A) Diastolic:</td>
<td></td>
</tr>
<tr>
<td>&lt;85</td>
<td>Normal</td>
</tr>
<tr>
<td>85-89</td>
<td>High normal</td>
</tr>
<tr>
<td>90-104</td>
<td>Mild Hypertension</td>
</tr>
<tr>
<td>105-114</td>
<td>Moderate</td>
</tr>
<tr>
<td>&gt; 115</td>
<td>Severe</td>
</tr>
<tr>
<td>(B) Systolic:</td>
<td></td>
</tr>
<tr>
<td>&lt; 130</td>
<td>Normal</td>
</tr>
<tr>
<td>130 - 139</td>
<td>High normal</td>
</tr>
<tr>
<td>≥ 140</td>
<td>High</td>
</tr>
</tbody>
</table>

Recent Classification of hypertension

<table>
<thead>
<tr>
<th>Category</th>
<th>systolic (mmHg)</th>
<th>Diastolic (mmHg)</th>
</tr>
</thead>
<tbody>
<tr>
<td>* Normal</td>
<td>&lt; 130</td>
<td>&lt; 85</td>
</tr>
<tr>
<td>* High Normal</td>
<td>130 - 139</td>
<td>85 - 89</td>
</tr>
<tr>
<td>* Hypertension:</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Stage 1 (mild)</td>
<td>140 - 159</td>
<td>90 - 99</td>
</tr>
<tr>
<td>Stage 2 (moderate)</td>
<td>160 - 179</td>
<td>100 - 109</td>
</tr>
<tr>
<td>Stage 3 (severe)</td>
<td>180 - 209</td>
<td>110 - 119</td>
</tr>
<tr>
<td>Stage 4 (very severe)</td>
<td>≥ 210</td>
<td>≥ 120</td>
</tr>
</tbody>
</table>
HYPOTENSION:

It is a decline of systolic blood pressure < 95-100 mmHg (supine hypotension)

Causes:
* Heart failure.
* Stenotic lesions of the heart.
* Hypovolaemia.
* Addison's disease.
* Drugs, e.g.: diuretics, nitrates, β blockers.
* Primary (essential hypotension !?) (orthostatic hypotension see later)

Pseudohypotension!? It can be seen in cases of shock as the high peripheral vascular resistance in some types of shock → very weak korotkoff sounds.

☆ Technique of measurement of blood pressure:
 archae Put the cuff around the upper arm with its lower edge 3 cm above the elbow.
 archae The width of the cuff is equal to 40% of the arm circumference (about 12 - 14 cm).
 archae The length of the cuff is equal to 80% of the arm circumference (about 25 cm).
 archae Too short or narrow cuff gives false high reading.
 archae A loose cuff gives false high reading.
 archae If the arm is not supported → false increase of diastolic blood Pressure about 10 mm Hg.
 archae Making sure that the cuff lies at heart level. If the brachial artery is much below heart level → false high pressure.
 archae Failure to remove tight clothes from the upper arm gives false low pressure.

Measurement of blood pressure. (1) no constricting garments; (2) apply cuff of the appropriate size; (3) palpate brachial pulse before applying stethoscope; (4) support arm at heart level; (5) inflate cuff until radial pulse is impalpable, check systolic pressure by auscultation, deflate slowly until diastolic pressure is reached.

Methods:

(1) Palpatory method:
The cuff is inflated until the pulse disappears, and then deflated slowly, the level at which the pulse reappears = systolic pressure. The value of this method is to avoid the auscultatory gap (a silent interval between systolic and diastolic pressures)

(2) Auscultatory method:
➢ The stethoscope is placed over the brachial artery (cubital fossa, medial to the tendon of biceps).
➢ The cuff is inflated above systolic then deflate slowly until the first Korotkoff sound heard this corresponds to systolic pressure and when sounds completely disappear this corresponds to diastolic pressure.

Rapid deflation of the cuff gives falsely low systolic and high diastolic pressure.
Problems and special techniques:

1. Indications of measuring blood pressure in L.L.?

Normally blood pressure in L.L. > U.L. with difference about 20 - 40 mmHg (Systolic pressure). Put cuff above knee and auscultate popliteal artery, in coarctation of aorta pressure in U.L > L.L.

Hill's sign means that L.L. Pr. > UL pr. with difference more than 20 - 40 mmHg (systolic pressure) in cases of aortic incompetence. In takayasu's disease the blood pressure in UL is low but it is normal in L.L.

2. What is the difference of blood pressure in both U.L.? Normally there is no difference or there is a difference up to 10 mmHg (Systolic). If there is significant difference, diagnosis of thoracic outlet must be considered.

3. How can you measure the blood pressure in patient with A.F? The best is to measure blood pressure 3 times and take an average.

4. How can you measure the blood pressure in patient without audible Korotkoff sounds? This is by palpatory method to determine the systolic blood pressure. Also during deflation inspect the column of mercury, the point at which the oscillations of mercury disappear corresponds to the diastolic blood pressure.

5. How can you measure the blood pressure in obese patient? Inflate the cuff around the forearm and auscultate radial artery. It is better to use the large sized cuff for obese patients.

6. Trousseau's sign in hypertensive patient i.e. hypertensive patient with carpal spasm after inflation of the cuff above systolic pressure = Conn's $ as this disease causes hypertension + tetany.

7. Orthostatic hypotension

It is decline in arterial blood pressure in upright position ± Postural dizziness (See below).

Causes of Orthostatic hypotension

* Hypovolaemia e.g. bleeding, dehydration.
* Autonomic neuropathy e.g. D.M. or chronic renal failure.
* Early Addison's disease.
* Weakness of the muscles of L.L.

Measure the blood pressure in supine position and then ask the patient to stand and re-measure blood pressure after 1-2 minutes.

Orthostatic challenge or the tilt test

* This is important to study the changes of heart rate or blood pressure when going from supine to standing position.
* 1-2 minutes after standing, about 7-8 ml/kg of blood shift to the lower body → ↓ COP, also increase of circulating catecholamines and systemic vascular resistance occur.
* Normally pulse rate is increased by about 10/m and stabilizes after 45 seconds and diastolic blood pressure increase by 3-8 mmHg and stabilizes within 1-2 minutes. Systolic blood pressure decrease only slightly by 3-5 mmHg and stabilizes within 1-2 minutes.
* Orthostatic hypotension means decrease of systolic pressure ≥ 20 mmHg or decrease diastolic ≥ 10 mmHg, also there is increase of heart rate of at least 30/minute.
A CASE OF HYPERTENSION

**Personal history:**
* Age.
* Sex: female (pills)
* Smoking

**C/O:**
* Asymptomatic, or presented with complications e.g:
  - Heart Failure. (Dyspnea)
  - Strokes (weakness of one side of the body)
  - Encephalopathy. (Loss of consciousness)
  - Angina. (Chest pain)

**History of present illness:**
* Ask about the causes (renal - endocrinial..........).
* Ask about complication (as above)
* Ask about drug use; e.g. steroids, pills, NSAIDs (Na & H2O retention)

**Family history:**
* Positive in essential hypertension.

**O/E:**

**Local examination:**

**Heart:**
* Left ventricular enlargement
* Loud, 2\(^{nd}\) heart sound aortic component.
* Loud S1 with Lt. V. ++.
* Ejection systolic click over A1
* Ejection systolic murmur over A1.

**Abdomen:**
* Bruit over flanks for renal artery stenosis.
* Renal swelling

**Neurological:**
* Examine for lateralization. (See neurology)

**Q. Uses of sphygmomanometer!?**

1. Measuring the B.P.
2. Diagnosis of pulses alternans.
3. Diagnosis of latent tetany.
5. Diagnosis of coarctation of aorta and Takayasu's disease.
6. Diagnosis of deep vein thrombosis.
7. Tourniquet in venesection.
8. Haemostasis.
9. Hill's sign in A.I.
Comment on the following points:


(1) Rate:
Normal heart rate at mental and physical rest = 60 - 90/minute. (90-100 high normal)

Tachycardia
(Rate > 100/m)
* Sinus tachycardia.
* Paroxysmal atrial or ventricular tachycardia.
* Atrial flutter.
* Atrial fibrillation with high ventricular response.

> Causes of sinus tachycardia:

- Physiological e.g. stress
- Heart failure,
  {Sympathetic drive (to maintain C.O.P) as C.O.P = stroke V. X H.R}
- Thyrotoxicosis.
- Drugs: sympathomimetics (Salbutamol)

Bradycardia
(Rate < 60/m)

Sinus bradycardia
Complete heart block
Second degree heart block

~ Causes of sinus bradycardia:

- Physiological e.g. sleep
- Drugs: Digoxin, ß Blockers.
- Hypothyroidism.
- Obstructive jaundice.
- ↑ I.C.T.

(2) Rhythm:

(A) Regular rhythm e.g. sinus rhythm and paroxysmal tachycardias:

(B) Irregular rhythm:

* Extrasystole: Usually it is occasional irregularity, sometimes it gives regular irregularity

> Causes of extrasystole:

- Functional e.g. stress, smoking.
- Rheumatic heart disease.
- Congenital heart disease.
- Digitalis toxicity.

> Regular irregularity describes certain types of extra systoles e.g.: pulsus bigeminy or trigeminy.

> Diagnosis of extrasystole:

C/O: irregular palpitation at rest e.g. occasional strong beat or extra beat.

O/E:

* You can count 4 successive, regular beats.
* Exercise decrease irregularity. * Pulse deficit < 10 / m.
* Neck V. A wave is present, to be differentiated from AF.
Atrial fibrillation (AF): It is a form of marked irregularity or irregular irregularity. Multiple, frequent extrasystoles and variable degree of heart block may give irregular irregularity.

- Diagnosis of A.F.: It gives irregular pulse in both timing and volume.
- C/O: Irregular palpitation at rest.
- O/E:
  - You can not count 4 successive regular beats.
  - Pulse deficit >10/m.
  - Neck V. —— Absent A wave.
  - Exercise —— increase irregularity why?
  - As this enhance A-V conduction —— Increase passage of impulses from atrium to ventricle (increased ventricular response)

- Causes of A.F.:
  - MVD (Rh. Heart) * ASD (CHD)  * Ischemic heart disease.
  - Thyrotoxicosis or thyrocardia. (Thyrotoxicosis with cardiac manifestations only)
  - Constrictive pericarditis.
  - Lone A.F. (without apparent cause).
  - Systemic Hypertension.
  - Infective endocarditis
  - Pulmonary embolism.

(3) Volume (amplitude):

☆ Big pulse volume (Bounding pulse):

i.e.: Big difference between systolic and diastolic pressure (wide pulse pressure) leads to increase pulse amplitude. The pulse pressure is wide when it is > 50% of the systolic pressure.

- Causes:
  - A.I.  * Heart block
  - Fevers.  * Atherosclerosis.
  - Thyrotoxicosis.  * L.C.F. (Vasodilatation)
  - Hypoxia. —— peripheral vasodilatation.

☆ Small volume (small amplitude - pulsus parvus):

The pulse is weak and felt with difficulty.

- Causes:
  - Stenotic lesions: M.S, or A.S.
  - Marked tachycardia.
  - Heart failure.
  - Shock (weak and rapid pulse) = thready pulse

Pulse varying in amplitude:

- A.F.
- Pulsus alternans.
- Pulsus paradoxus.
- Variable degree of Heart block.
- Extrasystoles.
(4) Special character:
The normal peripheral pulse is made up of four phases:
- Percussion wave (P wave) ejection of blood into aorta.
- Tidal wave (T wave) movement of blood from central aorta toward the periphery.
- Dicrotic wave (D wave) closure of aortic valve.
- Secondary wave due to elastic recoil.

(A) Water hammer Pulse or Collapsing or Corrigan pulse).

Ch.Ch.:
Rapid upstroke, rapid down stroke with ill defined crest, with large amplitude felt in forearm, while the upper limb is elevated.

Mech.:
The artery collapses completely between each beat, but distends abruptly due to large pulse pressure to give sudden shock. This occurs with pulse pressure about 80 or more with diastolic pressure < 50 mmHg.

Causes:
* severe A.I.
* P.D.A.

A water hammer is an evacuated glass tube half filled with water that was popular toy in the 19th century. When held in the hand and inverted, it delivers a short hard knock.

(B) Plateau pulse. (Anacrotic pulse) or (parvus – tardus):
* Slow upstroke
* Prolonged duration.
* Small amplitude.

Cause: aortic stenosis.

(C) Pulsus bisferiens (felt in carotid or brachial Ars.)
* Pulse wave has 2 distinct peaks (both located in systole).
* Upstroke is sharp and rises to a first peak then falls and rises again to a second peak.
* So a double pulse is felt in carotid arteries.

Causes:
* Severe A.I
* Double aortic lesion with dominant A.I.
**Pulsus alternans:**
There is alternate weak and strong beats, but the rhythm is regular. When severe it can be detected by the fingers, but sometimes it may only be proved by the sphygmomanometer.

**Causes:**
- Lt. ventricular failure which leads to difference in refractory period of the muscle fibers, the diseased fibers have long ARP.

**Pulsus paradoxus:**
It is an inspiratory decline in systolic Bl. pressure > 15 - 20 mmHg. Normally there is slight decline in systolic pressure during inspiration, so pulsus paradoxus is considered to be an exaggeration of a normal phenomenon.

**Causes:**
- Constrictive pericarditis.
- Bronchial asthma (severe asthmatic attack).
- Cardiac tamponade.
- Right ventricular failure.

**Mechanism:**
Normally the systolic blood pressure decreases during inspiration (mild decrease) because during inspiration the lung expands and accommodates a big volume of blood, this will leads to decrease of blood flow to the left side of heart → low cop, but this is compensated by the increase in the venous return during inspiration, so there is a slight decline in the systolic blood pressure.

In constrictive pericarditis or tamponade the compressed right side of the heart prevents the proper venous return and filling of the right side of the heart, meanwhile the expanding lung accommodates a greater amount of blood so, the amount, of blood reaching the left side of the heart decreased → ↓ COP → ↓ systolic blood pressure → weak pulse volume. Also severe asthmatic attack → ↑ intrathoracic pr → ↓ venous return → ↓ COP.

**Significance:**
- It indicates severity of the cause e.g.: (severe asthmatic attack)
- Optimal assessment requires sphygmomanometer.

**Equality:**
Both radial arteries must be examined at the same time for equality as regard volume.

**Causes of inequality:**
- Thoracic outlet $.$
- Embolus or thrombus in brachial or radial arteries.
- Dissecting aortic aneurysm.
- Atheroma in Subclavian artery.
(6) Peripheral pulsations:

Significance: (It is important to feel peripheral pulsations in the following conditions)
- Old age (atherosclerosis)
- D.M.
- Thromboembolic risk as in A.F., Infective endocarditis, and prosthetic valve.

(a) Radial artery: Felt lateral to the tendon of flexor carpiradialis.
(b) Brachial artery: felt at the elbow medial to the biceps tendon.
(c) Subclavian Artery: is felt from behind by pressing downward above the middle of clavicle.
(d) Carotid: Is felt medial to the sternomastoid muscle by index finger or thumb at the level of cricoid cartilage against the transverse process of 6th cervical vertebrae.
(e) Femoral: Is felt at the mid inguinal ligament while the thigh of patient is inflexion and abduction.
(f) Popliteal artery: Is felt in the middle of the popliteal fossa while the patient lies supine with the knee slightly flexed, place both thumbs on the patella and curl the fingers of both hands firmly into the popliteal fossa.
(g) Posterior tibial artery: Is felt behind the medial malleolus.
(h) Dorsalis pedis artery: Is felt lateral to the extensor hallucis longus tendon against navicular bone on the dorsum of the foot.

(7) Capillary pulsation (Aortic regurgitation):
- The capillary pulsation may be elicited by pressure on the finger nail just to cause blanching. The test is positive when the blanched area becomes alternately red/blanch with each heart beat.
- Also, we can examine for capillary pulsation by pressure with glass slide on the tongue or everted lower lip to produce an area of blanching. Capillary pulsation can be seen in uvula (Muller's sign), fundus of the eye and in the face (lighthouse sign).

(8) Tension is the minimal degree of compression required to feel the maximum pulse volume, this reflects the diastolic pressure.

(9) Force is the minimal degree of compression to obliterate the radial pulsation, this reflects the systolic pressure.
(10) Condition of the arterial wall:
Compress the radial artery by the index then roll the artery under the middle finger after emptying by the index, in young persons the arterial wall is so compliant that you can not feel it, but in old age it is felt as a cord like structure (arteriosclerosis).

Radial artery  Brachial artery  Femoral artery

Popliteal artery  Posterior tibial artery  Dorsalis pedis artery

Also, we can examine the pulse volume, tension and force by using the fingers of left hand to palpate the radial artery of the patient's right arm and use the right thumb to compress the brachial artery until the radial pulse is completely obliterated, then release the pressure gently over the brachial artery until you feel the radial pulse again.

VIII- Head and neck:

(1) Skull:
1- Shape.
   - Oxycephaly (vertically elongated head with pointed vertex)
   - Brachycephaly (flat back of the skull).
   - Dolichocephaly (Elongated head)
2- Depressed fractures.
3- Enlarged skull → Hydrocephalus and Paget's disease.
4- Small skull → Microcephaly and craniostenosis.
5- Enlarged supraorbital ridges in acromegally due to increase in the size of the frontal air sinuses, also there is prognathism.
6- Tender temporal artery in giant cell cranial arteritis.
7- Bruit on auscultation of the skull in cases of intracranial arteriovenous malformation.
(2) Eye:

Loss of hair in the outer 1/3 of eye brow:
- Myxoedema.
- Leprosy.
- Artificial.

Exophthalmos:
- Thyrotoxicosis.
- Cavernous sinus $.$
- Leukaemic deposits behind eye ball.
- Congenital glucoma.

Enophthalmos (sunken eye):
- Dehydration.
- Horner's syndrome.

Eyelid:
- Ptosis:
  - Congenital
  - Hysterical.
  - Mechanical.
  - Oculomotor nerve paralysis
  - Horner's syndrome.
  - Myasthenia gravis.
  ⇒ Retraction of eye lid in hyperthyroidism
  ⇒ Ectropion, entropion.
  ⇒ Blepharospasm in painful eye conditions
  ⇒ Stye and blepharitis

⇒ Oedema and puffy eye lids:
  - Renal, nephrotic or nephritic.
  - Myxoedema.
  - Mediastinal syndrome with S.V.C. obstruction.
  - Lack of sleep or excessive sleep !?.
  - Chronic cough.
  - Angioneurotic oedema.
  - Rarely as a part of generalized oedema, as liver cirrhosis and C.H.F.

Sclera:
⇒ Blue sclera:
  - Congenital glucoma.
  - Osteogenesis imperfecta.

Jaundice: See complexion.

Conjunctiva:
- Pallor. See complexion (conjunctiva is not reliable to diagnose pallor)
- Sub conjunctival hemorrhage (as in blood disease - trauma - cough severe hypertension), it usually has an upper limit to be differentiated from conjunctival congestion.
- Conjunctivitis is associated with photophobia, lacrimation and a sticky discharge of the eyelid.
Vitamin deficiency as Xerosis (Vit. A↓), vascularization (Vit. B2↓).

Pingueculae are triangular yellow deposits beneath the conjunctiva between the canthus and the edge of cornea, they develop with advancing years, and are of no clinical value. Pterygium is a patch of progressive fibrosis in the same area which may encroach upon the cornea.

**Cornea**
- Arcus senilis in senile patients and young with hyperlipidemia
- Opacitis due to trauma or infection.
- Kayser Fleischer ring in Wilson’s disease.

**Pupil:**
- See neurology.

**Lens:**
Cataract occurs in:
- Diabetes mellitus.
- Cretinism.
- Mongolism.
- Scleroderma.
- Myotonia atrophica.
- Hyperparathyroidism.

**The iris and ocular tension**
- Iritis (uveitis) is often a manifestation of systemic disease e.g. ankylosing spondylitis and Behcet's disease.
- The ocular tension can be tested digitally, it is tested in patients with headache or diminished visual acuity.

**Fundus examination:**
- Hypertensive patients.
- Diabetes M.
- Optic neuritis and its causes.
- Papilledema and its causes.

(3) **Nose and ears:**
- Redness of the tip of the nose: alcoholism.
- Sunken bridge (saddle nose) in congenital $,
  - Trauma, congenital or Wegner's granuloma.
- Working ala nasi (pneumonia or nervousness.)
- Rhinophyma (thickening of the nasal skin with induration and redness),
  - The nose may appear bulbous. It may be associated with alcoholism.
- Ochronosis (blue black pigmentation of the ear, nose and cheeks from binding of homogentisic acid to connective tissue and cartilage) this occur in Alkaptonuria.
- Lupus pernio (bluish red swelling of the nose and ear in cases of sarcoidosis).
- Nasal polyps with chronic atopic rhinitis and aspirin sensitive asthma.
- Large nose: congenital - acromegaly - myxoedema.
- Ears : Gouty tophi on the helix, discharge, F.B in the external meatus.
- Low set ear may be associated with congenital anomalies e.g. congenital H.D.

(4) **Cheeks:**
- Pale in anemia - Malar flush in M.S. and myxedema - Butterfly erythema in S.L.E. -
  - Bloated in cushing disease – ochronosis as above.
(5) Lips:
- Cheilosis is a reddening and cracking of one or both angles of the mouth (angular cheilosis or angular stomatitis), it occurs due to riboflavine or iron deficiency, it is also caused by candida.
- Cheilitis: painful vertical fissures mainly of lower lip caused by malnutrition or with Crohn's disease, it may occur with exposure to sunlight and wind.
- Pallor, syanosis (See complexion.)
- Angiocedema, herpes labialis.

(6) Teeth:
- Discoloration: Tobacco, poor hygiene or flourosis.
- Loosing of teeth: D.M.
- Wide spacing: Acromegaly.
- Notched: congenital syphilis (Hutchinson's teeth)
- Dental caries, tooth extraction for its relation to infective endocarditis.

(7) Gum:
- Bleeding: Vitamine C ↓, thromobocytopenia, chronic liver disease.
- Hypertrophy: Epanutin, monocytic leukaemia.
- Blue line: lead poisoning.

(8) Tongue:
The surface of the tongue normally varies as regard colour and appearance. Shades of pink and red or even yellow, brown or almost black may be of no medical significance though they are a potential cause of anxiety to the subject.

- Colour:
  - Black with iron therapy.
  - Brown with smoking.
  - Blue with cyanosis
  - Pale with anaemia.

- Atrophy: (Glazed red tongue)
  - With iron ↓ anaemia.
  - Hypovitaminosis e.g.: B12 ↓

- Leucoplakia:
  - Due to chronic irritation, it is precancerous.

- Moisture:
  - Dry tongue (under surface) = dehydration.

Dry mouth
- Dehydration, mouth breathing, drugs e.g. anticholinergics and sjogren syndrome.
**Ptyalism (increased salivation)**
- Neurosis - stomatitis
- Reflex from GIT diseases e.g. DU.
Tremors of the tongue:
- Thyrotoxicosis.
- Parkinsonism.
- Essential familial tremors.

Scrotal or fissured tongue:
- The tongue is covered by painless shallow or deep fissures, it may present in down syndrome and acromegally.

Strawberry tongue:
- Scarlet fever.

Percussion or tapping for:
- Fasciculations and myotome phenomenon

Cranial nerve examination:
- See neurology (12th cranial nerve)

Macroglossia:
- In myxoedema, acromegaly, amyloidosis and hemangioma.

Pseudomacroglossia:
- In Down $.

Oedema of the tongue occurs in angioneurotic oedema.

Buccal mucosa, palate, tonsils and pharynx:

Buccal mucosa:
- Pigmentation (Addison's disease).
- Aphthous stomatitis, they are ulcers on the inner sides of the lips, the edge of the tongue and the insides of the cheek.
- Koplik's spots.

Tonsillitis and pharyngitis: may be related to Rh. F and acute G.N.

Palate:
- Jaundice appears early in the soft palate.
- Petechial spots in thrombocytopenic purpura and leukemia.
- Pin point petechial spots also in infectious mononucleosis.
- High arched palate or cleft plate in congenital conditions, palatal movement, palatal and pharyngeal reflexes (see neurology).

Breath:
- Acetone smell in diabetic ketoacidosis.
- Ammonical smell in uremia.
- Foetor hepaticus in hepatic failure.
- Putrid smell in suppurative lung disease (Halitosis)

Parotid enlargement:
- Mumps.
- Liver cirrhosis.
- Stones & Tumor.
- Sjogren syndrome.
- Sarcoidosis.
- Endemic parotitis.
- Hypoproteinaemia.
(13) Neck:
- Torticollis: Hysterical, myositis of sternomastoid.
- Rigidity: Meningeal irritation & cervical spondylosis.
- Thyroid enlargement (see sheet of endocrinology).
- L.N. (see sheet of lymphadenopathy)
- Thrill: systolic thrill:
  - Thyrotoxicosis.
  - A.S.
  - Carotid shudder in A.I, it also occurs in A.S? (see CVS)
- Pulsation → Arterial & venous. (See later)

## NECK VEINS

(1) Venous Pressure

The venous pressure is measured most accurately by manometry. However, an adequate approximate of the venous pressure for clinical purposes can be obtained by inspection of the jugular pulsations. Internal jugular vein is preferred. The external jugular vein is visible but it is not preferred for examination because it is prone to kinking and partial obstruction as it traverses the deep fascia of the neck.

Right sided pulsations are preferred because, Left sided ones may be present falsely prominent due to Kinking of the left innominate vein.

The reference level for bedside evaluation of venous pressure is the sternal angle (the clinical zero level).

The vertical distance between the top of the venous column and the sternal angle represents the venous pressure, the normal upper limits for this distance is about 2cm when reclining the patient at an angle about 45 degree so venous pulsations can be seen normally in the lower neck. The distance is also 2cm in sitting position but the venous pulsations are hidden by the part of the thoracic cage above the sternum.

These values are less than those obtained by manometry because the true zero level is the right atrium.
Method of examination of Venous Pressure:

1. Examine the patient at sitting position, normally no venous pulsations will appear.
2. If no visible venous pulsations appear in sitting position, examine patient at 45 degree and measure the vertical distance between the upper limit of venous pulsations and sternal angle (normally the distance is about 2cm).
3. If there is visible venous pulsations at sitting position, measure the distance between the top of venous pulsations and the sternal angle (this means high venous pressure).

- If you begin to examine patient at 45 degree with no visible venous pulsations, examine him in sitting position, this is because when the venous column is very high the pulsations will not appear.
- The central venous pressure (CVP) can be determined by transvenous placement of a catheter in the right atrium. The normal CVP is < 9cm water (7mmHg).
- A jugular venous pressure 2 cm above the sternal angle is roughly equivalent to CVP of 7 cm, as the sternal angle is 5 cm above the right atrium.

General sheet:
- In cases of very high CVP the internal jugular is so full that pulsations may not be visible.
- In cases of low CVP the required angle is between 0 and 30.
- In cases of very low CVP the pulsations may not be visible at all.

(2) Venous Pulse (Waves):

- The normal visible jugular venous pulse consists of three positive waves (A, C and V) and two negative waves (X and Y).
- The A wave is normally the tallest wave exceeding both C and V waves in amplitude.
- The X wave is usually deeper than the Y wave.
- The heart sounds are preferable to the carotid pulse or apex beat for timing the venous pulsations.

Waves:

A Wave:
* Due to atrial contraction.
* It is a Presystolic wave.

C Wave:
* Due to elevation of the tricuspid valve at the start of ventricular contraction or it is just a transmitted pulsation of carotid artery at the onset of ventricular systole.

V Wave:
* Due to accumulation of venous blood in the right atrium during ventricular systole.
* It is a systolic wave.

Y Descent: (Diastolic collapse)
* It is due to descent of blood from right atrium to right ventricle (diastolic collapse)
X Descent: (Systolic collapse).
* It is due to right atrial relaxation.
* It is a systolic wave.

Abnormalities of the jugular venous pulse:

The A Wave:

1. Large or giant A waves:
   * T.S. * P.S. * P++
   * Ebstein's anomaly.
2. Absent A seen in atrial fibrillation.
3. Canon A waves? Occur in cases of complete heart block and PVT.

X Descent: (Systolic collapse)

It is obliterated in T.I.

V Wave:

It is large and prominent in T.I and right sided heart failure.

Y Descent (Diastolic collapse)

* Shallow Y descent in T.S.
* It is deepest rapid in constrictive pericarditis.

Special techniques:

★ Hepatojugular reflux i.e. The patient is positioned so that the jugular venous pulsations are properly monitored. An angle of 45 usually suffices. Abdominal compression for 15-30 seconds (20-35 mmHg), 35mmHg equivalent to a weight of 8 kg, when the venous pressure increased ≥ 4 cm, it is considered positive. This test is positive in R.V.F, T.I, T.S, constrictive pericarditis and pericardial tamponade.

★ Inspiratory filling in pericardial effusion. (Kussmaul's sign)
★ Expiratory filling in emphysema?

Causes of congested neck veins: (high venous pressure)

1. Non pulsating.
   a- S.V.C. obstruction due to thrombosis.
   b- Some cases of constrictive pericarditis (i.e. leading to SVC obstruction).
   c- Full venous column!?

2. Pulsating

1) Right sided heart failure, is the commonest.
2) Tricuspid valve disease as T.I & T.S.
3) Increased intrapericardial pressure.
   a- Pericardial effusion. b- Constrictive pericarditis.
4) Increased intrathoracic pressure:
   a- Massive pleural effusion. b- Tension pneumothorax.
   c- Emphysema.
5) Increased intra-abdominal pressure:
   a- Tense ascites. b- Pregnancy.
   c- Huge abdominal swelling.
6) Over transfusion especially in patient with renal insufficiency → hypervolaemia.

7) Hyperdynamic circulation?

**Difference between arterial and venous pulsations.**

i.e. By waveform and response to position, respiration, palpation and abdominal compression.

<table>
<thead>
<tr>
<th>Venous</th>
<th>Arterial</th>
</tr>
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<tbody>
<tr>
<td>1) Change by changing the position of the patient</td>
<td>1) No change</td>
</tr>
<tr>
<td>2) During straining the neck veins become engorged with diminished pulsation</td>
<td>2) No effect</td>
</tr>
<tr>
<td>3) Better seen than felt</td>
<td>3) Better felt than seen</td>
</tr>
<tr>
<td>4) Wavy</td>
<td>4) One wave (Jerk)</td>
</tr>
<tr>
<td>5) Internal to sternomastoid</td>
<td>5) Medial</td>
</tr>
<tr>
<td>6) Can be obliterated by pressure</td>
<td>6) Cannot be obliterated by pressure</td>
</tr>
<tr>
<td>7) Got an upper level</td>
<td>7) No upper level</td>
</tr>
<tr>
<td>8) Hepatoljugular reflex +Ve</td>
<td>8) -Ve</td>
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</tbody>
</table>

**N.B.:**
- When you obliterate the venous pulsation obliterate at the root of the neck to obliterate the external and internal Jugular veins.
- An alternative method to measure venous pressure by inspecting the veins of the dorsum of the hand in supine position with the arm is slowly raised. The level at which the veins collapse can be related to the sternal angle and then the CVP is measured.

**IX- Hands:**

1. **Temperature of hands:**
   a- Cold hands:
   - Low C.O.P. and shock.
   - Neurosis.
   - Associated with peripheral cyanosis.
   b- Warm hands:
   - Hot weather.
   - High C.O.P. states e.g. thyrotoxicosis.
   - Associated with central cyanosis.

|The hands usually warm in anaphylactic shock and in septic shock.|

2. **Tremors:**
   (Studied with the hand at rest and then outstretched)

   *Def.:* Rhythmic, oscillatory involuntary movement of the hands or other parts of the body around a fixed point

   a- Fine tremors: ( ↓ amplitude ↑ frequency)
   - Senility, thyrotoxicosis, neurosis, familial, fatigue or use of β₂ agonists (e.g.: Salbutamol)

   b- Coarse tremors: (↑ amplitude ↓ frequency)
   1) Flabbing tremors:
      - Hepatic failure, renal failure, and respiratory failure (with CO₂ retension)
   2) Parkinsonian tremors
   3) Intension tremors.

3. **Nails:**
   - Bitten nail in anxious personality.
Spooning (Koilonychia) in iron deficiency anemia.
Capillary pulsation in A.I.
Splinter hemorrhages e.g.: infective endocarditis - vasculitis - trauma - trichinella (horizontal)
Cyanosis: central or peripheral (see before).
Pallor (see before).
Clubbing (see before).
Striate Leuconychia (It is white transverse bands due to trauma, debilitating disease or in normal person.)
Onycholysis (separation of the nail plate from the nail bed. It may occur due to trauma, graves' disease and psoriasis.)
Beau's lines due to temporary arrest of nail growth, they are transverse grooves which appear at the same time on all nails a few weeks after an acute illness and move out to the free margins as the nail grows.
Half and half nail (white proximally and brown red distally)
Occur in patient with chronic renal failure. (Lindsay's nail)
Red lunulae occur with heart failure (Red half moon in nail bed)
Blue lunulae occur in Wilson's disease. (Blue half moon in nail bed)
Nail pitting occurs in psoriasis and rarely other conditions.
Nail bed infarcts occur in vasculitis e.g. in SLE and polyarteritis.
Terry's nail, it is whitening of the proximal 80% of the nail leaving a small rim of peripheral reddening. It occurs in liver cirrhosis, old age or with heart failure.
Yellow nail syndrome, it is a yellow color of nail plate due to abnormal lymphatic circulation.

4. Palms:
a) Palmer erythema in liver cell failure, alcoholics, rheumatoid disease or normal persons.
b) Sweating:  
Neurosis (palm)  
Thyrotoxicosis (palm and dorsum)
c) Wasting of the small muscles of the hands.
d) Temperature (see before):
  > Cold hand + sweating occur in anxiety.
  > Warm hand + sweating common with thyrotoxicosis.
  > Warm hand + cyanosis means ↓ arterial O₂ saturation
  > In heart failure the hands tends to be cold due to ↓ COP, if they warm the cause of heart failure may be hyperthyroidism or Cor pulmonale.
f) Hyperkeratosis in manual workers.
g) Jane way macules in infective endocarditis.
h) Nodules:
- Osler's nodules (Infective endocarditis).
- Heberden's nodules (Osteoarthritis).
- Tophi (Gout).
- Subcutaneous nodules (Rheumatoid disease).

5. Deformities:
   a) Polydactyly & Syndactyly as in Laurence Moon Biedle $.
   b) Arachnodactyly e.g. Marfan's $.
   c) Spade hand in acromegaly.
   d) Obestetric hand as in tetany.
   e) Dupuytren's contracture as in L.C.F. & alcoholism.

X- Lower Limbs:

1. Oedema: (see later)
   - Over bony prominence. You can press on the following areas. (Posterior to malleoli, dorsum of the foot and the chin of tibia).
   - Press for about 1/2 minute.
   - Determine whether it is pitting or non pitting edema.
   - Look for the extent of oedema.

2. Cyanosis - clubbing of toes.

3. Pellargic rash over the greater trochanter.(e.g: in malnourished farmers)

4. B.P. difference between upper and lower limbs in coarctation of the aorta or for Hill's sign in A.I. or in cases of takayasu's syndrome

5. Venous system for D.V.T & Varicose veins.

6. Deformity as in: Marfan's $, Duchenne myopathy, Friedriech's ataxia & other congenital diseases.

6. Ulceration At sides of ankle especially medially (chronic venous insufficiency). Toes or feet (Ischaemia), it is painful. Ulcers in pressure points e.g sole and heel (Neuropathic or trophic), it is painless.

**CLUBBING (Digital clubbing):**

* Definition:
It is a selective bulbous enlargement or swelling of the terminal phalanges of the fingers and toes particularly on the dorsal surface due to proliferation of the soft tissue of the nail (nail bed and the tissue at the nail base), interstitial oedema and vasodilatation of the arterioles and capillaries of the soft tissue of the nail due to toxemia, hypoxia, malignancy or other disorders.

* Causes:
1. C.V.S.:
   - Infective endocarditis.
   - Congenital cyanotic heart disease.

2. Chest:
   - Suppurative lung disease.
   - Interstitial pulmonary fibrosis.
   - Bronchial carcinoma, mesothelioma of the pleura.
   - Advanced chronic obstructive lung disease.
3. G.I.T.:
- Liver cirrhosis especially primary biliary cirrhosis!
- Ulcerative colitis.
- Crohn's disease.
- Bilharzial polyposis of colon.
- Coeliac disease.

4. Occupational:
   For example it is limited to thumb and index finger as in shoe makers.

5. Endocrinal e.g in graves' disease (thyroid acropachy), it occurs in 1% of cases.

6. Familial or congenital clubbing.

**Degrees of clubbing:**

1\(^{st}\) Degree: (Swelling of the tissue at the base of the nail) i.e. the s.c tissue over the base of nail.
- Obliteration of the angle of the nail (i.e. the angle between nail base and its adjacent skin, the angle of Lovibond).

2\(^{nd}\) Degree:
- The swelling involves the nail bed leading to increase of the curvature of the nail in its long axis
- The increase of convexity of the nail is called: (Parrot peak appearance).

3\(^{rd}\) Degree:
- (Swelling of the pulp of the finger in all its dimensions with drum stick appearance).

4\(^{th}\) Degree: (Hypertrophic pulmonary osteo arthropathy) i.e HOA
- Thickening of the distal ends of the long bones especially at wrists and ankles.
  It is due to subperiosteal new bone formation, so it is an X-ray finding.

* Clubbing of fingers is usually bilateral and nearly symmetrical and in both fingers and toes. (It less obviously in the toes)
**Causes of unilateral clubbing of one upper limb:**
- Thoracic inlet syndrome (cervical rib – pancoast tumor)
- Aortic aneurysm, Arteriovenous fistula in dialysis patients.

**Causes of clubbing in L.L. only:** PDA with reversed shunt.

<table>
<thead>
<tr>
<th>DD of clubbing:</th>
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<tbody>
<tr>
<td>* Increased nail convexity.</td>
<td>* Arthritis of ankles or wrists (like HOA)</td>
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<tr>
<td>* Pachydermoperiostosis</td>
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- The nail consists of a strong keratinous nail plate over the dorsal surface of the end of each digit protecting the finger tip.
- Recently it is believed that the clubbing is mediated by platelet growth factors or a humoral substance which dilate the vessels of the finger tip.
- Pale clubbing due to toxemia e.g.: infective endocarditis (Toxic clubbing).
- Blue clubbing due to hypoxia e.g. Cong. heart disease (Hypoxic clubbing)
- Diagnosis of early clubbing by observing the obliteration of the angle between the nail base and its adjacent skin. The angle can be visualized by resting a pencil over the nail, in normal persons there is a clear window below the pencil and above the nail, but in patients with clubbing there is no clear window as the pencil rests fully over the nail.
- Another sign to confirm is the loss of angles (schemroth's sign); it consists of disappearance of the diamond shaped window normally present when the terminal phalanges of paired digits are juxtaposed.
- Fluctuation of the nail bed is also an early sign of clubbing, the test is positive when the sensation of movements of the nail is greater than the very slight degree of fluctuation in normal persons.
- Clubbing usually occurs first in the thumb and index finger.
- Toxemia leads to clubbing within few weeks, but hypoxia leads to clubbing within several months and even years.

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**EDEMA**

**Definition:**
Edema means swelling of the tissues due to increase in the volume of the interstitial tissue fluid. In an adult weighing about 70 kg has about 45 litres of body fluid: 30 litres intracellular, 10 litres interstitial and about 5 litres in the circulating blood volume.

**Diagnosis:**
There may be a considerable increase in the interstitial fluid volume before it is clinically evident. The symptoms and signs of edema are the following:
1. Unexplained weight gain without any apparent tissue swelling clinically (occult edema).
2. Tightness of a ring or shoe.
3. Puffiness of the face, swollen extremities, enlarged abdominal girth, and persistence of indentation of the skin following pressure (pitting).
4. Examination of serous sacs may reveal pleural effusion, pericardial effusion or ascites.

To assess the state of hydration examine skin elasticity, tongue, intraocular pressure and blood pressure in supine and upright position. Also check for edema and venous pressure.
Generalized Edema

(1) Congestive heart failure (cardiac edema)

Ch.Ch.:
(1) Cardiac history is positive.
(2) Edema mainly in dependent parts.
(3) 0/E: (Signs of right sided heart failure)
* Congested neck veins.
* Congested liver (the liver is enlarged and tender)
* L.L. edema.
* Gallop on tricuspid area.

Causes of right sided Heart failure:
(1) Left sided heart lesion. E.g.: (M.S. → P++ → right sided heart failure)
(2) Chest diseases (cor pulmonale) e.g.: COPD. → hypoxia → pulm V.C → P++.
(3) P.S.
(4) Cardiomyopathy.

Pericardial effusion and constrictive pericarditis can lead to cardiac edema.

(2) Renal Edema

(A) Nephrotic $ (heavy proteinuria):

Ch.Ch.:
* Gradual onset, puffiness of eye lids.
* No oliguria, No hypertension, No haematuria or azotemia.

N.B.: Cardiac edema may occur in nephrotic $ (nephrotic $ may lead to pericardial effusion) → Cardiac edema, dyspnea, orthopnea.

(B) Nephritic $:

Ch.Ch.:
* Puffiness of eye lids.
* Acute onset.
* Usually there is oliguria, haematuria, azotemia and hypertension.

* In cases of renal of edema search for the cause of nephrotic or nephritic $.
* The cause of edema in nephrotic $ is mainly hypoproteinaemia.
* The cause of edema in nephritic $ is mainly salt and water retention.

(3) Hepatic edema:

Ch.Ch.:
(1) Ascites, and L.L. edema.
(2) Manifestations of liver disease.
(3) History of the causes as alcoholism, Hepatitis.

N.B.:
Other types of edema in a patient with liver disease e.g.:
cardiac edema in Bilharzian Cor pulmonale, renal edema due to glomerulonephritis secondary to Bilharziasis or viral hepatitis

In patient with ascites search for heart, liver or renal disease. if negative with no L.L. edema suspect local cause e.g.: T.B peritonitis, or malignant ascites.
Localized edema:

(1) D.V.T:
* Causes:
  1. Post partum.
  2. Post operative with prolonged recumbency.
  3. Nephrotic $?
  4. Malignancy.
  5. Other causes of thrombophilia (see hematology)
* O/E:
  Unilateral edema, pitting, tender calf muscle, +ve Homan sign. Examine heart for p++ or right sided heart failure (Thromboembolism) i.e. thromboembolic p++.

D.D. of DVT (Tender or swollen calf area):
  Cellulitis - Lymphedema - Osteomyelitis - Rupture plantaris - rupture baker cyst

D.D. of tender calf muscle:
  DVT- Rupture plantaris - Diabetic neuropathy - Cellulites.

(2) Lymphedema :( non pitting)
  Filariasis - Post mastectomy.

(3) Chronic venous insufficiency → L.L., signs of varicose veins, ulceration and pigmentation of the leg.

(4) Orthostatic edema: → L.L., diurnal variation.
  Occupational Factors

(5) Angioneurotic edema → sudden onset, self limited, mainly affecting the face (lips) asymmetrical, history of allergy.

Examination of a case of oedema:
The following points must be examined:
  1. The oedema is unilateral or bilateral.
  2. The oedema is pitting or non pitting.
  3. What is the level of edema e.g.: (below knee or reach above knee)
  4. Examine the serous sacs for ascites, pericardial or pleural effusion.
  5. Examine the heart and liver.

Pitting edema occurs in cardiac, renal or hepatic edema also occurs in DVT.
Non pitting edema occurs in Lymphedema and Angioneurotic edema.
XI. Lymph nodes (See later)

XII- Skin: inspect and palpate the skin for the following:

A. Colour and pigmentation:

1) Hyperpigmentation:

- Endocrinal
  - Addison's disease.
  - Cushing's.
  - Estrogen therapy.
  - Malnutrition.

- Metabolic
  - Chronic renal failure.
  - Primary biliary cirrhosis.
  - Haemochromatosis.

Localized pigmentation can occur in senility and neurofibromatosis. (Cafe au lait spots).

2-Depigmentation:

- Vitiligo (1st or 2nd degree) due to skin disease or with Grave's disease. Panhypopituitarism gives alabaster skin.

Colours as redness, pallor, cyanosis, jaundice and carotenaemia are discussed before.

B. Moisture: (dryness, sweating or oiliness)

- Dryness in dehydration and old age.
- Sweating in hyperthyroidism – anxiety - hypoglycaemia.

C. Texture:

- e.g. smoothness – roughness (hypothyroidism)

D. Elasticity:

- Decreased in old age – dehydration (poor skin turgor).
- Increased in inherited collagen diseases. e.g.: Ehler – Danlos syndrome.

Lift a fold of skin and notice the ease with which it is moved (mobility) and the speed with which it return into place (turgor)

E. Thickness:

- Increased in acromegaly, myxoedema & in localized areas as in Lymphedema.
- Decreased in old age, malnutrition.
F. Striae:
Rubrae & Alba in cases of Cushing disease.

G. Hair:

- Malnutrition.
- Myxoedema.
- Sheehan's $.
- Alopecia areata and its causes.
- Cytotoxic drugs.

- Fall of hair

- Hypertrichiosis: (Excessive hair growth in any site)
  - Idiopathic.
  - Familial.
  - Racial.
  - Cushing $.
  - Steroid & cyclosporine therapy.

- Hirsutism:

  (Male distribution of hair in females)
  - Idiopathic.
  - Polycystic ovary syndrome.
  - Adrenogenital syndrome.

H) Skin rash and its distribution.

I) Skin lesions of systemic diseases.

- Erythema nodosum (panniculitis or inflammation of subcutaneous fat) in Sarcoidosis –TB – Post streptococcal infections.
- Pyoderma gangrenosum in inflammatory bowel disease.
- Purpuric eruption with raised edge in vascular purpura, or without raised edge in thrombocytopenic purpura.
- Dermatitis herpetiformis in Gluten enteropathy.
- Xanthelasma i.e. subcutaneous deposits of cholesterol just medial to the eyelids which are suggestive to hyperlipedemia. Hyperlipedemia also manifested by xanthomata on the Achilles, patellar tendons or palms.
- Skin manifestations of endocrinal diseases and collagen diseases (see endocrine and rheumatology).

Terminology of skin lesions

- Macule: small flat area of altered colour.
- Papule: Elevated solid area ≤ 5 mm.
- Nodule: Elevated solid area > 5 mm.
- Plaque: Elevated flat-topped lesion > 5 mm.
- Vesicle: Elevated fluid-filled lesion ≤ 5 mm.
**General Sheet**

- Bulla: Elevated fluid-filled lesion > 5 mm.
- Blister: Common term for vesicle or bulla.
- Pustule: Discrete, pus filled raised area.
- Wheal: Pruritic, erythematous elevated area resulting from dermal edema.
- Petechiae: Pinhead-sized macules of blood in the skin.
- Purpura: A larger macule or papule of blood in the skin.
- Ecchymosis: A larger extravasation of blood into the skin.
- Haematoma: A swelling from gross bleeding.
- Telangiectasia: Visible dilatation of small cutaneous blood vessels.
- Ulcer: An area of skin from which the whole of the epidermis and at least the upper part of the dermis has been lost.
- Atrophy: Thining of skin due to diminution of the epidermis, dermis, subcutaneous fat.

**Xanthomas (xanthomata)**

Xanthomas are smooth surfaced yellow or orange papules or nodules in the skin due to focal dermal aggregations of lipid-loaded cells (foamy histiocytes). Different types of hyperlipidemia may induce varying patterns of xanthomas.

**Types or patterns:**
- Eruptive xanthoma, occur on the buttocks, posterior thighs (hyperlipidemia types I, IIb, III, IV and V). Also, developed with primary biliary cirrhosis.
- Tuberous xanthoma, over the joints especially knees and elbow (types IIA, III).
- Tendinous xanthoma, over the Achilles tendon and finger extensor tendons (Types IIA and III)
- Plane xanthoma, on palmar creases (Type III) and with primary biliary cirrhosis.
- Xanthelasma, with type IIA and III or without lipid abnormality.

**SUMMARY OF GENERAL EXAMINATION**

1. General condition.
2. Mental state.
4. Decubitus.
5. Facial expression.
6. Complexion (pallor, jaundice & cyanosis).
7. Vital signs (pulse, blood pressure & temperature).
8. Head & neck.
10. L.L.
11. L.N.
12. Skin.
1. The surface anatomy of the heart is represented by an irregular quadrangle bounded by 4 points:
   - Point (1) lower border of 2nd left costal cartilage 1.5 inch from the median plane.
   - Point (2) upper border of 3rd right costal cartilage 1 inch from the median plane.
   - Point (3) 6th costal cartilage 0.5 inch from the median plane.
   - Point (4) 5th left intercostal space about 3.5 inches from the median plane.

2. The border of the heart:
   - The upper border is a straight line between points (1), (2).
   - The lower border is a nearly horizontal line between points (3) and (4).
   - The right border is a slightly curved line between points (2) and (3).
   - The left border is a slightly curved line between points (1) and (4).
   - The atrio-ventricular sulcus:
     The surface anatomy of the atrio-ventricular sulcus is represented by a line (slightly convex upwards and to the right from the lower border of the 3rd left costal cartilage to the 6th right sterno-costal junction).

3. Surface anatomy of the cardiac valves:
   - P (pulmonary): Deep to the left 3rd sterno-costal junction.
   - A (aortic): Opposite the left 3rd intercostal space.
   - M (mitral): Deep to the left 4th sterno-costal junction. So the above 3 valves present behind the left border of the sternum.
   - T (tricuspid): Behind the center of the sternum opposite the Lt. 4th intercostal space.
CARDIAC CASE

A. Personal history: as before.

B. C/O: as before.

C. Present history:
   Ask about the following symptoms in a chronologically arranged manner and mention them whether positive or negative:
   1. Dyspnea, Orthopnoea and pasoxysmal nocturnal dyspnea (PND).
   2. Cough, expectoration and haemoptysis.
   4. Palpitation.
   5. Oedema of lower limb, ascites or pain in the right hypochondrium.
   6. Low cardiac output symptoms.
   7. Symptoms suggestive of hypertension.
   8. Embolic symptoms.
   10. Fever, jaundice.
   11. Any other symptoms suggestive of other system affection, e.g.: chest symptoms, neurological symptoms or symptoms of gastrointestinal tract.

Another Classification of Cardiac Symptoms:
1. Symptoms of pulmonary venous congestion.
2. Symptoms of systemic venous congestion.
3. Symptoms of low cardiac output.
5. Palpitation.
6. Cyanosis.
8. Embolism.
10. Other symptoms.

How can we ask about each symptom?

1. Symptoms of pulmonary venous Congestion:
   * It is due to stagnation of blood in the pulmonary veins of the lung due to failure of the left ventricle or mitral stenosis.
   * Lung congestion can manifest itself as:
     * Dyspnea on exertion (ask about its grades):
     * Orthopnoea (The patient trying to lie propped up e.g. using extrapillows).
     * P.N.D.
     * Dyspnea at rest (severe cases).
     * Cough and expectoration - Haemoptysis.
     * Acute pulmonary oedema.
2. Symptoms of Systemic Congestion:
   It is due to stagnation of blood behind a failed right ventricle or due to T.S. or T.I. or due to obstruction of the venous return to the heart e.g. Constrictive Pericarditis and Pericardial effusion.

   Systemic congestion can manifest itself as:
   a. Pain in the right hypochondrium.
   b. Swelling of lower limbs and abdominal distention due to oedema and ascites.
   c. Dyspepsia and vomiting due to congestion of the gastrointestinal tract.

3. Symptoms of low cardiac output: (The symptoms are usually exertional)
   Easy fatigue, coldness, dizziness, lack of concentration, sweating, syncopal attacks, oliguria, anginal pain and claudication pain.

4. Palpitation:
   • Ask about:
     a. Relation to exertion.
     b. Onset, duration and offset.
     c. Regularity.

5. Chest pain:
   • Ask about:
     (The following questions are asked for any pain at any site)
     a. Site and radiation.
     b. Type (character) and severity.
     c. Precipitating and relieving factors.
     d. Frequency & periodicity.
     e. Associated symptoms.

6. Cyanosis:
   • Ask about:
     a. Age of onset:
        ⊙ Since birth → Fallot's tetralogy.
        ⊙ Few years after birth → Fallot's triology.
        ⊙ In teenager → Eisenmenger's syndrome (reversed shunt).
        ⊙ Above age of 40 years → COPD with or without Cor-Pulmonale.
     b. Cyanotic spells and squatting → Fallot's tetralogy.
     c. Differential cyanosis → P.D.A with reversed shunt.
     d. Exertional cyanosis in cases of cardiac shunts or in cases of a cyanotic Fallot's and interstitial pulmonary fibrosis.

7. Symptoms of hypertension:
   ⊙ (Non specific): Headache (occipital), blurring of vision & epistaxis.
8. Symptoms of embolization:
   Hemiplegia, sudden blindness, painless haematuria, acute abdomen (mesenteric occlusion), or sudden onset of coldness of one limb (acute ischaemia)

9. Fever:
   ➔ Rheumatic fever or rheumatic activity, endocarditis, chest infection or D.V.T.

10. Ask about symptoms of other systems:
    ➔ As chest, G.I.T. or neurological symptoms.

D. Past history:
   Rh fever, Hypertension, DM, TB, Bilharziasis (Bilharzial cor pulmonale)

E. Family history:
   Hypertension, DM, Coronary heart disease. Rheumatic fever (i.e. the same environmental factors)

| Inherited disease with cardiac component: |
| ➔ Familial hypercholesterolemia. |
| ➔ Down’s syndrome → VSD. |
| ➔ Turner’s syndrome → coarctation of aorta. |
| ➔ Marfan’s $ → A.I or Mitral valve prolapse. |
| ➔ Myotonia → Cardiomyopathy. |
| ➔ Neurofibromatosis → Cardiomyopathy. |
| ➔ Friedreich’s Ataxia → Cardiomyopathy. |
| ➔ Noonan $ → Supravalvular aortic stenosis. |

CARDIAC SYMPTOMS

I. Dyspnea

Dyspnea is abnormally uncomfortable awareness of the act of breathing.

Pathogenesis of cardiac dyspnea:

A. Mechanical factors:

   1. Pulmonary congestion: (leading to ↓ lung complianc)

      Occurs in left sided heart failure or lesion leading to:

      1. Decreased lung compliance due to interstitial oedema (the most important factor).
      2. Diminished alveolar capacity with transudation in some alveoli.
      3. Congestion of bronchial mucosa with or without bronchospasm.
2. Infra-diaphragmatic causes:
   Pericardial effusion, constrictive pericarditis and right sided failure with SVC lead to enlarged liver and ascites which decrease diaphragmatic mobility.

3. Fatigue of respiratory muscles:
   Decreased respiratory muscles perfusion due to low cardiac output.

4. Pericardial effusion and pleural effusion (with heart failure):
   Leading to mechanical compression of the lungs.

B. Nervous factors:

1. Activation of Hering - Breuer reflex:
   This is a normally present reflex in which impulses arise from stretch of receptors present in the terminal air passages at the end of inspiration, this leads to reflex inhibition of inspiratory center and passive relaxation of the chest → expiration. In left sided failure, interstitial oedema activates this reflex, causing shallow rapid breathing.

2. Churchill - cope reflex:
   It occurs in pulmonary venous congestion which leads to reflex stimulation of respiratory center, through the juxtacapillary receptors of the lung which are stimulated due to pulmonary venous congestion (high pulmonary capillary pressure).

C. Chemical factors:

Pulmonary venous congestion and diminished tissue perfusion (↓ COP) lead to hypoxia, which stimulates respiration.

Grades of dyspnea:

<table>
<thead>
<tr>
<th>Grade I:</th>
<th>Dyspnea on severe exertion.</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Or: Dyspnea on more than ordinary activity.</td>
</tr>
<tr>
<td>Grade II:</td>
<td>Dyspnea on moderate exertion.</td>
</tr>
<tr>
<td></td>
<td>Or: Dyspnea on ordinary activity.</td>
</tr>
<tr>
<td>Grade III:</td>
<td>Dyspnea on mild exertion.</td>
</tr>
<tr>
<td></td>
<td>Or: Dyspnea on less than ordinary activity.</td>
</tr>
<tr>
<td>Grade IV:</td>
<td>Dyspnea even at rest.</td>
</tr>
</tbody>
</table>
Orthopnea:

Dyspnea on lying flat which is partially relieved by sitting, this occurs due to:
1. Increased venous return, which increases pulmonary venous congestion.
2. Elevation of the diaphragm by viscera.
3. Interference with mobility of the respiratory muscles.
So in laying flat the pulmonary venous congestion is increased → activation of Hering - Breuer reflex.

acja The most important 2 causes of orthopnea in a cardiac case are:
1. M.S.
2. Left ventricular failure.

N.B.: Orthopnea may occur due to a chest disease e.g.: severe asthmatic attack or increased intra-abdominal pressure e.g. tense ascites.

Paroxysmal Nocturnal Dyspnea (P.N.D)

☆ Definition: It is a Paroxysmal attacks of dyspnea that wakes the patient from sleep.
The patient wakes up 1-2 hours after sleep with marked dyspnea, cough with frothy expectoration. The patient usually sits upright, put his legs down and after a few minutes feels better and goes back to sleep.

The chest may be wheezy due to bronchial oedema, and even bronchospasm, giving a picture simulating bronchial asthma, this is called "cardiac asthma"(severe form of P.N.D with bronchospasm). Cardiac asthma is a state of impending pulmonary oedema.
The patient may pass into acute pulmonary oedema with excessive, frothy and blood tinged expectoration, cyanosis may occur with bubbling crepitations.

- P.N.D.: It is a characteristic symptom of left heart failure or lesion, and must be differentiated from nocturnal asthma.
- Orthopnea: It is breathlessness demanding the up right position. It is a symptom of persistent P.V.C. and indicates advanced left sided heart lesion.
- Breathlessness is not a common feature of right heart failure; however pulmonary hypertension may lead to dyspnea.

Pathogenesis of PND:
PND denotes transudation of fluid outside the pulmonary capillaries due to marked pulmonary venous congestion.
There are some explanations for being nocturnal:
1. Hypervolaemia theory (redistribution of blood volume):

Sleep decreases the venous pressure of the lower limbs leading to absorption of oedema fluid into the circulation. This will cause excessive increase in venous return leading to increase of pulmonary venous congestion leading to dyspnea.
2. Withdrawal of adrenergic help: (Sympathetic drive)

This occurs during sleep due to vagal predominance, and causes further weakness of the force of left ventricular contraction.

3. Bad dreams:

Will lead to tachycardia and increase of blood pressure.

4. Slipping of the head from over the pillow !?

Causes of PND, cardiac asthma and pulmonary edema.

1. L.V.F.: L.V.F is an aggressive process, so PND, cardiac asthma and pulmonary edema are common events.

2. M.S.: The above events occur only in some of cases (see below) because M.S. is a gradual process, so protective mechanisms will occur as follows:

   a. Opening of the anastomosis between pulmonary and bronchial veins, with shunting of the blood from the pulmonary veins to the bronchial veins with formation of bronchial varices which may rupture on coughing or straining → haemoptysis. (Pulmonary or bronchial apoplexy)

   b. An interstitial pulmonary barrier will be developed as chronic pulmonary venous congestion will lead to rupture of some small capillaries; this will lead to presence of RBCs in the interstitial tissue with deposition of iron causing haemosiderosis, which will prevent transudation into the alveoli.

   c. Vasoconstriction of the pulmonary arterioles will decrease pulmonary congestion but leading to pulmonary hypertension (PVC → reflex V.C of pulmonary arterioles).

   d. Increased thickness of the walls of pulmonary vessels decreasing the process of transudation.

* The previous (protective) mechanisms can explain why PND, cardiac asthma and pulmonary oedema are common in left ventricular failure than mitral stenosis.

* Pulmonary oedema can occur in mitral stenosis with a precipitating factor on top e.g.: Atrial fibrillation.
# DIFFERENTIATION BETWEEN CARDIAC AND BRONCHIAL ASTHMA

<table>
<thead>
<tr>
<th></th>
<th>CARDIAC</th>
<th>BRONCHIAL</th>
</tr>
</thead>
<tbody>
<tr>
<td>1. History:</td>
<td>Cardiac trouble</td>
<td>Chest trouble</td>
</tr>
<tr>
<td>2. Duration of attack:</td>
<td>Short</td>
<td>Long</td>
</tr>
<tr>
<td>3. Time of attack:</td>
<td>About 2 hours after sleep</td>
<td>Early in the morning</td>
</tr>
<tr>
<td>4. Dyspnea:</td>
<td>Inspiratory</td>
<td>Expiratory</td>
</tr>
<tr>
<td>5. Character of expectoration:</td>
<td>Frothy, blood tinged &amp; excessive</td>
<td>Thick pellets</td>
</tr>
<tr>
<td>6. Chest ex.:</td>
<td>Basal crepitations.</td>
<td>Diffuse rhonchi</td>
</tr>
<tr>
<td>7. Heart ex.:</td>
<td>Murmurs and gallop.</td>
<td>Normal</td>
</tr>
<tr>
<td>8. E.C.G.:</td>
<td>May be abnormal</td>
<td>Normal</td>
</tr>
<tr>
<td>9. Circulation time:</td>
<td></td>
<td></td>
</tr>
<tr>
<td>*Arm to tongue: (10 - 20 sec.)</td>
<td>++</td>
<td>Normal</td>
</tr>
<tr>
<td>*Arm to lung: (5 - 10 sec.)</td>
<td>Normal</td>
<td>++</td>
</tr>
<tr>
<td>10. Drugs:</td>
<td></td>
<td></td>
</tr>
<tr>
<td>*Morphine:</td>
<td>Improvement</td>
<td>Contraindicated</td>
</tr>
<tr>
<td>*Aminophylline:</td>
<td>Improvement</td>
<td>Improvement</td>
</tr>
<tr>
<td>11. Frequency:</td>
<td>Very low frequency</td>
<td>More frequent</td>
</tr>
</tbody>
</table>

Arm to lung circulation time = time between injection of ether in the cubital vein and smelling its odour. Arm to tongue circulation time = time between injection of a bile salt in the cubital vein and tasting bitterness. The circulation time can also be determined by isotopes.

Cardiac asthma is a severe form of P.N.D. with super imposed bronchospasm, it is considered as a case of impending pulmonary oedema. The bronchospasm is due to reactive bronchial tree or actually due to associated obstructive airway disease?

## Acute cardiogenic pulmonary oedema

**Definition:** Sudden transudation of fluid into the alveoli.

**Clinical picture:**
- Severe dyspnea, cough with excessive frothy blood-tinged expectoration, cyanosis, bubbling crepitations allover the chest.

**Cause:**
- Sudden increase in pulmonary venous pressure as in:
  1. Acute L.V.F.
  2. M S. with ppt factor as A.F.
American functional classification of heart diseases:

<table>
<thead>
<tr>
<th>Grade</th>
<th>Description</th>
</tr>
</thead>
<tbody>
<tr>
<td>I</td>
<td>No symptoms with ordinary exertion (no limitation).</td>
</tr>
<tr>
<td>II</td>
<td>Symptoms with ordinary exertion (slight limitation).</td>
</tr>
<tr>
<td>III</td>
<td>Symptoms with less than ordinary exertion (marked limitation).</td>
</tr>
<tr>
<td>IV</td>
<td>Symptoms at rest (inability to carry on any physical activity).</td>
</tr>
</tbody>
</table>

- This classification is a very easy method to assess and follows the cardiac cases.
- The symptoms in this classification could be dyspnea or chest pain.
- This classification is functional regardless the pathology of heart disease.

II. Haemoptysis

It is defined as coughing blood, blood tinged or streaked sputum.

☆ Haemoptysis in a cardiac patient may be due to:

1. Bronchial or Pulmonary apoplexy:
   - This is a profuse frank haemoptysis occurring mainly in M.S. due to rupture of bronchial varices with coughing or straining.

2. Chest infection:
   - This occurs more commonly with M.S. than with L.V.F as there is chronic congestion of the lung leading to decrease the lung defense, irritation and devitalization of bronchial mucosa occurs with super imposed infection with production of mucopurulent sputum streaked or tinged with blood due to rupture of bronchial capillaries.

3. Acute pulmonary oedema:
   - Acute left sided heart failure or M.S with aggravating factor causes transudation of fluid containing RBCs into the alveoli. This will cause severe dyspnea with expectoration of big amount of frothy, blood-tinged sputum.

4. Pulmonary infarction:
   - This occurs due to medium sized embolus in compromised lung. There will be sudden stitching pain in the chest increases by inspiration and associated with pleural rub.
   - There will be dyspnea, haemoptysis, fever, jaundice and haemorrhagic pleural effusion may also occur.

5. Associated chest conditions.
III. Cardiac Oedema

Oedema means swelling of the tissues due to an increase in the interstitial fluid.

Causes of cardiac oedema:
1. Right sided heart failure $\rightarrow$ S.V.C $\rightarrow$ transudation from capillaries.
2. Pericardial effusions and constrictive pericarditis $\rightarrow$ S.V.C

Characteristics of Cardiac Oedema:
1. Occurs in the dependent parts of the body:
   - Ankle oedema: in ambulant patients.
   - Sacral oedema: in bedridden patients.
2. Bilateral, but one side may be affected more due to:
   - Deep venous thrombosis.
   - Postural, in patients sleeping on one side.
3. It is always pitting oedema.
4. Oedema of lower limbs always precedes appearance of ascites, except cases of
   "Ascites praecox" where ascites occurs first, causes:
   (A) Constrictive pericarditis and pericardial effusion due to:
      - kinking of hepatic veins causes early liver congestion and ascites?
      - Obstruction of lymphatics passing through the diaphragm causes accumulation
        of lymph in the peritoneum?
   (B) Tricuspid incompetence (T.I):
      - Where regurgitation of blood causes early liver congestion and ascites?

5. Other features of cardiac disease.

Differential Diagnosis of Cardiac Oedema:
(1) From other causes of generalized oedema:

a. Renal Oedema:
   This occurs in nephritic or nephrotic syndromes. Oedema occurs first in the eye lids
   and is associated with features of renal disease.

b. Hepatic oedema:
   Oedema of lower limbs and ascites it is also associated with features of liver
   disease e.g.: cirrhosis, splenomegaly, jaundice.

c. Nutritional oedema:
   This occurs with severe nutritional deficiency or mal-absorption syndrome.
   There is long history of inadequate diet or diarrhea.
   The oedema occurs first in the lower limbs and is associated with features of
   nutritional deficiencies.
2. Local causes of oedema:

a. Angioneurotic oedema (non pitting oedema):
   This allergic oedema occurs with relation to certain factors, as eating certain foods or taking certain drugs, oedema occurs suddenly especially in the lips, eye lids. It is usually asymmetrical.
   If the oedema involving the tongue and larynx, it leads to life threatening respiratory obstruction.
   There is usually positive family history of oedema or other allergies, and the patient himself may have other forms of allergy. Rapid response of oedema to anti-allergic measures is characteristic.

b. Inflammatory:
   There are signs of inflammation as redness, hotness and tenderness e.g.: Cellulitis of the lower limb.

c. Lymphatic obstruction:
   Leading to non pitting oedema.

d. Venous obstruction:
   Dilated veins will be present over the limb. e.g.: in DVT of lower limb.

e. Orthostatic oedema:
   Due to prolonged sitting, or standing up (this may occur in normal persons.)

Pathogenesis of cardiac oedema:

1. Increased venous pressure: (S.V.C.)
   Due to accumulation of blood in systemic circulation in the cases of right sided heart failure or pericardial disease causing S.V.C.

2. Salt and water retention:
   Due to:
   (A) Diminished cardiac output causes diminished renal blood flow with diminished glomerular filtration. This will lead to increase obligatory water absorption by P.C.T.
   (B) Secondary hyper-aldosteronism:
   As diminished renal blood flow causes secretion of renin-angiotensinogen which stimulates aldosterone secretion.

3. Hypoalbuminaemia may occur as a result of long standing GIT congestion which leads to decreased intake and absorption, also leading to protein losing enteropathy.

4. Capillary permeability is increased due to tissue hypoxia.

5. Resistance to the action of the atrial natriuretic peptide (ANP)?
Causes of Unilateral Oedema in Cardiac Patient:
1. Positional (patient usually laying on one side).

IV- Low C.O.P. Symptoms

Easy fatigue - coldness - dizziness - oliguria - sweating - syncopal attacks, anginal pain and claudication pain. The symptoms are usually exertional.

Causes:
- Stenotic lesion of the heart e.g.: A.S, M.S.
- Heart failure.
- Pericardial effusion or constrictive pericarditis.

(Exertional syncope may be suggestive of stenotic heart lesion especially A.S.)

V. Symptoms of Hypertension

Usually it is asymptomatic so it is called the silent killer.
1. Headache usually occipital especially in the morning.
2. Blurring of vision:
   - These (1,2) symptoms are only suggestive and can occur in low C.O.P. states.
3. Manifestations of the cause or complications of hypertension.

The eye may be affected in various forms of C.V.S. diseases:
1. Sudden unilateral impairment of vision due to retinal haemorrhage in severe hypertension or from embolic manifestations.
2. Disturbance of vision in both eyes may be due to cerebral embolism.
VI. Embolic manifestations

1. Hemiplegia or hemiparesis.
2. Painless Haematuria.
3. Acute abdomen (mesenteric occlusion).
4. Sudden onset of coldness of a limb (acute ischaemia).

Causes: (The heart as a source of embolization)

- M.S. with A.F.
- Infective endocarditis.
- Mural thrombosis on top of myocardial infarction.
- Artificial valves.
- Dilated cardiomyopathy.

VII. Syncope

Definition: Sudden transient complete loss of consciousness due to reduced cerebral blood flow. If the ischaemia prolongs convulsions may occur. It is associated with postural collapse with spontaneous recovery.

N.B.: pre-syncope refers to lack of strength with sensation of impending loss of consciousness (faintness).

Causes:

A. Cardiac syncope:

1. Resistance to the blood flow e.g.: left ventricular outflow tract obstruction. e.g.: A.S. or HOCM. (Exertional syncope)
2. Ball and valve embolus and left atrial myxoma. (Positional syncope).
3. Adams-stocks, severe bradycardia or tachycardia. (Syncope at rest)
4. Acute diminution of venous return, as in hemorrhage, extensive burns or excessive peripheral vasodilatation.

B. Vasomotor syncope:

1. Vasovagal attack: (Simple faints)
   Exposure to fear, emotions or bad sights may cause excessive vagal stimulation leading to marked bradycardia and syncope. The patient will be pale with sweating, marked bradycardia and low BP.
2. Carotid sinus syndrome:
   Stimulation of hypersensitive carotid sinus e.g. during shaving or carotid massage, causes vagal stimulation and syncope.
C. Orthostatic syncope:

Normally, standing up causes, reflex vasoconstriction of the blood vessels of the lower limbs to prevent pooling of blood in lower limbs. This is mediated through sympathetic fibers. In orthostatic syncope, there is a disturbance in this mechanism.

Causes:
1. Autonomic neuropathy e.g. diabetic or uremic neuropathy.
2. Huge varicose veins.
3. Hypovolaemia. e.g.: Haemorrhage or dehydration.
4. Weakness of the muscles of the lower limbs (muscle pump).

D. Cerebral syncope:
1. Cerebral embolism and hypertensive encephalopathy.
2. Hyperventilation will wash CO₂ → Hypocapnia, leading to cerebral vasoconstriction and Syncope.
3. Transient ischaemic attacks.

E. Hypoxic syncope:

Due to marked hypoxia, e.g. in Fallot's tetralogy (cyanotic spells).

F. Cough and micturation syncope.
1. Cough syncope follows a prolonged paroxysm of coughing → increase the intrathoracic pressure → ↓ V.R.
2. Micturation syncope occurs in patients with senile prostate as the sudden relief of bladder wall pressure during micturation in upright position causes reflex peripheral vasodilation.

Blackouts: Some lapse of consciousness.

Causes:
1. Decreased blood flow to brain → syncope and its types.
2. Epileptic attacks.
3. Narcolepsy.
4. Psychiatric diseases e.g. fugue states.

VIII. Palpitation

Awareness of heart beats, terms such as thumbing, pounding, fluttering, jumping, racing and bumping are often used by the patient

Causes:
1. Rapid heart rate. e.g.: Sinus or paroxysmal tachycardia.
2. Forcible heart contraction (volume overload). e.g.: A.I or M.I
3. Irregular heart. e.g.: extrasystole or A.F.
4. Cardiac neurosis.

* Arrhythmia means a change of heart rate or rhythm.
* Arrhythmia may be manifested by palpitation, syncope or may be asymptomatic.
* Marked tachycardia or marked bradycardia usually leading to low C.O.P.
IX. Chest Pain
(Cardiovascular causes)

1. Coronary heart disease:
   a. Angina pectoris.
   b. Myocardial infarction.

2. Pericardial disease:
   a. Acute pericarditis.
   b. Pericardial effusion.

3. Pulmonary embolism:
   a. Massive pulmonary embolism.
   b. Pulmonary infarction.

4. Aortic aneurysm:
   a. Aortalgia: Retrosternal sawing pain due to stretch of sympathetic fibers around aorta.
   b. Neuralgia: Brachial or intercostal pain, due to compression of nerve roots.

5. Dissecting aneurysm of the aorta:
   a. Dissection of ascending aorta: Sudden severe retrosternal pain similar to myocardial infarction.
   b. Dissection of the arch: Sudden severe retrosternal Pain with radiation to the neck and UL.
   c. Intercostal arteries: Intercostal neuralgia.

The above 1, 3, 4, 5 are considered as vascular causes of chest pain.

6. Mitral valve prolapse (young female)

7. Pain of cardiac neurosis:
   * It occurs in neurotic patients usually with sighting respiration.
   * Left inframammary.
   * Stitching in character.
   * Not related to exertion.
   * Sometimes associated with local tenderness.
   * Cardiac examination is normal.

* Pain is defined as an unpleasant sensation localized to a part of the body accompanied by anxiety and the urge to escape or terminate the feeling.
* Pericordial pain can be caused by the above diseases and also by diseases causing retrosternal chest pain (see later).
**Anginal pain:**

It is usually in the form of heaviness, crushing or gripping, the patient may indicate the type of pain by clenching his fist or gripping his hands together. It is retrosternal, radiated to left shoulder, arm, jaw or epigastrium. It is usually precipitated by exertion and relieved by rest or nitrates. It is usually short lived for few minutes, but it may be prolonged in cases of unstable angina or myocardial infarction.

**The anginal pain:**
- Never to be localized.
- Never to be stitching or throbbing.
- Never to be < 30 seconds.

**Causes of anginal pain in young person:**
1. Aortic valve disease.
2. Prinzmetal angina.
3. Mitral Valve prolapse.
4. Vasculitis (males → PAN, females → SLE).
5. Pulmonary hypertension.
6. Familial hypercholesterolemia.

The typical anginal pain is usually exertional, retrosternal, relieved by nitrates. If one of the previous criteria is absent, it is considered as atypical anginal pain.

### Angina versus myocardial infarction

<table>
<thead>
<tr>
<th>(Angina)</th>
<th>(Myocardial infarction)</th>
</tr>
</thead>
<tbody>
<tr>
<td>(1) Site as before.</td>
<td>(1) As for angina.</td>
</tr>
<tr>
<td>(2) Precipitated by exercise or emotion.</td>
<td>(2) Often no obvious precipitant.</td>
</tr>
<tr>
<td>(3) Relieved by rest, nitrates.</td>
<td>(3) Not relieved by rest, nitrates.</td>
</tr>
<tr>
<td>(4) Mild/moderate severity.</td>
<td>(4) Usually severe (but may be silent).</td>
</tr>
<tr>
<td>(5) Anxiety mild or absent.</td>
<td>(5) Variable anxiety or angor animi.</td>
</tr>
<tr>
<td>(6) No increased sympathetic activation.</td>
<td>(6) Increased sympathetic activity.</td>
</tr>
<tr>
<td>(7) No nausea or vomiting.</td>
<td>(7) Nausea and vomiting are common.</td>
</tr>
</tbody>
</table>

### X. Fever in a Cardiac Case

1. Rh fever or Rh activity.
2. Infective endocarditis.
3. Chest infection.
4. Pulmonary infarction.
5. Myocardial infarction.
6. Acute pericarditis.
8. Associated conditions.

**Retrosternal chest pain:**
XI. Jaundice in a Cardiac Case

1. Hemolytic:
   In case of pulmonary infarction or due to mechanical haemolysis of RBCs on artificial valves.

2. Hepatocellular:
   Due to marked congestion of the liver, also late with cardiac cirrhosis.

3. Obstructive:
   Compression of bile canaliculi by the congested liver leading to cholestasis.

4. Associated:
   The commonest (e.g. viral hepatitis).

   Important symptoms of other systems:

   ▶ G.I.T:
     ✦ Nausea and vomiting
     ✦ Myocardial infarction.
     ✦ Digitalis toxicity.
     ✦ Gastrointestinal congestion with right sided failure.

     ✦ Diarrhea due to Digitalis toxicity
     ✦ Constipation due to Dehydration due to diuretics.

   ▶ Renal:
     ✦ Nocturia With heart failure.
     ✦ Acute polyurea may follow paroxysmal atrial tachycardia due to increase production of A.N.P.!

   ▶ Neurological:
     ✦ Embolic manifestations → hemiplegia.

GENERAL EXAMINATION

As before, see the general sheet.

1. General Condition.
2. Built.
3. Decubitus. e.g.: Orthopnea.
4. Face:
   ✎ Malar flush in M.S.
   ✎ Elfin facies in supravalvar A.S.
5. Complexion:
   1. Pallor (↓ COP - Anemia - Toxemia)
   2. Jaundice (as before)
   3. Cyanosis (as before).

- Pallor with jaundice e.g.: mechanical haemolysis of RBCs on artificial valves.
- Cyanoicterus (cyanosis and jaundice) occurs in cases of T.I.
6. Vital signs:
   1. Pulse.
   2. Blood pressure.
   3. Temp.

7. Neck:
   1. Arterial pulsation.
   2. Venous pulsation.
   3. Thrill (systolic).
   4. Thyroid swelling (hyperthyroidism → ↑ HR, hypothyroidism → ↓ HR).
   5. Lymph nodes with mediastinal $ causing obstruction of S.V.C.

8. Hands:
   - Temperature.
   - Tremors (due to anxiety or hyperthyroidism).
   - Capillary pulsation.
   - Splinter hemorrhage.
   - Osler nodules.
   - Jane way spot.
   - Cyanosis.
   - Clubbing.
   - Spooning or Koilonychia.

9. Lower limbs:
   - Oedema.
   - Cyanosis.
   - Clubbing.
   - Pulsations.

10. Any other + ve data.

11. Other systems:
   - Chest examination for bilateral fine basal crepitations e.g.: PVC.
   - Abdominal examination for liver enlargement in right sided failure, splenomegally in cases of infective endocarditis.
   - Neurological examination for lateralizing signs.

Mention any positive data in history and examination. Also mention the negative relevant data only.
LOCAL EXAMINATION OF THE HEART

Local Examination:

1. Inspection.
2. Palpation.
3. Percussion.
4. Auscultation.

we usually inspect and palpate at the same time to determine chamber enlargement

1. Inspection:

(A) Pericordial bulge:
Denote cardiac enlargement in early childhood due to either congenital or rheumatic heart disease. Pericordial bulge may also be due to pericardial effusion.

(B) Apex:

1. Site.
2. Extent

1. Site:
It is the lower most outer most visible and Palpable pulsation.
Normally it is in the Lt. 5th intercostal space just inside the mid clavicular line (M.C.L.). Its area is about one inch in diameter, 3.5 inch from the midline.

Abnormalities in site:

1) Shifted outwards:
   a- Right ventricular enlargement.
   b- Right Pleural effusion or Pneumothorax.
   c- Left sided lung fibrosis.

2) Shifted down and out in Left ventricular enlargement

3) Shifted to the right:
   a- Left pleural effusion or pneumothorax.
   b- Right sided lung fibrosis.
   c- Dextrocardia.

4) Shifted upward:
   a- Upper lobe fibrosis.
   b- Infra diaphragmatic causes as ascites, pregnancy.

5) Absent apex: (not visible or palpable)
   a- Thick chest wall.
   b- Apex under a rib.
   c- Emphysema.
   d- Pericardial effusion.
   e- Obese persons.
   F- Dextrocardia !?

2. Rocking movements: Apical bulge + left parasternal retraction at the same time is due to left ventricle ++ due to anti-clockwise rotation of the heart. In right ventricle ++ the reverse occurs due to clockwise rotation.

3. Double apex: It is a bulging around the apex due to ventricular aneurysm.

4. Double apical impulse: It is due to hypertrophic obstructive cardiomyopathy.
2. Extent of the apex:
Normally it is localized i.e. the apex diameter is less than one inch or presents in one space or its medial border is well defined.

a- Diffuse apex (Right ventricular pulsation):
The apex diameter is more than one inch or presents in more than one space or the apex medial border is ill defined, this indicates right ventricular enlargement, (not all cases of right ventricular enlargement causes diffuse apex).

b- Localized apex (Left ventricular pulsation):
The apex diameter is less than one inch or presents in one space or the apex medial border is well defined, this is present in normal persons and in cases of left ventricular hypertrophy.

The medial border of the apex is the most important to determine the extent of the apex (localized or diffuse apex).

C. Other pulsations:
I- Left 3rd and 4th parasternal space pulsation = right ventricular enlargement or dilated left atrium pushing the right ventricle forward?
II- Left 2nd space pulsation = pulmonary artery dilatation e.g. p ++
III- Right 2nd space pulsation = Ascending aorta dilatation e.g. systemic hypertension - aortic aneurysm - post stenotic dilatation.
IV- Epigastric pulsation = right ventricular enlargement - Aortic pulsation or liver pulsation.
V- Pulsation in the suprasternal notch = Hyperdynamic circulation, aneurysm of the arch of aorta or due to the kinking of carotid arteries in old age due to atherosclerosis.

D. Scars of previous operation:
➢ Midline sternotomy (open heart surgery) e.g.: valve replacement or coronary bypass.
➢ Infra mammary transverse scar (closed heart surgery) e.g.: mitral valvotomy.

II. Palpation:
A) Apex:
1- Confirm the site of the apex.
2- Character of the apex: (comment on amplitude and duration)
   a- Normally: the apex gives gentle tap i.e.: apex of no special character.
   b- Hyperdynamic apex: in (diastolic or volume overload) as M.I., A.I., V.S.D. & PDA.
   c- Heaving sustained: (Systolic or pressure overload) e.g.: A.S. Systemic hypertension.
   d- Slapping Apex: Palpable 1st sound with weak apex in M.S.
3- For Thrill (palpable murmur):
   a- Diastolic thrill over mitral area in M.S.
   b- Systolic thrill over mitral area in M.I.

4- Palpable sounds:
   1st H.S. palpable in M.S.

Many students are puzzled by the fact that the finger is lifted during systole, this is due to the rotatory movement of the heart during contraction, this leads to forward movement of the apex → apex beat.

(A) Use the hand to palpate the cardiac impulse. (B) Localise the apex beat with a finger. (C) If necessary, roll the patient into the left lateral position.

B) Left Parasternal area:
   - Pulsations, due to right ventricular ++ or pushed right ventricle due to dilated left atrium.
   - Left parasternal heave (pressure load on right ventricle) e.g.: P ++ or P.S.
   - Hyperdynamic pulsations. e.g.: T.I.
   - Systolic (Thrill) in V.S.D.

C) Epigastric pulsation (confirm the origin of pulsation):
   - Right ventricle: pulsation to the tip of the fingers coming from above downward, usually it is below left costal margin.
   - Aorta: From the back to the front slightly to the left of the mid line.
   - Liver: Expansile systolic pulsation in T.I. it is below the right costal margin.
The Method to examine for liver pulsation:
- Bimanual.
- Put the right hand away from the epigastrium.
- Ask the patient to stop breathing.

The Aortic pulsation is felt just above or below the umbilicus slightly to the left of the midline.

D) Base:
- Examine aortic and pulmonary area for pulsating pulmonary artery or aorta.
- Palpable 2\textsuperscript{nd} sound over pulmonary area (diastolic shock) in pulmonary hypertension.
- Systolic thrill in organic A.S.

Thrill of aortic valve-disease:
- A.S.: systolic thrill (is felt on the base in cases of organic A.S).
- A.I.: systolic thrill over neck only (carotid shuddering due to vibration of carotid during forceful ventricular systole). Carotid shuddering also can be felt in A.S due to vibration of the blood within carotid arteries.

III. Percussion: (Now it is not reliable)

1. Percussion of the right border of the heart:
   - We start on the right M.C.L. using heavy percussion from above downward. Normally there is dullness in the 4\textsuperscript{th} space (upper border of the liver) or it may be in the 5\textsuperscript{th} space. Precede one space above i.e. 3\textsuperscript{rd} space or 4\textsuperscript{th} and percuss parallel to the right border of the sternum. Normally no dullness to the right of the sternum, even the sternum itself is resonant, any dullness to the right of the sternum means right atrial enlargement, aortic aneurysm or pericardial effusion.

2- Percussion of the upper border (the base):
   - This is done by comparing the aortic with the pulmonary area. Normally both are resonant. Percuss each space from lateral to medial (from MCL to the sternum).
     - Dullness on the left 2\textsuperscript{nd} space = pulmonary artery dilatation.
     - Dullness on the right 2\textsuperscript{nd} space = Ascending Aortic dilatation or aortic aneurysm.

3. Percussion of the bare area of the heart:
   - We percuss with light percussion. The left parasternal line from above downward normally the 4\textsuperscript{th} and 5\textsuperscript{th} space are dull, they are resonant in cases of emphysema or pneumothorax. Also, the bare area becomes wide in pericardial effusion or in lung collapse.

4- Percussion of the left border of the heart:
   - By percussing from the outside of the apex to the apex. Dullness outside the apex = pericardial effusion.
We can percuss and compare right 3rd space with the left 3rd space outside the M.C.L. (for enlargement of left border of the heart) e.g.: left ventricular aneurysm or large left atrium!

IV. Auscultation:

We report on:

A- Heart sounds:
- The 1st and 2nd heart sounds and their intensity.

B- Additional sounds:
- Extra heart sounds (3rd and 4th heart sounds)
  - Opening snap.
  - Ejection systolic clicks.

C- Murmurs.
D- Pericardial rubs.

The bell of the stethoscope is best listening to low pitched sounds e.g.: 3rd and 4th heart sounds and also the murmur of mitral stenosis.

The diaphragm identifies high pitched sounds e.g.: normal heart sounds and the murmur of aortic incompetence.

HEART SOUNDS

1. First heart sound:

- Composed of 2 components:
  a- Valvular component: Closure of the Mitral and Tricuspid valve.
  b- Muscular component: (contraction of the ventricle)
    - Vibration in the chorda tendinae.
    - Vibration of blood during ventricular systole.

A- Causes of accentuated S1:

1- M.S. (decreased filling of left ventricle = closure of the valve from a lower position).
2- Hyperdynamic states.
3- Short P.R. interval. (Decreased Filling of the heart)
4- Tachycardia: due to shortening of the time of diastole = decreased filling of left ventricle.
5- Systemic hypertension with left ventricular hypertrophy (long standing hypertension) → ↑ muscular component of S1.

B- Causes of weak S1:

1- M.I - T.I.
2- Shock - hypotension.
3- Severe myocardial diseases or heart failure.
4- Mechanical factors e.g. thick chest wall, emphysema, pericardial effusion (Distant heart sound)
5- Calcific M.S.
C) Variable intensity of S1 occurs in atrial fibrillation.

| a- | S1 heard on the M & T area. |
| b- | The mitral closure precede the Tricuspid by 0.02 sec. |
| c- | The amplitude of S1 is related to the left ventricular systolic pressure. |
| d- | The mitral component is louder than the tricuspid component. |

II. Second Heart Sound:

Causes:

1. Closure of the semilunar valves. (Pulmonary and aortic valves)
2. Vibration of the blood in the great vessels.

- On the aortic area the aortic component of S2 is only heard so S2 over the aortic area never split.
- Aortic component louder & heard allover the pericordium.
- Pulmonary component heard on pulmonary area only.
- So the pulmonary component and aortic component heard over the pulmonary area, so, physiological splitting can be heard (increases during inspiration and decreases during expiration).

A- Causes of accentuated S2:

Pulmonary component:

1- Pulmonary hypertension.
   - M.S.
   - P.D.A.
   - V.S.D.
   - Cor-Pulmonale.

2- In children with thin chest wall.
3- Hyperdynamic circulation.

Aortic component:

- Systemic hypertension.
- Aneurysm of ascending aorta. (Magnification → ringing S2)
- Hyperdynamic circulation.
- Children with thin chest wall.

B- Causes of weak S2:

Aortic component:

- Aortic valve disease A.S., A.I.
- Hypotension and shock.
- Thick chest ......... etc.

Pulmonary component:

- Severe pulmonary stenosis.
- Thick chest ......... etc.
### C. Causes of splitting of S2 (Pulmonary):

- **Physiological splitting.** (audible inspiratory split)
- **Wide splitting** is due to delay of closure of pulmonary valve and the delay is increased during inspiration due to ↑ V.R. e.g. in VSD, PS, Rt BBB.
- **Wide fixed splitting** as wide splitting but is not affected by inspiration in ASD
- **Reversed splitting** is due to delay of aortic component, so the pulmonary valve closes before the aortic, it disappears during inspiration e.g. in Lf BBB, Tight A.S.
- **Closed splitting** in cases of pulmonary hypertension.

### III. Third heart sound:

It is due to vibration in the ventricle due to gush of blood from atrium into ventricle (during diastole). It is heard due to ↑↑ gush of blood from atrium to ventricle or due to flabby ventricular wall (in heart failure). The third heart sound is usually pathological after the age of 40 years.

**Causes:**

1. It may be present in children and young adults.
2. Pathological S3 is heard on the apex in diastolic overload of left ventricle as in M.I, VSD. Also, it is heard in left sided heart failure.
3. S3 heard on tricuspid area in T.I., A.S.D, and in right sided heart.
4. Third heart sound can also heard on the apex or tricuspid area in cases of hyperdynamic circulation. E.g.: anaemia, beri-beri and thyrotoxicosis.
5. S3 is difficult to be present on the apex in cases of M.S?
IV. Fourth heart sound:

It is due to vigorous atrial contraction due to ↑↑ the end diastolic pressure of the ventricle due to ↓↓ ventricular compliance on top of ventricular hypertrophy or ischemia.

**Causes:**

1- Systemic hypertension, ischemic heart disease & A.S. leading to S4 on the left side (apex).
2- P.S., pulmonary embolism & pulmonary hypertension and ischemic heart disease leading to S4 on the right side (tricuspid).
3- No 4th H.S in cases of atrial fibrillation.

Gallop:

Gallop signifies that 3 heart sounds are heard instead of the usual two plus tachycardia. The term should not be used to include splitting of the second sound, opening snaps or clicks.

Gallop is heard over tricuspid area in right sided heart failure or apex in left sided heart failure.

Gallop is usually heard when the third heart sound is accompanied by tachycardia with the 1st and 2nd heart sounds, the resulting triple rhythm is called gallop as it is resemble the sound of a galloping horse.

**Summation Gallop**

It is the presence of the third & fourth sounds in the presence of tachycardia e.g. in hypertensive heart failure, and ischemic heart disease, as these lesions produce atrial and ventricular gallop.

**Pericardial knock:**

This is a third sound heard in constrictive pericarditis due to the catching effect of the fibrosed pericardium on the relaxing ventricles.

Opening snap:

- It is a snapping sound due to opening of the rigid cusps of the mitral valve.
- It is heard after S2 and separated from it by the isometric relaxation phase.
- It is heard at the apex by the bell.
- It is best heard between the sternum and apex.

**Significance:**

1. It diagnoses M.S.
2. It indicates organic M.S.
3. It indicates mobile mitral valve.
4. It disappears in calcific mitral valve.
5. The early the opening snap to S2 the tight is the stenosis.
**Ejection clicks**

These sounds are heard in early systole just after S1 with opening of diseased semilunar valves.

**Significance:**

1. Aortic ejection click  
   ➢ (Valvular A.S.)  
   ➢ (Systemic hypertension.)

2. Pulmonary ejection click  
   ➢ (Valvular P.S.)  
   ➢ (P++)

**N.B.:**

➢ No ejection systolic click with subvalvular A.S. or P.S. or with calcific A.S.

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**Pericardial rubs**

- A pericardial rub is the characteristic physical sign of acute pericarditis.
- It is best heard at the left of the lower sternum with the patient breathing out using the diaphragm of the stethoscope.
- It is a superficial scratching sound which often has systolic and diastolic components.
- A pericardial rub requires being distinguished from a pleuro-pericardial rub. In both the sounds coincide with the cardiac cycle but the pleuro-pericardial rub is also influenced by respiration and is pleural in origin.

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**MURMURS**

**Pathogenesis of murmurs:**

Normally the blood flow within the circulation is smooth and passes in normal directions, thus normally there is no turbulence. Abnormal movement in velocity or direction (turbulence) will produce murmur.

**Mechanism of Turbulence (murmur):**

1. **Passage of blood through stenotic lesion** e.g. A.S., M.S and P.S

2. **Passage of blood through valvular or intravascular irregularity** e.g. congenital bicuspid aortic valve in the absence of significant stenosis.

3. **Over blood flow through non stenotic valves** → Relative or functional stenosis.  
   * e.g. ASD, blood is shunted to the right Atrium → ↑ Blood flow through the pulmonary valve → Functional P.S.
• Hemic murmur e.g. Anemia or other hyperdynamic states → functional murmur over the base of the heart.
• VSD: Blood from the left ventricle → Right ventricle → lung → left atrium → Functional mitral stenosis.
• Any incompetent valve → functional stenosis over the same valve e.g.: M.I. & A.I (i.e. severe A.I → functional A.S).

(4)Passage of blood into a relatively dilated structure:
• E.g. in P ++ the blood passes from right ventricle to dilated pulmonary artery → ejection systolic murmur (Functional murmur) at the pulmonary valve.
• In systemic hypertension the blood passes from left ventricle to relatively dilated aorta → ejection systolic murmur over aortic area.

(5) Abnormal direction of Blood e.g. M.I. and A.I.

(6) Shunting of blood through septal defect (from high pressure zone to lower pressure zone). e.g. VSD, P.D.A. In A.S.D. the shunting does not produce murmur due to the low pressure gradient between both atria.

Types of murmurs:

I. Organic murmurs:

Murmur due to structural lesion within the heart valves (the valve complex).
i.e. Lesion of cusps, Chorda tendinea or papillary muscles e.g. chronic rheumatic valvulitis or congenital heart disease.

Ch.Ch. of organic murmurs:
• Variable intensity. (Loud murmur indicates organic lesion)
• Thrills may be present. (Presence of thrill signifies organic lesion)
• Most of diastolic murmurs are organic.
• Associated symptoms and signs are + ve. e.g.: chamber enlargement, changes in the systemic blood pressure or associated symptoms related to the valve lesion.

II- Functional murmurs:

Murmur due to functional disturbance within the circulation with or without heart disease, common on the base of the heart due to high Pr. G across semilunar valves.
• Hyperdynamic circulation → ↑ Blood flow → functional stenosis heard on the base. e.g.: systolic murmur on aortic area.
• P++ → ejection systolic murmur over the pulmonary area.
• A.I. can produce functional murmur of A.S.
Ch. Ch. of functional murmurs:
- Usually they are soft, low intensity, not propagated with no thrill.
- Most of them are systolic.
- No chamber enlargement.
- No symptoms related to the auscultatory data.

III- Innocent murmur:
It is a type of murmur which is not associated with any functional or structural lesions (No lesions at all). e.g:
- Ejection systolic murmur: over the pulmonary area due to defect in the proportion between the size of the right ventricle and the pulmonary valve orifice.
- Venous hum: on the root of the neck on the right side in hyperdynamic circulation.
- Mammary soufflé: in pregnant and lactating women.

Areas to be auscultated:
- Mitral area: at the apex.
- Pulmonary area: left 2nd space.
- Aortic area:
  - 1st aortic area → right 2nd space.
  - 2nd aortic area → left 3rd space (Erb’s area)
- Tricuspid area: lower end of the sternum to the left.
- Left 3rd and 4th parasternal spaces.
- Left infra clavicular for PDA.
- Posterior thorax, T2-T6, 2-3 cm to either side of the spine for coarctation of aorta.
- Right to lower sternum for T.I.

We report on:
a) Timing.
b) Site of maximum intensity.
c) Character.
d) Propagation or radiation.
e) Intensity.

a) Timing of murmur:
Systolic:
- Pan Systolic: M.I, T.I & V.S.D.
- Ejection Systolic: A.S. & P.S.

Diastolic:
- Early diastolic: A.I or P.I.
- Mid diastolic: M.S. or T.S.
- Presystolic: M.S.

Continuous (machinery): P.D.A.

The duration of the murmur depends on the pressure gradient across the valve.
The loudness of the murmur is not related to the severity of the lesion.
The pitch of the murmur depends on the pressure gradient, the higher the pitch the greater the pressure gradient.
So the murmur of A.I. is high pitched but the murmur of M.S is low pitched.

b) Site of maximum intensity:
- Apex: M.S. & M.I.
- 2nd right space: A.S. / A.I. (syphilitic)
- 3rd left space: A.I. (Rheumatic).
- Left 3rd and 4th parasternal spaces: V.S.D.
- 2nd left space: P.S.
- 1st left space: P.D.A. (left infra clavicular).
- Tricuspid area: T.I. - T.S.

c) Character and pitch:
- Soft blowing → M.I. & T.I.
- Rumbling (low pitch) → M.S. & T.S.
- Harsh → A.S. & VSD.
- Soft (high pitch) → A.I.

d) Propagation:
- Localized to apex = M.S.
- Propagation to axilla = M.I.
- Propagation to carotid vessels = A.S.
- Propagation all over the pericordium = V.S.D.
- Propagation to the right of the sternum = T.I.

e) Intensity:

Intensity of a murmur is described in grades as follows:

- Grade I: Just audible in a quiet room. (Heard by an expert)
- Grade II: Quiet. (Heard by a non expert)
- Grade III: Loud without thrill. (Easily heard)
- Grade IV: Loud with thrill.
- Grade V: Very loud with thrill. (Heard over wide area)
- Grade VI: Audible without a stethoscope. (Extremely loud)

CLASSIFICATIONS OF MURMURS

A. Systolic murmurs:

1. Ejection systolic (Crescendo - Decrescendo):

Ch.Ch:
- Diamond shaped.
- Begins after short gap from S1.
Ends before second sound.
It increases to a crescendo about the middle of systole then diminished.

**Aortic stenosis (A.S) (Rh. - congenital - Calcific):**
- Harsh & loud (saw like).
- Propagates to the carotid and apex.
- Systolic thrill in aortic area and neck.
- Weak S2.

**Pulmonary stenosis (P.S.) (Mostly congenital - Carcinoid $):**
- Harsh & loud.
- 2nd left space.
- Systolic thrill.

**Atrial septal defect (A.S.D.):**
- Over blood flow across pulmonary valve → functional P.S.

**2. Pan systolic:**

**Ch.Ch:**
- Begins with 1st sound.
- Lasts throughout systole until after aortic sound.

**Mitral Incompetence (M.I):**
- Soft blowing.
- Radiate to the axilla or to the left parasternal area if there is posterior leaflet regurgite.
**Tricuspid Regurget (T.R):**
- Soft blowing.
- Thrill may be present.
- ↑↑ Intensity during inspiration.
- Heard over tricuspid area and right to the sternum.

**Ventricular Septal Defect (V.S.D):**
- Harsh & loud. (Tearing like)
- Over left parasternal area.
- Systolic thrill.
- It may propagate all over the pericardium.

**Pan systolic murmur (MI)**

**B. Diastolic murmurs:**

1) Early diastolic:

**Ch.Ch:**
- Starts immediately after S2.
- Soft.
- High pitched.
- Decrescendo quality.

**Aortic incompetence**
Aortic Incompetence {A.I} (Rh. - $):
- Soft & decrescendo.
- Maximum at A2 in Rh A.I and at A1 in syphilitic A.I.
- Positive peripheral signs in severe cases.
- ↑↑ When the patient leaning forward and holding the breath in expiration.

Pulmonary Incompetence {P.I} (P ++):
- It is usually occurs on top of P ++ (Graham Steell murmur).
- It is soft and heard at pulmonary area.

2. Mid diastolic:
Low pitched & rumbling in ch.ch. due to low velocity of blood flow as there is low gradient between atrial & ventricular pressure during diastole.

Mitral stenosis {M.S.} (Rh.):
- Mid diastolic rumbling.
- Pre-systolic accentuation.
- Low pitched.
- At the apex, not propagating.
- S1 ↑↑.
- It ↑↑ in left lateral position.

Tricuspid stenosis {T.S.} (Carcinoid - Rh.):
- Like M.S. but maximum at the tricuspid area and ↑↑ with inspiration.

3. Pre-systolic rumbling murmur:
- It starts at late diastole.
- Rises to a crescendo just before S1.
- It is due to atrial contraction. So, it disappears with A.F.
- It is caused by M.S. & T.S. (in mild lesions)

C. Continuous murmur:
Causes:

- P.D.A.
- A-V fistula in dialysis patients in the upper limbs.
- A-V shunting in the lung.
- Venous hum in the neck due to blood flow through the jugular veins. It is decreased by pressure with stethoscope.

Musical murmurs:

- Pan systolic murmur due to ruptured papillary muscle.
- Perforation of the cusps as in cases of endocarditis.
- Calcific A.S. like the cry of the sea-gull.
- Rupture chorda tendineae.

EXAMINATION OF OTHER SYSTEMS IN A CARDIAC CASE

1. Chest: For:
   - Bilateral fine basal crepitations in left sided heart failure.
   - Pleural effusion. (Systemic venous Congestion may lead to pleural effusion)
   - Signs of chest infection.
   - Wheezy chest in cardiac asthma.

2. C.N.S.: For: Signs of hemiplegia. (Lateralizing signs)

3. Abdomen: For:
   - Enlarged tender liver & ascites in right sided failure.
   - Spleen enlargement in cases of infective endocarditis.
Diagnosis:

1- Etiological:
   - Rheumatic heart disease (Multi valvular lesion, presence of M.S. or history of rheumatic fever)
   - Congenital heart disease. (Since birth, associated congenital anomaly, or septal defect)
   - Ischaemic or hypertensive heart disease.

2- Anatomical or Structural:
   - Valve lesion e.g.: mitral valve or aortic valve disease.
   - Pericardium e.g.: constrictive pericarditis.
   - Myocardium e.g.: cardiomyopathy.

3- Functional:
   - Compensated (no manifestations of left or right sided heart failure).
   - Non compensated (positive S&S of left or right sided heart failure).

4- Any complications:
   - A.F. or any arrhythmia.
   - Embolic manifestations e.g.: hemiplegia.
   - Infective endocarditis.
   - Chest infection.

Examples:
1- Rh. Heart disease (M.S. - MI), P++ compensated with no complications.
2- Rh. Heart disease (M.I. - A.I.), left sided heart failure complicated with infective endocarditis.
3- Congenital Heart disease (V.S.D.), compensated with no complications.

D.D. OF DULLNESS IN THE LEFT SECOND SPACE

a- cardiac causes:
   1- Pulmonary artery dilatation.
   2- Huge Aortic Aneurysm.
   3- Pericardial effusion (here the dullness change by sitting i.e. + ve shifting dullness).

b- Chest causes:
   Consolidations - fibrosis - Tumor.

c- Abdominal causes:
   Elevated diaphragm by any causes increasing the intra-abdominal pressure.
1) D.D. of systolic murmur over the mitral area:
   1- Mitral incompetence.  
   2- Tricuspid incompetence.  
   3- Aortic stenosis.  
   4- V.S.D.  
   5- P.S.  
   6- Functional murmur.

2) D.D. of diastolic murmur over mitral area
   1- M.S.  
   2- A.I.  
   3- Carey coomb's murmur.  
   4- Austin flint murmur.  

3) D.D. of systolic murmur over the base:
   Aortic area:
   - Organic A.S.  
   - Functional A.S. due to A.I.  
   - Hemic murmur.  
   - Bicuspid aortic valve.  
   - Sclerosis of aortic valve in old age.  
   - Systemic hypertension (long standing cases)

   Pulmonary area:
   - Organic P.S.  
   - ASD. → functional P.S.  
   - Hemic murmur.

Pressures in the normal heart in adults

1. Left sided
   a. Left atrial pressure (normal mean pressure ≤ 12 mmHg).  
   b. Left ventricular pressure
      - Systolic (100 – 150 mmHg)  
      - Diastolic (≤ 12 mmHg)  
   c. Aorta
      - Systolic (100 – 150 mmHg)  
      - Diastolic (60 – 90 mmHg)

2. Right sided
   a. Right atrial pressure (normal mean pressure ≤ 6 mmHg)  
   b. Right ventricular pressure.
      - Systolic (15 – 30 mmHg)  
      - Diastolic (≤ 6 mmHg)  
   c. Pulmonary artery
      - Systolic (15 – 30 mmHg)  
      - Diastolic (4 – 12 mmHg)
Prosthetic valves

* Diseased heart valves can be replaced with mechanical or biological prosthetic valves.

**Mechanical valves:**

1. Ball and cage
2. Tilting single disc and tilting bileaflet valves.

**Biological valves:**

Tissue valves from a big (xenograft) or human (homograft)

- The mechanical valves are durable and require long life anticoagulation because they may develop thrombus around the valve.
- The biological valve are less durable (degenerate within 10 years) but not requiring anticoagulants.
- Prosthetic mechanical valves produce 2 click sounds (opening and closing sounds).
- Disappearance or muffling of these sounds means malfunction of the valve.
- All prosthetic valves used in the aortic position normally produce a systolic flow murmur.

**Complications of prothetic valves:**

1. Infective endocarditis
2. Thrombosis.
3. Mechanical haemolysis
4. Mechanical failure (malfunction)

- Echocardiographic studies can aid in the evaluation of prosthetic valve function. Additional information can be obtained from trans esophageal echo if prosthetic valve endocarditis is suspected.

**Indications of valve replacement**

1. Severe A.S, severe A.I
2. Severe M.I, severe M.S with calcific mitral valve.

**Valvotomy:**

- It is indicated in severe M.S without calcification, it leads to symptomatic relieve for about 5-10 years then restenosis will occur.
- It is not efficient in cases of A.S, but can be done in cases of valvular P.S.
- It can be done surgically or by balloon valvuloplasty.
- It may be complicated by post valvotomy regurge or embolisation.
Surface Anatomy of the Chest

Bony landmarks and their vertebral levels:

(A) sternum:
- Upper border of manubrium → disc between T₂ & T₃.
- Sternal angle → disc between T₄ & T₅.
- Xiphi - sternal junction → T₉.

The sternal angle is the most important bony landmark on the front of the chest.

(B) Scapula:
- Superior angle of scapula → T₂.
- Root of spine of scapula → T₃.
- Inferior angle of scapula → T₇.

(C) Other landmarks:

In front:
- Feel the trachea in the suprasternal notch.
- The first rib and the last one are difficult to be palpated.
- If you want to count the ribs find the second rib and begin counting from it downward, the 2nd rib is the rib which joint the sternal angle.
- Each intercostal space lies below its rib.
- The position of the nipple in females is variable.
- The apex which is the lower most and outermost point at which the heart beats can be palpated, is found in the 5th intercostal space 3.5 inches from the middle line.

Behind:
- The first (upper most) spinous process which can be easily felt is that of C₇.
- Below C₇, you can palpate the spinous processes of all thoracic vertebrae.
Surface Anatomy of the Trachea:
The trachea begins in the neck where the larynx ends at the level of the lower border of the cricoid cartilage, and descends vertically downwards till it ends at the level of the manubrium sternal angle (T₄ & T₅), just to the right of the middle line (by dividing into right and left main bronchi).

Surface Anatomy of the Pleura:
Each lung is covered by a membrane called the pleura. The lung is conical in shape and has an apex, base (diaphragmatic surface) and three borders.

The apex of each lung reaches above the level of the first rib and projects upwards in the root of the neck for about an inch (2.5 cm); the apex lies behind the medial 1/3 of the clavicle. The part of the pleura which covers the apex of the lung is called the cervical pleura. The cervical pleura is defined by a curved line (convex upwards) extending from the junction of the medial and middle thirds of the clavicle to the sterno-clavicular joint.

Surface Anatomy of the Three Borders of the Pleura:
Each lung and each pleura has three borders anterior, inferior & posterior:

(A) The anterior border of the pleura:
- This border passes from behind the sterno clavicular joint downwards and medially from the sternal angle.
- Right pleura descends vertically downwards in the middle line to the back of xiphisternal junction.
- Left pleura descends vertically downwards only to the 4th costal cartilage. Here it curves to the left to reach the left border of the sternum and then passes downwards along the border of the sternum almost to the 6th costal cartilage. This means that a part of the pericardium on the left side is not covered by pleura and is directly related to the anterior wall of the thorax (bare area of the heart).

(B) The lower borders of the pleura:
- The surface anatomy of lower border of right and left sides is the same.
- It is represented by a line which is markedly convex downwards and crosses the following ribs.
• 8th rib → in MCL.
• 10th rib → in mid axillary line.
• The 12th spinous process → opposite the lateral border of the sacrospinalis muscle.

• The MCL → joint a point 1/2 way between the center of the suprasternal notch and the tip of the acromion process of the scapula.
• The mid axillary line is a vertical line which passes through the apex of the axilla.

(C) The post border of the pleura:
• The posterior border of the right and left pleura is the same.
• It extends vertically downwards along the vertebral column from the first to the last 12th thoracic vertebrae.

Surface Anatomy of Lungs:

➢ The surface anatomy of the lungs is less extensive than that of the pleura.
➢ Also the surface markings of the lungs differ greatly during inspiration and expiration, the surface anatomy of the lower borders of the lungs is described as midway between inspiration and expiration. The three borders of the lung are:

(A) The anterior border:
➢ Right lung like right pleura, but the lung descends only to the level of the 6th sternocostal junction.
➢ Left lung like left pleura till the 4th costal cartilage here the anterior border of the lung leaves that of the pleura and curves to the left about 1/2 an inch lateral to the left border of the sternum forming the cardiac notch. Then it curves downwards and medially to the 6th sternocostal junction.

(B) The lower border of the lung:
➢ It is the same on both sides.
➢ It is represented by a curved line which crosses the following structures:
   • 6th rib → in MCL.
   • 8th rib → in mid axillary line.
   • 10th thoracic spinous process where the lower border of the lung ends.
➢ In the extremes of respiration, the lower borders travel a journey of about 2 - 3 inches.

(C) The Posterior borders of the lung:
The posterior border of the two lungs is the same, each one runs downwards along the vertebral column from the apex of the lung to the level of the spinous process of the 10th thoracic vertebra.

Surface Anatomy of the Fissures of the Lungs:

Each lung contains an oblique fissure which is a complete fissure. It divides the lung into upper and lower lobes and horizontal fissure separates the upper from the middle lobe, it is only found in the right lung.
**Oblique fissure:**
Line drawn obliquely downwards and outwards from about 3 cm Lateral to the spinous process of third thoracic vertebra to the 6th costal cartilage about 3 fingers from the median plane.

**Horizontal fissure:**
Line which starts from the anterior border of the lung at the 4th costal cartilage and runs towards the right and slightly upwards till it meets the oblique fissure in the mid axillary line.
Present History:

Ask about the following symptoms in a chronologically arranged manner and mention them whether positive or negative.

1. Cough.
2. Expectoration.
4. Haemoptysis.
5. Dyspnea.
6. Toxic symptoms.
7. Asthmatic attacks.
8. Oedema of lower limb.
10. Cyanosis.
11. Pain in the right hypochondrium.
12. Any other positive symptoms of other systems.

How can we ask about each symptom?

(1 & 2) Cough and Expectoration:

Cough is the most frequent symptom of respiratory disease, ask about:

a- Time of cough:
   - Early morning, e.g.: In chronic bronchitis
   - Night, e.g.: in P.N.D (P.V.C)
   - All over the day, e.g.: in chest infection

b- Frequency, severity, character of cough (better observed by the physician) see later.

c- Dry or productive:
   - If productive ask about:
     - Amount: Large (> teacupful / day), Small (one or two spits /day)
     - Color.
     - Odor.
     - Relation to posture.
     - Consistency of sputum.
     - What increase and decrease it.
     - Associated condition as wheezes.

Cough of suppurative lung diseases or cavitory syndrome:
   - Cough with huge amount of yellow-greenish fetid sputum.
     (> teacupful / day)
   - Cough and expectoration are related to posture.

Cavitary or Suppurative Lung Disease Includes:

1. Bronchiectasis.
2. Lung abscess.
3. Empyema with broncho - pleural fistula.
(3) Chest Pain:

Ask about: Site - character - duration - radiation - what bring and what relieves & associated symptoms. The lung parenchyma is not sensitive to pain, but the parietal pleura and the tracheobronchial mucosa are sensitive to pain.

- The most important causes of chest pain of pulmonary origin:
  1- Non central:
    - Pneumonia
    - Pulmonary infarction.
    - T.B.
    - Malignancy.
    - Rib fracture.
    - Muscle strain.
    - Invasion of chest wall by a tumour.
    - Spinal nerve root involvement by vertebral disease or herpes zoster.
  2- Retrosternal:
    - Tracheitis.
    - Mediastinal emphysema.
    - Acute mediastinitis.
    - Mediastinal tumours.

(4) Haemoptysis:

Means coughing up blood or blood tinged or blood streaked sputum.

Most important causes are:
1- Chest infection
2- M.S.
3- T.B.
4- Pulmonary infarction.
5- Bronchial carcinoma and adenoma.
6- Bronchiectasis.
7- Blood disease as purpura - leukemia.

(5) Dyspnea:

see also C.V.S.

Any chest disease can cause dyspnea whether the disease is unilateral or bilateral.

- Dyspnea with acute onset (acute dyspnea): e.g. pulmonary edema, pneumothorax, foreign body, pneumonia, pulmonary embolism, Guillain-Barre syndrome and pleural effusion (rapidly accumulated).
- Dyspnea with gradual onset (chronic dyspnea): e.g. Emphysema & pulmonary fibrosis.
- Paroxysmal dyspnea: Bronchial asthma, cardiac asthma, allergic alveolitis, recurrent pulmonary embolism, carcinoid syndrome, myasthenia gravis & laryngismus stridulus.

1- Acute and paroxysmal dyspnea usually occurs at rest.
2- Chronic dyspnea usually starts as exertional and then progress to dyspnea at rest.
In cases of dyspnea ask about:-

- Onset, course and relation to exertion.
- Grading.
- Associated symptoms e.g. (chest pain, cough, wheezes or haemoptysis)
- Allergy.
- Heart diseases.
- Smoking.

(6) Toxic Symptoms:

Night fever - Night sweating - Loss of weight - Loss of appetite. These symptoms are suggestive of T.B or any chronic infection e.g. (lung abscess or bronchiectasis)

(7) Asthmatic attacks:

- Age of onset.
- Time of the attack and its frequency.
- Duration of the attack.
- History of allergy and family history.
- Precipitating factors and what relieves the attack.
- Paroxysmal nature i.e.: patient between the attacks is clinically free.

Causes of wheezy chest are bronchial asthma, asthmatic bronchitis, foreign body, cardiac asthma & carcinoid syndrome and certain types of vasculitides.

(8) Oedema of lower limb:

Causes of oedema of LL. in a chest case:

1. Cor-pulmonale with right sided heart failure.
2. Nutritional hypoproteinaemia (due to excessive loss of sputum and decrease intake) e.g. in cases of suppurative lung diseases.
3. Long standing suppurative lung diseases with amyloidosis of the kidney leading to nephrotic syndrome.
4. Repeated tapping of empyema.
5. In COPD due to CO₂ retention → respiratory acidosis → excessive reabsorption of NaHCO₃ from the kidney → sodium & water retention that leads to lower limb edema. Also hypoxia and hypercapnea → capillary permeability → oedema.

(9) Mediastinal compression:

a- Dysphagia:
b- Hoarseness of voice due to recurrent laryngeal nerve affection.

(10) Cyanosis: (See before)

Any advanced chest disease can leads to central cyanosis.
(11) Pain in right hypochondrium:
Due to enlarged liver in Cor-pulmonale.

(12) Any other positive symptom.

Upper respiratory tract symptoms.
1- Nasal obstruction e.g. in allergy.
2- Epistaxis → Unilateral is suggestive to a local disease. Bilateral is suggestive to systemic disease.
3- Hoarseness (dysphonia)
4- Laryngeal stridor which is a high pitched crowing sound occurring during inspiration.
5- Laryngeal pain due to laryngitis or laryngeal carcinoma
N.B.: tracheal stridor is lower in pitch than laryngeal stridor.

Past History:
1 Similar attack.
2 T.B or pneumonia
3 Asthma and recurrent bronchitis in childhood
4 Bilharziasis (Bilharzial cor pulmonale)- diabetes - hypertension.
5 Chest injuries.
6 Recent general anaesthesia, loss of consciousness (aspiration) or surgery (DVT → pulmonary embolism).

Family History:
1 Similar condition in the family.
2 History of chest infection especially tuberculosis (may be transmitted from one person to another).
3 History of different forms of allergy such as bronchial asthma, allergic rhinitis or urticaria.
4 History of D.M.

CHEST SYMPTOMS

HAEMOPTYSIS

Definition:
(Coughing up blood, blood tinged or blood streaked sputum).

Causes:

1- False haemoptysis:
(Blood originating above the level of the vocal cords.)
1- Bleeding from the gums e.g. scurvy.
2- Pharyngeal causes as: Inflammation - Tumors - Ulcers.
II- True haemoptysis:

(Below the level of vocal cords i.e. lower respiratory tract.)

1- Laryngeal causes:
   ♦ Acute and chronic laryngitis.
   ♦ Tumors.

2- Tracheal causes:
   ♦ Tracheitis.
   ♦ Tracheal tumours.

3- Pulmonary causes:
   ♦ T.B.
   ♦ Infarction.
   ♦ Pneumonia.
   ♦ Pulmonary congestion and oedema.
   ♦ Non specific chest infection.
   ♦ Bronchiectasis or lung abscess.
   ♦ Bronchial carcinoma or adenoma.
   ♦ Bronchoscopy or bronchial biopsy.

4- Cardio - vascular causes:
   ♦ M.S.
   ♦ Left ventricular failure.

5- Systemic disorder causes:
   ♦ Haemorrhagic blood disease as leukemia and purpura.
   ♦ Vasculitides e.g.: Wegener's granuloma.
   ♦ Good pasture syndrome.

The important causes of haemoptysis are:

1- Non specific chest infection (the commonest).
2- M.S.
3- Pulmonary T.B.
4- Bronchiectasis sicca haemorrhagica (apical bronchiectasis).
5- Bronchial carcinoma.
6- Pulmonary infarction (pulmonary embolism).
7- Blood diseases.

Causes of frank haemoptysis:

1- T.B.
2- Bronchial adenoma.
3- Bronchiectasis sicca haemorrhagica.
4- M.S. (bronchial apoplexy).
5- Bronchogenic carcinoma.

D.D.:

1- False from true haemoptysis:

1- History.
2- Blood on the surface of sputum in false haemoptysis.
3- E.N.T. examination.
2- **Haemoptysis from haematemesis:**

<table>
<thead>
<tr>
<th>Haemoptysis</th>
<th>Haematemesis</th>
</tr>
</thead>
<tbody>
<tr>
<td>1. History of chest or cardiac troubles.</td>
<td>1. History of dyspepsia, epigastric pain or vomiting</td>
</tr>
<tr>
<td>2. Blood is:</td>
<td>2. Blood is:</td>
</tr>
<tr>
<td>* Coughed.</td>
<td>* Vomited.</td>
</tr>
<tr>
<td>* Bright red in color.</td>
<td>* Dark brown (acid haematin)</td>
</tr>
<tr>
<td>* Mixed with froth.</td>
<td>* Mixed with food.</td>
</tr>
<tr>
<td>* Alkaline.</td>
<td>* Acidic.</td>
</tr>
<tr>
<td>3. Sputum remains blood tinged for few days.</td>
<td>3. Sputum usually normal colour.</td>
</tr>
<tr>
<td>4. No melena? Or (spurious melena)</td>
<td>4. Melena Usually occur ?</td>
</tr>
<tr>
<td>5. Examination for the cause.</td>
<td>5. Examination for the cause.</td>
</tr>
</tbody>
</table>

1- In **haematemesis** the blood may be bright red in color if:
   a- Bleeding is massive.
   b- In presence of achlorhydria.

2- In **haemoptysis** there may be melena if the patient swallows the blood.

3- Epistaxis may leads to melena (Spurious melena) if the blood swallowed, also it may give rise to apparent haemoptysis if blood in the posterior nares is inhaled and then coughed up.

**Diagnosis:**

1- Careful history and examination both general and local for the cause.
2- Nose, throat and gums must be examined to exclude a false origin.
3- X-ray chest – C.T chest.
4- Sputum examination. Microscopic ex may show hemosiderin laden macrophages.
5- Bronchoscopy and biopsy
6- Blood count, bleeding time and P.T.T.

**Treatment:** (see chest diseases).

**Q: Types of haemoptysis:**

- Blood stained or tinged sputum → blood & mucous mixed in various proportions.
- Blood streaked sputum → streaks of blood are present in mucoid or purulent sputum
- Frank haemoptysis.
- Pink frothy sputum e.g.: in pulmonary edema.
- Rusty sputum e.g.: in Pneumococcal pneumonia.
CHEST PAIN

Causes:

A- C.V.S. Causes:
1. Angina pectoris.
3. Dissecting aneurysm.
4. Pericarditis.
5. Pericardial effusion.
6. Aortic aneurysm.
7. Cardiac neurosis.

B- Pulmonary causes (pleural):
1. Pleurisy e.g. viral.
2. Pulmonary infarction → Pleurisy
3. Pneumothorax.
4. T.B → Pleurisy.
5. Pneumonia → Pleurisy.

C- Chest Wall:
1. Skin and subcutaneous tissues: Inflammation - trauma - neoplasm.
   I.e.: Coxsackie B infection (Bornholm disease).

D- Oesophagus:
1. Oesophagitis.
2. Oesophageal achalasia.
3. Oesophageal spasm.

E- Trachea:
Tracheitis.

F- Mediastinal pain:
Constrictive, retrosternal, may radiate into arms & neck. Lesions include tumors, Mediastinitis & mediastinal emphysema.

F- Abdominal conditions:
1. Perforated peptic ulcer?
2. Acute cholecystitis?
3. Acute haemorrhagic pancreatitis?

Causes of acute chest pain:
**Cough**

**Definition of cough**
It is an explosive expiration which provides a means of clearing the tracheobronchial tree of secretions and foreign bodies.

**Cough reflex:**
- Afferent through the vagus with all its branches. (Stimuli arising in the mucosa of the respiratory tract from the pharynx to the smaller bronchi).
- Also stimuli arising from the parietal pleura may produce cough e.g. due to pleurisy and during tapping of pleural effusion.
- Efferent through Vagus, phrenic and intercostals nerves.
- Center in the medulla.

**Causes and criteria:**

**A. Respiratory causes:**

1. **Pharyngeal:**
   - E.g.: pharyngitis or post nasal drip due to sinusitis
   - The cough is:
     - Non productive - explosive - persistent.
     - Painful with sore throat.

2. **Laryngeal:**
   - Due to laryngitis, laryngeal tumours, whooping cough and croup.
   - The cough is:
     - Persistent.
     - Painful.
     - Barking in character, may be associated with hoarseness or stridor.

3. **Tracheal causes.**
   - E.g.: Tracheitis associated with brassy painful cough.

4. **Pulmonary disease:**
   - Acute bronchitis → mucopurulent (day & night)
   - Chronic bronchitis → mucoid (worse at morning)
   - Bronchial Carcinoma → Change in ch.ch. of cough. (Persistent with haemoptysis)
   - Asthma: dry or productive (worse at night).
   - Bronchiectasis: productive, changes in posture induce sputum production.
   - Interstitial pulmonary fibrosis: dry, irritant and distressing.

5. **Pleural disease:**
   - Pleurisy → painful dry cough
   - Pneumothorax.

**B. Cardiac causes:**

1. Pulmonary venous congestion (often at night, frothy sputum).
2. Pulmonary infarction (painful, blood tinged sputum).
C. Abdominal causes: (Dry cough)
1- Sub diaphragmatic infections.
2- Vagal stimulation e.g.: gastroduodenal disease.

D. C.N.S. & others: (Dry cough)
1- Encephalitis - meningitis. (Meningeal cough)
2- Vascular accidents.
3- Aural causes.
4- Hysterical

| Paroxysmal cough: In Bronchial asthma, mediastinal $, whooping cough, extrinsic alveolitis, recurrent pulmonary embolism, and neurosis |

Character of sputum:

1- Frothy sputum: (serous)
It is a clear watery sputum but often blood tinged it is characteristic of pulmonary oedema (pink frothy sputum), pulmonary venous congestion, broncho alveolar carcinoma (rare)

2- Mucoid:
White or gray jelly like sputum characteristic of chronic bronchitis and bronchial asthma.

3- Purulent or mucopurulent:
Yellow or yellow green jelly like frank pus (purulent) occurs in abscess and bronchiectasis. Mucopurulent; occurs in all types of broncho pulmonary infection.

N.B.: Excessive eosinophils can cause sputum to appear purulent like (Yellow).

4- Rusty sputum
Reddish brown due to the presence of altered blood pigment, it occurs in Pneumococcal pneumonia.

5- Chocolate sputum (Anchovy Sauce):
Characteristic of amebic lung abscess.

6- Red-current jelly:
Combination of mucous, tissue debris and blood, it is characteristic of bronchial carcinoma.
7- Caseous sputum:
Characteristic of T.B (nannular sputum).

8- Black staining:
Inhalation of carbon.

Complications of cough:

1. Rupture emphysematous bullae → spontaneous pneumothorax.
2. Fractures of ribs.
3. Hernia.
4. Stress incontinence of urine and may be stools.
5. Retinal hge or retinal detachment in predisposed patients.
6. Cerebral hge and Subarachnoid hge in predisposed patients.
7. Cough syncope due to high intrathoracic pressure → ↓ V.R → ↓ COP.

☆ Bovine cough i.e.: the explosive ch.ch. of normal cough is lost due to paralysis of vocal cords due to malignancy.

Q: Causes of Dyspnea
- Physiological (exercise and high altitude).
- Pathological
  - Obesity
  - Anaemia
  - Respiratory diseases (Acute, chronic, paroxysmal).
  - Cardiac disease (Exertional, positional, at rest).
- Psychological: Hyperventilation.
- Pharmacological: Drug induced respiratory or cardiac disorders.

N.B.: Generally dyspnea can be also classified into
- Exertional
- At rest
- Paroxymal
- Positional (orthopnea, trepopnea, platypnea)

Causes of persistent or chronic cough
☆ Chronic bronchitis
☆ Bronchiectasis
☆ T.B
☆ Severe gastro oesophageal reflux
☆ Repeated aspiration
☆ Bronchial carcinoma
☆ Cough variant asthma
☆ Interstitial pulmonary fibrosis
☆ Drugs e.g. ACE inhibitors
☆ Psychogenic, it may be a habit
GENERAL EXAMINATION (See before)

1. Decubitus: e.g. lateral position in unilateral chest disease (e.g. Trepopnea).

2. Disturbed mentality, cyanosis (respiratory failure).

3. Congested neck veins: due to:
   - Cor - pulmonale.
   - Massive pleural effusion or pneumothorax.
   - Chronic obstructive pulmonary disease.
   - Mediastinal syndrome e.g. mediastinal L.N. → SVC obstruction

4. Enlarged lymph nodes due to:
   - Bronchogenic carcinoma (scalene lymph nodes).
   - Tuberculosis.
   - Sarcoidosis.

5. Clubbing of fingers, with:
   - Chronic suppurative lung disease.
   - Bronchogenic carcinoma.
   - Chronic obstructive pulmonary disease.
   - Interstitial lung fibrosis.
   - Mesothelioma.

6. Flabbing tremors in respiratory failure (CO₂ retension).

7. Oedema of L.L. may occur due to:
   - Right ventricular failure due to cor-pulmonale.
   - Renal amyloidosis due to suppurative disease (Nephrotic $).
   - Hypoproteinaemia due to loss of proteins in the sputum.

8. Liver enlargement may occur due to:
   - Cor-pulmonale.
   - Secondaries from bronchial carcinoma.
   - Amoebic liver abscess, Miliary T.B.
   - α1 anti-trypsin deficiency as it leads to liver cirrhosis.
   - Associated liver disease.

9. Splenomegaly in the following cases:
   - Amyloidosis, Sarcoidosis, miliary T.B.
   - Bilharzial Cor-pulmonale.

10. Ascites:
    - Cirrhotic ascites may lead to right sided pleural effusion.
    - Cirrhotic ascites with left sided pleural effusion suggestive to lung disease e.g.: T.B.
    - T.B peritonitis and right sided failure → ascites.
11. Skin lesions:
E.g.: Erythema nodosum in T.B or Sarcoidosis.
Herpetic vesicle may identify the cause of unilateral chest pain.

12. Neurological examination for neuropathy, myopathy (paramalignant $), pott's disease or signs of meningeal irritation (T.B.)

LOCAL EXAMINATION

I- Inspection:
   a- Shape and form.
   b- Symmetry.
   c- Movement.
   d- Dilated veins, swelling.
   e- Pulsation (apex and epigastrium).

A- Shape and Form:

Normal chest is:
☆ Symmetrical.
☆ Moves freely with respiration.
☆ Antero-posterior diameter equal 5/7 of the transverse diameter.
☆ Subcostal angle is nearly about 90.

Abnormalities in shape and form:
① Barrel Chest: (e.g. in patients with emphysema).
    ◆ Limitation of expansion.
    ◆ Antero-posterior diameter equal transverse diameter.
    ◆ Horizontal ribs with wide intercostal spaces - sternum is pushed forward.
    ◆ Wide subcostal angle.

N.B.: An increase in antero-posterior diameter may also be due to thoracic Kyphosis unrelated to respiratory disease.
Pigeon Chest: (pectus carinatum)
- Forward protrusion of the sternum.
- It denotes old rickets.
- The softened upper ribs bend inward, forcing the sternum forward.
- This is a common result of chronic respiratory disease usually asthma in childhood. Pigeon chest may also caused by rickets or it may be congenital.

Rachitic Chest:
- It is a pigeon chest plus:
  a- Rachitic rosary: bulging of the costochondral junction, the bulging is mostly inward and in severe cases there is outward bulging as knobs at the costochondral junction. It exists only during the activity of rickets in the 1st 2 years of life, healing obliterates the knobs without a trace.
  b- Harrison's Sulcus: a transverse groove extending from the xiphoid process towards the axilla. It is corresponding to the line of attachment of the diaphragm to the rib cage. It is due to inward pull of diaphragm on the soft ribs, it remains when rickets heals.

d- Funnel Chest: (Pectus Exacavatum)
The reverse of pigeon chest, the sternum (The lower end or the whole sternum) is retracted towards the spine, so it diminishes the antero-post diameter. It is usually asymptomatic, but when there is a very marked degree of depression the heart may be displaced to the left and the ventilatory capacity of the lungs will be restricted.
- It is either:
  a- Congenital.
  b- Acquired in shoemakers.

e- Kyphoscoliosis:
This type of chest deformity may alter the position of mediastinum and may reduce the ventilatory capacity of the lung causing hypoxia and then Core pulmonale. It is caused by tuberculous spondylitis, muscular dystrophy, Marfan syndrome or it may be congenital.

**B- Symmetry:**
The normal chest is symmetrical and the two sides of the chest move to an equal extent with respiration.

<table>
<thead>
<tr>
<th>Bulging</th>
<th>Retraction</th>
</tr>
</thead>
<tbody>
<tr>
<td>2. Pneumothorax.</td>
<td>2. Fibrosis.</td>
</tr>
<tr>
<td>5. Pericordial bulge.</td>
<td></td>
</tr>
</tbody>
</table>

To differentiate bulging from retraction look to the movement.
Chest sheet

N.B.: Notice also whether inspiration is noisy or not.

- **Wheeze**: a whistling sound in obstructive lung disease.
- **Stridor**: harsh, crowing inspiratory sound with obstruction of larynx, trachea or large bronchi.
- **Rattling**: intermittent coarse sound due to bubbling of secretions in trachea in dying or comatose patients.

**C. Movement:**

- Normal breathing is abdomenothoracic in males and thoracoabdominal in females (specially during pregnancy).
- Thoracic breathing occurs in peritonitis and abdominal distention (ascites-large ovarian cyst).
- Abdominal breathing occurs in a case of limited chest expansion due to lung disease or due to ankylosing spondylitis or pleural pain.

We also should comment on the following:

1. **Rate of respiration:**
   - 14 - 18/mm.
   - In the newly born it is about 44 and gradually it decrease till maturity.
   - **Tachypnea**: rapid rate of respiration as in fever - anemia - exertion - thyrotoxicosis - cardiac insufficiency and pneumonia.
   - **Bradypnea**: slow breathing e.g. increased I.C.T., excess alcohol or morphia.

2. **Respiratory depth:**
   - **Hyperapnea** i.e. increase depth of respiration e.g. in acidosis (air hunger).
   - **Oligopnea**: shallow breathing e.g. pneumonia, inhibition of respiratory center. In periodic or chyne - stokes breathing there is a cyclical variation in the depth of breathing (see later).

3. **Pulse - respiration ratio:**
   - Normally it is 4:1, this ratio is reversed in Pneumonia (it is an old term).

4. **Limitation of movement:** (Any chest disease may decrease movement)
   - It may be unilateral due to unilateral disease e.g. fibrosis, effusion or pneumonia.. or bilateral → e.g.: in obstructive lung disease.

5. **Abnormal inspiratory or expiratory movements:** (see later the mechanics of respiration)
   - **Suction of lower intercostal spaces**: (Inspiratory intercostals suction)
   - In chronic obstructive pulmonary disease (COPD) i.e.: Littin's sign.
   - It may be present in normal persons. It indicates diaphragmatic movement so it disappears in diaphragmatic paralysis and pleural effusion.
   - **Hoover's sign**: means inward movement of the ribs due to contraction of the diaphragm. It can be seen in C.O.P.D because the diaphragm is sufficiently flattened that it can pulls the ribs and both costal margins inward and medially. So the subcostal angle becomes more acute during inspiration.
7- Abnormal types of respiration: (see later)

**E- Pulsation (Apex-Epigastrium):**
For diagnosis of right ventricular hypertrophy (Cor pulmonale).

**F- Swellings or lesions of chest wall:**
- Subcutaneous emphysema, subcutaneous sarcoid nodules, bony swelling of ribs or spines.
- Skin eruption e.g. purpuric spots or herpes zoster.
- Engorged veins on the chest wall due to S.V.C obstruction.

II- Palpation:

**A- To confirm the movement:**

1- **Upper part:** (above the second rib) i.e. upper lung zone.
   - Put the palms in the infra clavicular fossa and the 2 thumbs are in the mid line at the level of supra sternal notch.

2- **Middle Part:** (from the second rib to the fourth rib) i.e. mid lung zone.
   - Put the palms in the middle part with the tips of thumbs in the mid-line.

3- **Lower Part:** (below fourth rib) i.e. lower lung zone.
   - Put the palms in the lower part with the tips of the thumbs in the mid-line.

Let the patient to inspire deeply and let your thumbs to follow the chest movement.

**Causes of limitation of movement:** (See inspection).

Chest expansion can be determined by measurement of the chest circumference using a tape at the level of fourth space, normally it is ≥ 5 cm if it is ≤ 2.5 cm it is definitely abnormal. Chest expansion is diminished in every type of diffuse lung disease and in ankylosing spondylitis.

**B- Palpable rhonchi or pleural rub (pleural fremitus)**

**C- Tactile Vocal Fermitus T.V.F.:**
- It is the palpable vibration of the vocal cords, the patient voice felt at the chest wall when the patient says 44 in Arabic or 99 in English.
- It is better to be felt by placing the hands over various areas of the chest and feeling the thrill transmitted through the chest.
- Examine both sides of the chest with comparison while the hands in the direction of bronchial tree and away from midline.
Conditions which increase T.V.F.:
1. Consolidation. e.g.: pneumonia
2. Cavitation, it must be:
   - Big cavity.
   - Superficial.
   - With an area of consolidation around it.
3. Pulmonary fibrosis.
   If it is markedly heterogeneous with pulling of trachea toward it.

Areas which normally got increased T.V.F.:
- Parasternal region on the right 2nd space (near tracheal bifurcation).
- Interscapular area.

Conditions which decrease T.V.F.:
1. Pleural effusion or pleural fibrosis (dense fibrosis).
2. Pneumothorax.
3. Emphysema.
4. Collapse, if the underlying bronchus is obstructed.

D. Position of mediastinum and examination of trachea:
1. Apex beat → examine the heart to exclude cardiomegaly.
2. Trachea: There are 2 methods to know the position of trachea:
   a. Insert the index finger into the pouch between the medial end of sternomastoid and the lateral aspect of the trachea. The patient sitting up with the head straight, you can find a little recess by the tip of your finger, the recesses should be equal on both sides.
   b. Trill's sign: i.e. bulging of the tendon of sternomastoid in front of deviated trachea (by inspection).

A. Identifying the position of the trachea.
B. Assessing the cricosternal distance.
**Causes of shift of Mediastinum:** (tracheal shift)

**Tracheal Shift to the right as an example:**

**a- Causes in the right:** - Collapse. - Apical fibrosis.

**b- Causes in the left:** - Pleural effusion. - Pneumothorax.

- The trachea can be palpated from the cricoid to the suprasternal notch (crico-sternal space/distance), this distance is 4 - 5 cm. this length may be shorten in COPD. To measure this space, insert one or two fingers between the cricoid cartilage and the suprasternal notch.

- Tracheal descent with inspiration (Campbell's sign) or tracheal tug may correlates with severity of COPD. It is not specific. It is felt by placing the tip of the index finger on the thyroid cartilage during inspiration. This is due to pulling on the trachea during inspiration due to strong diaphragmatic contraction in COPD.

- Vascular tracheal tug (Oliver's sign) i.e. descent of cricoid cartilage with ventricular contraction in cases of aortic aneurysm as the aortic arch overrides the left main bronchus. It can be felt by grasping the cricoid cartilage with gentle upward pressure with the thumb and index while the head is extended.

- Tracheal shift as above.

**E- Localized tenderness:**

e.g. fracture rib or tumour involving chest wall.

**III. Percussion:**

**Principle, technique:** Air is resonant, everything else is dull.

- We use the middle finger of the right hand; it struck sharply and quickly with the tip over the shaft of the middle phalanx of the left middle finger.

- Place the left hand on the chest wall with the fingers parallel to the ribs, the middle finger is placed firmly in the intercostal space chosen.

- Light percussion gives palpable rather than audible vibration.

- Heavy percussion gives palpable and audible vibration.

- Types of percussion notes:
  - **Tympamy:** hollow viscus. e.g. normal traub's area.
  - **Hyper - resonance:** emphysema and pneumothorax.
  - **Resonance:** normal lung.
  - **Impaired note:** pulmonary consolidation or fibrosis.
  - **Dullness:** pulmonary consolidation, pulmonary collapse & fibrosis.
  - **Stony:** pleural effusion.

- Percuss with two taps with comparison between right & left lung fields.
Methods of percussion:
The aim of percussion is to compare the degree of resonance over equivalent areas on the two sides of the chest, and to map out any area of abnormal note by percussing from a resonant to a dull area. Percussion of the lung is light percussion except the back by heavy percussion.

Sites of percussion:
- 3 lines
- 3 Areas
- 3 special zones

A- Lines: (percuss with comparison)
1. Starts to percuss both clavicles directly (medial third)
2. Percuss the infra-clavicular regions.
3. Percuss the intercostal space in MCL with comparison from second to sixth intercostal space. Normally, in the right we percuss from resonance to dullness & in the left we percuss from resonance to tympany.
4. Percuss the intercostal space in mid axillary line (from fourth to eighth space)
5. Percuss the scapular line by heavy percussion (from the above of spine of scapula and down to T₁₀.
6. Percuss the inter-scapular areas by heavy percussion with comparison.

B- Borders and bare area:
1. Upper border of the liver by heavy percussion of the right MCL from above downward.
2. Lower border of the lung by light percussion of right & left MCL, normally it is at 6th rib.
3. Bare area of the heart by light percussion for emphysema or pneumothorax.

C- Special Zones and tidal percussion:
(1) Kronig's Isthmus:
☆ It is limited medially by a line from the sterno-clavicular joining to 7th cervical vertebrae.
☆ It is limited laterally by a line joining the junction of medial 2/3 with the lateral 1/3 of the clavicle to the spine of the scapula at the junction of lateral 2/3 with the medial 1/3.
☆ It overlies the apex of the lung.
☆ Percuss from behind from dullness to resonance while the patient is sitting.

Causes of dullness in Kronig's Isthmus:
5. Apical bronchiectasis.
(2) **Traub's Area**: (it is bordered by the left 6th rib superiorly, the midaxillary line laterally and the left costal margin inferiorly), it overlying the fundus of the stomach.

**Boundaries:**
- **Left**: 9th to 11th rib in the mid axillary line.
- **Upper**: From the 9th rib to the apex.
- **Right**: from the apex to the tip of the left 8th costal cartilage (left lobe of liver).
- **Lower**: From the left 8th costal margin to the 11th rib mid axillary line.

☆ Normally it got a tympanic resonance due to the air in the fundus of the stomach.
☆ It can be examined by percussion of MCL & AAL from above downwards (from resonance to tympany)

**Causes of dullness in Traub's area:**
1. Splenomegaly.
2. Left Pleural effusion.
3. Pericardial effusion.
4. Hepatomegaly.
5. Full stomach and gastric tumor.
6. Infra diaphragmatic causes as ascites, pregnancy.

Its only importance in chest diseases is to differentiate pleural dullness (pleural effusion) from pulmonary dullness (fibrosis, consolidation, collapse)

(3) **Tidal Percussion**:
Used to differentiate supra from infra diaphragmatic dullness, also it can give an idea about the diaphragmatic movement by measuring the distance between the lower border of pulmonary resonance at the back of the chest in full inspiration and forced expiration.

* Percuss the scapular line from above downward from resonance to dullness, then compare the level of dullness during inspiration and expiration.

**Possibilities of tidal percussion:**
- **Normal**: dullness at $T_{10}$ inspiration → resonance
- **Supra diaphragmatic dullness**: dullness above $T_{10}$ inspiration → the same.
- **Infra diaphragmatic dullness**: dullness above $T_{10}$ inspiration → resonance.
- **Reversed tidal percussion** (diaphragmatic paralysis): Percuss from above downward till dullness, proceed one segment or space above (resonant) it becomes dull on inspiration (the diaphragm pulled by the negative pressure of the chest).
- Limited movement on both sides occurs in emphysema.

(4) **Shifting dullness**:
Used to differentiate pleural effusion, from hydro pneumothorax by percussing in 3 planes. In pleural effusion no change while in hydro pneumothorax it is changed.

- **1st plane**: Percuss the M.C.L from above downward till the dullness, proceeds one space above (resonant), ask the patient to sit, the resonance will be dull in hydro pneumothorax.
- **2nd plane**: Percuss from the sternum to the axillary line till the dullness, ask the patient to lie on the opposite side. The dullness will be resonant in hydro pneumothorax.
- **3rd plane**: Percuss the back (scapular line()) from above downward till the dullness, ask the patient to lean forward. The dullness will be resonant.
IV. Auscultation:

- We can use either diaphragm or bell.
- Most of the sounds reaching the chest wall from the bronchi and lungs are of low frequency so the bell is preferred.
- Stretching of the skin and hairs under the diaphragm during deep breathing produce sounds like pleural rub or crepitations so the bell is preferred.
- The patient is relaxed and breathing deeply and fairly rapidly through the mouth. Avoid prolonged deep breathing to avoid giddiness or tetany.

We report on:

A. Intensity of breath sounds  B. Type of breath sounds.
C. Adventitious sounds.  D. Voice sounds.

Methods of Auscultation: (auscultate and comparing both sides)

Auscultate anteriorly (MCL) from above the clavicle down to sixth rib, laterally (M.A.L) from the axilla to the eighth rib and posteriorly (scapular line) from above down to T10.

(A) intensity of breath sounds:

- Reduced air flow
  - Emphysema
  - Bronchial obstruction
  - Collapse.
- Reduced conduction
  - Pneumothorax
  - Pleural effusion
  - Thick chest wall.

Causes of low intensity:

(B) Types of Breath sounds:

The normal breath sound is called vesicular breathing characterized by:
- Related to vesicles. (Alveoli)
- It is soft & breezy, resembling the gentle rustling sound of tree leaves.
- Heard on inspiration.
- Expiration is short or absent.
- No pause between inspiration and expiration.
- Heard at any part on the lung especially in the axillary line.

\[ \text{Bronchial breathing} \quad \text{Harsh vesicular breathing} \quad \text{Vesicular breathing} \]

Abnormalities in breath sounds:

1- Bronchial or Hollow breathing:

Character:
- The expiratory sound is as long as the inspiratory sound and usually of high pitch.
- Hollow character.
- It is like the sound heard on the trachea.
- Bronchial breathing occurs when the relevant major bronchus remains patent and the normal lung tissue is replaced by a uniform conducting medium e.g.: consolidation, Cavitation or collapse (alveoli out of function)
Varieties of the bronchial breathing:

**a- Tubular breathing:** As blowing into a tube (it is a high pitched), it occurs in pneumonia.

**b- Amphoric breathing:** As blowing air through the mouth of a bottle.

Ex.: ★ Large empty cavity that communicate with a bronchus.
    ★ Open pneumothorax.

**c- Cavernous breathing:** As blowing into a cupped hand.

The pitch is low e.g.: in cases of cavity.

**2- Harsh vesicular breathing:**

Breath sounds characterized by prolonged expiration. It is heard in COPD. In cases of COPD the expiration is an active process, so it is prolonged; also this occurs in cases of bronchial asthma during the attack.

**C Adventitious Sounds:** Recent mechanisms (see later)

**1- Crepitations:** (Rales) Interrupted non musical short sounds with crackling quality heard mainly during inspiration.

A) Crepitations may be caused by the bubbling of air through secretions in dilated bronchi (bronchiectasis), pulmonary cavities and within the large bronchi in acute or chronic bronchitis or in resolving broncho pneumonia.

B) It is now believed that crepitations are also due to an alteration in the elastic properties of the lungs, also a much more frequent cause of crepitations is the explosive reopening of peripheral small airways during inspiration which become occluded during expiration.

C) Crepitations due to secretions either decreases or disappear temporarily after cough, they are widespread and bilateral in acute or chronic bronchitis or resolving broncho pneumonia while those audible over resolving lobar pneumonia, bronchiectasis or cavities are localized to the site of the lesion. In all these conditions they are audible throughout inspiration.

D) The crepitations which occur due to decrease the elastic properties of the lungs are audible in late inspiration, they are not influenced by coughing. They are more in the lower parts of the lung, because in the upright position small airway closure is more liable to occur than in the upper lobes, they occur in parenchymal lung conditions e.g.: interstitial pulmonary edema, early pneumonia and miliary T.B, pulmonary fibrosis and interstitial pulmonary diseases.
Types of Crepitations:

A- Fine crepitations:
- They are high pitched with high frequency.
- Audible at the end of inspiration, Causes are:
  - Early stage of pneumonia.
  - Bases of the lung in P.V.C.
  - Interstitial lung disease (crepitations are of leathery character and decreased on leaning forward).

B- Coarse crepitations: (↓ Pitch ↓ frequency)
- They are audible throughout inspiration, Causes are:
  - Bronchiectasis.
  - Chronic or acute bronchitis.
  - Resolving pneumonia, broncho pneumonia.
  - Lung abscess.
  - Pulmonary T.B. (pneumonia or cavity)

Crepitations usually the coarse type may be further classified into:
1. Consonating: e.g. presence of area of consolidation, ex. Pneumonia, lung abscess or bronchiectasis
2. Non Consonating: e.g. in acute or chronic bronchitis.

N.B.:
- Bubbling crepitations occurs in pulmonary edema.
- Crepitations can be also classified into:
  1. Early inspiratory with secretions (disappear temporarily after cough)
  2. Late inspiratory in PVC, interstitial pulmonary fibrosis and pneumonia. They are not influenced by cough.
- Medium sized capitations (old term) occur in bronchitis (nonconsonating) or pneumonia (consonating)

2- Rhonchi:
Continuous musical sounds due to passage of air in bronchi or bronchioles which are partially obstructed, either in:
- Wall → spasm - fibrosis - tumor.
- Lumen → secretions - foreign body.
- Pressure from outside → L.N. or aneurysm.
- Rhonchi heard during expiration are usually due to spasm or oedema of the bronchial wall, these rhonchi are usually persist after cough
- Rhonchi that accompany inspiration are usually due to secretions in the bronchi, these rhonchi may disappear or ↓ after coughing
- Rhonchi are heard diffuseely over both lungs in bronchial asthma and in most cases of COPD. In asthma, rhonchi are sibilant and are mainly heard during expiration which is prolonged. In COPD they are usually sonorous and both inspiratory and expiratory
Localized rhonchi may be heard over partially obstructed large bronchus. If the obstruction is caused by a fixed lesion e.g.: tumour or foreign body, the rhonchi are not altered by coughing and they are monophonic. If the obstruction is due to secretions, the rhonchi will disappear or change after cough.

Since the occurrence of rhonchi require a fairly high velocity of air, rhonchi may not be heard if ventilation is much reduced in severe airway obstruction e.g. severe asthmatic attack (silent chest).

Types of Rhonchi:

A- Sibilant rhonchi: (wheezes)
- High pitched - due to obstruction of small bronchi.

B- Sonorous rhonchi:
- Low pitched - due to obstruction of large bronchi.

3- Pleural rub:
- Leathery sound, commonly heard in mid & post axillary lines, it is produced due to movement of visceral pleura over the parietal pleura when both surfaces are roughed
- Related to respiration. (Mainly it is toward the end of inspiration)
- Usually localized.
- Increased by pressure of stethoscope.
- Decreased or absent by effusion, but it may remain audible above an effusion
- If pleurisy involves the pleura adjacent to the pericardium, a pleuro pericardial rub may also be heard, this is a misleading term as the pericardial element is not due to pericarditis, but it is caused by the roughened pleural surface adjacent to the pericardium due to movement across one another by cardiac pulsation.

• A pneumothorax click:
It is a rhythmical sound, synchronous with cardiac systole, due to left pneumothorax, it is produced when there is air between the two layers of pleura overlying the heart.

• Hamman sign:
Crunching sound due to pneumomediastinum.

(D) (Voice sounds)
- Voice sounds conducted through consolidation, Cavitation and lung collapse associated with patent bronchus resemble more closely those produced at the larynx than those heard over normal lungs in that they are louder and more distinct (abnormal vocal resonance) i.e (bronchophony)
- Transmission of whispered voice almost without distortion, so that the individual syllables can be clearly recognized (whispering pectoriloquy)

Voice sounds can be detected when the patient say ninety-nine or 44 in arabic several times while auscultating the site of lung lesion.
Causes of abnormal voice sounds (abnormal vocal resonance and whispering pectoriloguy):

- Pneumonia.
- Collapse with patent bronchus.
- Lung cavitation.
- Extensive fibrosis.

> Egophony: (Greek word for the voice of a goat)
In which the syllables of voice sounds have a peculiar nasal quality or bleating above the level of pleural effusion or in some cases over an area of consolidation
> E to A changes are a variant of egophony where the five vowel sounds (A, E, I, O, U) become A in effusion or consolidation. Now we refer to them as the E to A change.

Special Tests:

1- **Post - tussive suction:**
Suction sound heard over a cavity after cough, indicates that the cavity is collapsible (old sign).

2- **Succussion splash:**
Heard in cases of hydropneumothorax.

3- **Coin test:**
+ Ve in tension pneumothorax.

4- **Auscultatory percussion:**
Tapping lightly over the manubrium of the sternum with the tip of one finger while auscultating the chest posteriorly. A decrease in sound amplitude is considered as sign of lung abnormality. Up till now it is not proved.

5- **Auscultation for mediastinal L.N:** see examination of L.N.

6- **Auscultation of trachea:** to allow recognition of stridor.

**EXAMINATION OF THE BACK**

A- Inspection:
For any deformity in the spine:
- Lateral deviation (scoliosis).
- Kyphosis.
- Lordosis due to increase in the anterior convexity of the lumbar spine.

**N.B.:** Extensive deformity of the spine may leads to pulmonary hypoventilation and Cor pulmonale. (e.g.: Kyphoscoliosis)

B- Palpation:
- T.V.F. in the infrascapular regions.
- Movement: place the fingers on each axilla and the palms are applied firmly to the chest, so as your fingers are one rib below the Inferior angle of the scapula. Thumbs extend till meat in the middle line over the vertebral spines. Let the patient to inspire deeply.
C- Percussion:
We use heavy percussion comparing the right and left scapular lines as before.
   a. Supra scapular area.
   b. Scapular area.
   c. Infra scapular area.
   d. Inter scapular area.
   e. Tidal percussion
   f. Direct percussion on the spines e.g. for Pott’s disease.

D- Auscultation: As in front (see before).

D.D. OF DULLNESS ON THE RIGHT BASE

1. Pulmonary causes:
   1. Pneumonia.
   2. Lung abscess (Aspiration, amebic).
   4. T.B, tumours.

2. Pleural:
   1. Pleural effusion.
   2. Pleural fibrosis.
   3. Hydro or pyoneumothorax.
   4. Empyema.

3. Infra diaphragmatic causes:
   1. Sub diaphragmatic abscess.
   2. Amebic liver abscess.
   3. Hepatomegaly and other abdominal tumors.
   4. Ascites.

4. Cardiac causes:
   Massive pericardial effusion.

5. Chest wall causes:
   Tumors of skin, rib or subcutaneous tissue and muscles.

* Describe the diagnostic feature of the above causes.
* Mention how to differentiate between supra diaphragmatic (1&2) from infra diaphragmatic (3) i.e. tidal percussion.
* Mention Investigations and treatment of each disease.


DIAGNOSIS

- Aetiological if possible e.g. T.B
- Anatomical (The affected lobe).
- Functional i.e. presence of respiratory failure or core pulmonale.

**Examples:**
- Case of COPD (chronic bronchitis/emphysema) no manifestation of core pulmonal or respiratory failure.
- Case of suppurative lung disease mostly, bronchiectasis, there is core pulmonal, but no respiratory failure.
- Case of left sided pulmonary fibrosis mostly, post tuberculous with right sided compensatory emphysema, no manifestations of respiratory failure or core pulmonal.

SEGMENTS OF THE LUNGS

(A) The right lung: Composed of 3 lobes:
1. Upper lobe: (3 segments) Apical, anterior and posterior.
2. Middle lobe: (2 segments) Medial and lateral.
3. Lower lobe: (5 segments) Superior, anterior, posterior, medial and lateral.

(B) The left lung: Composed of 2 lobes:
1. Upper lobe:
   - Proper: (2 segments) apico-posterior and anterior.
   - Lingula: (2 segments) superior and inferior.
2. Lower lobe: (4 segments).
   Superior, antero basal, lateral basal and post basal.

TYPES OF BREATHING

1- Cheyne - stockes breathing: it is a type of periodic breathing

There are periods of apnea for about 1/2 minute followed by hyperapnea for 8 - 10 cycles then apnea, and so on. It is due to depression of respiratory center with sensitivity to CO₂.

Hyperventilation → washes CO₂ → apnea → CO₂ retension → Hyperventilation
and so on with a crescendo-decrescendo pattern

Causes:
1. Depression of R.C. by opiates, barbiturates or alcohol.
2. Depression of R.C. by hypoxia or ischaemia as in: vertebro-basilar insufficiency, advanced H.F. pontine damage and cerebral infarction.
3. It occurs during sleep in some normal persons?

II- Biot's Breathing: It is a variant of cheyne-stokes breathing

Occurs in meningitis, head injuries and medullary compression.
There is periodic breathing but it lacks the typical crescendo-decrescendo pattern.
**III- Apneustic Breathing:**
Post inspiratory pause in brain stem lesions especially pontine.

**IV- Neurogenic Hyperventilation:**
Mid brain lesion and upper pontine lesion.

**V- Ataxic Breathing:**
Lesion in medulla oblongata. It is a sort of fibrillation of respiratory centers.

**VI- Grunting Breathing:**
Pneumonia.

**VII- Stridulous:**
In laryngeal obstruction.

**VIII- Kussmaul’s breathing:** in metabolic acidosis

**MECHANISM AND ORIGIN OF BREATH SOUNDS**

Breath sounds are normally produced by 2 elements:

1. **Laryngeal element:**
Vibrations of the vocal cords caused by the rapid flow of air during inspiration through the glottis (opening between vocal cords). This will result in sound waves with high and low frequency named glottis hiss, these sound waves then pass through the trachea and lung tissue, during this traveling all high frequency sounds are absorbed by normal lung tissue, which allows only the low frequency waves to filter across chest wall.

2. **Vesicular or lung element:**
Due to vibrations of the peripheral lung tissue by inspiration only, so the normal breath sounds composed of:
- Inspiration = low frequency sounds of the glottis hiss + high frequency sound at the periphery.
- Expiration = low frequency sounds of the glottis hiss only.

Bronchial breathing is glottic due to transmission of the glottic hiss through bronchial tree to an area with alveoli out of function e.g. consolidation, because the normal breath modify the bronchial sound into vesicular.
MECHANICS OF RESPIRATION

- Lung compliance refers to the ease with which the lungs can expand under pressure.
- Elasticity of the lung refers to the tendency of the lung to return to its initial size after being distended.
- Normal quiet inspiration results from contraction of the diaphragm. Inspiration is aided by contraction of external intercostals.
- Abnormal inspiratory movements produced by contraction of the sternomastoids, scaleni and trapezii. Indrawing of the supraclavicular, suprasternal fossae, and the intercostals spaces occur in severe airway obstruction.
- Normal quiet expiration is a passive process by recoil of the muscle of inspiration (diaphragm and external intercostals) and lung tissue.
- Abnormal expiratory movements are produced by powerful contractions of the abdominal muscles and Latissimus dorsi. This occur when the elastic recoil of the lungs become insufficient to complete the expulsion of air from alveoli as in cases of emphysema, COPD and bronchial asthma.

Recent mechanisms of production of adventitious lung sounds

1. Rupture of fluid films or bubbles when there is rapid air flow through secretions coating the large airways, this mechanism is responsible for the generation of coarse crepitations e.g. in cases of acute and chronic bronchitis.
2. Sudden equalization of intra airway pressure occurs during sudden reopening of partially collapsed small airways. This mechanism occurs when small airways are compressed by high interstitial pressure due to scarring (interstitial fibrosis) or fluid in the interstitium. This mechanism is responsible for the production of fine prepitations in cases of interstitial pulm fibrosis, or interstitial edema due to pneumonia or PVC.
3. Fluttering of the airway wall, this occurs whenever air flows rapidly through a narrow airway e.g. bronchospasm or bronchial secretions, this simply sucks in the airway wall itself producing rhonchi.
4. Rubbing of inflamed pleural surfaces when the two pleural layers become rough by an inflammatory process and covered with fibrinous layers producing leathery sound (pleural rub) which can be heard during inspiration and expiration.

- The crepitations of bronchiectasis is not generated due to the bubbling of air through thin secretions. In bronchiectasis the airways are abnormally dilated as a result of the destruction of elastic and muscular components of the wall. So the bronchial wall tends to collapse in expiration and then reopen suddenly in inspiration producing the crepitations that do not clear with coughing.
- Early or mid inspiratory crepitations are produced by mechanism 1 (as above), while late inspiratory crepitations are produced by mechanism 2 (as above).
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<th>1. Consolidation:</th>
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<td>Shape</td>
<td>Expansion</td>
<td>Mediastinum</td>
<td>T.V.F</td>
<td>Note</td>
<td>Breath. S</td>
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<td>Normal</td>
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<td>Central</td>
<td>+++</td>
<td>Dull</td>
<td>Bronchial</td>
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<td>2. Cavitation:</td>
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<td>Normal or retracted</td>
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<td>Central or pulled</td>
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<td>3. Collapse:</td>
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<td>Retracted</td>
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<td>4. Fibrosis:</td>
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<td>Pulled</td>
<td>+ or -</td>
<td>Dull</td>
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<td>Normal</td>
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<td>Central</td>
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<td>Normal</td>
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<td>6. Emphysema:</td>
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<td>Barrel</td>
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<td>Hyper resonant</td>
<td>Harsh. V</td>
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<td>7. Pleurisy:</td>
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<td>Central</td>
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<td>Normal</td>
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<td>8. Effusion:</td>
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<td>Bulge</td>
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<td>Stony dull</td>
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<td>9. Pneumothorax:</td>
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<td>Bulge</td>
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<td>Pushed</td>
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<td>Tympanitic</td>
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<td>10. Hydropneumothorax:</td>
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<td>Bulge</td>
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<td>Pushed</td>
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<td>Shifting dullness</td>
<td>Diminished</td>
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- --- (Decreased or negative)
- ++ (Increased)
THE ABDOMEN

Surface Anatomy of the Abdomen

The superficial limits of the abdomen are formed by:

☆ The lower margin of the thorax above.
☆ The inguinal ligament below.
☆ The iliac crests below and on each side.

However, the real cavity of the abdomen extends upwards beyond the costal margin into the arch of the diaphragm under cover of the lower ribs and the lower cartilage.

The costal margin:

➢ The costal margin is formed by the fused costal cartilages of the 7th to the 10th ribs.
➢ The 11th and 12th ribs are not long enough to reach the front, their tips can be palpated posterolaterally.

Iliac crest:

➢ Feel the iliac crest, trace it anteriorly and notice that it ends in front at the anterior-superior iliac spine.
➢ The tubercles of the iliac crests are the most prominent lateral points of the iliac crests. They lie 5 - 6 cm behind the anterior superior iliac spines.

The inguinal ligament:

➢ It passes downwards and medially from the anterior superior iliac spine to the pubic tubercle.
➢ The mid inguinal point (midway between the anterior-superior iliac spine and the middle line (symphysis pubis).
➢ The midpoint of the inguinal ligament (midway between the anterior superior iliac spine and the pubic tubercle.
➢ Deep inguinal ring lies opposite a point one cm above the mid point of the inguinal ligament.
➢ The superficial inguinal ring lies just above the pubic tubercle.
➢ The linea semilunaris is a line which corresponds to the lateral border of the rectus abdominis muscle, it extends from mid inguinal point towards the mid clavicular point.

IMPORTANT VERTEBRAL LEVELS

☒ Xiphisternal Joint → T9/T10.
☒ Transpyloric plane → L1 (midway between the suprasternal notch and symphysis pubis).
☒ Subcostal plane → L3
☒ Plane of the highest points of iliac crests (supracristal plane) → L4.
☒ Intertubercular plane → L5.
Liver:

- **The inferior border:** Passes along a line which extends from a point one finger's breadth below the right costal margin at the side of the trunk (10th rib) to the point of the apex of the heart. (5th left intercostal space 3.5 inches from middle line just below the nipple).

- **The superior border:** 5th I.C. space in the M.C.L. 5th rib right M.C.L. 7th rib in the right M.A.L. 9th rib in the scapular line.

- **The right surface of the liver** extends from the level of the 9th rib in the mid axillary line to the level of the 11th rib in the mid axillary line (or just below 10th rib).

- **Normally the left lobe** is one patient's hand breadth below xiphisternum.

Gall bladder:

The gall bladder has a fundus, a body and a neck. The fundus of the gall bladder comes in contact with the anterior abdominal wall close to the tip of the 9th costal cartilage. This corresponds to the upper end of the right linea semilunaris or to the point where the transpyloric plane cuts the right costal margin.

Spleen:

It underlies the left 9, 10, 11 ribs posteriorly its long axis is parallel to the long axis of the posterior part of the 10th rib.

Pancreas:

- It has a head, a neck, body and a tail. The neck lies in the transpyloric plane.
- From the neck, the head passes downwards and to the right while the body passes upwards and to the Left.

Kidneys:

(A) **Anterior surface markings:**

- As a rule, the right kidney is about 1.5 cm lower than the left so the upper end of the right kidney reaches the 11th intercostal space while the upper end of the left kidney ascends higher to reach the 11th rib itself.
- The hilum of each kidney 6 cm from the median plane.
Abdomen sheet

- Hilum of right kidney lies little below the transpyloric plane.
- Hilum of left kidney lies a little above transpyloric plane.
- The lower end of right kidney 5 cm above iliac crest.
- The left kidney 6 - 6.5 cm above the iliac crest.

(B) Posterior surface markings:
The kidney lies in an area bounded by 4 lines called morris's parallelogram.
- Two vertical lines about one inch, 3.5 inches from the median plane.
- Two horizontal lines at the level of T₁₁ and L₃.

Ureter: (25 – 30 cm long)
- Anterior, from point on the transpyloric plane about 6 cm from the median plane (hilum of the kid) to the pubic tubercle.

The different parts of viscera in different regions of the abdomen:
- The abdomen is divided into nine regions by the intersection of imaginary planes, two horizontal and two sagittal,
- The upper horizontal plane is the transpyloric plane (L₁).
- The lower horizontal plane is the intertubercular plane (L₅).
- The sagittal planes drawn vertically from the mid-inguinal points towards the mid – clavicular points (linea semilunaris)

(1) Epigastric region:
- Left lobe of the liver.
- Part of right lobe of the liver.
- Gall bladder.
- The 2 orifices of the stomach.
- Part of the stomach.
- 1st and 2nd parts of the duodenum.
- Pancreas.
- Inner end of the spleen.

(2) Right hypochondrium:
- Greater part of the right lobe of the liver.
- Right hepatic flexure of the colon.
- Part of the right kidney

(3) Left hypochondrium:
- Part of the stomach.
- Splenic flexure of the colon.
- The greater part of the spleen.
- Tail of the pancreas.
- Part of the Left kidney
(4) Umbilical region:
- Transverse colon.
- Coils of the jejunum and ileum.
- Third part of the duodenum.
- Greater omentum and mesentery.

(5) Right lumbar region:
- Ascending colon - Right kidney.

(6) Left lumbar region:
- Descending colon, part of Left kidney.

(7) Right iliac fossa:
- Caecum.
- Appendix and the end of the ileum.

(8) Left iliac fossa:
- Sigmoid colon - coils of the jejunum and ileum.

- Loin is apart of the back between costal margin (superiorly), iliac crest (inferiorly) and the lateral border of sacrospinalis.
- Groin is the area between lower limb and abdomen.
- Flank is the side of the body between ribs and iliac bone.
- The abdomen can be divided by vertical and horizontal planes through the umbilicus into four quadrants, left and right upper quadrants, left and right lower quadrant.

ABDOMINAL CASE

Present history:

Ask about the following symptoms in a chronological arranged manner and mention them whether positive or negative:

Symptoms:

1- Upper GIT symptoms:
- Appetite.
- Nausea.
- Haematemesis.
- Halitosis.
- Xerostomia.
- Dysphagia.
- Vomiting.
- Melena.
- Ptyalism.
- Epigastric pain.
- Belching
- Water brash

2- Lower GIT symptoms:
- Diarrhea.
- Constipation.
- Flatulence.
- Dysentery.
- Bleeding per rectum.

3- Other symptoms:
- Jaundice
- Hiccough
- Dyspepsia (it can be considered as an upper GIT symptom)
- Acoria
- Genitourinary symptoms.
- Parorexia
HOW CAN WE ASK ABOUT EACH SYMPTOM?

(1) Pain:
Ask the same questions as before + the relation of pain to meals and to vomiting.

Types of pain:
1- Colicky pain:
   - Renal or ureteric colic.
   - Biliary colic.
   - Intestinal colic.
   - Uterine colic.

2- Throbbing pain:
   - Inflammation with pus under tension.

3- Sawing pain:
   - Rheumatic disease of bones and joints.
   - This may affect the thoracic cage → upper abdominal pain.

4- Burning pain:
   - Reflux oesophagitis.
   - Peptic ulcer.

5- Dull aching pain:
   - Kidney swelling.
   - Splenomegaly or hepatomegaly.

6- Constricting pain (Tightness or compressing):
   - Ischaemic heart disease. (It may radiates to epigastrium)

7- Heaviness or dragging pain:
   - Splenomegaly or hepatomegaly.

8- Stitching or pricking:
   - Pleurisy. (It may radiates to upper abdomen)

(2) Appetite:
Ask if it is:
- Normal.
- Decreased.
- Increased
- Ask about weight gain or weight loss.
- Perverted appetite (parorexia): desire for unusual food, this may occur during pregnancy and in cases of Ankylostoma.

Causes of increased appetite with loss of weight:
- a- Diabetes mellitus.
- b- Thyrotoxicosis.
- c- Parasitic infestation.
- d- Malabsorption syndrome.
Loss of appetite (anorexia):
   a- Anorexia nervosa.   b- Depression.
   c- Viral hepatitis.    d- Uremia.
   e- Gastritis.      f- Malignancy

3. Nausea and vomiting:
   ⊗ Frequency of vomiting/day.
   ⊗ Relation to meals.
   ⊗ Is vomiting heralded by nausea or does it occur without warning.
   ⊗ Does it relieve the pain or not.
   ⊗ Amount (huge in pyloric obstruction).
   ⊗ Color e.g coffee grounds in as in haematemesis, yellow colour indicates bile in severe vomiting.
   ⊗ Odour e.g. foul smell in ulcerated cancer stomach.

The combination of nausea and vomiting usually suggests an upper gastrointestinal disorder, but this may be a prominent feature of non alimentary disorders. In intracranial tumours vomiting usually occur without warning.

4. Haematemesis and melena:
   ⊗ Haematemesis = vomiting of blood. (Ask about color, amount, number of attacks and occurrence of hypotension or shock)
   ⊗ Melena = Passage of dark tarry soft offensive stool.
   - Spurious melena due to ingestion of blood of epistaxis or haemoptysis.

Important causes:
   a- Peptic ulcer.
   b- Esophageal varices.
   c- Acute gastritis especially caused by drugs as aspirin - cortisone.
   d- Cancer stomach, cancer oesophagus.
   e- Blood diseases as leukemia - purpura.

5. Diarrhea:
   Too frequent or too loose stool or both.
   a- Frequency / day.
   b- Relation to meal (secretory diarrhea occurs even with fasting, see later).
   c- If associated with pus, mucous, blood or tenesmus = dysentery.
      Tenesmus means sense of incomplete evacuation.
   d- Colour of stool e.g.: bloody as in ulcerative colitis, pale as in steatorrhoea, tarry (melaena)
   e- If the stool is of large volume and not excessively frequent suggesting small bowel disease.
Abdomen sheet

f- If the stool is of small volume and excessively frequent suggesting large bowel disease.
g- Is the diarrhea is acute or chronic or intermittent.

(6) Constipation:

Definition:
Passage of too hard stool or evacuation less often than every other day.

Ask about:

a- What is the usual habit. (Has constipation been life long or of recent onset?)
b- If alternating with diarrhea or not e.g.: chronic dysentery.
c- Absolute constipation i.e.: no feces or flatus e.g.: intestinal obstruction.
d- Is there is associated abdominal pain or anal pain or rectal bleeding.
e- Has the shape of the stool changed e.g.: (become pellet-like) in spastic colon.
f- Drug history.

(7) Jaundice:

1. Age

- Young age:
  - Viral hepatitis.
  - Haemolytic jaundice.

- Old age:
  - Obstruction:
    - Stone.
    - Cancer head of pancreas.

2. Sex:

- Females are more liable to gall stones leading to obstructive jaundice.

| Female: fourty - fertile - fatty - filthy. |

3. Color of sclera. e.g (Lemon yellow in hemolytic, olive green in obstructive)

4. Color of urine. e.g (Dark and frothy in obstructive), no discoloration of urine (acholuric jaundice) in hemolytic jaundice.

5. Color of stool. e.g (Dark in hemolytic, clay in obstructive)

6. Abdominal Pain: In right hypochondrium

- Dull ache: Hepatitis
- Colicky: Stone.
- Colicky + fever + rigors (Ascending cholangitis).

7. Itching or pruritis: With obstructive jaundice.

8. Bleeding tendency: Due to lack of vitamin K absorption in cases of obstructive jaundice or hepatocellular jaundice.

9. History of:

Drug or alcohol intake, blood transfusion - contact with a case of viral hepatitis, skin tattooing. Ask about family history (spherocytosis).
10. In case of viral hepatitis ask about:
- Fever.
- Anorexia - nausea - vomiting.
- Pain in right hypochondrium.

(8) Dyspepsia and flatulence:
Feeling of abdominal discomfort or indigestion together with distention following meals, it is usually marked in gall bladder and pancreatic diseases especially with fatty meals.

(9) Heart burns:
(Pyrosis)
Sensation of pain behind the lower end of the sternum, it is due to reflux oesophagitis.

Causes:
1- Associated with Peptic ulcer or gastritis.
2- Hiatus hernia.
3- Hyper acidity.
4- Smoking and excessive coffee intake.
5- Nitrates, anticholinergic therapy or calcium channel blockers.

(10) Edema of lower limb & ascites:
Causes:
1- Liver cirrhosis.
2- Mal absorption syndrome.
3- Nephrotic syndrome.
4- Malnutrition.
5- Protein in losing enteropathy.
6- Bilharzial cor pulmonale.

(11) Dysphagia:
(Ask about)
Onset, course, duration & type of food either fluids or solids, painless or painful. Also ask about the level at which food appear to stick (see later).

(12) Genitourinary symptoms (see nephrology)
- Dysuria: pain of discomfort during the act of micturation.
- Strangury: severe suprapubic pain associated with inability to pass urine with acute bladder neck obstruction e.g.: stone or blood clot.
- Frequency: passing urine more often than usual.
- Urgency: a sudden need to pass urine.
- Polyuria: passing a larger volume of urine than normal (> 3L/D).
- Oliguria: passing a smaller volume of urine than normal. (< 400 ml/D)
- Nocturia: the need to pass urine during the night or the sleeping hours.
- Haematuria: blood in urine.
- Pneumaturia: Air bubbles in the urine.
- Stress or urge urinary incontinence, common in females.
- Hesitancy: Delay in initiating urine flow.
- Impaired force of urinary stream e.g.: due to prostatic hypertrophy.
- Impotence.

(13) Any other positive symptoms.

Past and family history as before with especial attention to Bilharziasis, viral hepatitis, abdominal surgery or blood transfusion in the past.

**IMPORTANT ABDOMINAL SYMPTOMS**

**GIT bleeding**

Classification of GIT bleeding

1) **Upper GIT bleeding** is due to lesions proximal to the ligament of Treitz (duodenojejunal junction) i.e. oesophagus, duodenum, and stomach. It is presented mainly by haematemesis, melena and sometimes by bleeding per rectum.

2) **Small bowel bleeding** from the ligament of Treitz to the ileocecal valve. It is presented mainly by melena and sometimes by bleeding per rectum.

3) **Lower GIT bleeding** from lesions of the anorectum and colon. It is mainly presented by bleeding per rectum, but right colonic lesions may be presented by melena.

Causes of upper GIT bleeding:

2) Gastric: Gastric erosion – peptic ulcer – cancer stomach.
3) Duodenal ulcer.
4) Blood disease or vascular malformations.

Causes of lower GIT bleeding:

- Inflammatory bowel disease.
- Hemorrhoids.
- Angiodysplasia.
- Anal fissure.
- Cancer colon.
- Diverticulitis.
- Ischaemic colitis.

Presentations of GIT bleeding:

GIT bleeding presents by five ways i.e. hematemesis, melena, bleeding per rectum, occult blood loss (Anaemia) or shock.

**Haematemesis:**

Means vomiting of blood due to lesion above the ligament of Treitz (duodeno – jejunal junction), it is bright red or in the form of coffee grounds when blood has had an appreciable period of time in contact with gastric acid.
Melena:
Means presence of altered blood in stool giving it the soft tarry black coloration with offensive odour due to lesions from areas proximal to and including the caecum. Also lesions in the right hemi colon can produce melena so, melena usually means that the site of bleeding is proximal to the mid transverse colon. It is usually indicate upper GIT bleeding.

Bleeding per rectum: (Hematochezia)
- It is the passage of bright red blood from the anus.
- It is mainly due to lesions of the anorectum and colon (lower GIT) e.g. cancer colon, diverticulitis, hemorrhoids, anal fissure, inflammatory bowel disease, angiodysplasia and ischemic colitis. Sometimes it is due to upper GIT lesions or small bowel lesions with brisk bleeding and rapid transit time.

Clinical picture of GIT bleeding:
- Clinical picture of the cause.
- The presentation of bleeding depends on the site of lesion so, bleeding can be presented by haematemesis, melena. (50-100 ml blood needed to produce melena) or bleeding per rectum.
- Upper GIT bleeding may be presented by bleeding per rectum with brisk bleeding with rapid transit time.
- In massive bleeding the classical picture of shock will developed.
- Occult bleeding presented with anaemia.

D.D:
(1) Melena should be differentiated from black stool due to Iron or charcoal therapy?
Dilute the stool by water, in melena it is red and in others it is grayish.
(2) Haemoptysis from haematemesis (see chest sheet)

Investigations:
1 - Liver function tests  
2 - P.T, P.T.T.  
3 - Upper endoscopy.  
4 - Ba study and abdominal sonar.  
5 - Lower endoscopy.

* H & M may occur together with a sudden profuse bleeding.  
* Melena alone indicates that the bleeding is slower and less in amount.  
* Postural hypotension = loss of > 20% of the blood volume.  
* Faintness and sweating = severe bleeding.

How to detect occult blood in stool:
1) Guaiac test: Stool + ether + guaiac reagent → Blue color in + ve cases.  
2) Benzidine test: Less reliable as it gives + ve results with iron therapy.  
3) Isotopic method: RBCs labeled with Cr 51
Abdominal pain often heralds gastrointestinal disease and brings the patient to the attention of the internist or surgeon.

Mechanism and causes of abdominal pain:

1. Distension and stretching of the wall of a hollow organ e.g.: colonic distension.
2. Stretching of the capsule of a solid organ e.g. splenomegaly, hepatomegaly and kidney enlargement.
3. Spasm of viscous organ e.g.: intestinal colic, ureteric colic or biliary colic.
4. Mucosal inflammation e.g.: Gastritis, oesophagitis
5. Peritoneal irritation e.g.: peritonitis
6. Ischaemia e.g.: ischaemic colitis.

So abdominal pain may be visceral (Gut organs), parietal (parietal peritoneum) which is innervated by somatic nerves, referred pain e.g. gall bladder pain → right shoulder or it may be psychogenic.

Non-alimentary causes of abdominal pain:

1. Myocardial infarction.
2. Dissecting aortic aneurysm.
4. Pleurisy.
5. Herpes zoster.
6. Diabetic ketoacidosis.
7. Torsion testis/ovary.

D.D. OF EPIGASTRIC PAIN

1) Diseases of lower end of esophagus:
   - Hiatus hernia - oesophagitis - carcinoma.
2) Gastro - duodenal:
   - Peptic ulcer - gastritis -gastric carcinoma - pyloric stenosis.
3) Gall bladder:
   - Acute and chronic cholecystitis - gall stones.
4) Pancreas:
   - Acute and chronic pancreatitis – cancer pancreas.
5) Hepatic:
   - Congested liver - amebic hepatitis – viral hepatitis - carcinoma.
6) C.V.S.:
   - Angina - infarction - pericarditis - dissecting aneurysm.
7) Chest:
   Diaphragmatic pleurisy.

8) Miscellaneous:
   Herpes Zoster - fatty hernia of linea Alba - myositis - diabetic ketoacidosis.

**Analysis of some causes of abdominal pain:**

- **Peptic ulcer:**
  - Epigastric.
  - Nocturnal.
  - With stress, aspirin and smoking.

- **Biliary colic:**
  - Epigastric / right hypochondrium.
  - Colicky.

- **Renal colic:**
  - Loin.
  - Colicky.
  - May follow periods of dehydration.

- **Acute pancreatitis:**
  - Epigastrium / left hypochondrium.
  - Severe.

- **Colonic Pain:**
  - Colicky.
    - By the defecation or passing flatus.
    - Lower abdomen on either sides.

- **Appendicitis:**
  It is initially localized around the umbilicus (visceral pain) then it shifts to the right iliac fossa with peritoneal irritation (parietal or somatic pain).

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<thead>
<tr>
<th>Medical conditions which may mimic acute abdomen.</th>
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<tbody>
<tr>
<td>* Myocardial infarction</td>
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<td>* Pleurisy</td>
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<td>* Irritable bowel §</td>
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<tr>
<td>* Diabetic ketoacidosis, Addisonian crises</td>
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<td>* Henoch schonlein purpura</td>
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DIARRHEA

Definition:
➤ Passage of excessively liquid or excessively frequent stool.

(> 300 gm / 24 hrs.)
➤ It is mainly due to increased amount of fluid content of the stools.

Causes:
(A) Acute diarrhea:
1. Food as spices - alcohol.
2. Food poisoning.
3. Drugs e.g.: purgatives, antibiotics.
4. Food allergy.
5. Acute infection e.g.: Typhoid - cholera - bacillary dysentery - rota virus
6. Parasites e.g. Giardia - entamoeba - strongloides.

(B) Chronic diarrhea:
1. Irritable bowel syndrome.
2. Inflammatory bowel disease.
3. Malabsorption syndrome (enumerate its causes).
4. Thyrotoxicosis.
5. Autonomic neuropathy.
6. T.B. enteritis.
7. Drugs as Laxatives, digitalis.
8. Parasitic gut infections.
9. HIV.
10. Colorectal malignancy

Other classification:
(1) Osmotic diarrhea:
Less than 500 ml / day during fasting, it is related to meals.
Due to: Lactase deficiency - use of lactulose.

(2) Secretory diarrhea:
More than 500 ml / day during fasting, it can occur during fasting.
Due to: Carcinoid - Viral - Giardia - Cholera - Shigella - Salmonella & E. coli.

(3) Disturbed colonic motility:
Due to: Irritable bowel disease - Autonomic neuropathy & thyrotoxicosis.
Abdomen sheet

Investigations:
1. Cause, e.g.: thyrotoxicosis.
2. Stool culture and examination for parasites and fecal fat.
4. Colonoscopy, small intestinal biopsy.

Treatment:
(A) Correct any disturbances caused by diarrhea:
1. Fluid loss and dehydration.
2. Electrolyte depletion as hyponatraemia - hypokalaemia.
3. Malnutrition and vitamin deficiencies.

(B) Diet:
In acute diarrhea:
Food should be withheld at first.

Then allow small soft feeding.
Avoid spices - alcohol, raw vegetables - milk and its products.

In chronic diarrhea:
As acute diarrhea, also give fat soluble vitamin as A, D and K.

C - Antidiarrheal agents:
1. Pectin - kaolin compounds 30 cc T.d.s.
2. Diphenoxylate (lomotil) 2.5 mg T.d.s
3. Opiates: Better avoided in chronic diarrhea for fear of addiction.

D - Specific treatment according to the cause.

CONSTIPATION

Definition:
Passage of either too hard stool or evacuation less often than every other day.

Causes of constipation:
1. Decrease intake, pregnancy.
2. Disturbed conditioned reflex e.g.: (unfamiliar places).
3. Dietary fibers deficiency.
4. Drug therapy e.g.: opiates, anticholinergic, calcium carbonate, Iron.
5. Irritable bowel disease.
7. Intestinal obstruction.
8. Metabolic disorders e.g.: hypothyroidism, DM, uraemia, hyperkalemia and hypokalemia.
10. Major depression.
11. Anal pain conditions.
Treatment:
A) Re-establishment of regular evacuation:
Trial for defecation at a regular period after meal for a bowel movement, even when
the urge to defecate is not present especially 20 - 30 minutes after breakfast( peak of
gastrocolic reflex)
B) Diet:
1- Adequate volume with adequate bulk or residue, e.g.: vegetables, bran.
2- Adequate fluids, a glass of hot water taken 1/2 hour before breakfast got a mild
laxative effect.
C) Exercise: Is important as the tone of the external abdominal muscles is important
D) Laxatives
E) Prokinetics
F) Enemas:
Specially in chronic constipation either with warm tap water or saline enema.
F) Specific treatment of the cause.

VOMITING

Definition:
It is forceful oral expulsion of the gastric contents.
Mechanism:
A reflex composed of an afferent through the vagus to the vomiting center in the
medulla and efferent through the vagus leading to:
 a- Closure of the glottis.
b- Contraction of diaphragm.
c- Closure of the pylorus and relaxation of the stomach and the cardiac orifice.
d- Contraction of the abdominal muscles.

Causes:
I. Gastric:
• Acute gastritis – Peptic ulcer - Pyloric obstruction - Cancer stomach.
II. Intestinal:
• Obstruction – Intestinal colic - Appendicitis.
III. Renal:
• Pyelonephritis – Renal colic – Renal Failure
IV. Gynecological:
• Pregnancy - Dysmenorrhea - Salpingitis - Torsion of ovarian cyst.
V. Hepatic and biliary:
• Acute cholecystitis – Gall stones - Hepatitis - Acute pancreatitis.
VI. Cardiac:
• Myocardial infarction - Congestive heart failure - Digitalis toxicity.

VII. Ocular:
• Glaucoma.

VIII. Metabolic:
• Uremia - Diabetic acidosis - Addison's disease - Hyperparathyroidism.

IX. Miscellaneous:
• Neurosis, Anorexia nervosa.
• Increase I.C.T., Migraine.
• Drugs as apomorphine - Digitalis.
• Disease of vestibular apparatus e.g.: vertigo.

Complication of severe vomiting:
1. Dehydration.
2. Alkalosis - tetany.
3. Subconjunctival or subarachnoid haemorrhage.
4. Pulmonary aspiration.

Treatment:
1. Cause
2. Antiemetic.
3. Severe cases: fluid therapy.

Non alimentary causes of vomiting:
1. Drugs.
2. Neurological.
3. Labyrinthine disorders.
4. Psychological.
5. Metabolic.
7. Renal

DYSPHAGIA

Definition: Difficulty in swallowing with a sensation of sticking or obstruction of the passage of food through the mouth, pharynx or the oesophagus.

Causes:
Oropharyngeal causes:
- Stomatitis.
- Oral malignancy.
- Pharyngitis.
- Myasthenia gravis.
- Pharyngeal malignancy.
- Aphthous ulcer.
- Tonsillitis.
- Bulbar and pseudo bulbar palsy.
- Pharyngeal diverticulum.
- Pharyngeal pouch.

In cases of painful inflammatory conditions it is better to be called odynophagia.
**Oesophageal causes:**
(>Food sticking during swallowing is an important symptom of oesophageal disease>

- **Causes in the wall:**
  - Globus hystericus.
  - Oesophageal spasm & achalasia.
  - Myopathy.
  - Strictures.

- **Causes in the lumen:**
  - Atresia.
  - Carcinoma.
  - Webs.
  - Foreign body.

- **Mediastinal causes:**
  - Goitre & bronchogenic carcinoma.
  - Dilated left atrium.
  - Mediastinal L.N. ++.

**Examples**

- **Oesophageal achalasia:**
  - Female, no marked weight loss.
  - Long duration.
  - Intermittent.
  - More to fluids.

- **Cancer oesophagus:**
  - Old age, male.
  - Short duration.
  - Progressive.
  - More to solids.

---

**ODYNOPHAGIA**

It is a painful swallowing which is characteristic of inflammatory disorders.

- Tonsillitis.
- Pharyngitis.
- Oesophagitis.

- Dysphagia localized to the level of the cricoid cartilage may result from tumour, stricture or pharyngeal pouch.
- Dysphagia localized to the level of the lower sternum suggests disease of the lower oesophagus, e.g. tumour, achalasia or peptic oesophagitis with or without stricture.

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**DYSPEPSIA**

**Definition:** An upper abdominal discomfort, nausea, heartburn or distension in relation to meals, it may be described as a sense of indigestion. It is usually originate from the upper GIT.

**Causes of Dyspepsia:**

1. Upper GIT disorders e.g. peptic ulcer diseases, acute gastritis, motility disorders e.g. oesophageal spasm and functional dyspepsia (non-ulcer dyspepsia and irritable bowel syndrome).
Non ulcer dyspepsia is in the form of early satiety, nausea and fullness, it may be associated with H pylori infection.

HICCUP

Definition:
It is a sharp inspiratory sound caused by contractions of the inspiratory muscles terminated abruptly by closure of the glottis.

Causes:
1. Stimulation of the diaphragm by:
   (1) Inflammation near by diaphragm
       Pneumonia.
       Oesophagitis.
       Subphrenic abscess.
       Pancreatitis.
   (2) Gastric distension.
   (3) Cardiac diseases
       Myocardial infarction.
       Pericarditis.

2. Metabolic e.g. Uraemia.
4. Idiopathic.

Treatment:
1. Simple home remedies:
   ☆ By diverting the patient's attention by distracting conversation, fright, painful or unpleasant stimuli.
   ☆ Ask the patient to perform the following:
      * Breath holding.
      * Inhaling fumes.
      * Sipping ice.
      * Dry cane sugar.
2. Medical treatment:

- Diazepam, chlorpromazine & haloperidol.
- Stimulation of nasopharynx by a catheter.
- Lignocaine orally.
- Metoclopramide or domperidone.
- CO₂ inhalation (rebreath into a paper bag).
- Antacid.

3. Surgery:

- Phrenicotomy !?

MISCELLANEOUS SYMPTOMS

Retching:

Rhythmic contraction of the stomach, diaphragm and abdominal muscles with contraction of the cardia, nothing is expelled to the mouth, it frequently precedes or accompanies vomiting e.g in cases of gastritis.

Regurgitation:

Effortless regurge of the gastric contents into the mouth without nausea or abdominal diaphragmatic muscular contraction in cases of achalasia & hiatus hernia.

Water brash:

It is a reflex salivary hypersecretion which occur in response to GIT lesion with filling of the mouth with saliva. It may occur in D.U.

Anorexia:

It is loss of appetite or lack of desire to eat. It must be differentiated from fear of eating (sitophobia) because of painful condition in mouth or gut. e.g. (gastric ulcer)

Parorexia (an appetite for unusual food):

- Pagophagia (ingestion of ice)
- Geophagia (Eating earth)

These may occur in neurosis and pregnancy.

Acoria:

Excessive ingestion of food due to loss of the sensation of satiety.

Nausea:

Sense of thickness with imminent desire to vomit frequently associated with salivation and sweating e.g in pregnancy, peptic ulcer, uremia and neurosis.

Belching:

Excessive eructation of gases as in aerophagy (air swallowing) in neurotic persons.
Wind (flatulence):
Repeted belching, excessive or offensive rectal flatus, abdominal distension may all be called (wind). The normal volume of flatus per rectum 200 - 2000 ml / Day. It consists of mixture of swallowed air (aerophagia) with gases derived from colonic bacterial fermentation of poorly absorbed carbohydrates. Excessive flatus occurs in lactase deficiency and malabsorption. Absence of flatus is a feature of intestinal obstruction.

Ptyalism (Excessive salivation):
Causes:
- Neurosis
- Reflex due to disease of stomach or duodenum.
- Inflammatory conditions in the mouth.
- Bulbar paralysis.
- False Ptyalism may occur in facial paralysis due to difficulty of swallowing.

Xerostomia:
Causes:
- Mouth breathing.
- Dehydration.
- Anticholinergic drugs.
- Sjogren's $.

Halitosis (Bad odour of breath):
Causes:
- Oropharyngal conditions. e.g.: Dental caries & tonsillitis.
- E.N.T. e.g. sinusitis.
- Pulmonary e.g. Suppurative lung disease.
- Certain types of foods.
- Dyspepsia.

N.B.: Special odours:
- Acetone odour : Diabetic ketoacidosis.
- Ammonical odour : Chronic renal failure
- Foetor hepaticus : liver cell failure.

CAUSES OF SPLENOMEGALY

I. Bacterial infection:
A- Acute:

B- Chronic:
- $ - Miliary T.B. - HIV.
Abdomensheet

II. Parasitic:
Malaria - kala azar - Bilharziasis.

III. Blood diseases:
Leukemia - hemolytic anemia - I.T.P - Polycythaemia rubra Vera and Lymphoma.

IV. Metabolic:

VI. Miscellaneous:
Felty’s syndrome - still’s disease - Sarcoidosis.

Causes of just palpable spleen:
- Infective endocarditis.
- Infecious mononucleosis.
- Typhoid.
- Viral hepatitis.
- Brucellosis.

Causes of moderate splenomegaly:
- Chronic hemolytic anaemia.
- Lymphoma.
- Portal hypertension.
- Leukemia.

Cause of huge spleen:
- Chronic malaria.
- Chronic myeloid leukemia.
- Gaucher’s disease & amyloidosis.
- Mylofibrosis.
- Kala - azar.

CAUSES OF HEPATOMEGALY

(A) Tender liver: (Tender hepatomegally)

(B) Minimal or no tenderness:
1- Infections:
   - T.B. - Sarcoidosis - Brucellosis - Weil’s disease – Infecious mononucleosis
2- Parasitic:
   - Malaria - kala azar - Hydatid disease – Bilharziasis.
3- Cirrhosis with its types. (Early cirrhosis)
4- Tumor (primary or secondary).
5- Metabolic:
Fatty liver - Amyloidosis - Gaucher's Disease - Neimann pick Disease.

6- Blood diseases:
Leukemia - haemolytic anemia - Lymphoma.

Causes of nodular liver:
1- Bilharzial (with coarse nodularity).
2- Post necrotic cirrhosis (post hepatitis).
3- Malignancy.
4- Gumma (Heparlobatum).
5- Hydatid disease.

GENERAL EXAMINATION

1. General condition.
2. Level of consciousness for hepatic encephalopathy.
3. Decubitus & fascies.
4. Complexion:
   - Pallor → GIT bleeding & hypersplenism.
   - Cyanosis → Opening of intrapulmonary shunt in liver cell failure.
5. Vital signs:
   - Pulse: Hyperdynamic circulation with big pulse volume in L.C.F.
     H.R. ↓ → obstructive jaundice.
     ↓ Volume, ↑ rate in hypovolaemia (bleeding).
   - Blood pressure: ↓ In advanced L.C.F.
     Shock in cases of G.I.T. bleeding.
   - Temperature: ↑ In viral hepatitis.
     ↑ In spontaneous bacterial peritonitis.
6. U.L.:
   - Clubbing → Cirrhosis especially biliary cirrhosis.
   - Ulcerative colitis.
   - Bilharzial polypi.
   - Mal-absorption $( coeliac )$
   - Flabbing tremors → L.C.F.
   - Palmar erythema → Normal person, L.C.F., alcohol.
7. L.L.:
   - Edema.
   - Ascites without edema suspect local cause as T.B. peritonitis or malignant ascites
8. Head & Neck:

- Wasting of temporalis in chronic liver disease.
- Thyroid swelling with Lupoid hepatitis.

- **Spider naevi (Arterial spider)**: they are present in the distribution of S.V.C.
  An arterial spider consists of a central arteriole, radiating from which numerous small vessels resembling a spider's legs. They may be present in 1% of population, if > 5 or increasing in size and number it is a pathological. It is present in chronic L.C.F. or fulminant L.C.F. D.D. are insect bite, Purpuric eruption, Campell de Morgan spot or venous stare. Common sites of spider naevi are the neck, the face, forearms, chest wall above nipple line and the dorsum of the hand. They are rarely found in the mucous membrane of the nose, mouth and pharynx.

9. Signs of Hypovitaminosis:

- **B1** → Neuropathy.
- **B2** → Angular stomatitis, vascular cornea, Sulphur granules.
- **B6** → Neuropathy.
- **Nicotinic acid** → Pellagric rash over greater trochanter.
- **A** → Bitot's spot. Night blindness.
- **B12, Folic acid** → Pallor due to megaloblastic anaemia.
- **K** → Ecchymosis.

10. Lymph nodes & skin:

**Q. Skin manifestations of chronic liver disease**

- White nail.
- Spider naevi.
- Itching marks.
- Jaundice.
- Palmer erythema.
- Paper mony skin.
- Purpuric eruption.
- Hyperpigmentation.

**LOCAL EXAMINATION**

A. Inspection:

1- General form and contour.
3- Subcostal angle.
5- Divarication of recti.
7- Hernial orifices.
9- Dilated veins.
11- Striae & pigmentations.
2- Movement.
4- Epigastric pulsation.
6- Umbilicus.
8- Hair distribution.
10- Visible peristalsis.
12- Scars of operation.
A) Inspection:

1- General form and contour:

A- Bulging:

1. Localized → organ swelling (organomegaly) e.g.:
   - Bulge of lower half e.g. ovarian tumour, distended bladder or pregnancy.
   - Bulge of upper half e.g. pancreatic cyst, or gastric dilatation.
   - Bulge of lower third e.g. ovarian tumour, distended bladder uterine fibroid or pregnancy.

2. Diffuse → 6 F's: fat, fluid, flatus, foetus, feces, and fatal growth.
   - In ascites → bulging more in the flanks, while in other causes the bulging is more antero-posterior.

b- Retraction (sunken abdomen or scaphoid abdomen): as in dehydration & also in T.B. peritonitis, starvation or wasting disease.

2- Movement:

Normally the abdomen moves freely with respiration (bulge on inspiration and retraction on expiration). In cases of:

a- Peritonitis → no movement at all.

b- In paralysis of diaphragm:
   1. Unilateral paralysis → one side moves only. (Se-saw movement)
   2. Bilateral paralysis → paradoxical movement i.e. opposite the normal.

3- Subcostal angle:

Normally it is about 90°. It is wide in upper abdominal swelling, the commonest is hepatosplenomegaly. Also it is wide in ascites

4- Epigastric pulsation:

Right V++ in Bilharzial cor pulmonale, hepatic pulsation in T.I and alcoholic liver disease?

5- Divarication of recti:

Ask the patient to raise his head from the pillow, this cause the abdominal recti to contract and reveal the separation of the pair. It denotes weak abdominal muscles e.g.: Congenital, Multiparous, or any other conditions producing increase of the intra abdominal pressure or loss of muscular tone.

6- Umbilicus:

Normally it is mid way between xiphisternum and symphysis pubis, and slightly inverted.

a) Shifting of the umbilicus downwards = upper abdominal swelling e.g.: Hepatosplenomegaly, or ascites.
b) Shifting upwards in lower abdominal swelling e.g.: Ovarian cyst, full bladder, and uterine masses (pelvi abdominal swellings).

c) Everted umbilicus = increased intra abdominal pressure e.g. ascites.

d) Everted nodular umbilicus = secondaries from abdominal malignancy (sister Joseph's nodule).

e) Bluish umbilicus (Cullen's sign) due to hemoperitoneum.

f) Dilated veins radiating from the umbilicus (caput medusae) in cases of portal hypertension. It is due to patent umbilical vein connecting the left branch of the portal vein to veins around the umbilicus. Normally the umbilical vein is obliterated forming the ligamentum teres.

g) Inflammatory reddish swelling of the umbilicus occur with inflamed Meckel’s diverticulum.

h) Insipissated desquamated epithelium and debris in the umbilicus (omphalolith)

i) Discharge e.g urine (patent urachus), intestinal contents (patent vitello-intestinal duct, pus (pilonidal sinus), stool (faecal fistula), blood during menstruation (endometrioma).

j) Umbilical granuloma or adenoma.

- Hernial orifices:

Hernias denote weak abdominal muscles associated with increased intra- abdominal pressure. The patient examined while standing so abdominal contents bulge through the hernial ring. Let the patient to cough. Many hernias are encountered during routine examination of the abdomen. Ex. incisional hernia - Epigastric hernia - umbilical hernia -inguinal hernia - femoral hernia.

(8) Hair distribution:

In male the hair distribution reaching a level up to the umbilicus. In females it ends on a horizontal line. The male distribution change to female distribution in liver cirrhosis.

(9) Dilated veins:

* Normally the blood flow in the lower 1/2 of the abdomen is from above downwards and in the upper 1/2 it is from below upwards (away from umbilicus).

* Normally no apparent veins are seen in the abdominal wall, except in thin persons, where there may be small venules in the subcostal region which are not distended so they are not significant.

* The pathological veins are dilated and tortuous (distended or engorged).

  a) In I.V.C. obstruction the engorged veins draining from below upward.(in both flanks)

  b) In S.V.C. obstruction the engorged veins draining from above downwards.

    (on the chest wall and upper abdomen)
c. In portal hypertension: veins radiate from the umbilicus, draining away from umbilicus.

⇒ How to differentiate portal H from I.V.C obstruction:

1. **Distribution**: in portal obstruction the veins are around the umbilicus or more commonly in the epigastrium, while in caval obstruction the veins are situated laterally in both flanks. In I.V.C obstruction colateralls may appear on the back.

2. **Direction**: in portal obstruction the blood flow is away from the umbilicus, while in caval obstruction it is towards the umbilicus.

3. **Oral glucose test (Old test)**: glucose absorbed from the intestine to the abdominal veins i.e. portal venous blood, which now has higher glucose concentration than that of systemic veins. In portal hypertension the engorged veins have higher glucose than that of the anticubital vein. In caval obstruction, no difference.

*Caput medusae may need infrared photography to be visualized more clearly. Tense ascites may cause functional I.V.C obstruction and cause difficulty in interpretations.*

In cases of IVC obstruction, and portal hypertension the visible collateral veins are usually tortuous dilated superficial epigastric veins.

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10. **Peristaltic waves:**

1. Peristaltic waves in the region of the epigastrium, running from left to right denote pyloric obstruction.

2. Peristaltic waves around the umbilicus in a ladder pattern due to ileocaecal or small intestinal obstruction.

11. **Striae:**

Striae are stretch marks usually present on the flanks or lateral aspects of the abdomen. They are white or pink linear marks on the abdominal skin produced by gross stretching of the skin with rupture of the subcutaneous elastic fibres and indicate a recent change in size of the abdomen e.g. pregnancy, ascites, wasting diseases, weight gain or loss. Wide purple striae are characteristic of cushing's disease or excessive steroid therapy.

12. **Scare of operation:**

- Scare of splenectomy.
- Scare of nephrectomy.
- Scare of cholecystectomy.

What are the indications!?

B) **Palpation:** You must ensure the following:

- Patient lie flat on bed.
- Patient flexing his legs.
- Patient opens his mouth and breathes quietly.
- It should be done with warm gentle hands.
- Gentle pressure is at first exerted (superficial palpation) and gradually increasing it (as this can easily make the abdomen rigid).
- Start away from the area of pain or tenderness.
1) Superficial palpation:

For any:
- Tenderness
- Abdominal guarding/rigidity
- Superficial masses

Abdominal guarding/rigidity:
- It is a defence mechanism over an inflamed organ, viscus or peritoneum.
- If it is localized with absence of respiratory motion in those areas of abdominal wall plus rebound tenderness, indicates a focal area of peritonitis.
- If it is generalized (board like) i.e. never to be relaxed, indicates generalized peritonitis.
- Guarding/rigidity not due to peritonitis e.g. tender organ or viscus characterized by variability in muscular contraction and relaxation during examination with no rebound tenderness, this is also occurs in nervous patients (generalized nervous guarding/rigidity).

2) Deep palpation:

For organ enlargement, palpate the abdomen more deeply with the flat of the hand.

If any tumor or organ is felt you must examine:

a- If it is intra or extra abdominal by asking the patient to raise his head and trunk without using his arms. If the mass is in the abdominal wall it tends to become more prominent, on the other hand it tends to disappear if it is intra-abdominal.

b- Describe any swelling i.e.: site-size-surface-shape-edge-consistency-movement with respiration.

- Bimanual palpation: obtained by using the 2 hands, it is useful in palpating the kidneys, spleen, liver (Expansile pulsation).
- Dipping method: This is used in presence of massive ascites to detect hepatic or splenic swelling. Press suddenly with the tips of the fingers while the hand placed gently on the abdomen. The sudden displacement of liquid gives a tapping sensation over the surface of the liver or spleen.

N.B.: In any type of palpation start at a point away from the site of any pain.

Structures normally palpable:
1- Contracted muscles of the abdominal wall may be mistaken for an intraperitoneal mass.
2- Caecum and descending colon, when it is full of gases or fluids.
3- Abdominal aorta.
4- Liver edge may be felt 1 - 3 cm. below the costal margin on deep inspiration.
5- Lower pole of right kidney especially in females with lax abdominal wall.
METHODS OF PALPATION of DIFFERENT ORGANS:

**Palpation of Liver:**

1. The hand should be placed flat with the fingers pointing upward and positioned so that the sensing fingers (index & middle) are lateral to the rectus muscle.
2. The hand should be firmly pressed inwards & upwards, and it should be kept steady while the patient takes a deep breath through the mouth.
3. At the height of inspiration the inward pressure on the front hand is released while the upward pressure is maintained. With this movements the tips of the fingers should slip over the edge of a palpable liver.
4. Trace the surface of a palpable liver across the abdomen for irregularities using the fingertips.

**Two common mistakes should be avoided:**

1. One is to feel for the liver with the hand placed horizontally; in this position the palm of the hand may press backwards the edge which is desired to feel.
2. The second error is to start feeling too high up.

**Comment on (Left lobe – Right lobe – bimanual examination)**

- Size → ↑↑ or ↓↓.
- Pulsations → TI (Systolic expansile).
- Consistency → Firm, soft, hard.
- Tenderness → +ve or -ve.
- Border → Sharp or rounded.
- Surface → Smooth or nodular.

**Palpation of spleen:**

- Bimanual examination

The left hand is placed over the left lower most rib cage posterolaterally and the right hand is placed flat with the fingers at right angle to the left costal margin, starting from the right iliac fossa, pressing inwards & upwards like the liver examination. Repeat this process along the entire rib margin as the position of the enlarging splenic tip is variable.

**Examples**

- In liver cirrhosis, the liver is firm, nodular with sharp border.
- In hepatitis or congested liver, the liver is enlarged, soft and tender.
- In leukaemia or lymphoma the liver is firm, smooth with rounded border.
Abdomen sheet

- If the spleen is not palpable, position the patient in the right lateral position with flexion of left knee and hip.
- **Splenic percussion sign**: We percuss the last intercostal space anterior axillary line normally tympanic then, ask the patient to hold inspiration, if the tympany becomes dull = slightly enlarged spleen.
- **Hooking method**

![Diagram of palpation methods](image)

(A) Palpation of the spleen. (B) Alternative method for palpation of the spleen (Hooking).

**Kidney palpation:**

- **Bimanual examination:**
  - Place the left hand posteriorly below the lower rib cage in the loin and feel the renal angle and the right hand over the lumbar region.
  - Push the two hands together firmly but gently as the patient breathes out.
  - Feel the lower pole moving down between the hands as the patient breathes in deeply.
  - Push the kidney back and forwards between the two hands this is known as ballotting to differentiate it from the spleen.
  - Assess the size, surface and consistency of a palpable kidney.
  - Examine the left kidney from either side (see later).

<table>
<thead>
<tr>
<th>Splenic swelling</th>
<th>Renal Swelling</th>
</tr>
</thead>
<tbody>
<tr>
<td>Can cross mid line.</td>
<td>Can not cross mid line except in cases of horseshoe kidney.</td>
</tr>
<tr>
<td>Moves freely with respiration downward &amp; medially.</td>
<td>Limited mobility with respiration (up &amp; down).</td>
</tr>
<tr>
<td>There is a notch.</td>
<td>There is no notch.</td>
</tr>
<tr>
<td>Does not fill the renal angle.</td>
<td>Fills the angle.</td>
</tr>
<tr>
<td>You can insinuate your hand between it &amp; the costal margin, but you can't reach to the upper pole.</td>
<td>You can, and also you can reach to the upper pole.</td>
</tr>
<tr>
<td>No post ballottement.</td>
<td>post ballottement is positive.</td>
</tr>
<tr>
<td>Dull, its dullness continous with splenic dullness.</td>
<td>There is a band of resonance overlying the renal swelling.</td>
</tr>
</tbody>
</table>
Abdomen Sheet

Palpation of the left kidney. (A) From the same side and (B) from the opposite side.

**Palpation of the gall bladder:**
- We palpate in the same method of liver but:
  - Normally gall bladder is not palpable.
  - Enlarged gall bladder ch. ch. by:
    - Globular swelling with well defined border.
    - Lateral to the edge of rectus near the tip of 9th costal cartilage.
    - Directed down and medially.
    - Dull, and its dullness continuous with hepatic dullness.
    - The surface is smooth and the consistency is cystic.

**Murphy's kidney punch**
- While the patient is sitting, examine for tenderness in the renal angle with the thumb with comparing both sides.
- It may allow distinction between renal tenderness or tender abdominal wall or muscles of the back.

**Murphy's sign:**
Place the examining fingers over the gall bladder area and ask the patient to take a deep breath. Inspiration may be sharply arrested with tensing of the abdominal muscles because of a sudden accentuation of pain suggesting acute cholecystitis.

**Palpation of urinary bladder:**
- Normally it is not palpable, if there is retention of urine it is felt as smooth, firm, regular oval swelling in the suprapubic region. Its upper border may reach the umbilicus, but its lower border can't be felt (pelvi-abdominal mass). In women, it must be differentiated from gravid uterus, fibroid or ovarian cyst (usually present on left or right).

**Percussion of the abdomen:**
- Percuss from resonance to dullness.
- Place the finger used for percussion on the abdomen parallel to the direction to the anticipated note change.
- Percuss lightly for the liver and more firmly for deeper structures e.g. upper border of the liver or the urinary bladder.
- The principal value of percussion is to distinguish between different swelling: gases, ascites, cystic or solid swelling. Also it is important for the spleen and urinary bladder.
Abdomen sheet

**Ascites:**
1) Shifting dullness for moderate ascites.
2) Fluid thrill in tense ascites.
3) Knee elbow position in mild ascites.
4) Puddle sign for minimal ascites. In patient with knee elbow position, the examiner places his diaphragm over the most dependent part of the abdomen and starts flicking a finger over a flank, then gradually moves the diaphragm over the opposite flank, a positive sign is a sudden increase in intensity of the sound just as the diaphragm moves beyond the edge of the peritoneal fluid.

**Percussion of spleen:**  (Splenic percussion sign) see before.

**Percussion of liver:**
- Lower border: Light percussion.
- Upper border: Heavy percussion.

**Causes of reduced hepatic dullness:**
- Pulmonary hyperinflation.
- Shrunken liver.
- Air under diaphragm.

**Fist percussion**
Place the ball of one hand in the costovertebral angle (for kidney tenderness) or in the right lower intercostal spaces and ribs (for liver tenderness e.g amoebic liver abscess) and strike it with the ulnar surface of the fist of the other hand.

**Percussion of bladder:** with retention of urine (suprapubic dullness)

**Percussion of any other swelling:** this is according to its site.

**Shifting dullness:**
- Examine the patient supine and percuss from the centre of the abdomen into the flank until a dull note is obtained.
- Mark the level or keep the finger in place as the patient rolls on to the other side.
- Pause for at least 10 seconds. Ascites is suggested if the note becomes resonant; the shifting here must be bilateral.

**Fluid thrill:**
- Place a detecting hand on the patient's flank, flick the skin of the abdominal wall over the other flank using thumb or fore finger with the patient's hand placed on the abdomen along the midline sagittal plane to prevent any possible thrill transmitted via the abdominal wall.

D) Auscultation:

1. **Intestinal sounds** or borborygmi:
   (Gurgling sounds heard every 5 - 10 seconds)
   - They occur more frequently after meals.
   - Increased in mechanical intestinal obstruction, carcinoid syndrome and malabsorption.
   - Absent in paralytic ileus.

   **Auscultate for peristalsis bowel sounds for at least 3 minutes before deciding that they are absent**

2. **Venous hum** too and fro continuous murmur heard in the epigastrium or in the region of umbilical process or umbilicus in cases of portal hypertension due to refluxing of umbilical veins (cruevillier – Baumgarten murmur).
   - The association of dilated abdominal wall veins and a loud abdominal venous murmur at the umbilicus with normal liver is termed cruevillier – Baumgarten syndrome. It indicates intrahepatic portal hypertension (cirrhosis).

3. **Fibrin rub** over the liver or spleen (perihepatitis or perisplenitis) i.e inflamed visceral surfaces of the liver or spleen.
Abdomen sheet

4- **Harsh systolic murmur** over the flanks in renal artery stenosis.

5- **Succussion splash**: A splashing sound over the stomach in case of pyloric obstruction, to elicit a Succussion splash, place the hands over the lower ribs and shake the patient quickly and rhythmically from side to side. It is a sound like shaking a half filled water bottle.

6- **Hepatic bruit** over hepatic carcinoma.

7- **Arterial bruit** which is usually systolic over aortic aneurysm.

8- **Peritoneal friction rub** exactly as the pleural rub, it may be palpable, also friction rub may indicate infarcts or tumour of the liver and spleen (heard over the right upper and left upper quadrants respectively).

9- **Foetal heart sounds**.

10- **Uterine soufflé**.

11- **Puddle sign for ascites**.

12- **Scratch sign** for organomegaly e.g.: liver enlargement, splenomegally and urinary bladder, see below.

| Urinary bladder: | The diaphragm of the stethoscope is placed just above the symphysis pubis in the midline. Scratches are done by moving the finger along the vertical midline from the umbilicus downward. The point at which the scratching sound intensifies indicates a change in the underlying tissue and thus locates the upper edge of the bladder. |
| Liver: | The stethoscope is placed either beneath the xiphoid or over the liver just above the costal margin in the MCL, scratches are then done by moving the finger from the right lower quadrant toward the costal margin alone MCL. The point at which the scratching sound intensifies indicates a change in the underlying tissue and therefore the presence of the lower liver edge. |

**Diagnosis:**

1- **Anatomical**: i.e.: Liver - spleen - kidney.

2- **Pathological**: e.g.: Cirrhosis.

3- **Etiologic**: e.g.: Bilharzial liver, post hepatitis cirrhosis.

4- **Functional**: i.e.: Compensated or not (L.C:F.).

As so comment on the presence or absence of portal hypertension.

**Examples:**

1. Case of hepatosplenomegaly, mostly post hepatitis liver cirrhosis with portal hypertension, No manifestations of liver cell failure.

2. Case of splenomegaly for D.D. mostly due to liver cirrhosis with shrunken liver with portal hypertension and manifestations of L.C.F.
ASCITES

Definition:
Excessive accumulation of fluid in the peritoneal cavity.

Causes:

(A) Cardiac:
- R.V.F.
- Tricuspid valve diseases T.I. T.S.
- Constrictive pericarditis (Ascites precox)

(B) Hepatic:
- Cirrhosis with all its causes.
- Veno occlusive disease.

(D) Renal:
- Nephrotic syndrome.

(E) Nutritional

(F) Chylous ascitis:
Due to thoracic duct obstruction by tumour or due to filariasis, it is milky white- dissolve
in ether- give orange color with sudden III.

(G) Hemoperitoneum:
Traumatic - malignant - blood disease.

(H) Miscellaneous:
- Meigs's syndrome (ovarian tumour – Ascites – pleural effusion)
- Polyserosities:leading to Ascites - pleural effusion - pericardial effusion.

(I) Local peritoneal diseases:
- T.B. peritonitis.
- Malignancy 1ry or secondary.

Signs of Ascites:
I. Inspection:
- Bulging more in the flanks.
- Divercation of recti.
- Shift of umbilicus downward.

II. Percussion:
- Sinking dullness in moderate ascites.
- Fluid thrill in tense ascites.

III. Displacement of organs:
- Abdomen pushed upward producing dullness in the pulmonary area which
  becomes resonant on sitting.
- Venal percussion on chest (infradiaphragmatic dullness).
Abdomensheet

D.D.:  
1) Abdominal gases:  
   Abdomen is resonant all over.

2) Ovarian cyst:  
   a- The swelling is central or to one side.  
   b- Resonant flanks, no shifting dullness.  
   c- Umbilicus pushed upward.  
   d- Upper border of the dullness is convex.  
   e- Blaxland's ovarian sign or ruler's test:  
      After emptying the bladder a ruler is transversely placed above the pelvis and  
      pressed backwards with both hands, you feel the transmitted aortic pulsation in  
      ovarian cyst but not in ascites.  
   f- P.V

3) Pregnant uterus.  
4) Distended bladder.  
5) Obesity.  
6) Other huge abdominal Swelling.

Investigations for Ascites:  
1- Urine for Bilharzial ova and albuminuria.  
2- Stool for Bilharzial ova.  
3- X-ray heart, chest, and echocardiography.  
4- Liver function tests, kidney function tests.  
5- Ba meal for esophageal varices, pelviabdominal sonar  
6- Examination of ascites fluid, "remember the differences between exudate and  
   transudate".  
7- Pelvi-abdominal sonar.  
8- Laparoscopy + peritoneal biopsy for malignancy and T.B. peritonitis.

Q: Patient with ascites without lower limb edema, we can suspect:  
   - Ascites precox  
   - Local causes in the abdomen, e.g.: T.B. peritonitis & malignant ascites (e.g.:  
     Mesothelioma)  
   - In patient with cirrhotic ascites the diuretic therapy may cause relieve of  
     the lower limb edema with persistence of ascites? See also theories of  
     ascites in liver cirrhosis.
IMPORTANT CAUSES OF PALPABLE ABDOMINAL MASSES

1) Right iliac fossa masses:
- Appendix mass.
- Cancer colon.
- Crohn's disease.
- Psoas abscess.
- Transplanted kidney (Note any overlying scar)
- Ileocaecal T.B.
(Adherent bowel loops)

2) Left iliac fossa masses:
- Diverticular abscess.
- Transplanted kidney.
- Psoas abscess.
- Cancer colon.

3) Midline masses:
- Aortic aneurysm.
- Cancer, stomach.
- Ovarian cysts.
- Bladder (Retention of urine).
- Pancreatic pseudocyst.
- Cancer pancreas
- Horseshoe kidney
- Uterine masses.

DD of tender abdomen
- Tender abdomen may originate from abdominal wall, viscus organ or solid organ or peritoneum.
- When the patient raises head and shoulders, the tenderness of abdominal wall will persist but tenderness from a deeper lesion will decrease due to protection by the tightened muscles. Abdominal wall tenderness may be due to muscular strain, viral aches or trauma.
- Pain, with rebound tenderness are associated with peritoneal inflammation.
- Absence of rigidity and rebound tenderness suggestive to tender viscus or solid organs so try to localize the site of tenderness according to the surface anatomy of the abdominal areas to determine the affected viscus or solid organ.
REFERENCES

- Harrison text book (principles of Internal Medicine).
- Cecil Textbook (Textbook of Medicine).
- Kumar (Clinical Medicine).
- Davidson's (Principles and Practice of Medicine).
- Henry/Thompson (Clinical Surgery).
- Cecil Essentials of Medicine.
- The National Medical Series for Independent Study (Medicine).
- MacLeod's clinical examination.
- Hutchison's clinical methods.
- Chamberlain's symptoms and signs in clinical medicine.
- Problem – oriented medical diagnosis.
- Barbara Bates, a guide to physical examination and history taking.

AUTHOR'S AVAILABLE BOOKS

1- Hepatology.
2- Gastroenterology.
3- Endocrinology.
4- Rheumatology.
5- Cardiology.
6- Nephrology.
7- Hematology.
8- Neurology and psychiatry.
9- Infectious diseases, tropical diseases, immunology, nutrition, genetics, geriatric, toxicology and therapeutics.
10- Respiratory diseases.
11- Principles of history-taking and physical examination.
   - Cardiology
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