# Causes of the eight examinable parameters at your finger tips

# A Handbooklet for Clinical Medicine Students

By Daniel K. Kimwetich

**KMTC Nakuru Campus** 

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# **Table of Contents**

1. Pallor	
2. Jaundice	9
3. Oral Thrush	
4. Cyanosis	
5. Finger Clubbing	
6 Dehydration	17
7. Lymph nodes	
8 Oedema	22

# 1. Pallor **Definition**

allor is a state in which the level of circulating red blood cells, the amount of haemoglobin (Hb) or haematocrit (packed cell volume) is below the normal expected range, taking into account both **age**, **sex** and **race**.

# The Hb in males is 13-18gm/dl, while in females it is 12-16gm/dl. The Hb of a baby at birth is 15-18gm/dl.

NB: the presence of symptoms in anaemic patient depends on how quickly the anaemia has developed i.e. in a sudden drop in Haemoglobin, the patient will present with signs and symptoms. Anaemia developing slowly over a prolonged period may be asymptomatic.

#### Causes

# 1) Blood loss

- Acute blood loss onset is sudden e.g. epistaxis, Oesophageal varices, ruptured aneurysm
- Chronic blood loss prolonged persistent haemorrhage e.g. hook worm infestations, Menorrhagia, recurrent epistaxis, peptic ulcers, haemorrhoids, ulcerative colitis, frequent blood donation.

# 2) Anaemia due to inadequate production of red blood cells.

- i) Deficient essential factors necessary for erythropoiesis e.g. iron, folic acid, vitamin B12, protein, ascorbic acid, nicotinic acid, riboflavin, copper.
- ii) Chronic inflammatory diseases e.g. infections like TB, non infectious diseases e.g. rheumatoid athritis, systemic lupus erythematosis.
- iii) Chronic renal diseases production of erythropoietin is reduced.
- iv) Chronic liver diseases e.g. liver cirrhosis.
- v) Endocrine abnormalities e.g. hypothyroidism, hypopituitarism, hypoadrenalism. There will be low tissue absorption of  $O_2$  > reduced metabolic activity in the bone marrow > reduction in RBC Production.

# VI) Impairment of bone marrow activity (Bone Marrow failure) e.g.

- Aplastic/hypoplastic anaemia.
- BM infiltration with malignant cells e.g. leukemia, multiple mycloma, /metastasis of a malignant disease in BM.
- Toxic effects resulting from chronic infections, malignancies, uremia or collagen disorders.

# 3) Excessive destruction of the red blood cells.

- □ Intrinsic (Hereditary)
  - Membranopathies spherocytosis and elliptocytosis
  - o Enzymopathies Glucose-6-phosphate deficiency (G6PD) and pyruvate kinase deficiency
  - o Haemoglobinopathies Sickle cell and Thalassaemia

- □ Extrinsic (Acquired)
  - o Immune
    - Autoimmune haemolytic anaemia
    - Iso-immune haemolytic anaemia (Rh, ABO incompatibility)
  - o Non-immune
    - Trauma
      - Microangiopathy
      - Mechanical damage like burns
    - Hypersplenism
       – spleen enlarges, traps RBC, & destroys RBCs.
    - Physical agents
      - Drugs
      - Chemicals e.g. phenacetine,pb,cu<sup>2+</sup>
      - Toxins e.g. septicaemia, uraemia
    - Bacterial infections
    - Parasitic disorders e.g. malaria
    - Membrane defects e.g. liver disease
    - Defective red cell maturation

# **NOTE**

# MORPHOLOGICAL CLASSIFICATION

# 1) Normocytic normochromic anaemia.

The RBC are of normal size, shape, & contain normal amount of haemoglobin.

The index -MCHC-Normal,

-MCV-Normal

# **Causes**

- i) Chronic infections.
- ii) Any chronic debilitating disease e.g. malignancies.

# 2) Macrocytic normochronic anaemia.

RBC are too large but contain normal amount of Hb.

Blood for full haemoglobin- MCV is increased.

# **Causes**

Deficiency of vitamin B12 and folic acid deficiency.

# 3) Microcytic hypocromic anaemia.

The *RBC* will be small in size and contain less than Normal amount of pigment.(Hb)

Blood for full haemogram.

- *MCV* reduced.
- *MCH/MICHC* reduced.

# Causes

iron deficiency

# **REMEMBER**

#### 1. Bone Marrow Failure

This can be absolute, mechanical or relative bone marrow failure.

#### Causes

Absolute	bone	marrow	failure can	result from: -
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- □ Can be congenital
- Idiopathic
- Drugs
  - o Analgesics
  - o Antibiotics e.g. Chloramphenicol
  - o Anticonvulsants epanutin
  - o Antidiabetic oral hypoglycaemics
  - o Anti-thyroid
  - o Cytotoxics
  - o Industrial chemicals
- Radiation induced
- Tumours
- □ Heavy metal compounds

# **Mechanical (Bone marrow replacement)**

- □ Malignancy
- □ TB bone marrow
- Leishmaniasis
- □ Gaucher's disease

# Relative (Symptomatic anaemia)

- Chronic bacterial infections
- □ Chronic renal failure
- □ Chronic liver disease
- □ Chronic parasitic infestation Leishmaniasis
- Malignancy
- Connective tissue disorders
- □ Chronic endocrine failure
- □ Nutritional disorders malnutrition

# 2. Microcytic Anaemia

Is due to iron deficiency anaemia whose causes are: -

- □ Low dietary intake
  - Malnutrition
- □ Chronic blood loss
  - o From the G.I.T
    - Oesophageal varices
    - Peptic ulcer (duodenal ulcer & gastric ulcer)
    - Hookworm infestation (A. duodenale –0.2 mls/worm/day; N. americanas 0.03 mls/worm/day)
    - Ca stomach, Ca colon, Ca rectum
    - Haemorrhoids
  - o From the G.U.T
    - Bleeding from Ca cervix or endometrium
    - Haematuria
    - Bladder polyps
    - Severe urinary tract infection
    - Malignancy
    - Schistosomiasis
  - Malabsorption of iron
  - Physiological increased demand
    - Infants
    - Aldolescents
    - Pregnancy

# 3. Megaloblastic anaemia

#### Causes

- □ Vitamin B12 deficiency
  - o Dietary e.g. in vegetarians
  - Malabsorption
    - Gastric ulcers lack of intrinsic factor
    - Intestinal causes
    - Stagnant loop syndrome
    - Tropical sprue
    - Transcobalamin II deficiency
    - Drugs
- □ Folic acid deficiency
  - Dietary
  - Malabsorption
  - o Increased demand

- Physiological e.g. pregnancy, prematurity and infancy
- Pathological
  - Blood disorders
  - Malignancies
  - Inflammatory
  - Metabolism
- Urinary loss
  - Congestive cardiac failure
  - Acute liver failure
- o Drugs
- Liver disease

# 4. Haemolytic Anaemia

Due to heamolysis of the RBCs - can be intrinsic (inherited) or extrinsic (acquired)

# 5. Haemorrhagic Disorders

Fall in 3 groups

- 1. Platelet disorders
- 2. Blood vessel disorders
- 3. Coagulation disorders

#### Platelet Disorders

- Platelet deficiencies
  - Reduced production
    - Marrow infiltration
    - Infections
    - Marrow hypoplasia
    - Defective maturation
  - o Reduced survival
    - Increased destruction
      - Drugs
      - Immune mechanisms
      - Infections
    - Consumption
      - D.I.C
      - Infections
    - Sequestration
      - Spleenomegally
      - Hyperspleenism

# □ Abnormalities of platelets

#### **Blood Vessel Disorders**

This can occur in aneurysms, injury, arteriosclerosis and phlebitis.

# **Coagulation Disorders**

- Hereditary
  - o Haemophilia A (Classical)
  - o Haemophilia B (Christmas disease)
  - Von Willibrand disease
- Acquired
  - Auto antibodies to clotting factors
  - Vitamin K deficiency
  - o Vitamin K antagonism
  - o Vitamin C deficiency
  - Liver disease
  - o Alcohol
  - Haemolytic uraemia syndrome

#### 2. Jaundice

# **Definition**

aundice is the yellow colouration of the sclerae, mucous membranes and the skin caused by accumulation of bilirubin or bilirubin complexes. The sites for eliciting jaundice are the sclera, mucous membranes and the skin.

It is usually detectable clinically when the plasma bilirubin exceeds 50  $\mu$ mol/l (~3 mg/dl) but recognition is often dependent on the ambient light available.

#### Causes

- □ Pre-hepatic / haemolytic causes
  - a. Congenital
  - Haemoglobinopathies SCD, Thalassaemias
  - Red cell membrane defects spherocytosis, elliptocytosis.
  - Metabolic disease G6PD deficiency, pyruvate kinase deficiency.

# b. Acquired

- i) Autoimmune as in:
- Warm antibodies
- Cold antibodies
- Paroxysmal nocturnal haemoglobinuria

- ii) Infections e.g. malaria, septicemia, Viral infections.
- iii) Alloimmune
  - Transfusion Rxns
  - After organ transplant
- iv) Mechanical
  - MAHA
  - Valve prosthesis
- v) Systemic diseases
  - Renal failure
  - Liver failure
- vi) Drug induced
  - S'namides
  - Rifampin
  - Ribavirin
- vii) Ineffective erythropoiesis
  - Cobalamine, folate, severe iron deficiency
- Hepatic causes
  - o Acquired
    - Infections
      - Viral hepatitis (ABCDE etc), Leptospirosis, Relapsing fever,
        Typhus fever
      - Severe sepsis (septicaemia) from bacterial infections
    - Hepatocellular toxins e.g. Fungi aminata toxins
    - Alcoholic hepatic injury, liver cirrhosis, Hepatoma
    - HIV cholangiopathy
    - Hodgkins disease
    - Cystic fibrosis
    - Snake bites
    - Drugs
    - Post-operative
    - Recurrent -idiopathic
    - Pregnancy
  - Congenital
    - Criggler-Najjer syndrome
    - Rotor syndrome
    - Dubin-Johnson syndrome
    - Gilberts syndrome
- □ Post-hepatic (Obstruction)
  - Common bile duct (CBD) stones choledocholithiasis
  - Gallstones
  - Cancer of bile ducts

- Cancer head of pancreas
- Cancer ampulla of Vatae
- Cancer bile duct
- Sclerozing cholangitis
- Biliary atresia
- Pancreatic/Pancreatic pseudocyst
- Congenital malformation
- Roundworm migration

#### 3. Oral Thrush

#### **Definition**

ral Thrush (monolial stomatitis) is a fungal infection due to *Candida albicans* that presents with white patches slightly raised above the surface that is redder than normal. It may form extensive sheets throughout the mouth.

Thrush may be confused with milk curds but the curds are easily removed while thrush is removed with difficult leaving a raw surface.

Check for oral thrush at the sides of the tongue, soft palate and the hard palate.

Causes of oral thrush include: -

- 1. Sepsis
- 2. Immunosuppression
- 3. Prolonged use of cytotoxic drugs
- 4. Prolonged use of broad-spectrum antibiotics
- 5. Debilitating diseases
- 6. Unclean dentures

The common causes of ulcers in the mouth are: -

- 1. Inflammatory bowel disease
- 2. Leucoplakia
- 3. Lichen planus
- 4. Thrush (oral candidiasis)
- 5. Idiopathic apthous ulcers
- 6. Koplick's spot (measles)
- 7. Malignant erosion

# 4. Cyanosis

#### **Definitions**

yanosis is the bluish discolouration of the skin and mucous membranes due to excessive amounts of reduced haemoglobin in circulation. It is evident clinically when more than 5 gm% of haemoglobin is deoxygenated.

Cyanosis is of considerable diagnostic value in respiratory and cardiovascular conditions. The sites for examining cyanosis are:

- 1. Oral mucosa
- 2. Frenulum of the tongue
- 3. Lips
- 4. Nose
- 5. Extremities the fingers and toes
- 6. Ears

Cyanosis can be classifies *as peripheral cyanosis*, *central cyanosis* and cyanosis due to abnormal pigments.

Causes of cyanosis

# i) Peripheral Cyanosis

This is due to poor local circulation and it is seen at the extremities (hands and the feet). The pulses are weak and impalpable but the arterial pulse pressure of oxygen is normal. The cyanosis results from diminished capillary blood flow allowing more time for oxygen removal by the tissues.

#### Causes

- 1. Low cardiac output as seen in mitral stenosis "malar flush" (mitral stenosis with severe pulmonary hypertension) and shock.
- 2. Local peripheral vasoconstriction
  - a. Extreme cold
  - b. Ergot poisoning
  - c. Raynaud's phenomenon
  - d. Vasospasms

# ii) Central Cyanosis

- □ There is excess of desaturated haemoglobin in the blood leaving the aorta and it is best seen on the mucous membranes of the tongue and frenulum of the tongue.
- ☐ There are 3 types of central cyanosis
  - I. Due to deficient oxygenation of blood in the lungs mainly because of respiratory conditions.
  - Inadequate ventilation e.g. pneumonia, chronic bronchitis and asthma.

- Reduced amount of total ventilating air e.g. poliomyelitis.
- Impaired oxygen transfer across the alveolar capillary membrane e.g. fibrosing alveolitis.
- Absolute excess of desaturated haemoglobin with a normal percentage saturation e.g. primary polycythemia.
- II. Right-to-left shunting of blood by-passing the lungs (the cardiovascular causes).
  - The 5Ts (Cyanotic Heart Disease)
    - Tetralogy of Fallot
    - Truncus arteriosus
    - Tricuspid atresia
    - Total anomaly of pulmonary venous drainage
    - Transposition of great vessels
  - Right-to-left shunts where there is an abnormal communication between pulmonary artery and vein (arteriovenous aneurysm).
- III. Cyanosis due to abnormal pigments.
  - Methamoglobin
  - Sulphaemoglobin

# 5. Finger Clubbing

The Nails

ail consists of the nail plate, nail folds (proximal and lateral), matrix (lunula) and the nail bed. Nail disorders occur in many skin conditions or in isolation. The nail disorders can be congenital or acquired disorders.

Nail disorders seen in skin conditions include: -

Onycholysis and pitting in psoriasis





- □ Discolouration and crumbling in fungal infections
- □ Pitting and ridging in chronic dermatitis
- □ Severe dystrophy in lichen planus



Causes of finger clubbing

# • Lung disease:

- Lung cancer, mainly non-small-cell (54% of all cases), not seen frequently in small-cell lung cancer (< 5% of cases)</li>
- o Interstitial lung disease most commonly idiopathic pulmonary fibrosis
- Complicated tuberculosis
- Suppurative lung disease: lung abscess, empyema, bronchiectasis, cystic fibrosis
- o COPD
- o Mesothelioma of the pleura
- o Arteriovenous fistula or malformation

#### • Heart disease:

- Any disease featuring chronic hypoxia
- o Congenital cyanotic heart disease (most common cardiac cause)
- o Subacute bacterial endocarditis
- Atrial myxoma (benign tumor)
- Tetralogy of Fallot
- Gastrointestinal and hepatobiliary:
  - Malabsorption
  - o Crohn's disease and ulcerative colitis
  - o Cirrhosis, especially in primary biliary cirrhosis
  - Hepatopulmonary syndrome, a complication of cirrhosis

#### • Others:

- o Graves' disease (autoimmune hyperthyroidism) in this case it is known as *thyroid acropachy*
- Familial and racial clubbing and "pseudoclubbing" (people of African descent often have what appears to be clubbing)
- Vascular anomalies of the affected arm such as an axillary artery aneurysm (in unilateral clubbing)

# Congenital Nail Disorders

- 1. The Nail-patella syndrome
  - ☐ The patella and some of the nails are rudimentary or absent (thumb nails are usually involved)
- 2. Pachyonychia congenita
  - ☐ These are mis-shapenned and hypertrophic nails.

# **Acquired Nail Defects**

#### 1. Beau's lines

These are transverse ridges on the nails due to temporary interference with nail formation and it is self-timing. They are commonly seen during convalescence from a variety of severe diseases causing prolonged fever.



# 2. Koilonychia

□ Is the loss of the normal nail contour resulting in flat or even depressed nail surface ("spoon-shaped" nails). The nails are slow growing and brittles. It is commonly associated with hypochromic (iron deficiency) anaemia.



# 3. Finger Clubbing

- □ Is an exaggeration of the normal nail curve with loss of the normal angle between the nail and the posterior nail fold (see finger clubbing
- 4. Paronychia
- 5. Herpetic whitlow
- 6. Paronychial tumours
- 7. Iatrogenic Nail Abnormalities
- 8. Leuconychia

# Clubbing may be present in one of five stages: [10]

- 1. **No visible clubbing.** Fluctuation (increased ballotability) and softening of the nail bed only. No visible changes of nails.
- 2. **Mild clubbing.** Loss of the normal <165° angle (Lovibond angle) between the nailbed and the fold (cuticula). <u>Schamroth's window</u> is obliterated. Clubbing is not obvious at a glance.
- 3. **Moderate clubbing.** Increased convexity of the nail fold. Clubbing is apparent at a glance.
- 4. **Gross clubbing.** Thickening of the whole <u>distal</u> (end part of the) finger (resembling a drumstick)
- 5. **Hypertrophic osteoarthropathy.** Shiny aspect and <u>striation</u> of the nail and skin

# Grading of finger clubbing

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Schamroth's test or Schamroth's window test (originally demonstrated by South African cardiologist Leo Schamroth on himself) is a popular test for clubbing. When the distal phalanges (bones nearest the fingertips) of corresponding fingers of opposite hands are directly opposed (place fingernails of same finger on opposite hands against each other, nail to nail), a small diamond-shaped "window" is normally apparent between the nailbeds. If this window is obliterated, the test is positive and clubbing is present.

# 6. Dehydration

ehydration results from fluid imbalance due to excessive fluid loss as in: -

- 1. Increased fluid loss
  - a. Diarrhoea
  - b. Vomiting
  - c. Diuretic therapy
  - d. Burns
  - e. Fever (pyrexial states)
  - f. DKA
- 2. Decreased fluid intake
  - a. Very ill patients
- 3. Redistribution of fluids from plasma to tissues

# 7. Lymph nodes

Examine for any masses in the neck region and note the following characterises: -

- 1. Site
- 2. Size
- 3. Consistency
- 4. Tenderness
- 5. Mobility

# Causes of Lymphadenopathy

- 1. Infections (lymphoid and/or phagocytic hyperplesia)
  - a. Viral
    - i. Herpes virus
      - 1. CMV
      - 2. EBV
      - 3. VZ
    - ii. Rubella
    - iii. HIV
    - iv. HAV
  - b. Bacterial
    - i. Streptococcus
    - ii. Staphylococcus
    - iii. Brucella
    - iv. Tularaemia
    - v. Listera
    - vi. Pastearella pestis
    - vii. Haemophillus ducreyi
    - viii. Syphilis
    - ix. Leptospirosis

- x. Mycobacterium
  - 1. Tuberculosis
  - 2. Leprosy
- xi. Chlamydial
  - 1. Trachoma
  - 2. LGV
- c. Fungal
  - i. Histoplasmosis
  - ii. Cocidiomyocosis
- d. Parasitic
  - □ Toxoplasmosis
  - □ Trypanosomiasis
  - Filariasis
- 2. Malignancies
  - a. Haematological
    - i. Lymphomas
    - ii. Hodgkin's disease
    - iii. Leukaemia
  - b. Metastatic (infiltrative)
    - i. Ca breast, lung, kidney, prostate, head and neck, G.I.T. melanoma, sarcoma, seminoma, neuroblastoma
- 3. Autoimmune disorders/inflammation
  - a. Rheumatoid arthritis
  - b. Sarcoidosis
  - c. Systemic lupus erythromatosis (SLE)
  - d. Dermatomyositis
  - e. Immune complex disease/serum sickness
- 4. Drug side effects
- 5. Infiltration e.g Gaucher's disease
- 6. Endocrine (lymphoid hyperplasia) hyperthyroidism

# Localized Lymphadenopathy

Moderate enlargement of the cervical and inguinal lymph nodes may be a normal finding representing residual inflammation from prior infection. The physical characteristics are important clues to the diagnosis. Soft, discrete, mobile lymph nodes suggest residual inflammation, whereas hard, fixed nodes are consistent with malignancy.

#### Generalized Lymphadenopathy

Generalized lymphadenopathy is commonly seen with neoplasms of the immune system, infections, reactive processes, tumours, collagen vascular diseases and drug reactions. Genearlized lymphadenopathy involves at least three separate sites.

#### Causes

1. Infectious

- a. Bacterial
  - i. Brucellosis
  - ii. Leptospirosis
  - iii. Mycobacterium/Tuberculosis
  - iv. Syphilis (secondary)
- b. Fungal
- i. Histoplasmosis
- ii. Coccidiodomycosis
- c. Viral
  - i. Measles
  - ii. Rubella
  - iii. EBV
  - iv. CMV
  - v. Hepatitis B
  - vi. HIV
  - vii. Influenza
- d. Protozoa
  - i. Toxoplasmosis
  - ii. Kala azar
  - iii. Leishmaniasis
  - iv. Trypanosomiasis
- e. Helminths
  - i. Filariasis
- 2. Noninfectious
  - a. Malignancy
    - i. Lymphoma
    - ii. CML
    - iii. CLL
    - iv. acute Leukeamia
    - v. metastasis
  - b. Sarcoidosis
  - c. Collagen vascular diseases
  - d. Hyperthyroidism
  - e. Myeloproliferative disorders
  - f. Lymphoproliferative disorders
  - g. Drug side effects (hydralazine, isoniazid)

# Causes of Cervical Lymphadenopathy

- 1. Infectious causes
  - a. Infections of the head, neck, sinuses, ear, eyes, scalp and pharynx
  - b. Bacterial cervical adenitis (unilateral)
    - i. Staphylococcal

- ii. Streptococcal
- iii. Tuberculosis (often suppurative)
- c. Viral respiratory tract infections
  - i. Influenza virus
  - ii. Adenovirus
  - iii. Rhinovirus
  - iv. Rubella
  - v. Measles
  - vi. Herpes simplex virus
  - vii. Epstein-Barr virus (EBV)
  - viii. Cytomegalo virus (CMV)
  - ix. HIV (acute)
- d. Pharyngitis
  - i. Streptococcal
  - ii. Diptheria
  - iii. Yersinia
  - iv. Corynebacterium haemolyticum
- e. Oral and odontogenic infections
  - i. Ludwig's angina
  - ii. Vincent's angina
  - iii. Lemiere syndrome
- 2. Noninfectious causes
  - a. Lymphoma
  - b. Lymphoproliferative disorders
  - c. Nasopharyngeal cancer
  - d. Metastatic carcinoma
  - e. Thyroid cancer
  - f. Sarcoidosis
  - g. Malignancy of the head and neck

# Preauricular Lymphadenopathy

Is frequently seen in association with conjuctival infections and otitis externa.

# Causes

- 1. Oculoglandular syndrome (conjunctivitis)
  - a. Viruses
    - i. Adenoviruses (follicular conjunctivitis)
    - ii. Papillomaviruses (corneal erosion)
    - iii. Picornaviruses (haemorrhagic conjunctivitis)
    - iv. Herpes simplex (corneal ulcerations)
    - v. Herpes zooster (corneal ulcerations)
    - vi. Measles
    - vii. Mumps (parotitis)

# viii. Inflenza (flu-like illness)

- b. Bacteria
  - i. N. gonorhoea
  - ii. F. tularenis
  - iii. S. aureus
  - iv. S. epidermidis
  - v. S. pnemoniae
  - vi. H. influenzae
- 2. Mycobacterium
- 3. Spirochetes
- 4. Otitis externa

# Scalenes/supraclavicular

- 1. Thoracic or retroperitoneal infections
  - a. Bacterial
  - b. Fungal
- 2. Malignancy
  - a. Lung
  - b. Retroperitoneal
  - c. G.I.T e.g. Virchow's node (enlarged left supravicular lymph node)
  - d. Lymphoma

# Epitrochlear

- 1. Infections of hand or forearm (unilateral)
- 2. Secondary syphilis
- 3. Lymphoma (unilateral)
- 4. Sarcoidosis (bilateral)
- 5. Tularaemia (bilateral)

# Axillary

- 1. Infectious causes
  - I. Bacterial infections of the upper extremity
  - II. Plaque
  - III. Tularemia
- 2. Noninfectious causes
  - I. Lymphoma
  - II. Breast cancer
  - III. Metastatic carcinoma
  - IV. Melanoma

#### V. Brucellosis

# Inguinal

- 1. Infectious (leg and feet)causes
  - a. Lower extremity infections
  - b. Sexually transmitted disease
    - i. Syphilis
    - ii. LGV
  - c. Plaque
  - d. Tularemia
  - e. Pasteurella pestis
- 2. Noninfectious causes
  - a. Melanoma
  - b. Lymphoma
  - c. Pelvic malignancy

# Hilar lymphadenopathy

- 1. Sarcoidosis
- 2. Tuberculosis
- 3. Systemic fungal infections
- 4. Ca lung (unilateral)

# Mediastianal lymphadenopathy

- 1. Mononucleosis syndromes
- 2. Sarcoidosis
- 3. Tuberculosis
- 4. Histoplsamosis
- 5. Ca lung
- 6. Lymphoma

# Abdominal/retroperotoneal

- 1. Mesenteric lymphadenitis (tuberculosis)
- 2. Lymphoma
- 3. Germ cell tumours/seminoma
- 4. Prostatic cancer
- 5. Other malignancies

#### 8. Oedema

- Ankle/sacral swelling.
- Can be generalized (Anarsaca).

- ♦ Systemic
- Causes
  - 1] Cardiac
    - Congestive Cardiac Failure(CCF)
    - Constrictive pericarditis
  - 2] Renal
    - Acute Glomerulonephritis(AGN)
    - Nephrotic Syndrome
    - Renal failure
  - 3] Hepatic
    - Liver cirrhosis
    - Hepatoma
    - Liver failure
  - 4] Malnutrition
    - Kwashiorkor
    - Beri beri
- ♦ Localized

#### Causes

- 1] Inflammation/Cellulitis
- 2] Venous obstruction due to:-
  - Tumours
  - Parasites e.g. W. Bacrofti and Ankylostoma
  - Pressure
  - Thrombosis (DVT)
- 3] Infections Lymphogranuloma Venerium (LGV)
- 4] Lymphatic obstruction
- 5] Allergic/Hypersensitivity reactions

# The mechanism of Oedema formation in cardiac failure:-

- ♦ Impaired renal flow
- Reduced renal blood flow lowers GFR promoting excessive re-absorption of water and salt by the renal tubules.
- ♦ Increased venous pressure
- ♦ Effects of aldosterone hormone
- ♦ Effects of anti-diuretic hormone

♦ Lymphatic factors e.g. Lymphangiectasis, incompetent valves and poor drainage have been demonstrated in cardiac failure.

# Oncotic pressures changes:-

- Congestion of the liver reduces albumin synthesis
- Loss of appetite reduces intake of proteins
- Loss of proteins in oedema fluids

# Vasodilatation e.g. in Beriberi

- Abnormal accumulation of carbohydrates causes peripheral vasodilatation and high output failure occurs.