Diseases of Blood

A)Introduction to Blood

a)Plasma

b)Cell types

B)Diseases of RBC

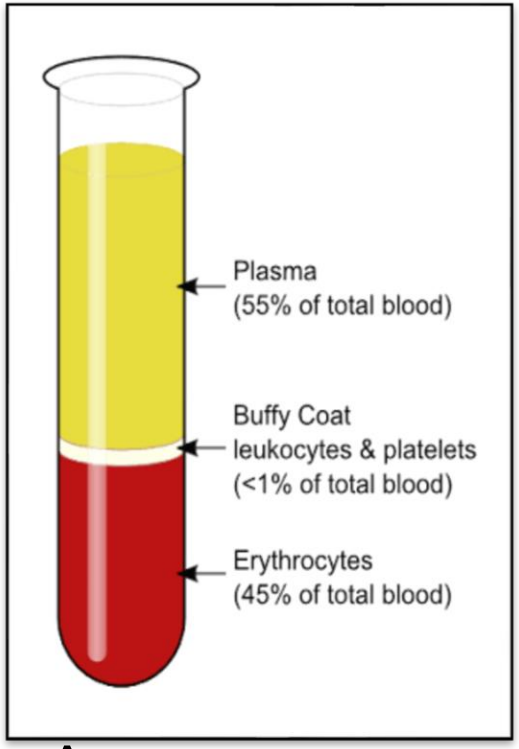
C)Diseases of WBC

D)Diseases of Platelets

Basics about blood and cells

Blood

- Blood consists variety of cells suspended in a fluid medium called plasma
- Plasma is composed of
  - 90% water
  - 8% protein
  - 1% organic salts
  - 0.5% of Lipids
  - 0.1% Sugar
  - Other components in lesser Amount



Proteins in plasma

- Blood Coagulation proteins
- Albumins
- Globulins

Globulins

Alpha Globulins

- Proteases
- Antiproteases
- Transport proteins

Beta Globulins

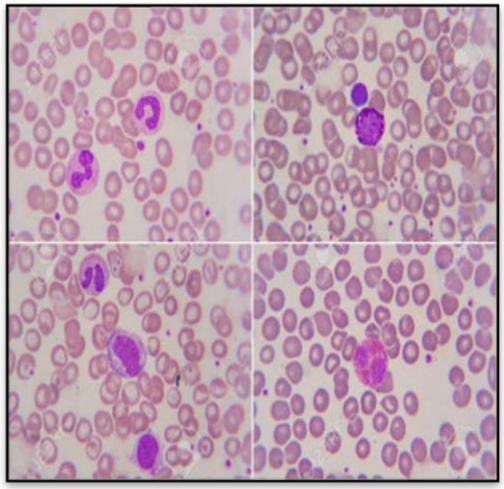
- Transferrin
- Other transport proteins

Gamma Globulins

- Immunoglobulins

Blood Cell Types

- Red Blood Cells (Erythrocytes)
- White Blood Cells(Leucocytes)
- Platelets(Thrombocytes)



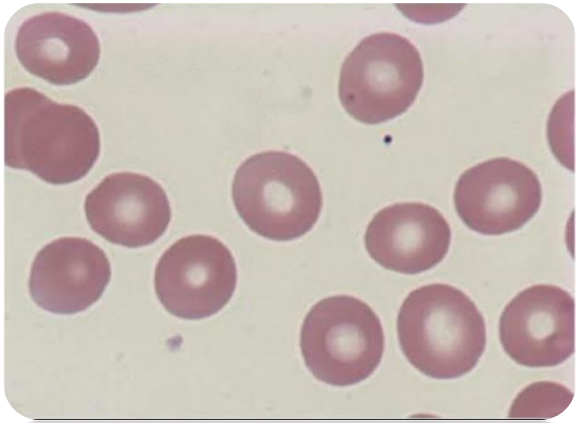
- All these cells are formed in Bone Marrow
- Process of Blood Cell formation is called as Haemopoiesis

Diseases Of RBC

Basics of About Erythrocytes

Red Blood Cells (Erythrocytes)

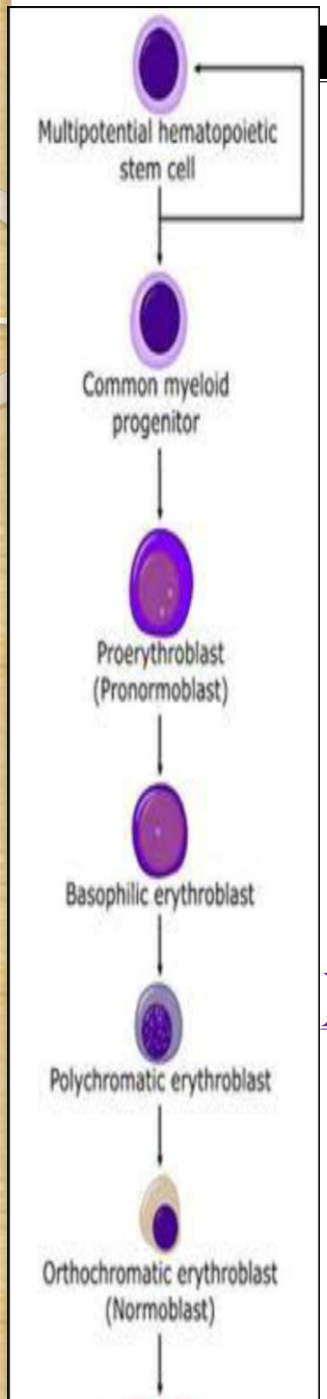
- Cells containing large amount of oxygen - carrying haemoglobulin
- Primary function is to transport oxygen and carbondioxide
- Functions are exclusively within the vascular system



I)Development Of Erythrocytes

- Entire process of development of erythrocytes is called as Erythropoiesis

- Whole mass of red blood cells and their precursors in the bone marrow is called as Erythron



PLURIPOTENTIAL STEM CELL

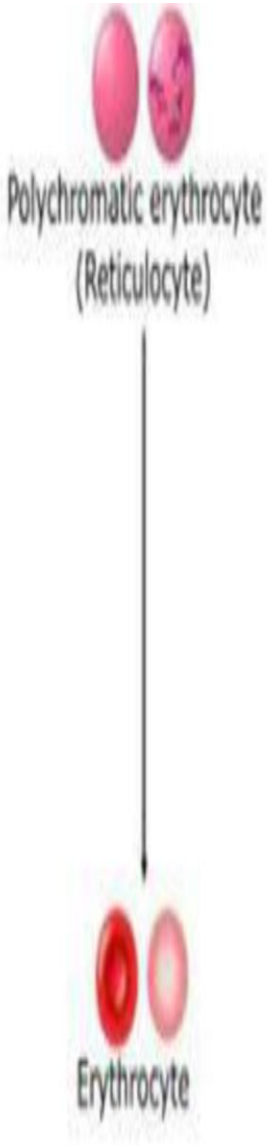
UNIPOTENTIAL STEM CELL

PROERYTHROBLAST

EARLY NORMOBLAST

NTERMEDIATE NORMOBLAST

LATE NORMOBLAST



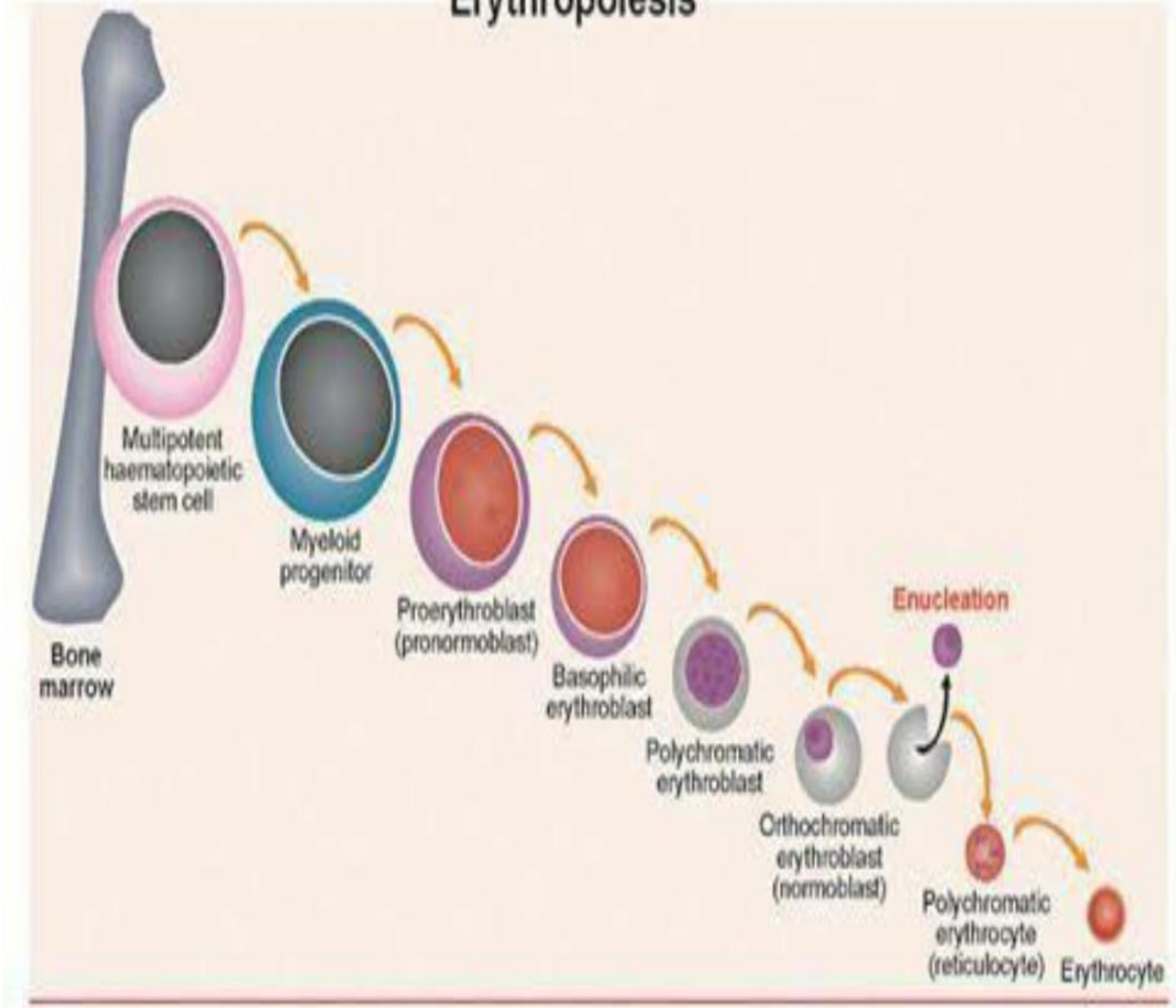
RETICULOCYTE (Released into blood stream which will differentiate only after 24 to 48 hours)

ERYTHROCYTE

TABLE 10.1: Important events during erythropoiesis

Stage of erythropoiesis	Important event
Proerythroblast	Synthesis of hemoglobin starts
Early normoblast	Nucleoli disappear
Intermediate normoblast	Hemoglobin starts appearing
Late normoblast	Nucleus disappears
Reticulocyte	Reticulum is formed. Cell enters capillary from site of production
Matured RBC	Reticulum disappears Cell attains biconcavity

Erythropoiesis

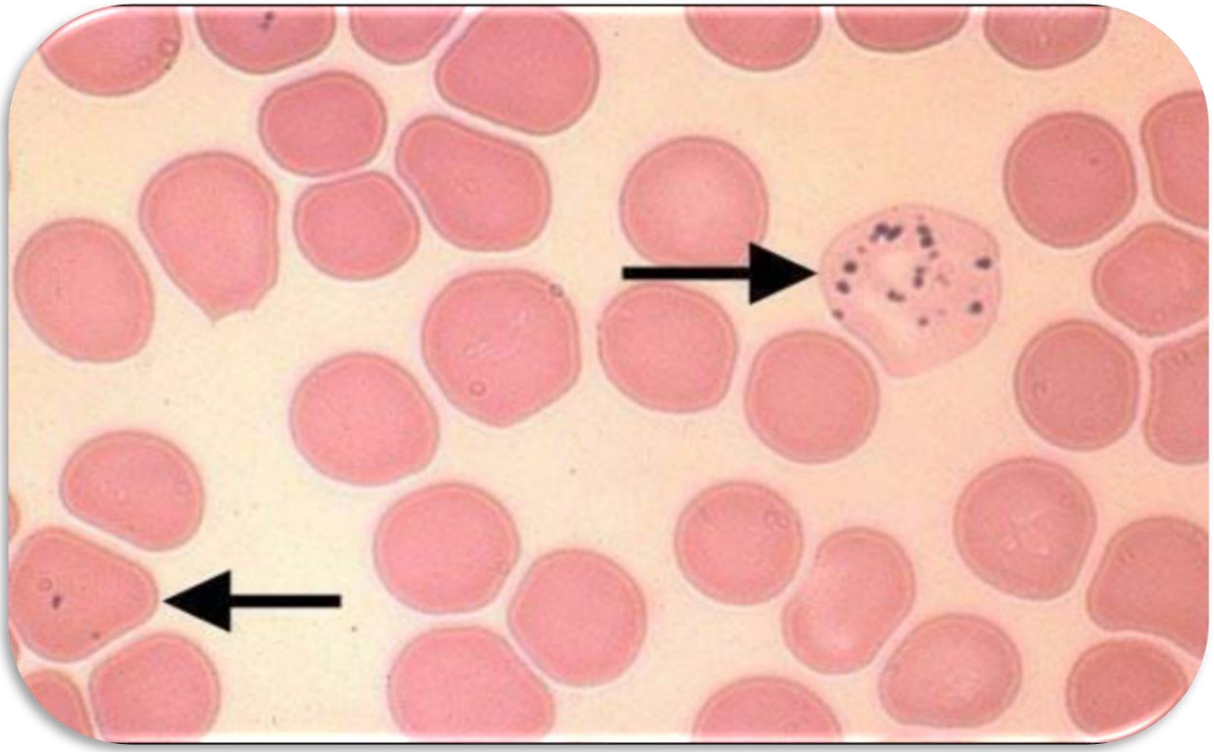


Other components needed for red cell production are :

- Iron
- Folic acid
- Vitamin B12
- Protein precursors (Erythropoietin)

- Mature erythrocytes are the cells without nucleus

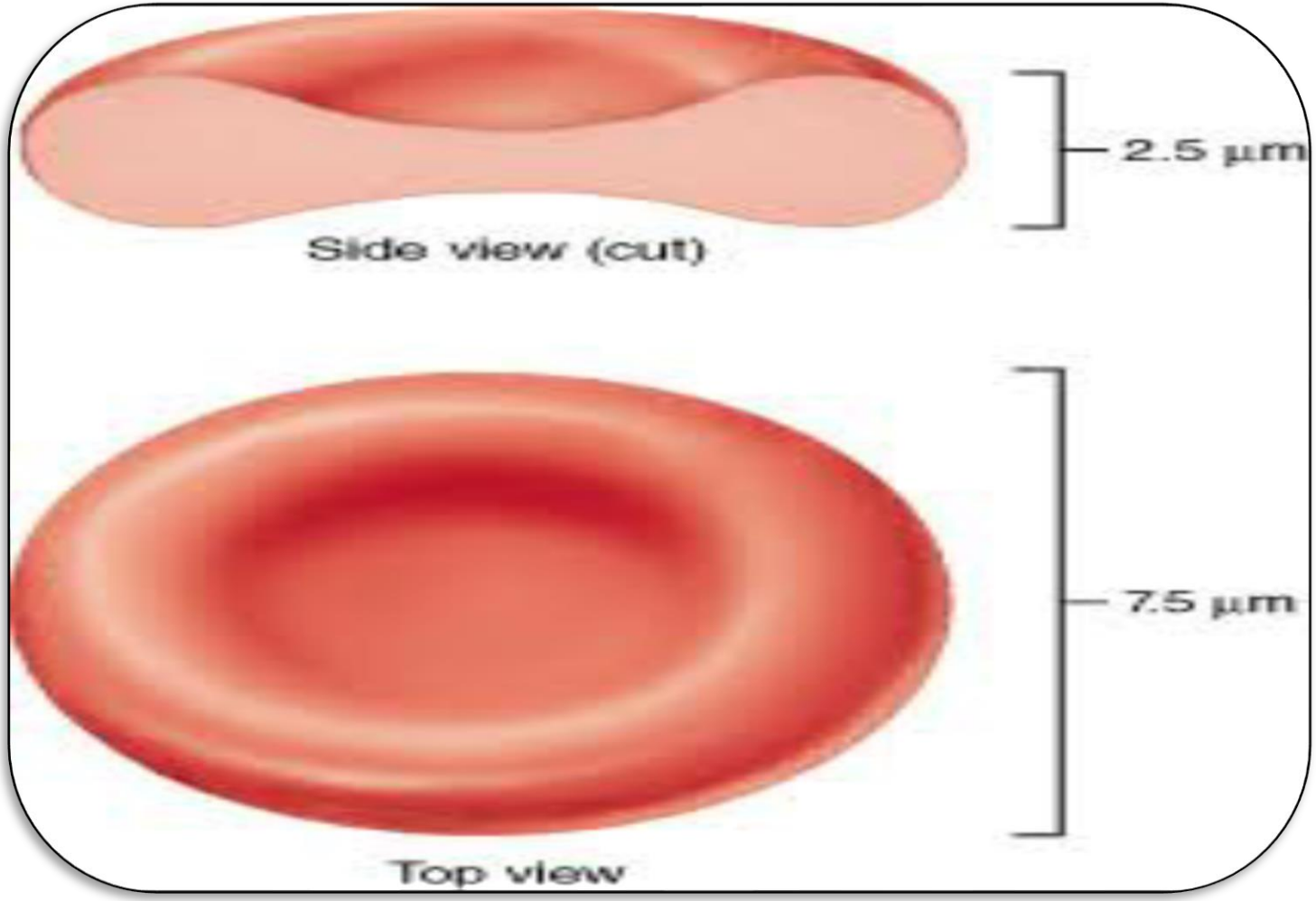
- Remnants of nucleus seen in erythrocytes after nuclear extrusion is called as Howell-Jolly Bodies



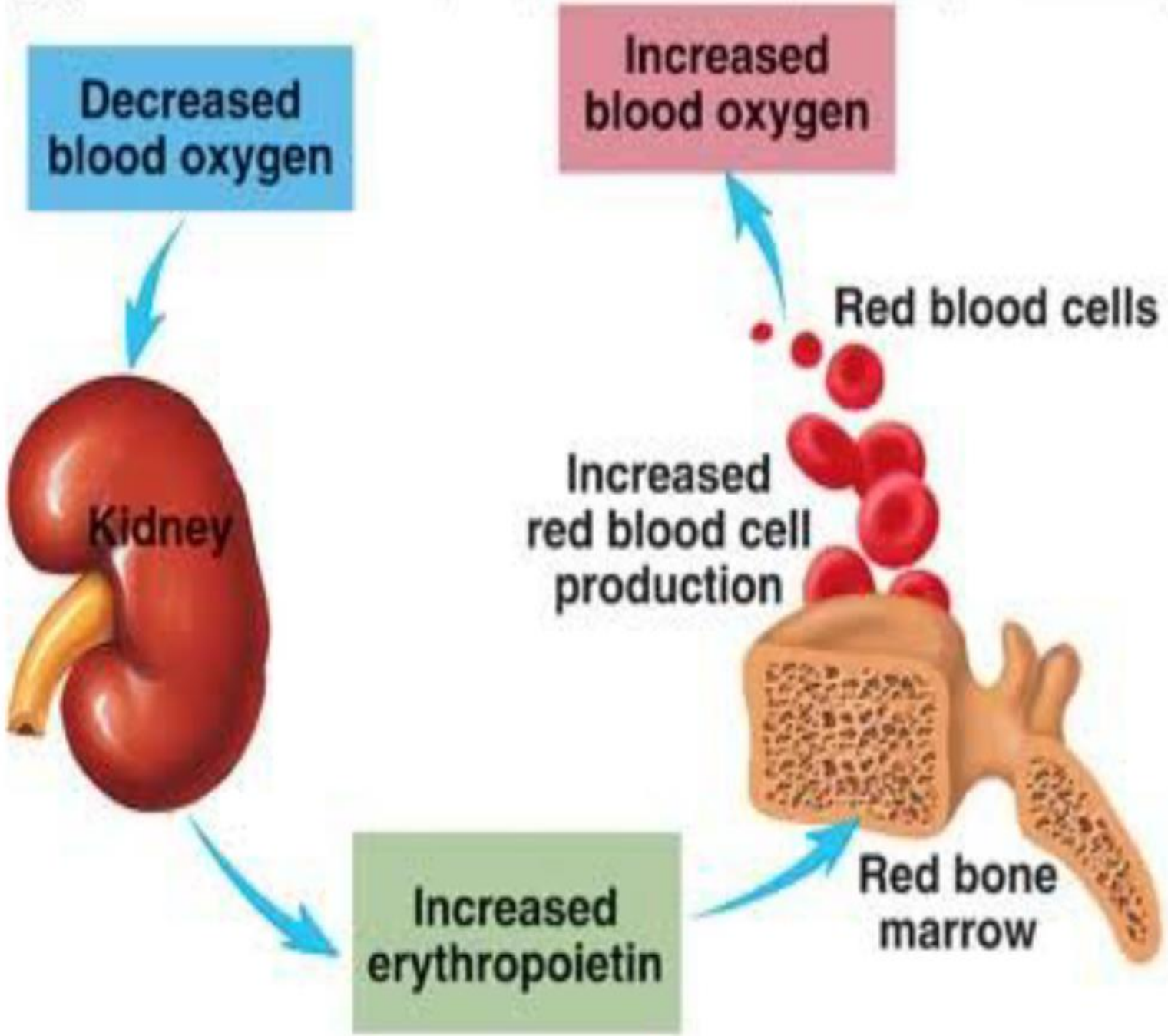
2)Size and Shape of Erythrocytes

- Matured Red blood cells
- Highly adapted for its principal function of oxygen and carbondioxide transport
- Fully developed erythrocytes contain an outer plasma membrane enclosing haemoglobulin and limited about of enzymes for maintaining the cell.
- Absence of Nuclues
- Outer lipid bilayer incorporating various globulins
- Main skeletal protein is Spectrin

Shape	Biconcave disc shape
Size	6.7-7.7micrometer
Number per liter	3.9-6.5 x 10^{12}
Duration of Development	5-7 days
Lifespan of Mature cells	120days


2.5 µm
Side view (cut)
7.5 µm
Top view

3)Production of Erythrocytes



Haemoglobulin

Haemoglobulin

- Complex conjugated protein of molecular weight 64,000 consists of two pairs of polypeptide chains each of which a haem is attached
- Two components heme and globulins
- Heme has iron-porphyrin compound
- Heme gives red color to the blood
- Each polypeptide chain is composed of a number of aminoacids

- Based on the number and sequence of amino acids,polypeptide chains are classified into six types

1. Alpha chains
2. Beta chains
3. Gamma chains
4. Delta chains
5. Epsilon chains
6. Zeta chains

- Haemoglobin A(Hb-A) which comprises 97% of hb in adult red cells, consists of two alpha and two beta chains

- Haemoglobin F (Hb-F) which accounts for about 70-90% of fetal haemoglobin consists of two alpha and two gamma chains(later during development gamma chains replaced by beta chains)

- Normal Hb in adult- Hb-A & Hb- A_2

- Normal Hb in fetus are Hb-F and Hb-Bart's

Anaemia

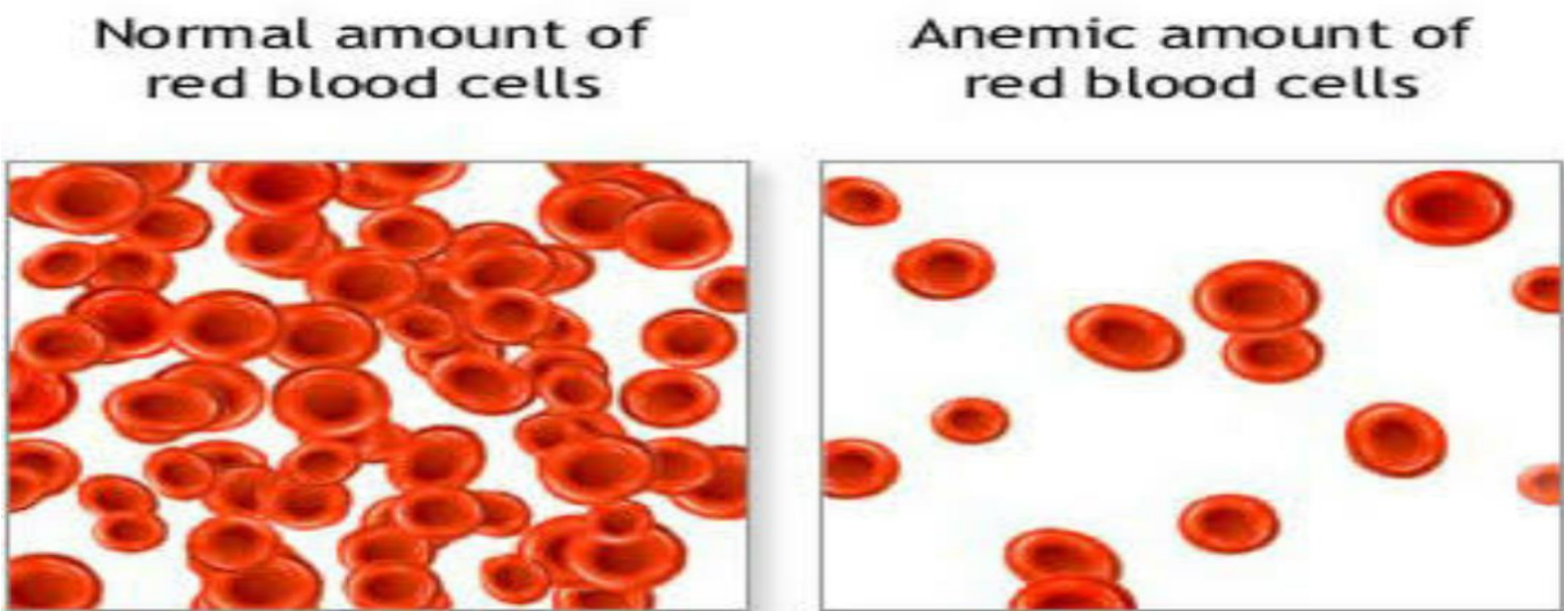
Definition

- Defined as a state in which the blood haemoglobulin level is below the normal range for the patient's age,sex and altitude of residence. (General medicine)

- Anaemia is defined as an abnormal reduction in number of circulating red blood cells,the quantity of haemoglobin and the volume of packed red cells in a given unit of blood.(Shafer)

- Normal adult haemoglobulin limits

1. Male – lies between 13-16 g per dLitre
2. Female – lies between 11.5 – 15 per dLitre



Classification of Anaemias

- Based on the cause of anaemia
- Based on the morphology of red cells
- Kinetic classification of Anaemia

A) Based on causes of Anaemia

Mechanism	Common Cause
Loss of Blood	
Acute	Trauma
Chronic	Gastrointestinal tract lesions, gynaecological causes, tumours
Decreased Red Cell Production	
Nutritional deficiency	Iron, vitamin B_{12}, folate
Inflammation mediated	Iron deficiency anaemia of chronic disease
Erythropoietin deficiency	Renal disease
Immune-mediated	Aplastic anaemia
Bone marrow infiltration	Primary haematopoietic neoplasms, metastatic neoplasms
Endocrine failures	Thyroid, pituitary
Increased Red Cell Destruction	
Membrane alterations (genetic)	Hereditary elliptocytosis, hereditary spherocytosis
Membrane alterations (acquired)	Hypophosphataemia, paroxysmal nocturnal haemoglobinuria
Enzyme deficiencies	G6PD, pyruvate kinase, hexokinase
Haemoglobin abnormalities	Thalassaemia syndromes sickle-cell disease, haemoglobinopathies
Immunologic abnormalities	Haemolytic disease of newborn, transfusion reactions, drug-induced reactions
Mechanical trauma	Disseminated intravascular coagulation, thrombotic thrombocytopenic purpura, defective cardiac valves, repeated physical trauma
Infections of red cells	Malaria
Toxic /chemical injury	Snake venom, lead poisoning
Splenic Sequestration	Reticuloendothelial hyperactivity with splen

B)Based on Morphology of Red cells

- Haemotocrit-ratio of packed erythrocytes to the total blood volume and decreased haemoglobulin (Hb) concentration below the normal levels for the individual based age and sex
- Mean Cell volume(MCV)
- Mean cell haemoglobulin (MCH)
- Mean cell Haemoglobulin concentration(MCHC)
- Red cell distribution Width (RDW)

Table 15. 3. Adult reference values of red blood cells

Parameter	Men	Women
Haemoglobin	14–17.5 gm/dL	12.3–15.3 gm/dL
Haematocrit	41.5–50.4%	36.9–44.6%
Red cell count	4.5–5.9 million cells/µl	4.5–5.1 million cells/s
Reticulocyte count	0.5–1.5%	
Mean cell volume (MCV)	80–96 fL	
Mean cell haemoglobin (MCH)	27.5–32.2 pg	
Mean cell haemoglobin concentration (MCHC)	33.4–35.5 g/dL	
Red cell distribution width (RDW)	11.4–14.5	

Morphologic classification :

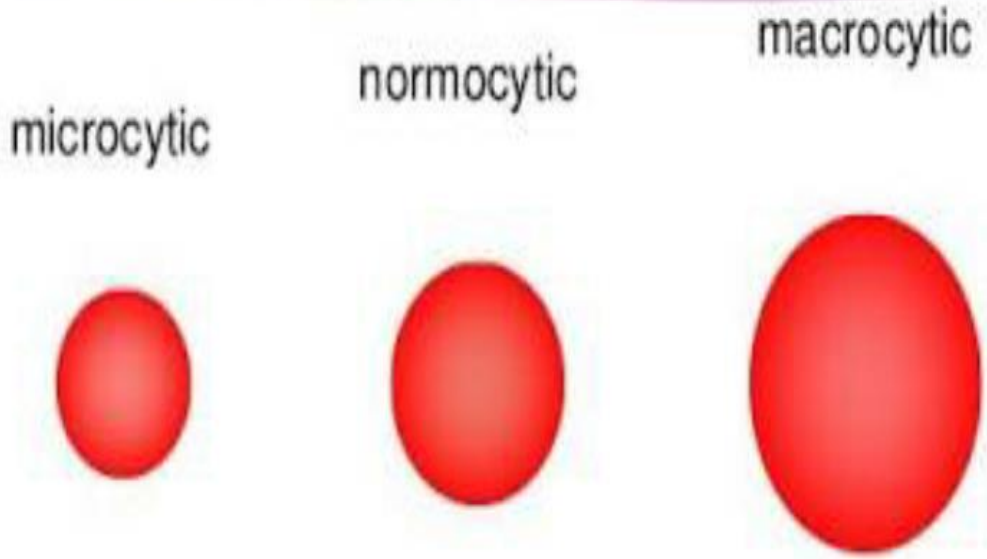


Table 15. 2. Morphologic classification of the anaemia

Type of Anaemia Based on RBC Size and Haemoglobin Content	RBC Morphology	Peripheral Smear	Red Cell Indices	Most Common Caus
Normocytic Normochromic	Normal cell Normal Hb		Reduction in RBC number; normal MCV, MCH & MCHC	Haemorrhage; haemolysis; lack of blood formation; d tion of blood with fluid
Macrocytic Normochromic	Large cell Normal Hb		Increased MCV and MCH; normal MCHC	Lack of erythrocyte-ma factors ('extrinsic' and 'intrinsic' factors)
Microcytic Normochromic	Small cell Normal Hb		Reduced MCV and MCH; normal MCHC	Thalassaemia syndro
Microcytic Hypochromic	Small cell Reduced Hb		Reduced MCV, MCH and MCHC	Iron deficiency

C)Kinetic Classification of Anaemia

I)Impaired erythrocyte production(Reticulocyte production index less than 2):

A.Hypoproliferative:

i)Iron-deficient erythropoiesis

- Iron deficiency anaemia
- Anaemia of chronic disorders

ii)Erythropoietin deficiency:

- Renal disease
- Endocrine deficiences

iii)Hypoplastic Anaemia:

- Aplastic Anaemia
- Pure red cell aplasia

iv)Infiltration

- Leukemia
- Metastatic Carcinoma
- Myelofibrosis

B) Ineffective:

i)Megaloblastic

- Vitamin B12 deficiency
- Folate deficiency
- Other causes

ii)Microcytic

- Thalassemia
- Certain sideroblastic anaemia

iii) Normocytic

II)Increased erythrocyte production(reticulocyte production index greater than 3):

i)Hemolytic anaemia:

- Heriditary
- Acquired

ii)Treated nutritional deficiences

Types of Anaemia

Iron deficiency Anaemia

Megaloblastic Anaemia

- a)Vitamin B12 deficiency anaemia
- b)Folate deficiency Anaemia

Aplastic Anaemia, Hemolytic Anaemia

Sickle cell anaemia

Thalassaemia

Polycythaemia

Iron deficiency Anaemia

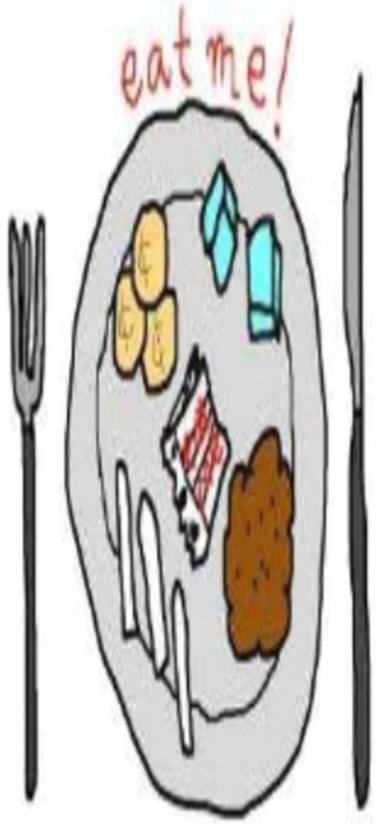
- Occurs due to excessive loss of iron
- Iron is the essential part in Hb
- Low iron levels result in decreased incorporation of haemoglobulin into red blood cells

Causes:

1. Insufficient dietary iron intake
2. Absorption of iron
3. Losses due to diseases
4. Bleeding lesions of GIT
5. Parasitic infections

PICA

When what you crave is not fit for human consumption



Plummer –Vinson Syndrome

- Also called as Sideropenic dysphagia/ Patterson- Kelly syndrome
- Occurs due to long standing cases of iron deficiency
- Characteristic features are:

1. Iron deficiency anaemia
2. Glossitis
3. Koilonychia
4. Postcricoid web results in dysphagia.(dysphagia is intermittent process more for solid substances than liquids

Sideroblastic Anaemia

- This type of anaemioa occurs due to iron overload
- They are either inherited or acquired type
- Inherited(hereditary disorder) due to disturbance in X-linked disorder
- Acquired due to

1. Idiopathic
2. Drugs
3. Alcohol abuse
4. Lead toxicity
5. Other diseases(rheumatoid arthritis / Carcinoma)

Megaloblastic Anaemia

- Occurs due to deficiency of either Vitamin B12 ,Folic acid or both
- Deficiency of these two substances red bone marrow produces abnormal rbc's called as Megaloblast
- Vitamin B12 is essential for maintanence of normal integrity of the nervous system

Pernicious Anaemia

- Inability of stomach to absorb vitamin b12 in small intestine due to the lack of intrinsic factor needed for the absorption
- Probably autoimmune disorder with genetic predisposition producing blocking antibodies for intrinsic factors
- Also found to be associated with human leucocyte antigen and helicobacter pylori

Aplastic Anaemia (Bonemarrow by itself is unable to produce cells)

- Aplastic Anaemia is defined as a condition in which an acellular or markedly hypocellular bonemarrow results in pancytopenia(deficiency of all three components of blood)
- No abnormal cells are seen

Classification

- Congenital - Fanconi'sAnaemia,
  -Dyskeratosis congenita

- Acquired –Primary or Idiopathic (no definite cause)

  -Secondary (definite or likely agent can be identified)

Secondary- Etiological agent

- Drug induced
  - *Cytotoxic Drugs*-Alkylating agents,antimetabolities
  - *Antibacterial drugs*-*Sulphonamides,Isoniazide Arsenicals*
  - *Antirheumatic-phenylbutazone,diclofenac,gold salts*
  - *Transquillers-promazine,meprobamate*
  - *Miscellaneous drugs-Benzine,lindane,hydralazine*
- Chemicals-Benzene,lindane,DDT
- Viral infections-Viral hepatitis,infectious mononucleosis,HIV,Paravovirus
- Miscellaneous-pancreatitis,Radiation,Eosinophilic fascitis

Classification

Due to intra-erythrocyte defect:

- Congenital- Membrane defect(Spherocytosis, elliptocytosis)

  -Enzyme defects(pyruvate kinase,G6PD)

  -Haemoglobin defects(sickle cell anaemia,thalassaemia)

- Acquired-paroxysmal nocturnal hemoglobinuria

- Due to extra-erythrocytic defect:
  - Antibodies
  - Drugs
  - Infections
  - Inflammatory and neoplastic disease

Haemorragic Anaemia(Blood loss leads to loss of RBC's)

- Acute blood loss anaemia, also called haemorragic anaemia

- It's a specific type of anaemia that sufficient decrease in red blood cells due to acute hemorrage (bleeding)

Hemolytic Anaemia(Lytic activity of produced RBC's)

- This type of anaemia results from an increased rate of red cell destruction which are not in normal shape and rigidity due to various defects in process called extravascular hemolysis

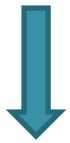
- The normal life span of RBC's (90-120 days) are shortened

Sickle cell Anaemia(Genetic alternation)

- Heriditary disease worldwide,transmitted through autosomal dominant triat
- Single point mutation occurs in beta globulin gene that alters the structure of hb produced
- Abnormal variant produced is called sickle cell haemoglobin (Hb –S)
- Sickle cell Triat –Hetrozygous state (Hb AS)

Thalassaemia

Inherited defects occurs due to the mutation occurring in hemoglobin genes



leads to reduced and defective haemoglobin production

- Chromosome 16 regulates the controlled production of alpha chains
- Chromosome 11 regulates the controlled production of beta chains
- Both alpha and beta globulin chains are produced in equal propotion

- Mutation occuring in these chromosome leads to dispropotion of alpha and beta chains
- Alpha thalassaemia-Complete absence of alpha globulin or particular copy of gene is absent
- Beta thalassaemia

1. Also called Cooley's anaemia
2. Inherited recessive pattern
3. Mutation occurs in beta globulin gene leads to complete loss or reduced amount of beta globulins

Polycythemia

- Its an abnormal increase in number of erythrocytes and haemoglobulin

1. Relative polycythemia- concentration of red cells becomes grater than normal(total cell mass remains the same)
2. Absolute polycythemia-increase in the total red cell mass.Two forms

- Primary
- Secondary

Polycythaemia	Polycythaemia Vera
• Only increase in rbc's	• Clonal stem cell disorder characterized by increased production of all myeloid elements

Erythroblastosis Fetalis

Congenital type of hemolytic anaemia due to Rh incompatability results from the destruction of foetal blood brought about by the reaction between maternal and foetal blood factors

Summary of all anaemias

- Deficiency Anaemia

1. Iron deficiency
2. Vitamin b12 deficiency
3. Folic acid deficiency

- Hemolytic Anaemia(Mutation in globulin chains)

1. Sickle cell anaemia
2. Thalassaemia

- Bone marrow defect

1. Aplastic anaemia

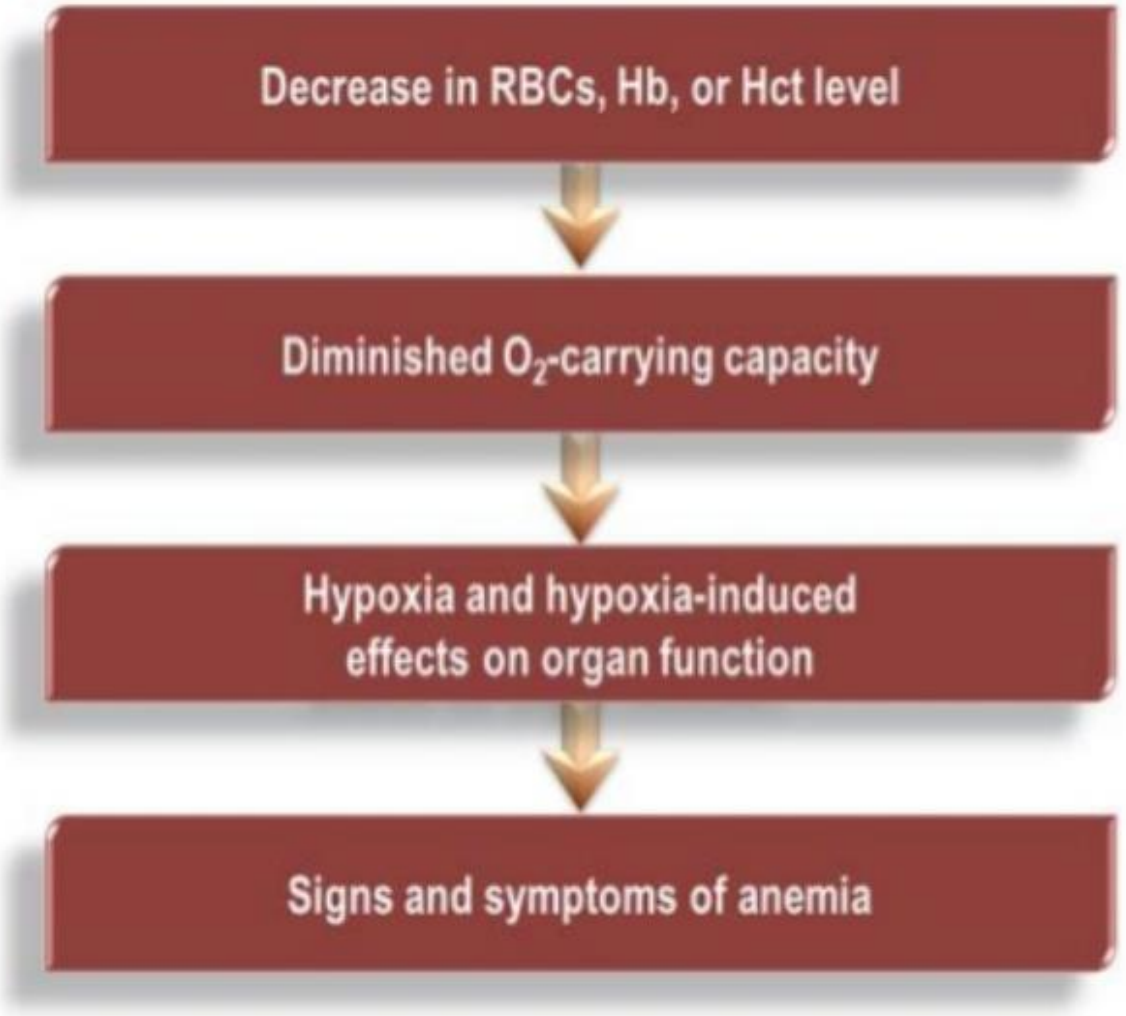
- Increased production of RBc's and Haemoglobin

1. Polycythemia

Other Names of Each Anaemia

Iron deficiency Anaemia and Plummer-Vinson Syndrome	• **Patterson –Brown Kelly syndrome** • **Paterson – kelly syndrome** • **Sideropenic dysphagia**
Vitamin B_{12} Deficiency	• **Pernicious Anaemia** • **Addisonian anaemia** • **Biermer anaemia** • **Hunter-addison anaemia** • **Lederer anaemia** • **Biermer-Enrlich anaemia** • **Addison-Biermer disease**
Thalassemia	• **Cooley's anaemia** • **Mediterranean Anaemia** • **Erythroblastic Anaemia**
Sickle cell Anaemia	• **Sickle cell disease**
Polycythemia Vera	• **Polycythemia rubra vera** • **Erythremia** • **Vaquez's Disease** • **Osler's disease**

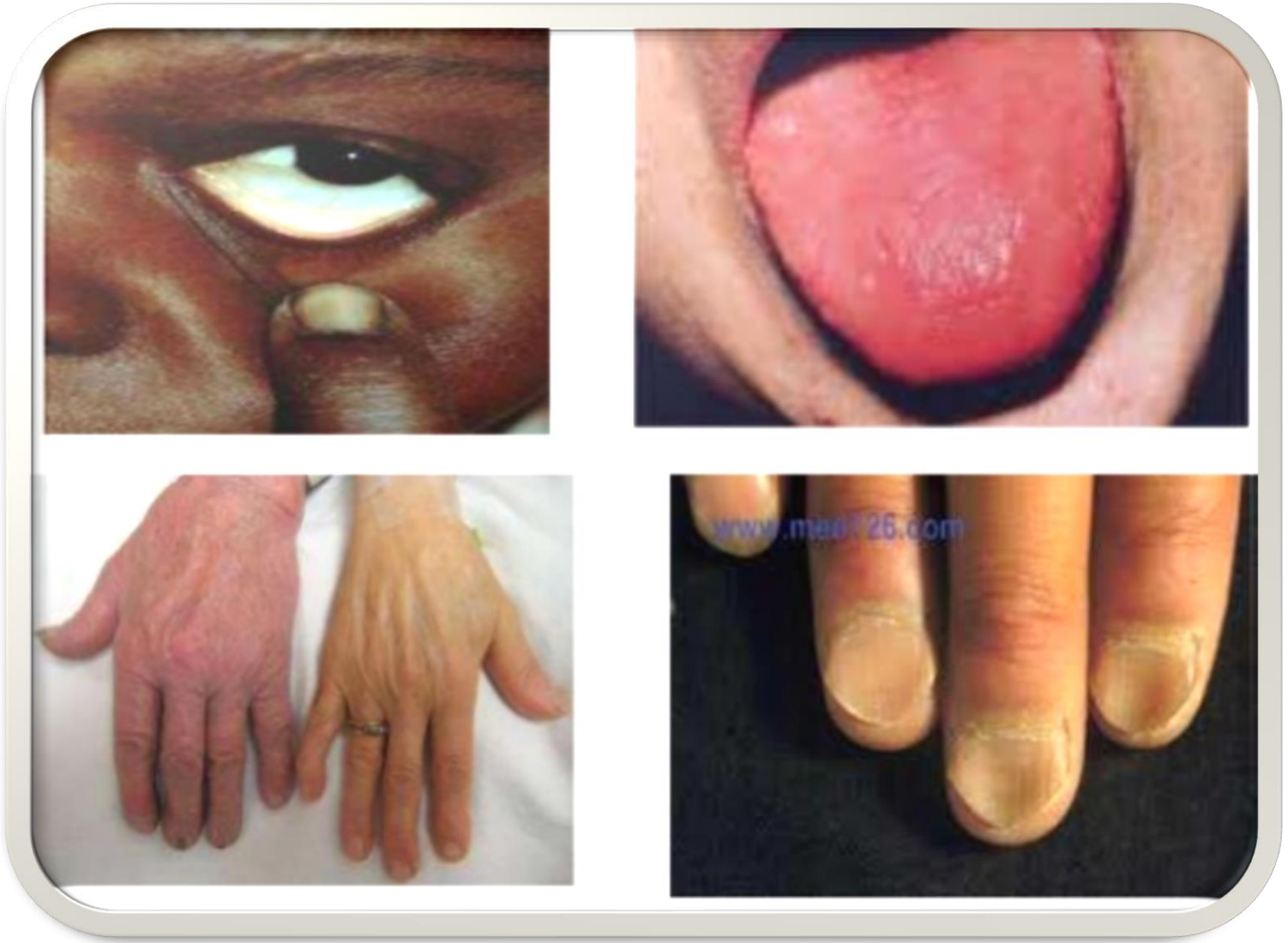
Pathophysiology of Anaemia



Signs And Symptoms

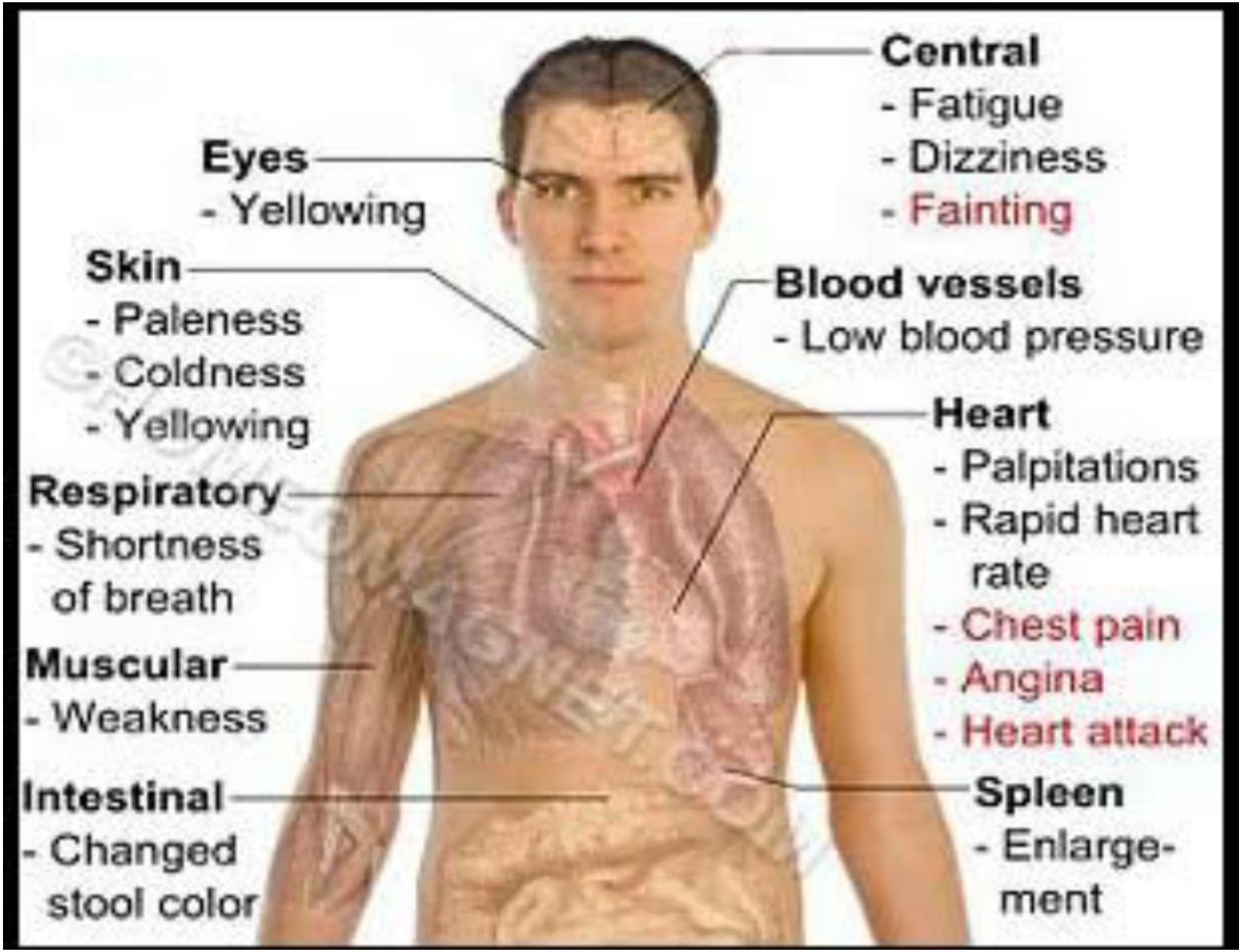
Signs of Anaemia

- Brittle nails
- Koilonychia (spoon shaped nails)
- Atrophy of the papillae of the tongue
- Angular stomatitis
- Brittle hair
- Dysphagia and Glossitis
- Plummer vinson/kelly patterson



Symptoms of Anaemia

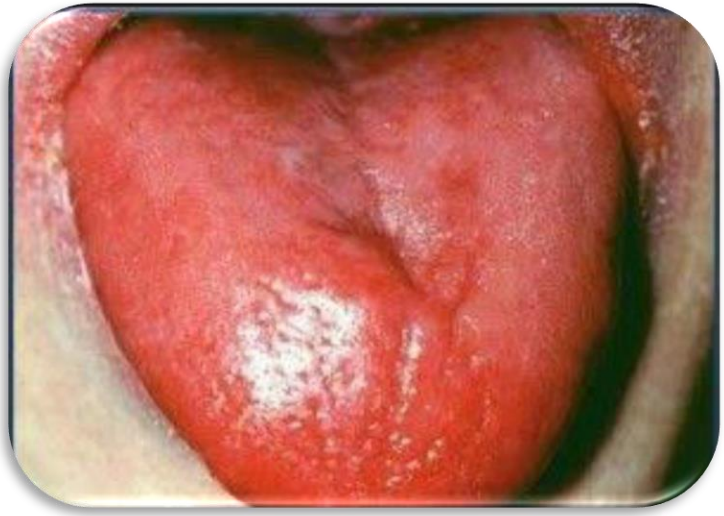
- Easy fatigue and loss of energy
- Unusually rapid heart beat, particularly with exercise
- Shortness of breath and headache, particularly with exercise
- Difficulty concentrating
- Dizziness
- Pale skin
- Leg cramps
- Insomnia


Central
- Fatigue
- Dizziness
- Fainting
Eyes
- Yellowing
Skin
- Paleness
- Coldness
- Yellowing
Blood vessels
- Low blood pressure
Heart
- Palpitations
- Rapid heart rate
- Chest pain
- Angina
- Heart attack
Respiratory
- Shortness of breath
Muscular
- Weakness
Intestinal
- Changed stool color
Spleen
- Enlarge-ment

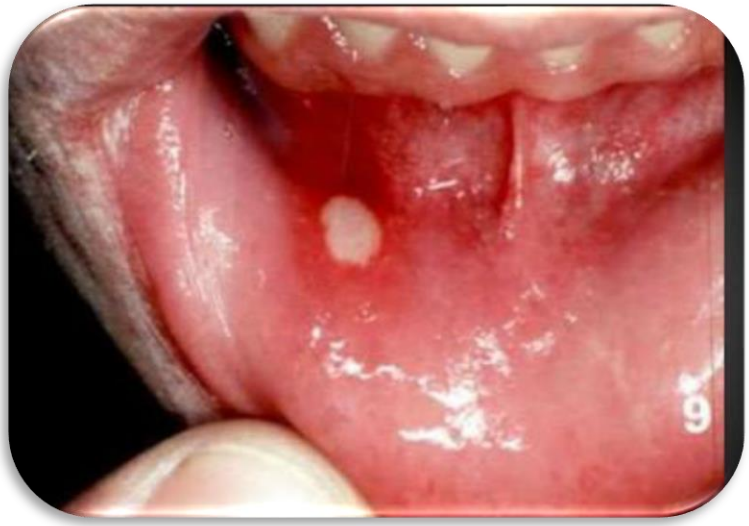
Oral Manifestations

Variants of Anaemia	Oral manifestations
Pernicious Anaemia/ Celiac Sprue	• Painful tongue • Glossitis (Intially beffy red tongue gradually leads to atrophy of papilla called Bald tongue- Hunter's glossitis or Moeller's glossitis • Burning sensation extend from tongue to oral mucosal sites • Small,shallow apthous ulcers
Aplastic Anaemia(Lack of inflammatory response)	• Petechiae • Purpuric spots • Frank haematomas • Haemorrage into oral cavity • Spontaneous gingival bleeding • Severe form of ulcers (Gangrene)
Thalassemia	• Prominence of pre-maxilla • Obvious Malocclusion • Pallor of mucosal surface
Sickle Anaemia	Bony changes evident

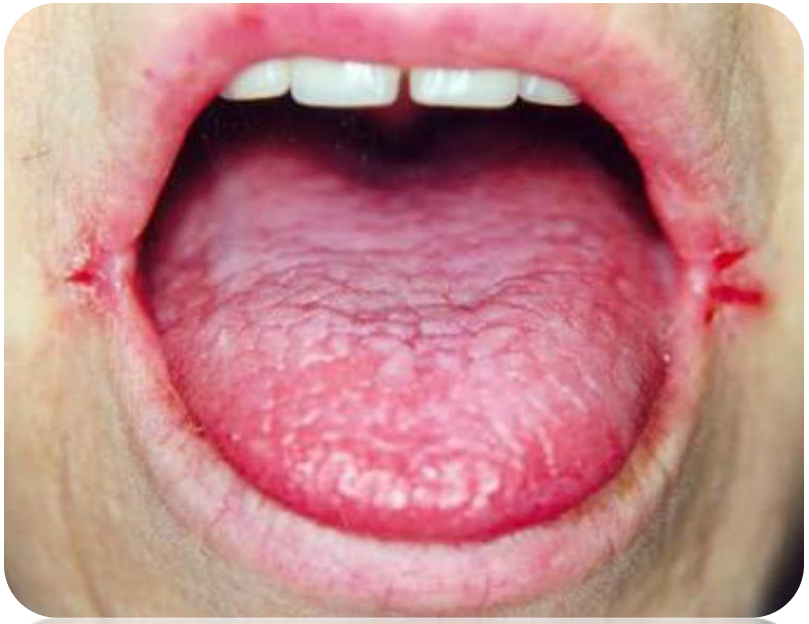
Iron deficiency anaemia	• **Cracks or fissures at the corner of mouth-Angular chelitis** • **Glossitis** • **Depapillation of tongue**
Polycythemia Vera	• **Oral mucous membrane appears deep purplish red** • **Gingiva are engorged and swollen bleeds spontaneously on slight provocation** • **Submucosal petechiae,ecchymoses,hematomas** • **Intercurrent infections occcur** • **Cyanosis 9Due decreased haemoglobin)**
Erythroblastosis Fetalis	• **Deposition of blood pigment on teeth giving them green,brown or blue hue** • **Enamel hypoplasia also present involving** **a)anterior tooth incisal edge** **b)Middle portion of deciduos cuspid and first molar crown** **In region characteristic ring like defect occurs called as Rh hump by Watson**



Glossitis

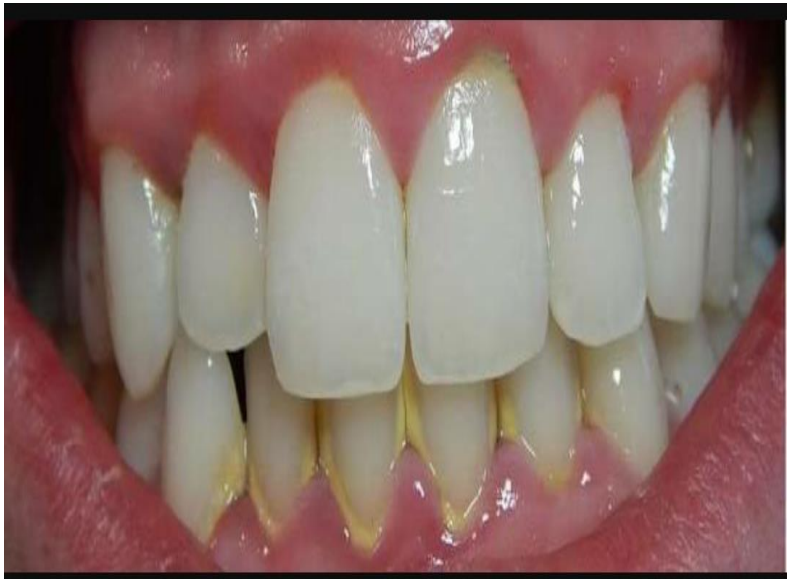


Apthous Ulcer

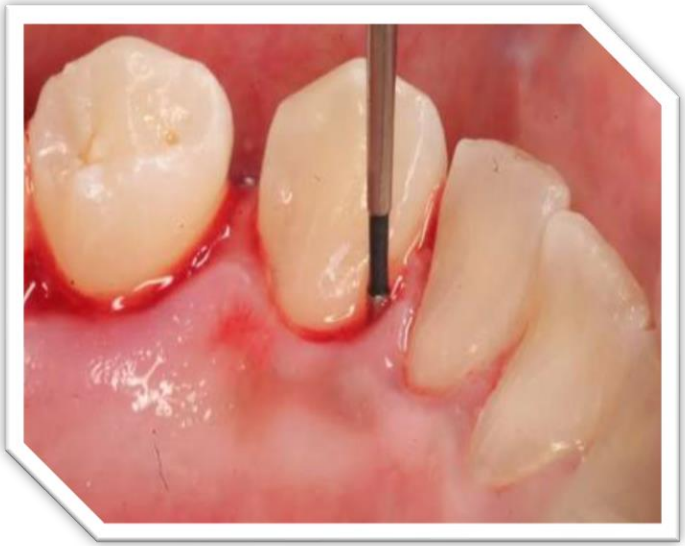


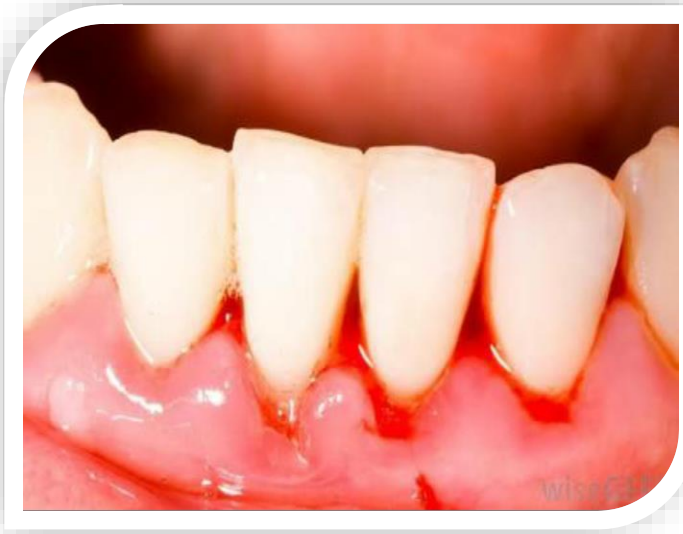
Angular chelitis

BOP is positive associated periodontal problem

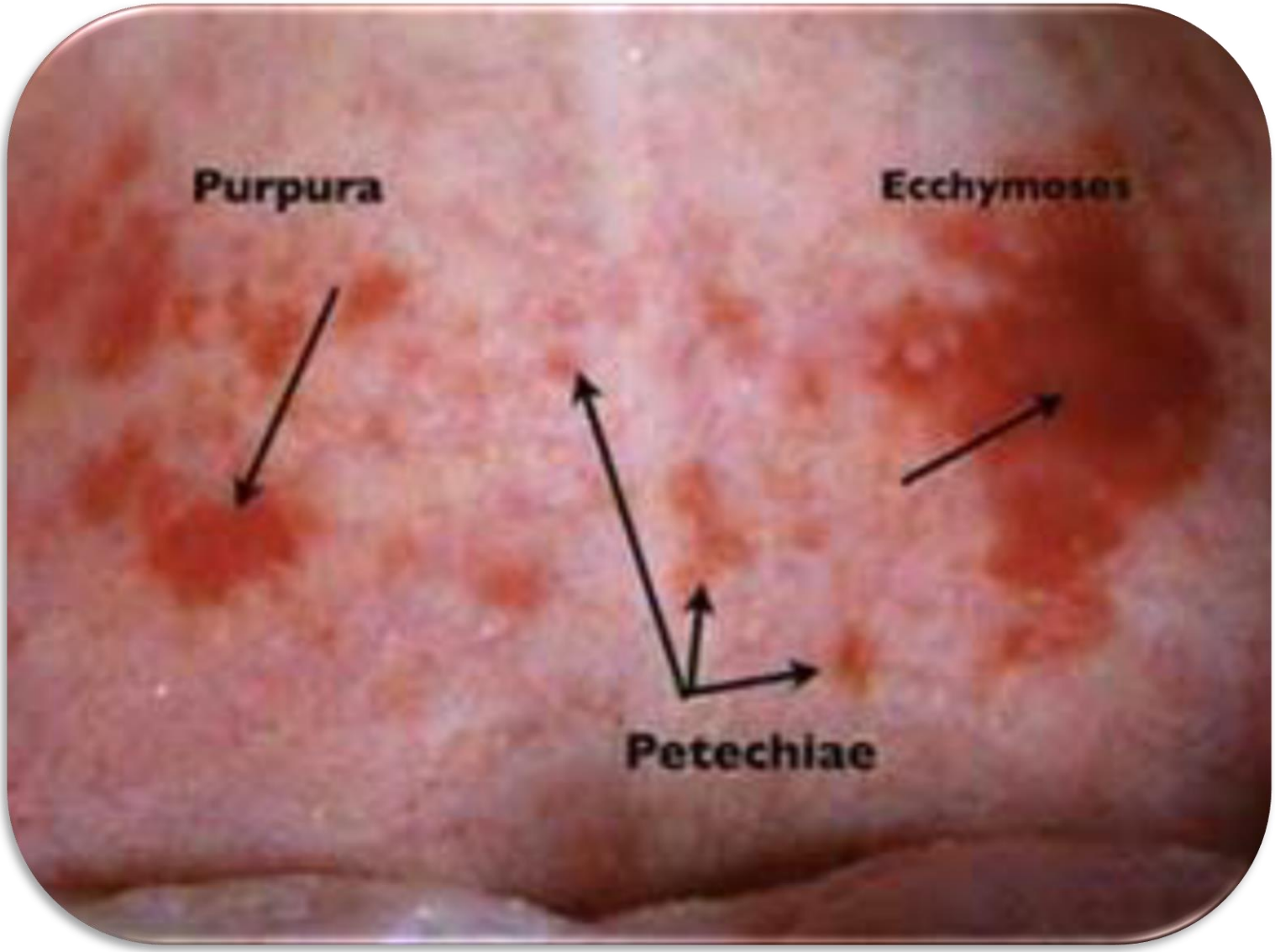


Periodontal problem





Healthy gingiva with spontaneous bleeding


Purpura
Ecchymoses
Petechiae

Radiographic features

- Bony deformities are present in Sickle cell Anaemia and Thalassaemia they have unique Radiographic Features like

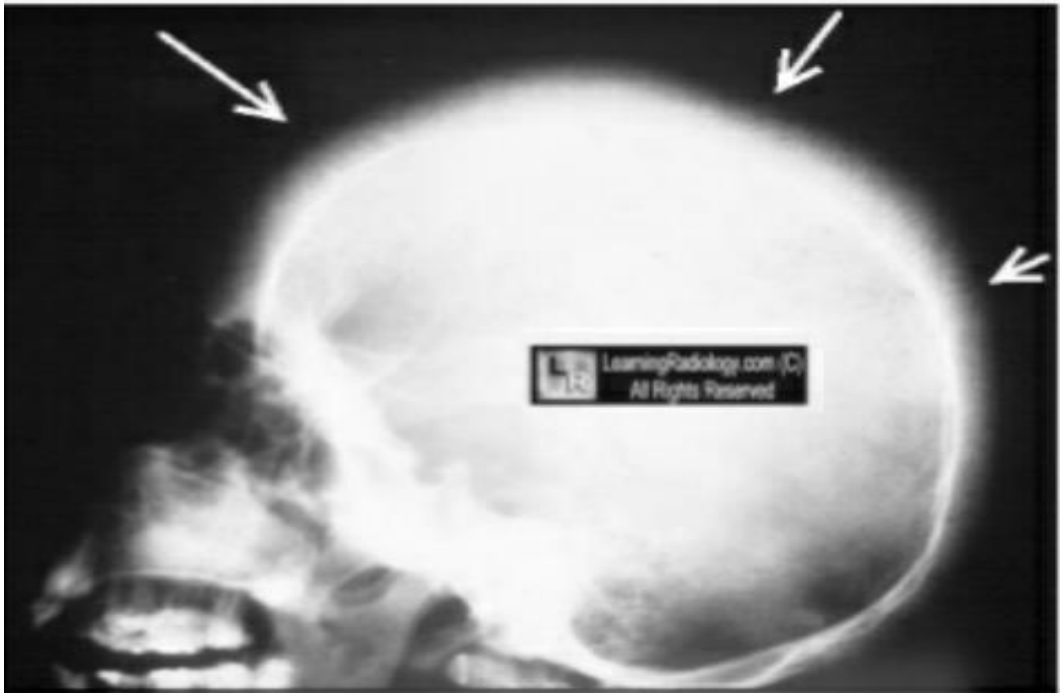
1. Hair on end appearance in skull radiograph

2. Rib within-a- rib in chest radiograph

3. Step-ladder appearance in mandibular intra-oral radiographs

a)Hair on end appearance

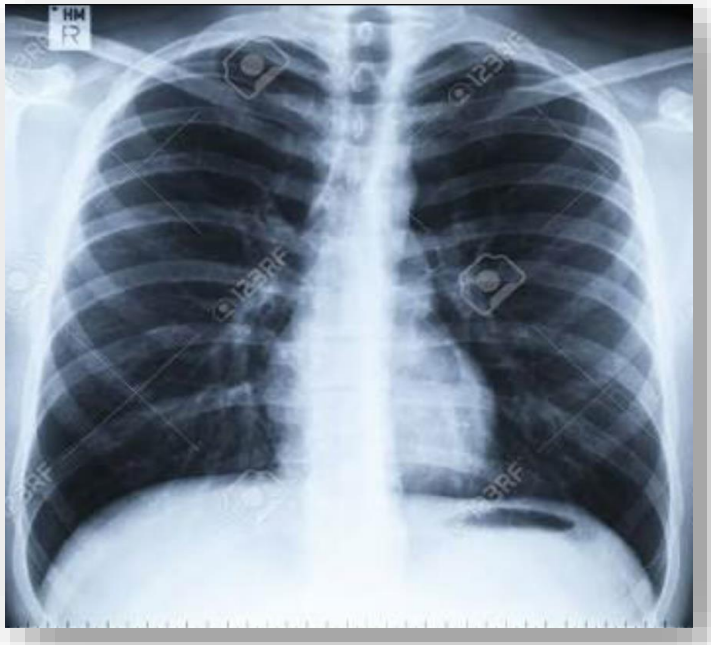
This appearance is due to the extreme thickening of dipole which leads to the poor differentiation of outer and inner plates of skull with elongation of inbetween trabecule



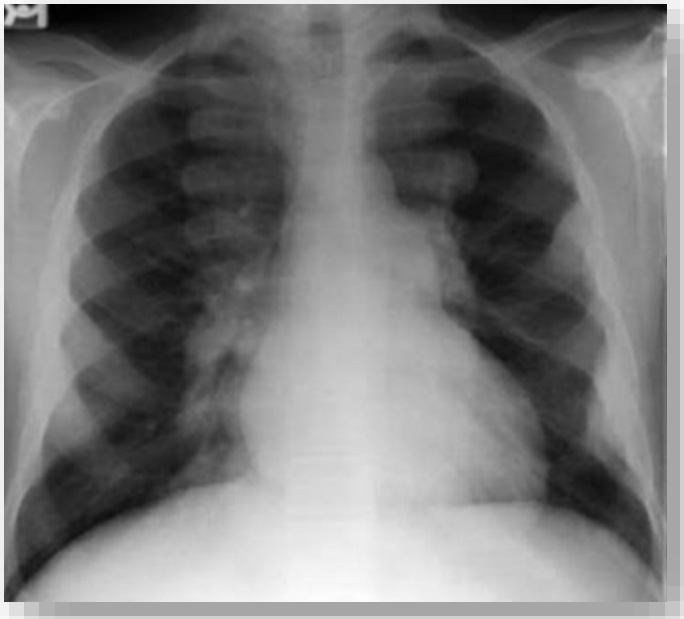
b) Rib within- a- Rib

- This feature occurs due to the overlapping of medullary spaces within the ribs
- This is noted in the middle and anterior portion of ribs

Normal

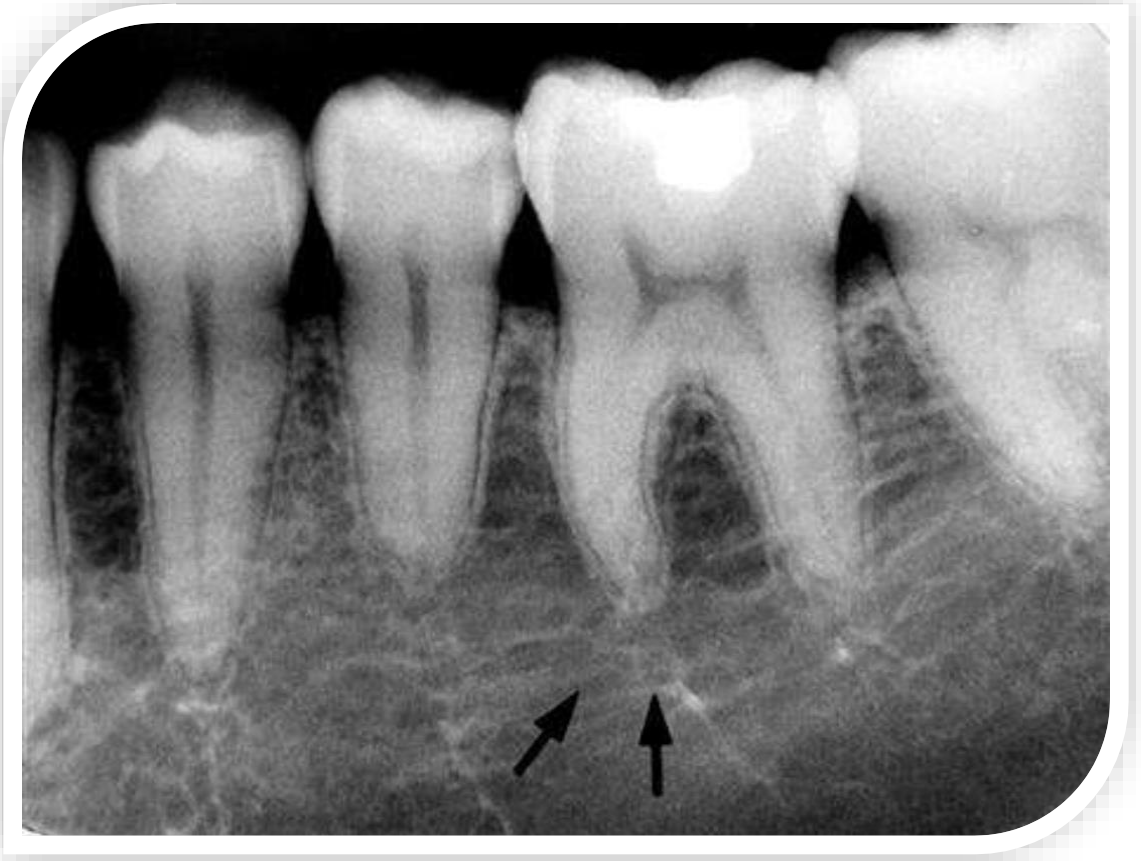


Pathological



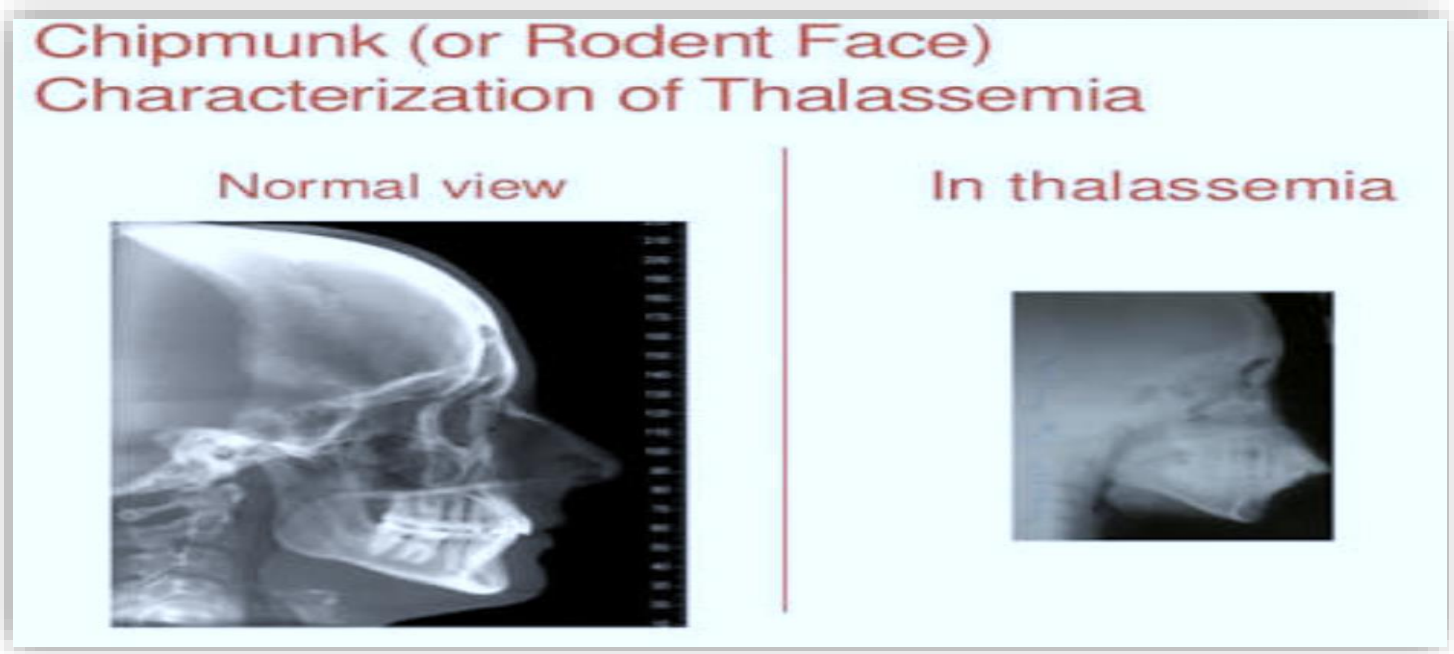
c) Step-Ladder appearance

- This is due to the increased intertrabecular spacing



Mongoloid Feature of Thalassaemic patient

- Due to the prominence of cheek bones,protrusion or flaring of maxillary anterior teethand the depression of the bridge of the nose



Other features of sickle cell anaemia

- Bone within bone appearance-Area of decalcification surrounded by reactive sclerosis that is subsequently demarcated from remaining bone by a radiolucent area
- Mandibular inferior border is thinned out

Laboratory Investigations

- Blood investigations
- Peripheral blood smear
- Exfoliative cytology
- Bone marrow Biopsy

A)Blood investigations

The red cell population is defined by

1.Quantitative parameters:

- Volume of packed cells i.e. the hematocrit
- Hemoglobin concentration
- Red cell concentration per unit volume.

2.Qualitative parameters:

- Mean corpuscular volume
- Mean corpuscular hemoglobin
- Mean corpuscular hemoglobin concentration.

- Hematocrit (Packed cell volume): It is the proportion of the volume of blood sample that is occupied by RBCs.
  - Men -42-52%
  - Women -36-48%

- Cell Volume Hemoglobin Concentration: It is the amount of hemoglobin per unit volume of blood.(Gms/Dl)
  - Women - 12-16gms/dl
  - Men - 14-17 gms/dl

- Red Cell Count: Total number of Red Cells per unit volume of blood sample. [No.of RBC/ cu.mm]
  - Men - $4.2\text{-}5.4*10^{6}/mm^{3}$
  - Women- $3.6\text{-}5.0* 10^{6}/mm^{3}$

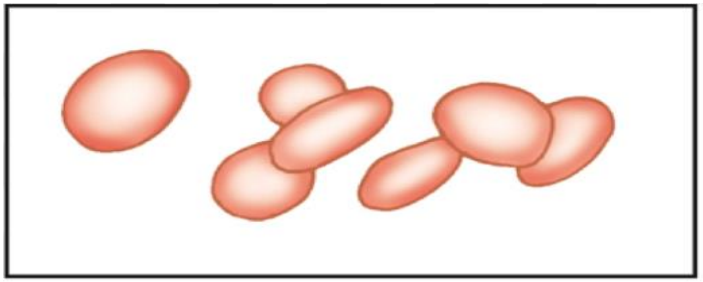
- Mean Corpuscular Volume: It is the average volume a RBC. [fL]
  - Normal 82-98mm^3or 82-98fL

- Mean Corpuscular Hemoglobin: It is the average hemoglobin content per RBC.
  - Normal value is 27 to 31 pL

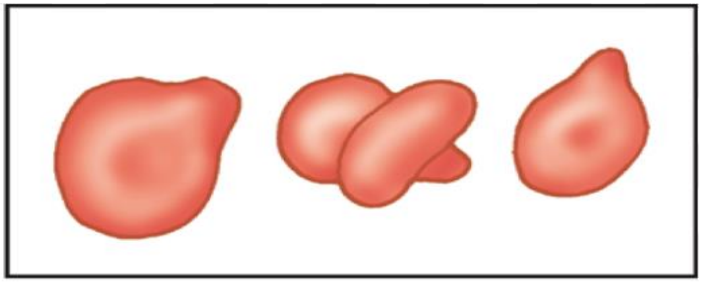
- Mean Corpuscular Hemoglobin Concentration: It is the average concentration of hemoglobin in a given Red Cell Volume. [Gms/ dL]
  - Normal 32-36 g/Dl

B) Peripheral Blood Smear

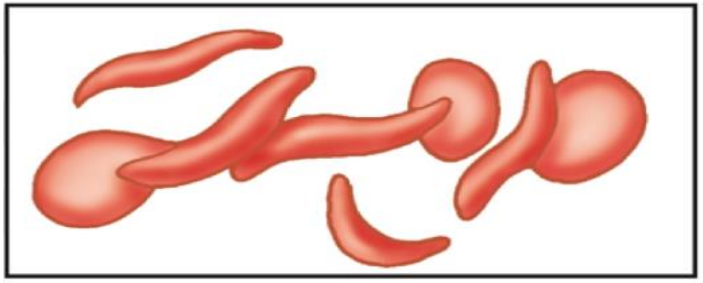
- This is the specific method done to diagnose the type of anaemia using the morphological structures of rbc's



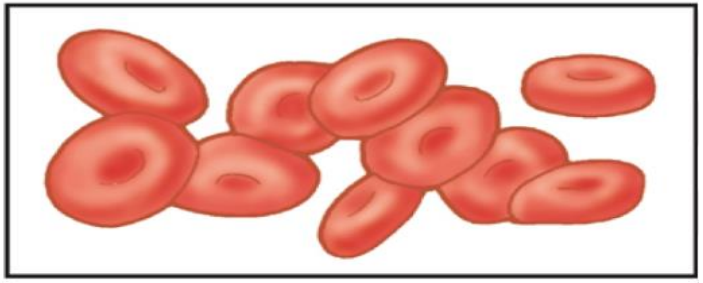
A Iron-deficiency anemia



B Megaloblastic anemia



C Sickle cell disease



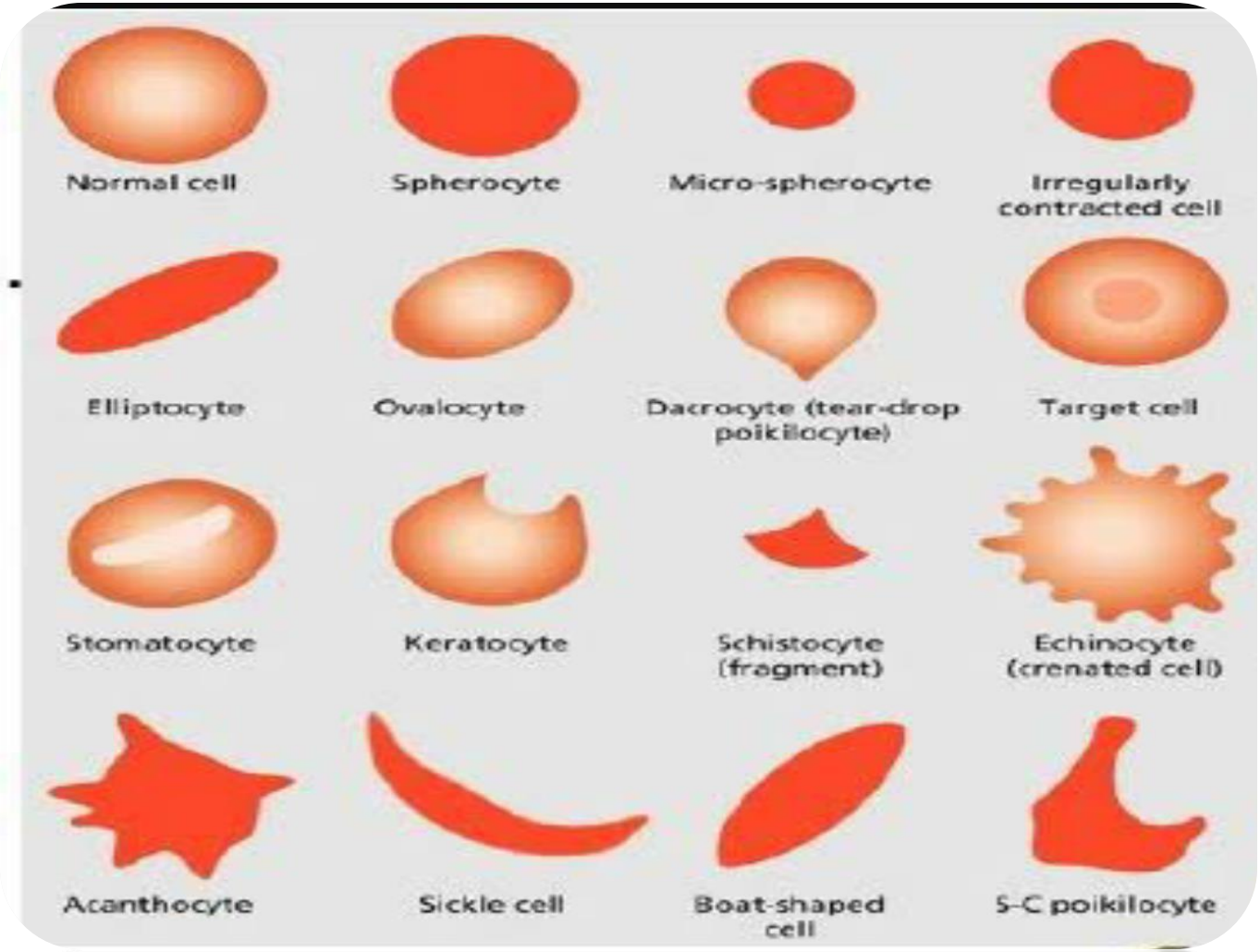
D Normal

Degenerative forms of RBC

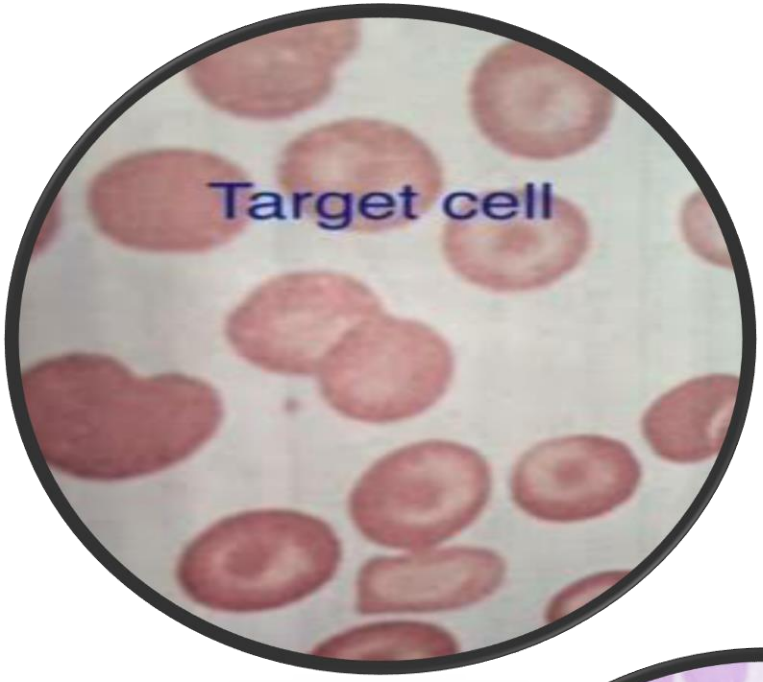
Poikilocytosis – abnormal variation in shape

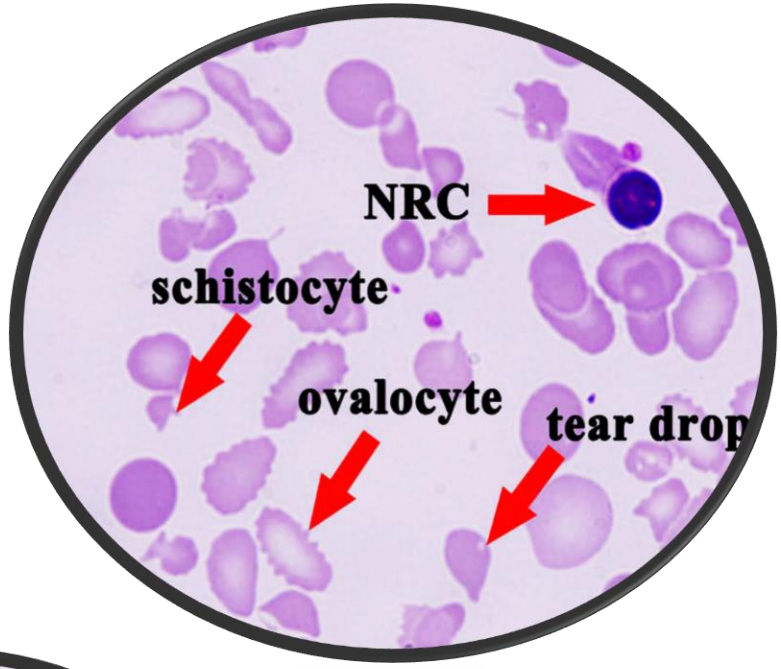
Anisocytosis – abnormal variation in size

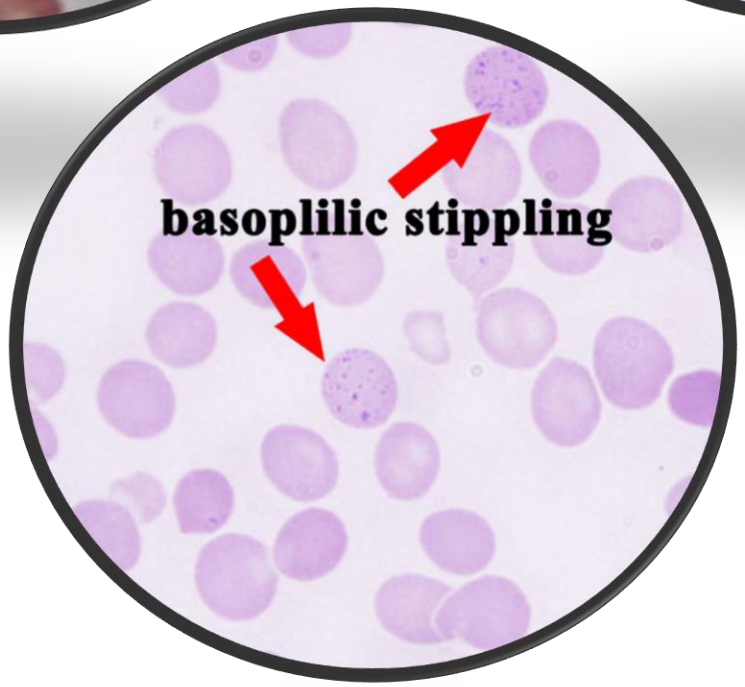
Different types of Poikilocytosis



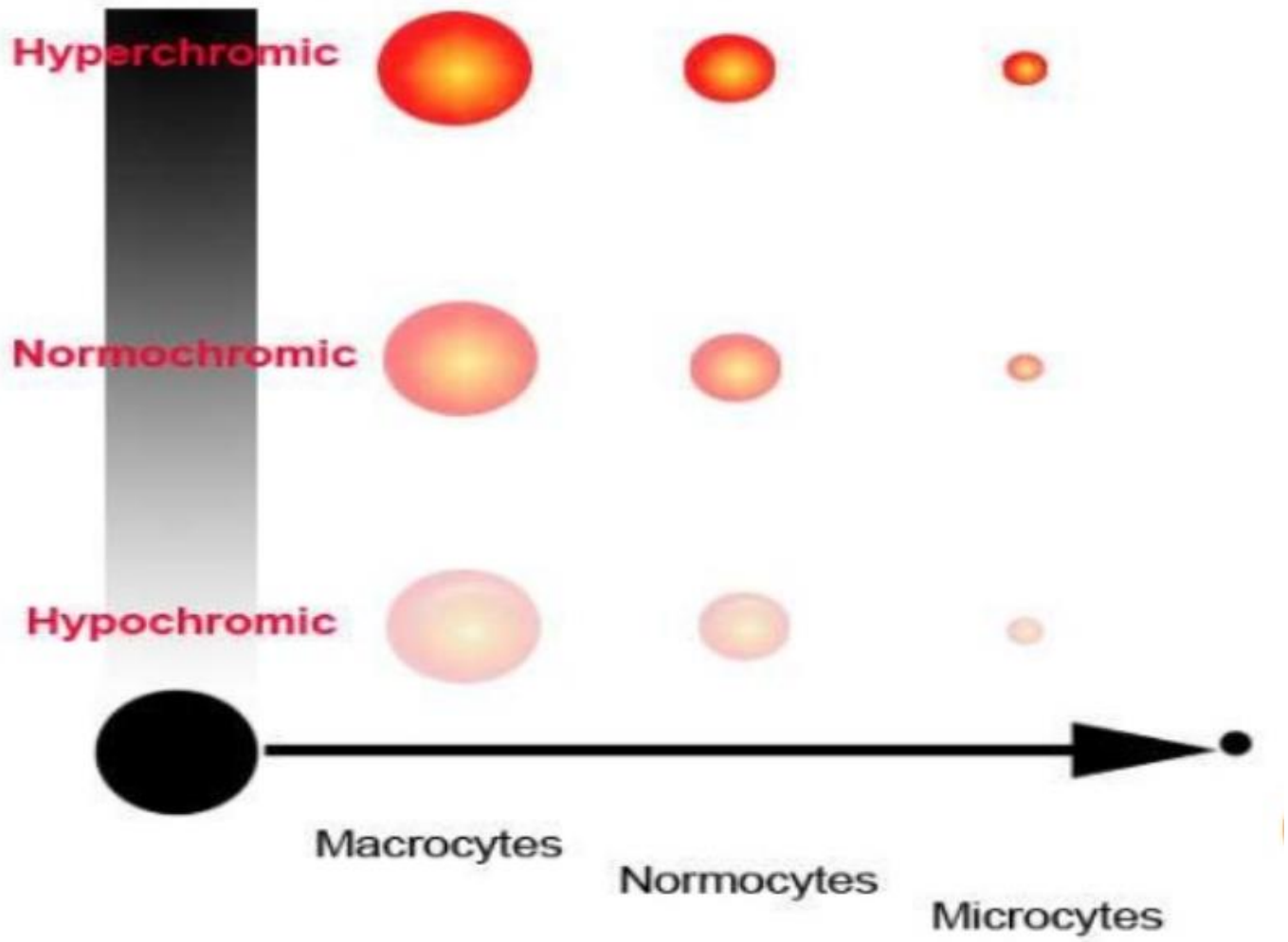
- Iron deficiency Anaemia-pencil like erythrocytes and certain other types are seen
- Vitamin B12 deficiencies- Ovalocytes type is seen.Howell-Jolly bodies seen
- Sickle cell Anaemia- Sickle cells are seen
- Thalassaemia – Basophilic stippling,fragmented cells,target cells are seen
- Sideroblastic Anaemia- Sideroblast type


Target cell


NRC
schistocyte
ovalocyte
tear drop


basoplilic stippling

Anisocytosis



C)Exfoliative Cytology

- Scrapings from buccal mucosa of anaemic patients shows significant alternation in nuclei of epithelial cells

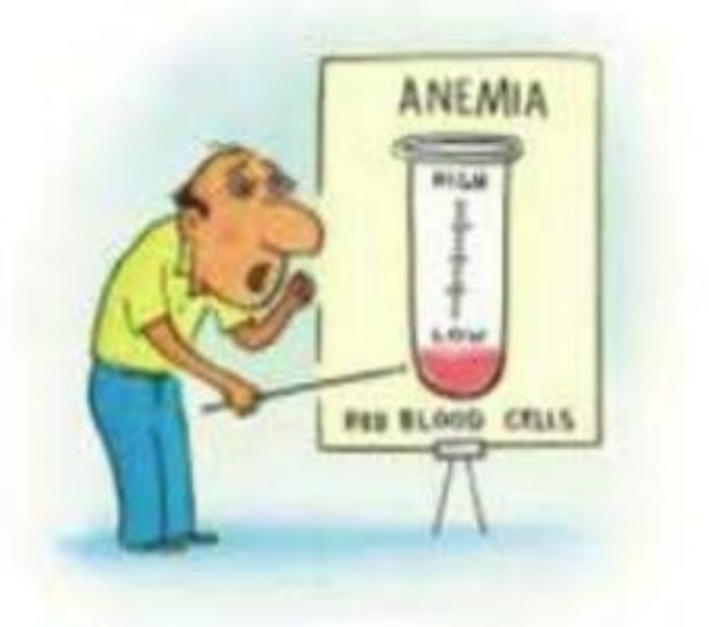
- Features like:

1. Nuclear enlargement
2. Binucleation
3. Abnormal chromatin distribution

Diagnosis

Anemia Diagnosis

- complete blood count.
- thorough evaluation of the patient
- Physical examination and medical history



WHO Grading of anemia

- Grade 1 (Mild Anemia): 10 g/dl
- Grade 2 (Moderate Anemia): 7-10 g/dl
- Grade 3 (Severe Anemia): below 7 g/dl

Treatment

- Treatment of anaemia depends on the type of anaemia
- In deficiency anaemia supplement of deficient compound treats the anaemia
- Whereas in case of other types like problem arising in bone marrow such as aplastic anaemia,sickle cell anaemia
- Thalassaemic anaemia, haematopoietic stem cell transplantation will be the definitive treatment

Pharmacological Management

TREATMENT OF MILD &MODERATE

GROUP	DOSAGE/day
Children 2-5 years	20-30 mg iron
Children 6-11 years	30-60 mg iron
Adolescents and adults	60 mg iron

- Anemia will correct within 2 to 4 months if appropriate iron dosages are administered and underlying cause of iron deficiency is corrected.
- Continue iron therapy an additional 4 to 6 months (adults) after the hemoglobin normalizes to replenish the iron stores.

TREATMENT OF SEVERE ANEMIA

AGE GROUP	DOSE	DURATION
<2 years	25 mg iron + 100-400 ug folic acid daily	3 months
2-12 years	60 mg iron + 400 ug folic acid daily	3 months
Adolescents and adults, including pregnant women	120 mg iron + 400 ug folic acid daily	3 months

- After completing 3 months of therapeutic supplementation, pregnant women and infants should continue preventive supplementation program

TREATMENT OF PREGNANT WOMEN

Prevalence of anemia in pregnancy	Dose	Duration
>40 % in population	60 mg iron + 400 ug folic acid daily	6 months in pregnancy, and continuing to 3 months postpartum

Non Pharmacological Treatment

- Tea and coffee inhibit iron absorption when consumed with a meal or shortly after a meal.
- Vitamin C (ascorbic acid) is also a powerful enhancer of iron absorption from nonmeat foods when consumed with a meal. The size of the vitamin C effect on iron absorption increases with the quantity of vitamin C in the meal.
- Germination and fermentation of cereals and legumes improve the bioavailability of iron by reducing the content of phytate, a substance in food that inhibits iron absorption.
- Promote and support exclusive breastfeeding for about 6 months followed by breastfeeding with appropriate complementary foods, including iron-rich through the second year of life.

Conclusion

"Anaemia should be taken seriously. It is more than just tiredness; it affects the entire body, especially in growing children"

Thank you