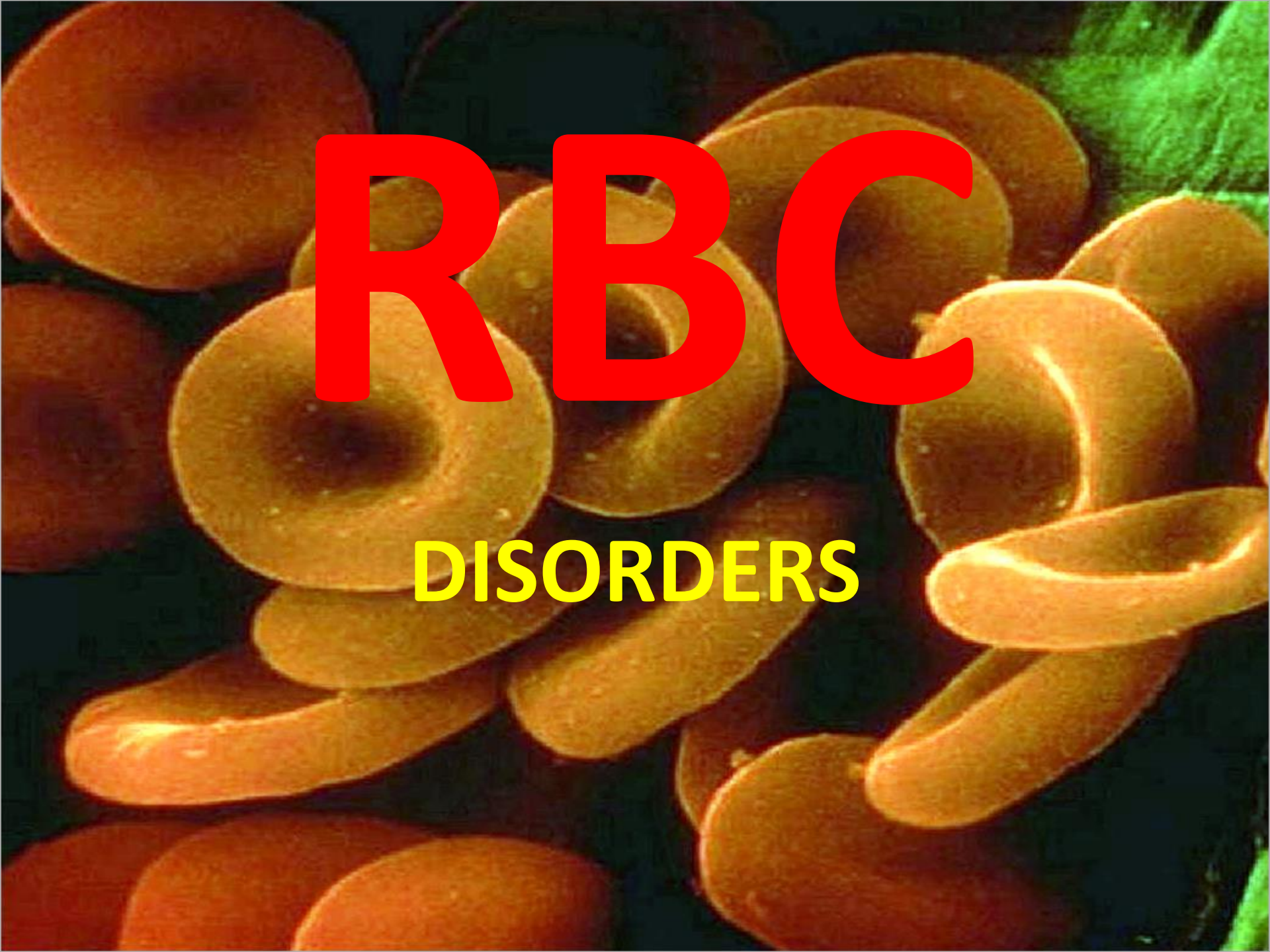

RBC
DISORDERS

ANEMIA

Ancient greek "anaimia" = ' ***lack of blood****"*

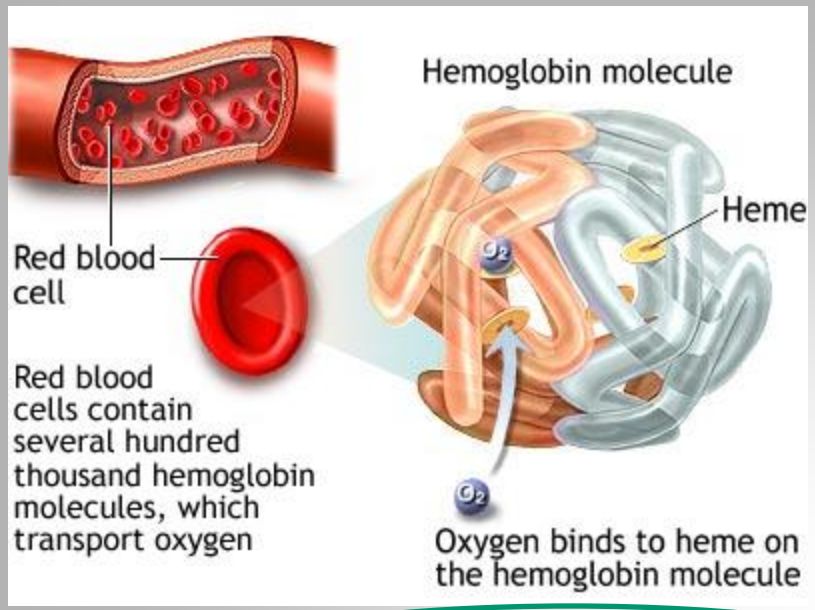
Blood hemoglobin level is below the normal range for the patient's age and sex

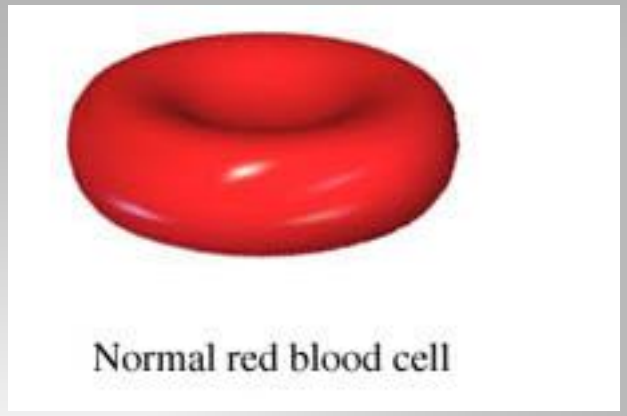
decrease in normal number of red blood cells (RBCs) or less than the normal quantity of hemoglobin in the blood.

↓

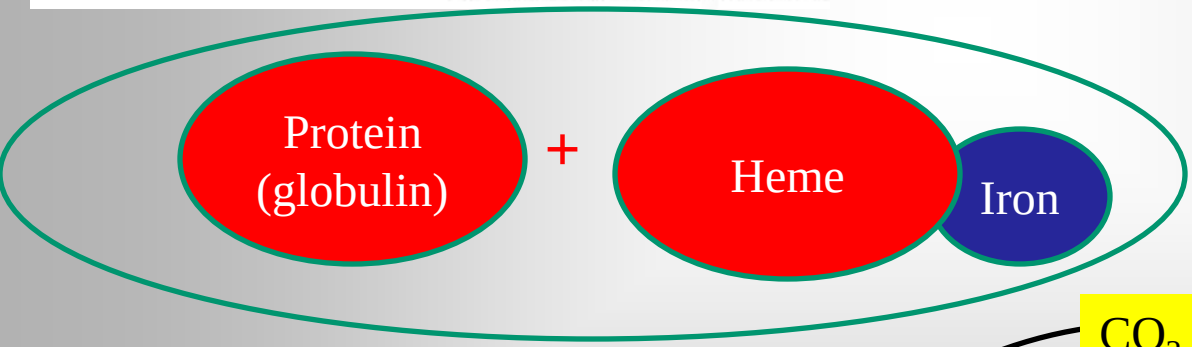
Decrease in the concentration of red blood cells or Hb in the peripheral blood

HEMOGLOBIN

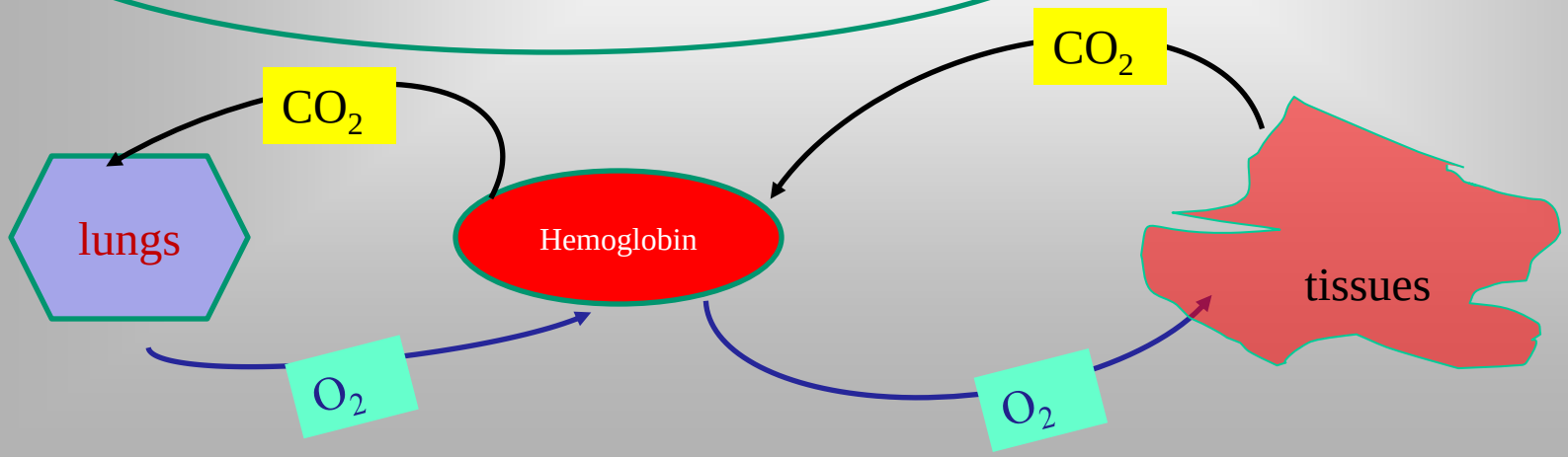




About 280 million Hb molecules



= Hemoglobin



Adult Reference Ranges for Red Blood Cells

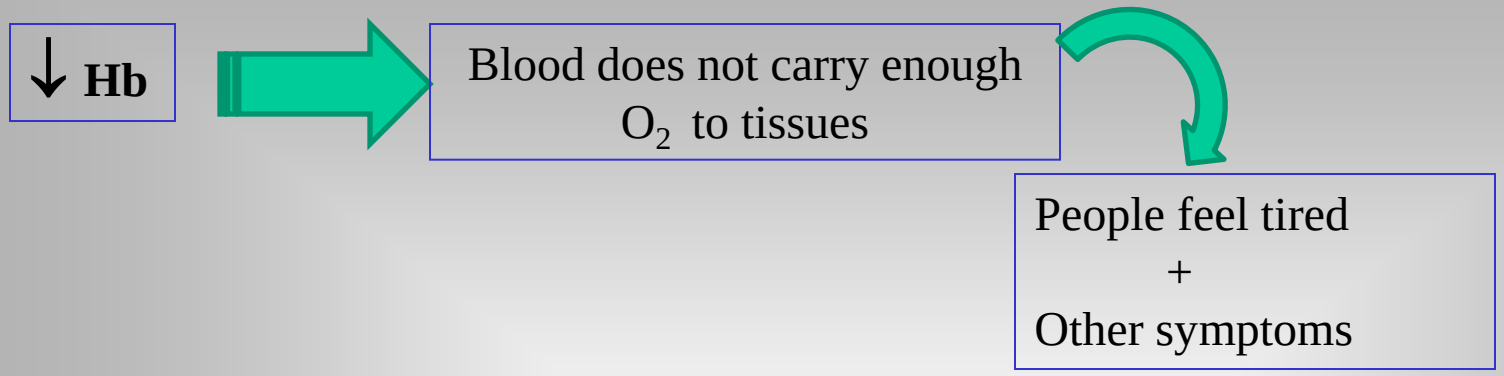
Measurement (units)	Men	Women
Hemoglobin (gm/dL)	13.6–17.2	12.0–15.0
Hematocrit (%)	39–49	33–43
Red cell count (10^6 /µL)	4.3–5.9	3.5–5.0
Reticulocyte count (%)	0.5–1.5	
Mean cell volume (μm^3)	82–96	
Mean corpuscular hemoglobin (pg)	27–33	
Mean corpuscular hemoglobin concentration (gm/dL)	33–37	

11 - 15

↓ **Hb** → Blood does not carry enough O_2 to tissues → People feel tired + Other symptoms

Hematocrit - The hematocrit level measures **how much** of the blood is made up of RBCs. The normal range for hematocrit levels for the general population is 32–43 percent. **A low** hematocrit level is another **sign** of **anemia.**

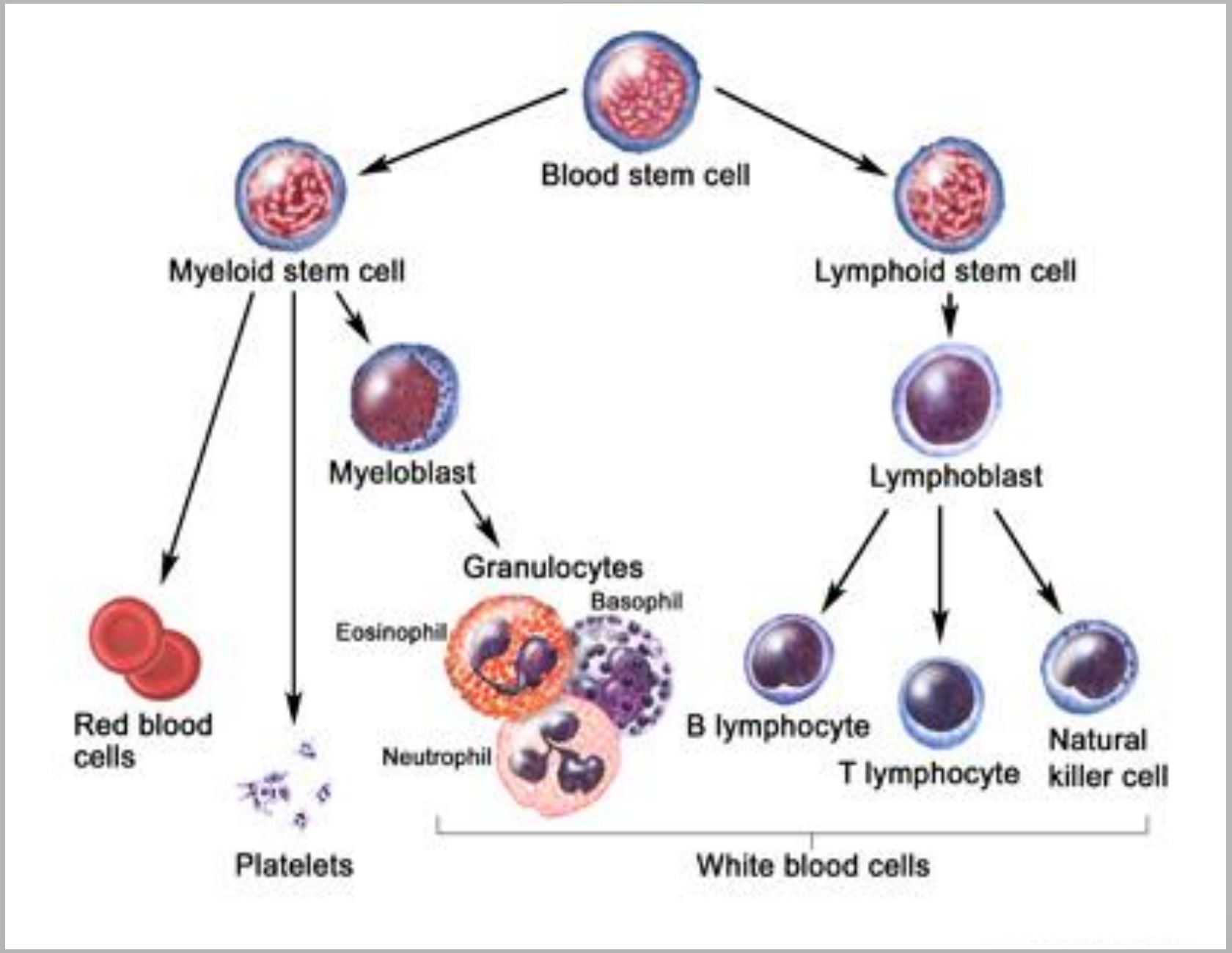
The number of RBCs - Too few RBCs means a person has anemia. A low number of RBCs is usually seen with either a low hemoglobin or a low hematocrit level, or both



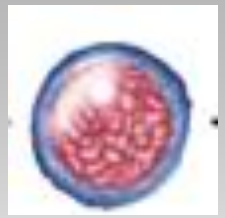
The mean cell volume - measures the **average size** (volume) of RBCs. In iron-deficiency anemia, the RBCs are usually smaller than normal. This is called **microcytosis.**

Reticulocyte count - Reticulocytes are young RBCs. This test measures the number of new RBCs in your blood. The reticulocyte test is used to determine whether your bone marrow is producing RBCs at the proper rate. A higher than average count usually indicates **either blood loss or destruction of RBCs earlier** than their normal life of 120 days. A **lower than average count indicates a decreased production of RBCs** by the bone marrow. People with pernicious anemia have low reticulocyte levels.

SOURCE


Blood stem cell
Myeloid stem cell
Lymphoid stem cell
Myeloblast
Lymphoblast
Granulocytes
Basophil
Eosinophil
Neutrophil
Red blood cells
Platelets
B lymphocyte
T lymphocyte
Natural killer cell
White blood cells

SOURCE



Erythroid stem cell

↓

Early normoblast

↓

Intermediate normoblast

↓

Late normoblast
(still a nucleated cell)

Extruded into circulation

Reticulocyte
(no nucleus, but ribosomes + mitochondria present)

→ **Erythrocyte**

Normal R.B.C

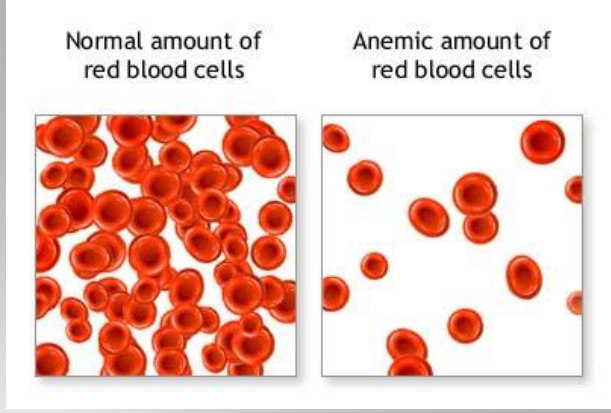
Normal size – **7 – 7.2 μ**

Biconcave, disc like non-nucleated cell

Normal life 120 days

Flexible cell membrane, destroyed in spleen, when it no longer has the ability to be flexible

Classification of **anemia**



I

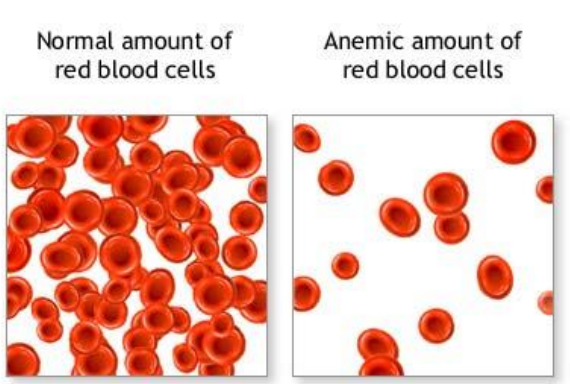
1. Due to blood loss, acute/chronic
2. Inadequate production of normal RBC's by bone marrow (hypoplasia, aplasia)
3. Excessive destruction of RBC's

II

On basis of morphology of RBC

1. Normocytic
2. Microcytic
3. macrocytic

Classification of **anemia**



III

a. Due to **deficency** of factors essential for normal blood formation

1. Iron
2. Cyanocobalamin (Vit B12) and folic acid

b. Anemia due to **excessive** blood destruction (hemolytic anemia)

I. Due to intracorpuscular factors

i. Abnormal shape of erythrocytes
ii. Abnormal hemoglobins
iii. Erythrocyte enzyme deficencies

II. Due to extracorpuscular factors

c. Anemia due to aplasia or hypoplasia of bone marrow

Signs & Symptoms

Symptoms

- Lassitude
- Fatigue
- breathlessness on exertion
- Palpitations
- Throbbing in head and ears
- Diziness
- Tinnitus
- headache
- Dimness of vision
- insomnia
- Parasthesia in fingers and toes
- Angina

Signs

- Pallor of
a) Skin
b) Mucous membrane
c) palms of hands
d) conjunctiva
- Tachycardia
- Cardiac dialatation
- Systolic flow murmurs
- oedema

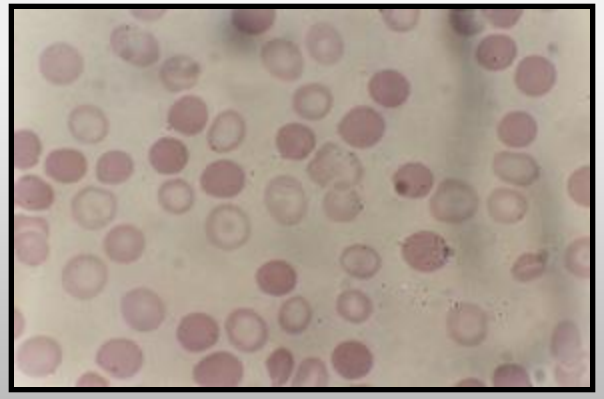
All of these signs and symptoms can occur because the heart has to work harder to pump more oxygen-rich blood through the body

Iron deficiency Anemia

1. Increased utilization- growth spurt
2. Physiologic iron loss- menstruation, pregnancy
3. Pathologic iron loss- GIT bleeding, genitourinary bleeding
4. Decreased iron intake

Consequence of deficiency

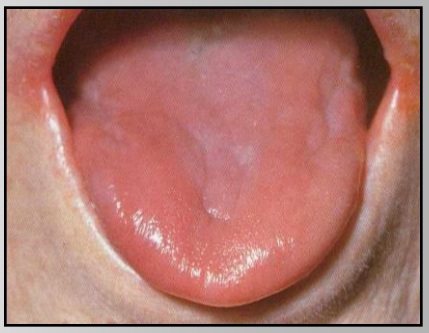
Cell growth & **proliferation** impaired by iron **deficiency.** Bone marrow cells have highest proliferative activity, especially **RBC** precursors, followed by **GIT** cells



Iron deficiency Anemia

Clinical features

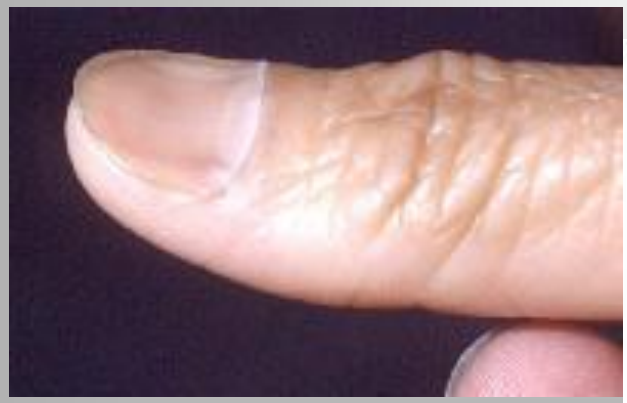
1. General features- weakness, lassitude, palpitations, exertional dyspnoea
2. GIT

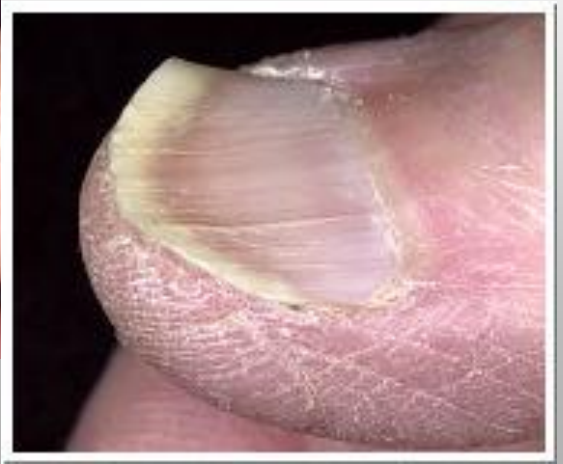


- Glossitis- smooth, reddened , swollen, shiny and tender tongue
- Angular stomatitis- erosion, tenderness at corners of mouth
- Gastric atrophy- sometimes gastritis and gastric bleeding

3. Nails-

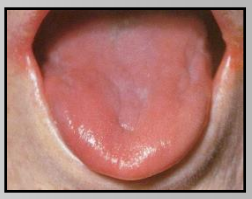
- Initial drying and brittleness
- Followed by flattening and brittleness
- Finally concavity (koilonychia)





Iron deficiency Anemia

Clinical features



4. Menorrhagia in iron deficent woman
5. Pica- eating and craving for strange foods

- Starch - amylophagia
- Ice- pacophagia
- Clay- geophagia

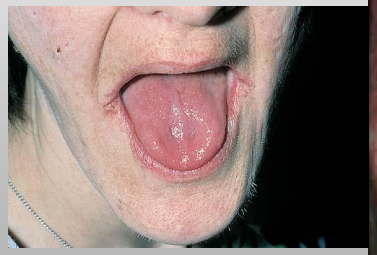
6. Plummer Vinson syndrome

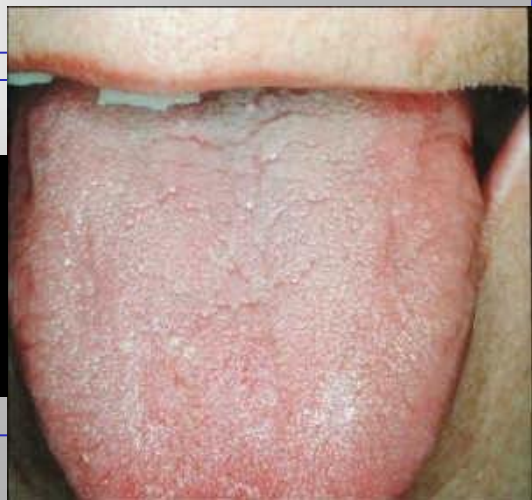
- Dry mouth
- Angular stomatitis
- Atrophic changes in mucosa of mouth, pharynx, upper esophagus

Pharyngeal and I.O Ca more common in these patients

Oral features of Fe def.-

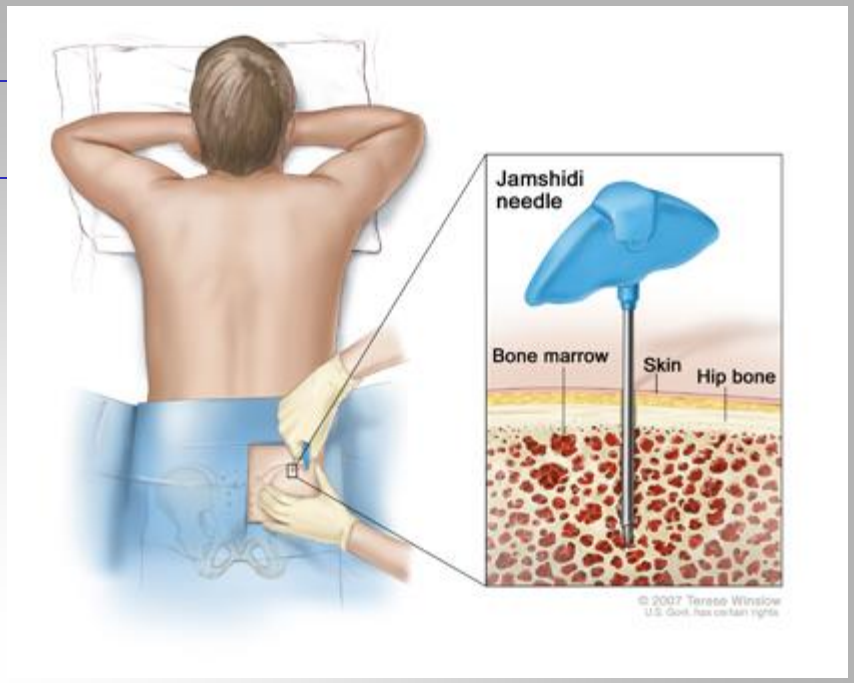
- glossitis, angular cheilitis,
- slow healing after oral surgical procedures,
- pallor of soft palate and tongue,
- papillary atrophy





Iron deficiency Anemia

Diagnosis



1. Blood investigations
- ↓ Hb
- Microcytic , hypochromic cells
- ↓ MCV, ↓ MCH, ↓ MCHC
2. Serum studies
- Low serum ferrtin
- Increased TIBC
3. Bone marrow- absence of stainable iron
4. Investigation for source of bleeding

Treatment of Fe def.-

•Oral iron tablets- 325 $FeSo_4$, 3 times daily, Fe gluconate or lactate

•Parenteral iron

a) Iron –sorbitol given 1.5 mg/ kg body wt given I.M

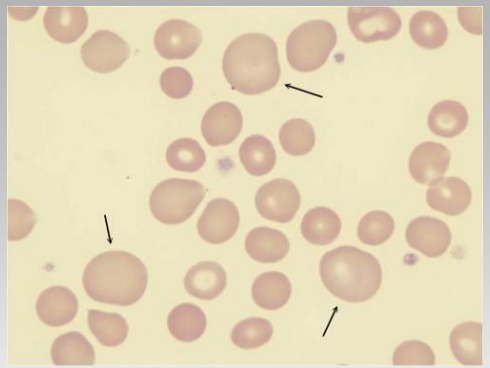
b) Iron dextran given I.V route

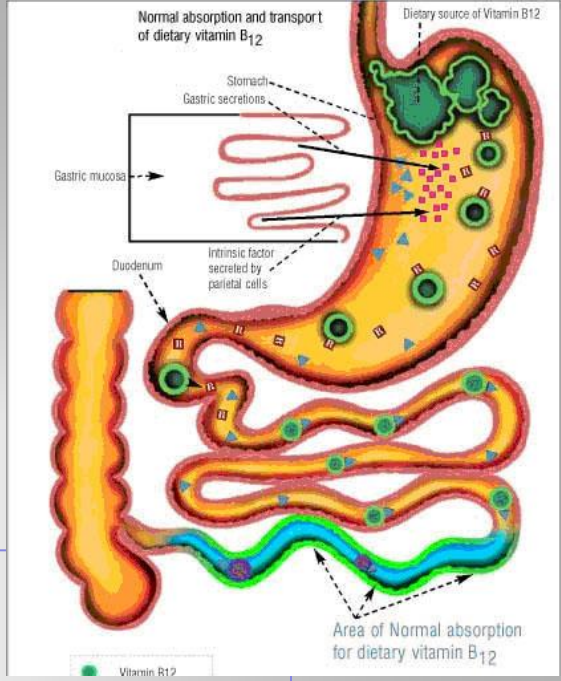
Megaloblastic Anemia

Megaloblasts- correspond to normoblasts of normal erythropoesis

↓

Macrocyte





Why are megaloblasts formed ?

•Both Vit B_{12} and folic acid are essential for DNA synthesis

• When DNA synthesis is impaired time between cell divisions increases

• When time between divisions increases, more cell growth occurs and the cell becomes larger

• the synthesis of Hb is unimpaired

• Hb production appears to be a factor limiting proliferation.

• Once a certain Hb level has stopped , cell division stops.

Megaloblastic Anemia

- B_{12} - exclusively from animal foods. Daily requirement 2.5 µg.
- For B_{12} absorption from GIT, "**Castle's intrinsic factor**" secreted by gastric parietal cells **(IF)**
- If no **IF** present - no entry of B_{12} into ileum → def of B_{12}
- Def of B_{12} due to lack of **IF called** Addisonian or **PERNICIOUS** anemia

- B12 – 2 mg stored in liver, 2 mg elsewhere, daily requirement 2.5 µg, **3 yrs** supply present in liver
- Folic acid -5-20mg stored in liver, daily requirement 400 µg

Megaloblastic Anemia

Pernicious anemia

- Mostly females between 45 and 65 yrs of age
- Mostly due to autoimmune reaction against parietal cells

Clinical features – affecting 1.blood 2. GIT 3. nervous system

a) Due to anemia- weakness, lightheadedness, palpitation, tinnitus

b) GIT- sore tongue (smooth and beefy red). Anorexia with moderate wt. loss, diarrohea and constipation

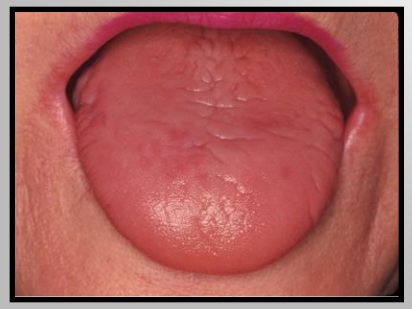
c) Neurologic changes- due to neuronal death. Numbness, parasthesia in extremities, weakness, ataxia, reflexes ↑ or ↓

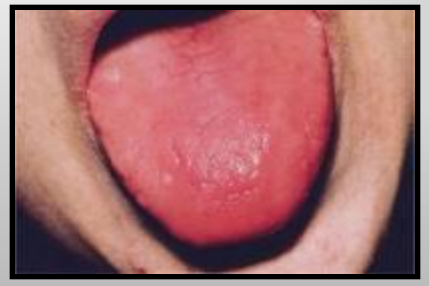
Megaloblastic Anemia

Pernicious anemia

Oral manifestations

- Painful glossitis and glossopyrosis
- Glossitis- fiery red color of tongue with distribution to tip and margins, with papillary atrophy of tongue
- Advanced cases- loss of normal muscle tone, loss of taste sensation
- Difficulty in wearing dentures- mucosa cannot tolerate





Megaloblastic Anemia

Diagnosis of pernicious anemia
- Blood smear- ↑ed MCV, ↑ed MCH, MCHC normal
- Bone marrow examination- megaloblatic marrow changes

Common to both B12 an folic acid def.

Serum assay for Vit B12 and folic acid

Schilling test

Small amount rad. act B12 by mouth
↓ *Followed by*
Large amt. non r.a parenteral B12

Normal pt. will excrete 7-30% of r.a B12 in 24 hrs, pernicious pt. will excrete only **3%** in 24 hrs *

If **pernicious** pt. given r.a B12 + **IF** orally, then normal absorption & urinary excretion

Megaloblastic Anemia

Folic acid anemia

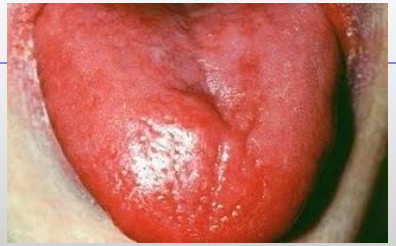
- in pts. with diet inadequate in leafy vegetables
- pts with ↑ed requirement, e.g pregnant, called “Anemia of pregnancy”

Clinical features

- GIT- diarrohea, glossitis, cheilitis
- Hematological changes
- No specific neurologic symptoms- difference with **pernicious**

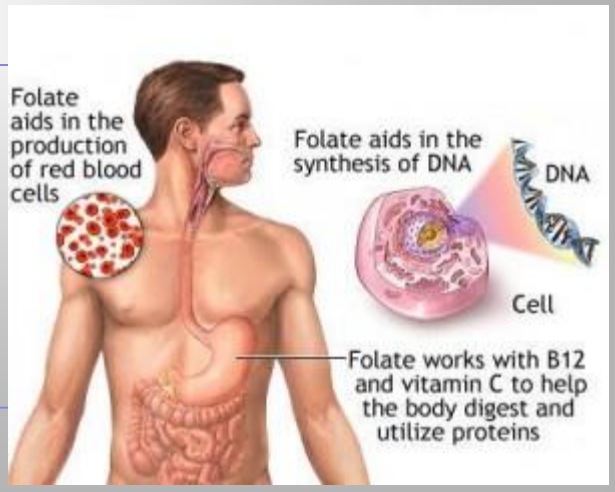
Oral manifestations

- angular cheilitis, severe ulcerative stomatitis, pharyngitis



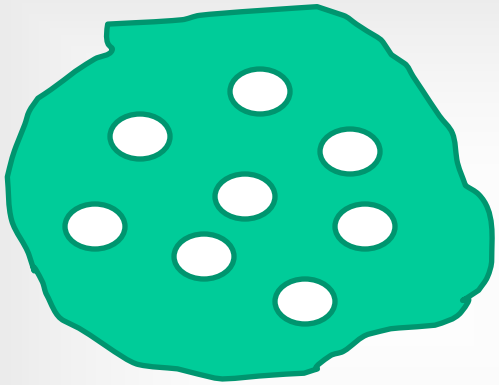
Diagnosis

- Dietary history
- Intestinal biopsy
- Normal Schilling and B12 serum assay
- low serum assay of folic acid



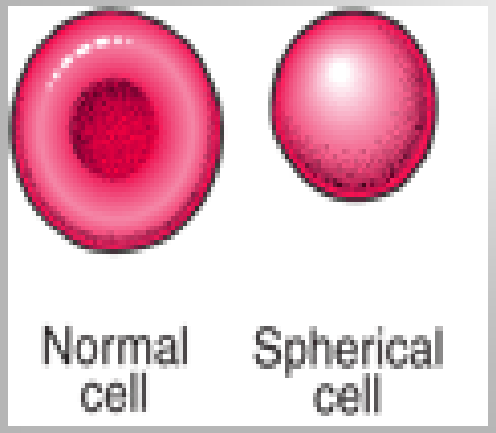
Hemolytic Anemia

Excessive destruction of erythrocytes caused by either
- intracorpuscular defects in erythrocyte
- extracorpuscular factors



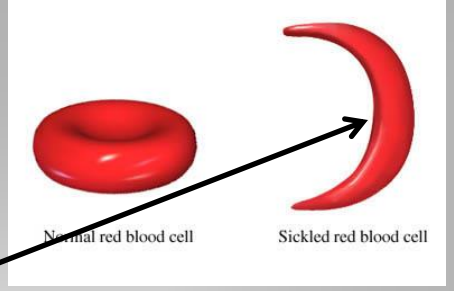
Fine filtering system of spleen

Hereditary spherocytosis



Clinical features
- episodic jaundice, splenomegaly
- Diagnosis- spherocytes in blood smear
- Management- splenectomy

Hemolytic Anemia



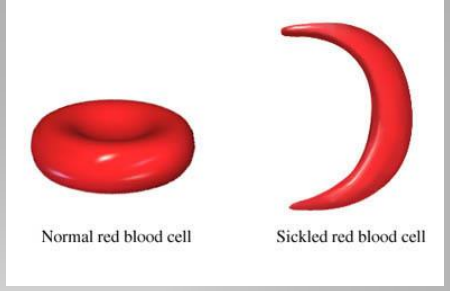
Abnormal hemoglobins - **Hemoglobinopathies**

- Normal hemoglobin - Hb A - **£ $_2$ ß$_2$ (alpha 2, beta 2)**
- In sickle cell disease- Hb S instead of Hb A
- Hb S has one protein different in **ß** chain
- HbS as it releases oxygen, polymerizes and aggregates with other HbS molecules, making the red cell stiff and distorted

- These distorted, sickle-shaped red cells , get destroyed in the spleen → hemolytic anemia

- Clump together, especially in small vessels, ischemic tissue damage

Hemolytic Anemia



Abnormal hemoglobins - **Hemoglobinopathies**

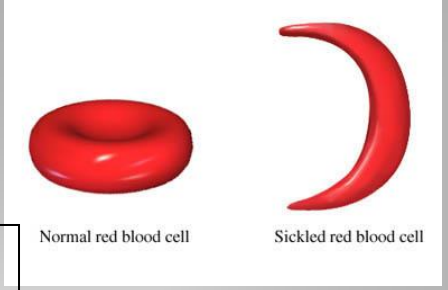
£ $_2$ ß$_2$ (alpha 2, beta 2)

- Sickle cell anemia (homozygous) – both beta chains abnormal
- Sickle cell trait (heterozygous) – only one one beta chain abnormal

Clinical features- related to
- chronic anemia, due to reduced erythrocyte survival
- tissue infarction following stasis of blood

- jaundice, pallor, fatigue, reduced exercise tolerance
- splenic infarction , chronic leg ulcers, painful attacks of bone and abdominal pain

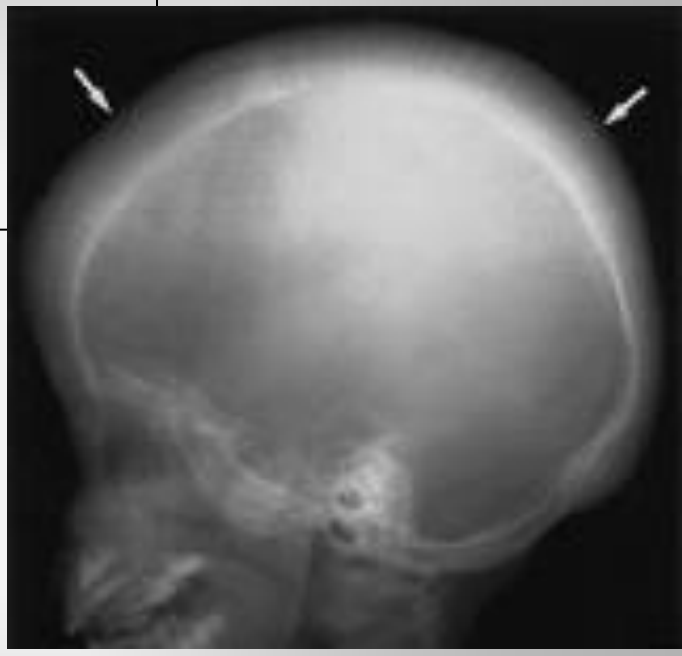
Hemolytic Anemia



Abnormal hemoglobins - **Hemoglobinopathies**

Oral manifestations

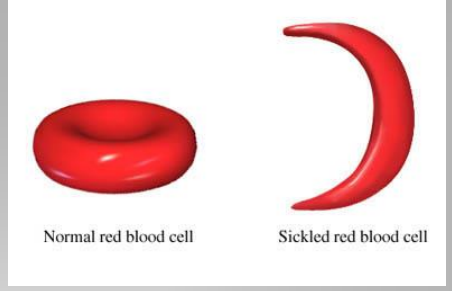
- jaundice & pallor of oral mucosa
- delayed eruption and hypoplasia of dentition *
- ↑ed radiolucency seen in jaws on dental rg
- skull "**hair on end"** appearance



Diagnosis

- Demonstation of sickling
- hemoglobin electrophoresis

Hemolytic Anemia



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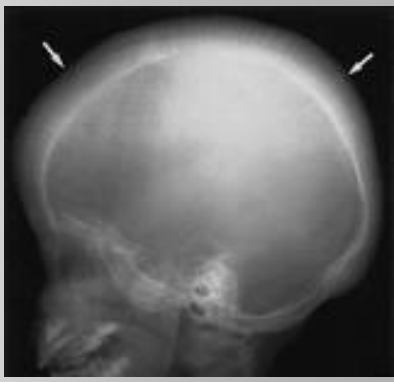


Dental considerations

- Chronic anemia and slow healing, so minimal tissue manipulation
- Keep dentition healthy, as infection might precipitate aplastic crisis
- Avoid using G.A

Hemolytic Anemia

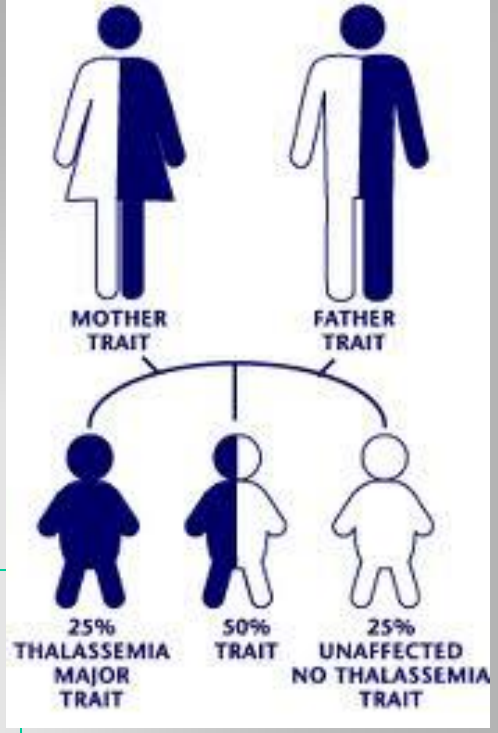
Abnormal hemoglobins - **Hemoglobinopathies**

Thalassemia

£ $_2$ ß$_2$ (alpha 2, beta 2)

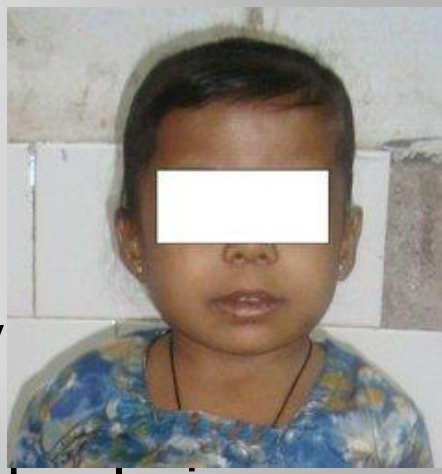
Defect in the synthesis of either £ or ß chain of Hb

- £ thalassemia- impaired production of £ chain
- ß thalassemia- impaired production of ß chain



ß thalassemia- heterozygous or homozygous

- intra medullary erythroid destruction
- shortening of the life span of circulating rbc's

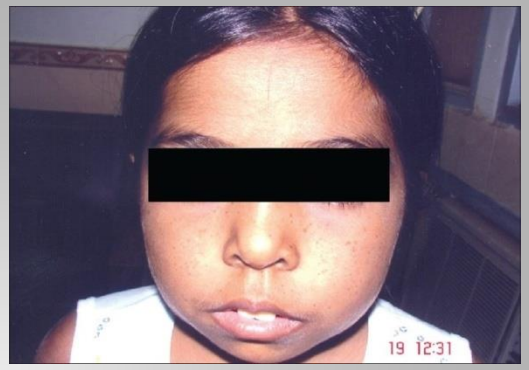


ß thalassemia major- Cooley's anemia

Clinical manifestations after 4 to 6 months

- Pt severely anemic and have short life expectancy
- pt appears wasted and malnourished
- peculiar skin color due to combination -icterus, pallor & ↑ed melanin
- cardiomegaly with signs of CCF
- radiographs show osteoporosis

Hemolytic Anemia



Abnormal hemoglobins - **Hemoglobinopathies**

Thalassemia

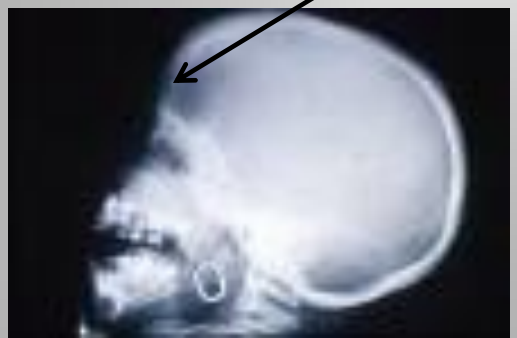
£ $_2$ ß$_2$ (alpha 2, beta 2)

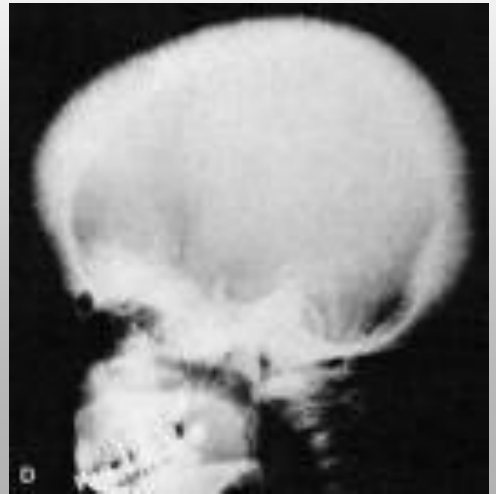
Defect in the synthesis of either £ or ß chain of Hb

- £ thalassemia- impaired production of £ chain
- ß thalassemia- impaired production of ß chain

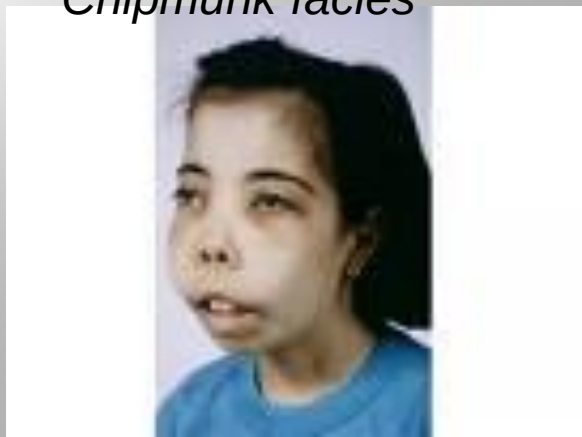
Oral manifestations

- by 2nd yr, mongoloid appearance, frontal and parietal bossing
- overdevelopment of maxilla, malocclusion, open bite, spacing
- pale oral mucosa
- Skull radiographs, “hair on end”, ↑ed size of maxilla

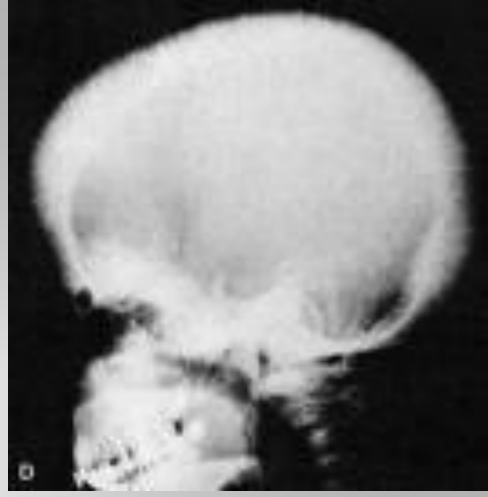




Chipmunk facies



Hemolytic Anemia



Abnormal hemoglobins - **Hemoglobinopathies**

Thalassemia

£ $_2$ ß$_2$ (alpha 2, beta 2)

Defect in the synthesis of either £ or ß chain of Hb

- £ thalassemia- impaired production of £ chain
- ß thalassemia- impaired production of ß chain

Diagnosis

- Family history
- clinical signs
- blood smear- hypochromic, microcytic anemia
- Hb F upto 90%

Dental consideration- remember poor wound healing after dental procedure

Treatment

- Life long transfusion
- folic acid supplements

Aplastic Anemia

Normocytic, normochromic anemia

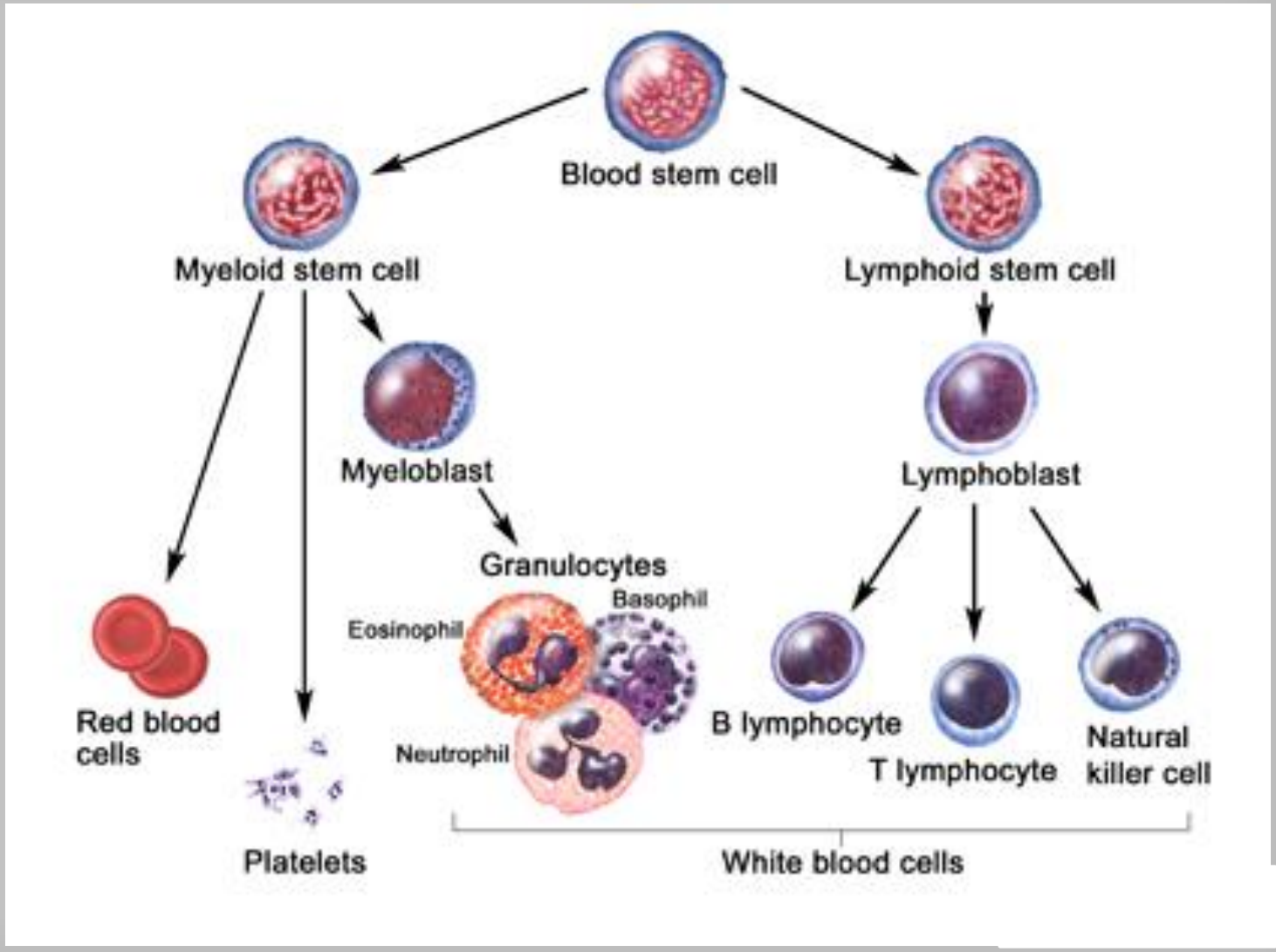
All the cellular elements of blood involved

Primary aplasia	Secondary aplasia
Unknown etiology	Idiosyncrasy to certain drugs
	Cancer chemotherapy
	Exposure to radiation
	Viral infections
	Severe def. of folate or B12

Oral manifestations
- Pallor of oral mucosa
- Petechiae and purpura of oral and pharyngeal tissue
- oral bleeding with no apparent cause
- ulcers having little or no surrounding erythema

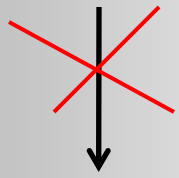
Dental considerations
- prevent dental and oral infections and oral bleeding
- keep the number of dental procedures to minimum
- Beware of potential of any procedure to cause hemorrhage
- Eliminate potential sites of infection

W B C Disorders


Blood stem cell
Myeloid stem cell
Lymphoid stem cell
Myeloblast
Lymphoblast
Granulocytes
Basophil
Eosinophil
Neutrophil
Red blood cells
Platelets
B lymphocyte
T lymphocyte
Natural killer cell
White blood cells

Myeloid stem cells

Myeloblasts (***leukemic cells)***

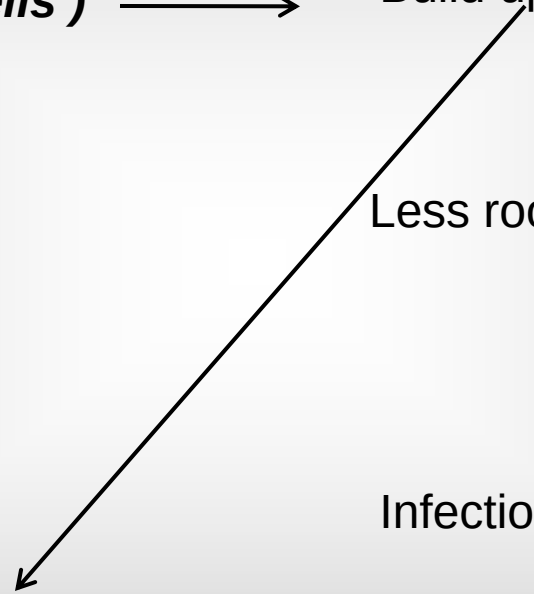


Healthy white blood cells

Build up in blood and bone marrow

Less room for normal WBC, rbc and platelets

Infection, anemia & easy bleeding



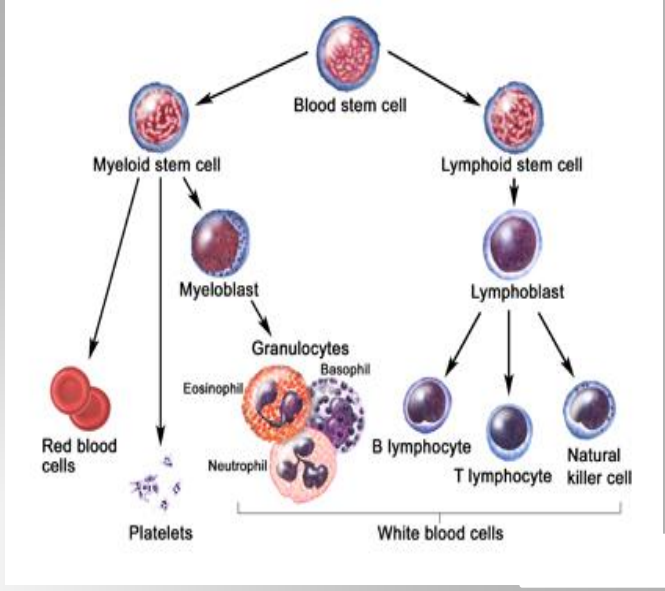
Can spread outside blood to CNS (*brain and spinal cord),* skin & gums

LEUKAEMIA → lymphoblastic

LEUKAEMIA → Non lymphoblastic

acute

chronic



ORAL MANIFESTATIONS OF LEUKEMIA

- spontaneous gingival bleeding or oozing
- petechiae formation
- oral soft tissue or gingival infection
- pharyngitis
- lymphadenopathy

DENTAL CONSIDERATIONS

- prevention of infection
- prevention of bleeding

Always **check whether gingival bleeding is proportional to local factors !!!**