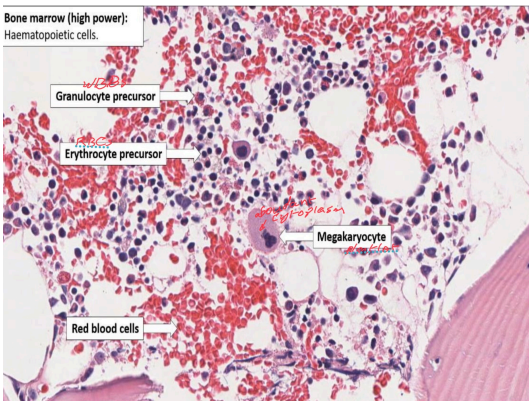
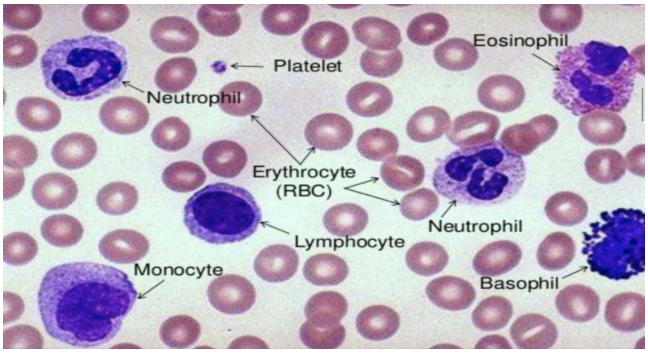
• By birth: the bone marrow in whole of the skeleton is haematopoietically active and is the chief source of blood cells and it remains so till puberty.
• After puberty: the red marrow is present in vertebrae, ribs, sternum, skull, pelvis and proximal epiphyseal regions of humorous and femur.



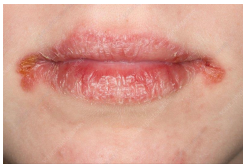



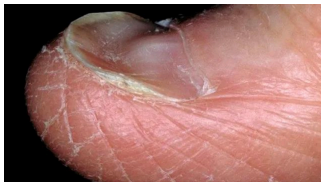

# ANEMIAS

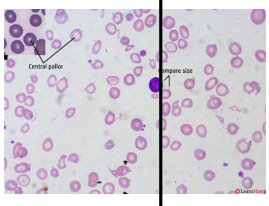
Definition : HB <13 g/dl Male, <12 g/dl Femal
- Morphologic classification: according to the shape and amount of Hb
1. Normocytic (MCV 80-100 fl)
2. Microcystic (MCV < 80 fl)
3. Macrocystis (MCV > 100 fl)
- COMPLETE BLOOD COUNT (CBC)
• Normal reticulocyte count: less than 1.5%
• In anemia one should correct the percentage of reticulocytes.

## #IRON DEFICIENCY ANEMIA (IDA)

• Deficiency of iron is the most common nutritional disorder in the world.
- functional body iron is present in hemoglobin, myoglobin and iron-containing enzymes.
- The storage pool represented by hemosiderin and ferritin.
• Transported in plasma by an iron-binding protein called transferrin,
• The absorption of dietary iron in the proximal duodenum.
Clinical features:
- Fatigue
- Impaired growth and development
- Pica (eating mud in children)
- Koilonychia (spoon shaped nails)
- Angular stomatitis (ulceration at the angle of mouth)
- Palpitation.
• Findings on CBC and peripheral blood:
Microcytic and hypochromic RBCs

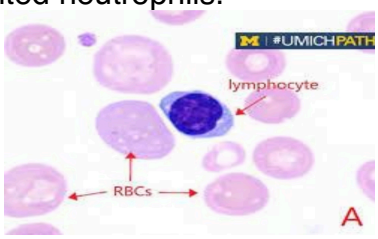






## # MEGALOBLASTIC ANEMIA

- Due to vitamin B12 and folate deficiency.
• Results in the formation of megaloblasts.
- Causes of deficiency:
Impaired absorption:
B12: intrinsic factor deficiency, gastrectomy, intestinal diseases, ilial resection.
Folate: intestinal diseases, anticonvulsant.
-Vitamin B12 absorption mechanism:
• Intrinsic factor produced in the stomach.
• Without sufficient intrinsic factor, vitamin B1 absorption is impaired, which can lead to pernicious anemia
• Site of B12 absorption is terminal ilium
• Folate deficiency during pregnancy causes neural tube defects in babies
• Folate supplementation is mandatory before during pregnancy
• Findings on CBC and peripheral blood:
- Macrocytic, normochromic RBCs.
Hypersegmented neutrophils.



# APLASTIC ANEMIA

* Findings on CBC, Blood film and Bone marrow:
- Pancytopenia: meaning there are low numbers of red blood cells, white blood cells, and platelets. A reticulocyte count is also low.
- Normocytic, Normochromic RBCs
- Dry tap bone marrow aspirate
- Bone Marrow biopsy: markedly hypocellular bone marrow is largely devoid of hematopoietic cells; often
only fat cells, fibrous stroma, and scattered lymphocytes and plasma cells remain

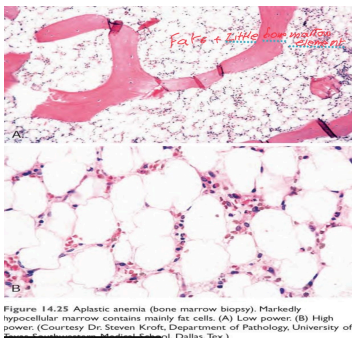
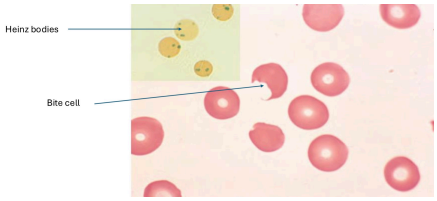


Figure 14.25 Aplastic anemia (bone marrow biopsy). Markedly hypocellular marrow contains mainly fat cells. (A) Low power. (B) High power. (Courtesy Dr. Steven Kroft, Department of Pathology, University of

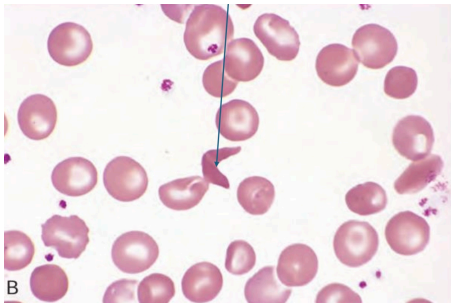
# GLUCOSE-6-PHOSPHATE DEHYDROGENASE DEFICIENCY

- Recessive x-linked trait, placing males at much higher risk for symptomatic disease
- The episodic hemolysis that is characteristic of G6PD deficiency is caused by exposures that generate oxidant stress:
- The most common triggers are infections
- The other important initiators are drugs and certain foods (fava beans)




# SICKLE CELL ANEMIA

• Common hereditary hemoglobinopathy caused by a mutation in β-globin
• Pathophysiology: deoxygenated HbS polymerizes, causing sickle-shaped
• Findings in CBC and peripheral blood: cells that hemolyze and block blood vessels
• Normocytic, Normochromic RBCs
• Clinical features:
• Autosplenectomy: in adolescence or early adulthood.
• Vaso-occlusive crises cause severe pain in the affected region, mostly, lung, bone, skin, and others.
- replacement of a charged glutamate residue with a hydrophobic valine residue.




# THALASSEMI

- Germline mutations that decrease the synthesis of either α-globin or β-globin.
? Findings on CBC and peripheral blood:
- Microcytic Hypochromic anemia.
? Clinical features:
- Crewcut skull and distinct facial features: Wide forehead, prominent cheekbones, a flattened nasal bridge, and a protruding upper jaw, due to the expansion of bone marrow.

# BLEEDING DISORDERS

## DEFECTIVE COAGULATION

### 1.Hemophilia A

• Caused by absolute reduction of factor VIII or normal amount but defective factor VIII.
• Sites of bleeding: large joints and soft tissue.
• laboratory tests: prolonged PTT.

### 2. Hemophilia B

• Caused by deficiency in blood clotting factor IX.
• Bleeding sites: similar to hemophilia A.
• Laboratory tests: prolonged PTT.

### 3. VON WILLEBRAND DISEASE

• The disease result from: Quantitative or qualitative defects of plasma vWF.

## PLATELET DISORDERS

### #ITP (IMMUNE THROMBOCYTOPENIC PURPURA)

| Acute ITP | Chronic ITP |
|---|---|
| Mostly children | Mostly women |
| Preceded by infection | No recent history of infection |
| Self limited | Not self limited |
| | Megakaryocyte increased in bone marrow |

### # THROMBOTIC THROMBOCYTOPENIC PURPURA (TTP)

• Characteristic pathologic feature: Hyaline microthrombi (platelet rich) in microcirculation (capillary of skin and gingiva)
• Clinically: Acute thrombocytopenia, fever, microangiopathic hemolytic anemia, neurologic abnormalities, renal dysfunction.

### # HEMOLYTIC UREMIC SYNDROME (HUS)

• E. Coli O157:H7 (toxin induced endothelial damage)
• Clinically: Bloody diarrhea followed by acute renal failure., Platelet microaggregate (hyaline microthrombi) formation (usually limited to the glomerular capillaries), Acute thrombocytopenia, microangiopathic hemolytic anemia.

Schistocytes