Pathology Study Notes:

1: Anemia

Hematopoetic Stem Cells:

- **Myeloid:**
  - Myeloblast
  - Immature monocyte
  - Megakarocyte
  - Pronmoblast (RBC)
- **Lymphoid:**
  - Lymphocytes

LAB TESTS:

Complete Blood Count (CBC): looks at RBC, WBC, platlets
RBC: number of cells
Hemoglobin: the amount of hemoglobin you have (Anemia you don't have enough)
Hematocrit: volume of RBC's you have

Complete Blood Count (CBC):

- MCV: Mean Cell Volume = microcytic, normocytic, macrocytic
- MCHC: Mean Cell Hemoglobin Concentration = hypochromic, normochromic (tells you how much hemoglobin each cell is carrying around)

Size variations: Anisocytosis
Shape variations: Poikilocytosis

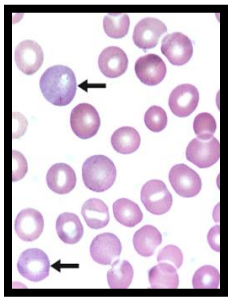
ANEMIA:

- A reduction below normal in hemoglobin or RBC number
- Symptoms: pale skin and mucous membranes, jaundice, tachycardia, breathlessness, dizziness, fatigue

SPECIFIC TYPES OF ANEMIA: 3 WAYS TO GET ANMEIA =

LOSS OF BLOOD

- Cause may be trauma, acute blood loss. At first the hemoglobin is normal and after 2-3 days you see reticulocytes (young RBC precursors which are bigger than normal). Chronic blood loss is different because it causes iron deficiency anemia.



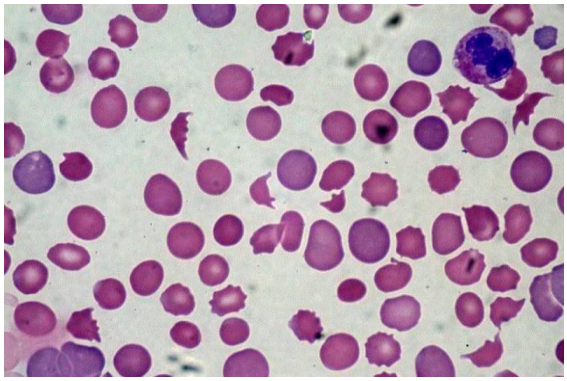
- Reticulocytes: bigger, younger, have some RNA in them giving them their bluish/purple color instead of the red in normal RBC

DESTROY TOO MUCH BLOOD (Hemolytic Anemias)

- Chronic vs Acute
- Chronic: inherited, not too bad, can become acute if something happens
- Acute: suddenly, not inherited (ex: antibodies)
- Signs of destruction: increase bilirubin, increase Lactate dehydrogenase enzyme, low haptaglobin (carrier molecule of free hemablobin)

Extracorpuscular reasons

MICROANGIOPATHIC HEMOLYTIC ANEMIA:



RBC's get ripped up in small blood vessels, physical trauma to red cells, SCHISTOCYTES (funny shaped RBC's) and find out why cause some causes are very serious. There is activation of the coagulation cascade causing fibrin strains in small vessels where passing RBC's get snagged as they rush thru and end up looking weird called : Schistocytes (which is a medical emergency, always pathologic, pointy shaped RBC). There is a special kind called Triangulocyte

Causes of MAHA:

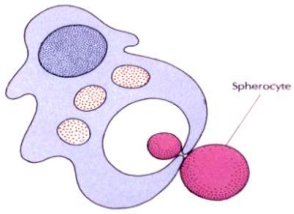
- A MOST
- Artificial heart valve, malignancy, obstetric complications, sepsis, trauma

AUTOIMMUNE HEMOLYTIC ANEMIA:

Temp at which antibody binds: can do DAT (Direct Antiglobulin Test)

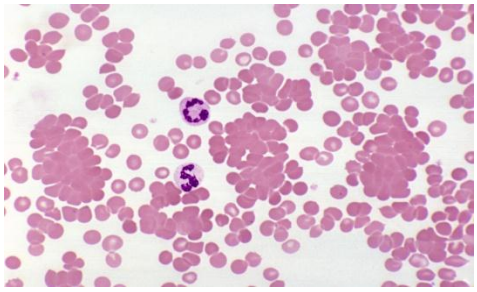
Warm AIHA (WARM GISS)

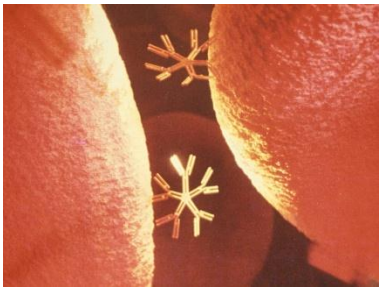
- IgG, Spleen, Spherocytes



Cold AIHA (COLD CIMA)

- IgM, complement, Intravascular hemolysis, Agglutination





Intracorpuscular reasons

SICKLE CELL ANEMIA

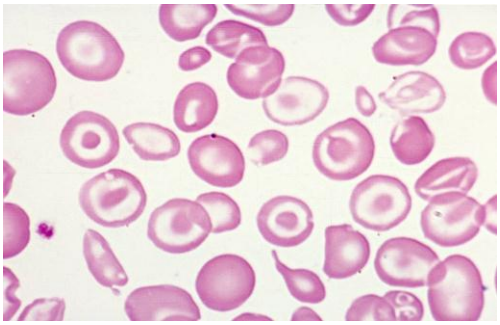
- **Hemoglobinopathy** (qualitative defect in hemoglobin, point defect in beta chain)
- Single amino acid substitution (point mutation) in beta chain of hemoglobin of valine to glutamate
- Can be heterozygous (sickle trait but no symptoms) or homozygous (double hit and have symptoms)
- Sickle cells are nasty, fragile (burst easily) and get stuck to vessels and clog up vessels, aggregates and polymerizes (sticks together) on deoxygenation
- Lesions on hands/feet common due to blood vessel clogged up (infarct distal to clog)
- **In spleen**: infarct, heal and form scar, over and over again, no more spleen (spleen gets rid of encapsulated bugs)

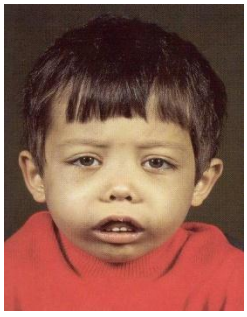
Clinical Findings:

- Blacks, 8% heterozygous
- Severity variable
- Chronic hemolysis, vaso-occlusive disease, and increase infections (autosplenectomy)
- Treatments: prevent triggers, vaccinate, transfuse (wt normal blood)

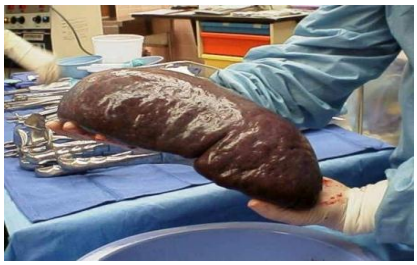
THALASSEMIA

- Quantitative defect in hemoglobin,
- Cant make enough alpha and beta chains
- Variable disease severity
- Hypochromic (low hemoglobin), microcytic (small in size) anemia with increased RBC and TARGET cells
- Alpha more serious cause beta can get help from delta
- Medullary expansion





HEREDITARY SPHEROCYTOSIS

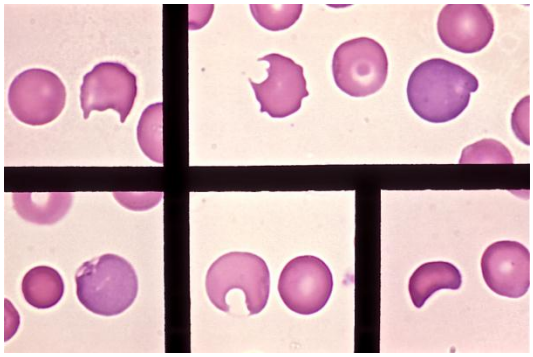
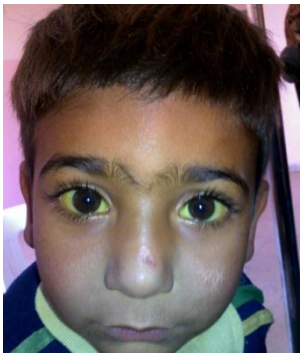


- Problem with RBC membrane
- Lots of spherocytes
- Spectrin defect (proteins that attack cytoskeleton to the membrane)
- Splenectomy is curative (symptoms may go away)

Glucose 6-Phosphate Dehydrogenase Deficiency (G6PD Deficiency)

- Low G6PD (helps detox the cells) leads to high peroxides which causes cell lysis
- Oxidant exposure
- Bite cells (removal of Heinz bodies)
- Self limiting

Clinical Findings:

- Some asymptomatic, some episodic hemolysis
- Triggers: broad beans, drugs
- Spontaneous resolution
- Jaundiced sclera
- RBC's die because they can't reduce nasties, nasties attack hemoglobin bonds, heme breaks away from globin, globin denatures and sticks to RBC membrane (Heinz body) and spleen bites out Heinz bodies
-  

MAKE TOO LITTLE BLOOD

Too few bldg blocks:

Iron-Deficiency Anemia:

- GI bleeding is most important cause (not most common cause)
- Microcytic, hypochromic anemia (little cells with low hemoglobin)
- Must find out why (menstruation, child birth, colon cancer
- Atrophic glossitis (bald, shinny tongue with no papilla…need iron for that), Koilonychia (concavity in nail)

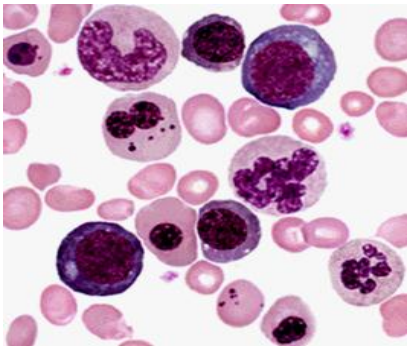
Causes: decreased iron intake (bad diet, bad absorption), increased iron loss (GI bleed worry most about, menses, hemorrhage), increased iron requirement (pregnancy)

ANEMIA OF CHRONIC DISEASE (can be confused with iron deficiency but in this case you cant metabolize iron correctly due to chronic disease)

- Infections, inflammation, malignancy
- Iron metabolism disrupted
- Normal looking cells
- Lab values low, anemia usually mild

MEGALOBLASTIC ANEMIA

- Defective DNA synthesis
- Nuclear/cytoplasmic asynchrony (different sizes)
- Low B12/folate
- Macrocytic anemia (MCV number high) with oval macrocytes and hypersegmented NEUTROPHILS
- Retarded DNA synthesis, unimpaired RNA synthsis = BIG cells, immature nucleus, mature cytoplasm
- Atrophic glossitis



Too few erythroblasts

APLASTIC ANEMIA

- Pancytopenia (everything decreased)
- Empty marrow (all fat and no hematopoetic tissue…just lymphocytes)
- Most idiopathic
- Causes: Idiopathic, drugs, viruses, pregnancy, Fanconi anemia (congenital disease)

Not enough room

- Bone marrow full of fibrosis