Chapter 17: Blood

I. OVERVIEW

Formed elements in plasma.
Separated in hematocrit = % RBCs as volume.
Ph between 7.35 and 7.45.
Five times more viscous than water.
Approximately 38C.
Men with 5-6L;    women with 4-5L.

II. PLASMA

Approximately 90% water.

8% proteins: albumin maintains osmotic pressure; globulins include antibodies; fibrinogen for clotting.

Remainder: nutrients, electrolytes, gases, hormones….

III. FORMED ELEMENTS

(A) Erythrocytes = Red Blood Cells = RBCs

-1- Structure:
Small, biconcave discs.
Anucleate.
Huge surface area.
More than 97% of volume is hemoglobin.
Only anaerobic metabolism.

-2- Function:
Transport O2, some CO2.
Hemoglobin with globin portion = four polypeptides
Two alpha and two beta chains;
Four hemes, with iron,
Each heme carries one O2.
One billion O2/ RBC.

Oxyhemoglobin (OxyHb) w/ O2
DeoxyHb     w/o O2
CarbaminoHb w/ CO2
CarboxyHb   w/ CO
-3- Production:

Called **erythropoiesis**.
From red marrow.
Reticulocytes enter blood, 1-2% of circulating RBCs.
Production rate ~ 2 million/ second.
Hormonally regulated by EPO.
Requires dietary iron, B12 and folate.

-4- Destruction:

After 100-120 days.
RBCs become frayed, often removed in spleen and liver,
Phagocytized.
Iron and amino acids recycled.
Rest of heme becomes bilirubin,
then a sequence of bile pigments.
Processed by liver.

-5- Disorders:

**Anemias**

Hemorrhagic
Hemolytic
Aplastic- destruction of erythropoietic tissue.
Iron Deficiency
Pernicious- lack of B12
Thalassemia- genetically malformed globin.
Sickle Cell- genetically malformed beta chain.

**Polycythemia** elevated hematocrit

Polycythemia Vera: bone marrow cancer.
Secondary: at high altitudes, response to CO (smokers), blood doping.

(B) **Leukocytes** = White Blood Cells = WBCs

-1- General Characteristics:

Less than 1% of blood volume.
Body defense.
Mobile, **diapedesis** (leave vessels).
Positive **chemotaxis**.
**Leukocytosis** (increased #); **Leukopenia** (decreased #).
-2- Granulocytes
Visible granules in cytoplasm when stained with Wright Stain.

(a) Neutrophils
Most numerous WBCs.
Polymorphonuclear- variable multilobed nuclei.
Fight bacteria.
Phagocytic,
Destroy surrounding cells with respiratory burst.

(b) Eosinophils
Reddish granules.
Fight parasitic worms.
Involved in allergic reactions.

(c) Basophils
Large dark blue granules.
Release histamine.

-3- Agranulocytes

(a) Lymphocytes
Second most abundant WBCs.
Most not in blood.
T & B Cells.

(b) Monocytes
Largest leukocytes.
Transform into macrophages.

-4- Production and Lifespan

Leukopoiesis regulated by interleukens and CSFs (colony stimulating factors).
All produced in bone marrow.
Myeloid and lymphoid (lymphocyte) stem cells.

-5- Disorders

Leukemias: large variety.
Acute vs. chronic.
Acute more serious, in children.
Mononucleosis: Epstein-Barr virus infects agranulocytes.
(C) **Platelets**

Fragments of *megakaryocytes*. Involved in *hemostasis*. Production enhanced with hormone *Thrombopoietin*.

**IV. HEMOSTASIS**

Control of blood loss. Regulated sequence.

(A) **Vascular Spasm** = vasoconstriction.

(B) **Platelet Plug** formation. Triggered by exposed collagen. Positive feedback; platelets release chemicals to attract more platelets.

(C) **Coagulation** - Goal is a mesh of fibrin strands.

Phase I and II lead to conversion of prothrombin into thrombin. Phase III: Thrombin converts fibrinogen to fibrin.

*Intrinsic Pathway* initiated from blood- slower. *Extrinsic Pathway* from damaged endothelium- faster.

Calcium required for several steps.

(D) **Clot Removal & Prevention**

Clot retraction & repair- function of platelets, during vessel repair.

Fibrinolysis- done by enzyme plasmin.

Unnecessary clots prevented by quick removal of clotting factors and their inhibition, e.g. by heparin. Process inhibited by healthy, intact endothelium.

(E) **Hemostatic Disorders**

-1- **Thromboembolic Conditions**

Inappropriate clots called *thrombi*. An *embolus* when traveling, An *embolism* when lodged. Risk increased with *atherosclerosis*, poor circulation. Therapies include aspirin, heparin, warfarin, coumadin.
-2- Bleeding Disorders

**Thrombocytopenia**: reduced platelet #,
Liver Disease,
Vitamin K deficiency,
**Hemophilia**: genetic, usually X-linked. Lack of a clotting factor.

V. CLINICAL ASPECTS

(A) **Transfusions**
May be whole blood or packed red cells.
Blood type based on genetically produced **agglutinogens**.
Most attention paid to ABO and RhD markers.
Antibodies reacting to agglutinogens called **agglutinins**.

(B) **Hemolytic Disease of the Newborn = Erythroblastosis Fetalis**:
Baby with RH+ blood, mother Rh-. Woman’s agglutinins pass placenta, destroy fetal blood.

Blood typing based on reaction of blood mixed with antisera (agglutinins).
Clumping indicates the matching agglutinogen.

(C) **Blood Tests**

Hematocrit
Glucose
Differential WBC Count
Prothrombin Time
Complete Blood Count
SMAC – blood chemistry.

VI. DEVELOPMENT

Fetal blood develops first from yolk sac, then liver and spleen.
All marrow is red at birth.
Fetal hemoglobin with stronger O2 affinity.