Cardiovascular Blood Hemostasis

I. Blood Groups

A. ABO Blood Group
B. Agglutinogens – cell membrane / agglutinins – plasma
C. Blood typing ABO Rh groups produce most serious problems
D. All humans have one of four blood types:

Blood Type	Antigen	Antibody
A	A	Anti B
B	B	Anti A
AB	A and B	Neither
O	Neither	Anti A and B

E. Preferred types for transfusion:

Blood Type	Preferred	Permissible
A	A	O
B	B	O
AB	AB	A,B,O
O	O	None

F. Rh Factor
1. If Rh factors present = positive
2. If Rh factors not present = negative
3. Antibodies form in Rh negative person by stimulation
4. *Erythroblastosis fetalis*

II. Hemostasis

A. Blood Vessel Injury:
1. Smooth muscle in vessel contracts
2. Platelets release ADP and Serotonin which make platelets stick together
3. Platelets release Epinephrine and Serotonin which increase blood vessel contraction
4. Platelets release a Prostaglandin called Thromboxane A2 (TXA2) which acts for 30 seconds to cause platelet aggregation
5. Arterial epithelial cells release Prostacyclin (PG12) which acts for 2 minutes or more to prevent platelet aggregation. This also causes vasodilation.
6. PG12 protects the body from emboli caused by TXA2

B. Clotting Factors:

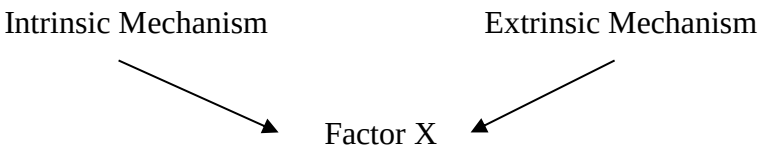
1. Several synthesized in the liver with the aid of vitamin K
2. Most are proteins, some being protease enzymes that activate other factors
3. Some are cofactors that activate enzyme complexes

C. Extrinsic Mechanism

1. Tissue damage activates the extrinsic
2. Injured tissue releases tissue Thromboplastin. With the addition of calcium ions (Ca+2) Thromboplastin and other factors activate Factor VII.
3. Factor VII activates Factor X and the clotting mechanism begins
4. Any injured cell can activate this system

D. Intrinsic Mechanism

1. Blood contacts negative charged surface (collagen, glass, plastic, etc.) which activates the intrinsic system
2. Factor XII converts Prekallikrein to Kallikrein
3. Kallikrein + Kininogen from platelets accelerate Factor XII
4. Factor XI activated
5. Factor XI activates Factor IX
6. Factor IX + Factor VIII and Calcium activates Factor X
7. After Factor X activated pathways converge



E. Thrombus Formation

1. Factor X converts Prothrombin to active Thrombin
2. Thrombin converts soluble Fibrinogen to insoluble Fibrinogen
3. Factor XIII or Fibrin Stabilizing Factor causes Fibrin cross links
4. Clot is formed – blood cells and platelets are caught in fibrin mesh
5. Crossovers between intrinsic and extrinsic mechanisms
    - **a)** Activated Factor XII from intrinsic
    - **b)** Activates Factor VII of extrinsic
    - **c)** Factor VII of extrinsic can activate Factor IX of intrinsic
    - **d)** Crossovers prevent uncontrolled bleeding by activating whichever system is functional

F. Clot Destruction

1. Clots are temporary
2. After 30 minutes clot retracts and becomes smaller
3. When clot is formed Plasminogen is deposited in the clot
4. Plasminogen Activator from epithelium plus Factor XII slowly digests clot

III. Other Blood Criteria

A. Blood cell count

1. Erythrocyte count: number of rbc in 1cc of blood
2. Leukocyte count: number of wbc in 1cc of blood
3. WBC differential: percent of each wbc in the sample
4. Absolute count: total number of each wbc type in sample

B. Terms:

1. Hematocrit: packed cell volume, percent of cells compared to total blood volume in centrifuged blood
2. Hemoglobin: grams per deciliter of the hemoglobin molecule
3. Mean Corpuscular Hemoglobin Concentration (MCHC): (hemoglobin/hematocrit) X 100
4. Mean Corpuscular Volume: (hematocrit/rbc count) X 10

5. Macrocytic Anemia: MCV above maximum range, MCHC normal, typically indicative of folic acid deficiency
6. Pernicious anemia: condition of anemia that results as the failure to produce adequate intrinsic factor by the stomach (vitamin B12 deficiency)
7. Normocytic Normochromic Anemia: MCV and MCHC normal, caused by acute blood loss or damage to bone marrow (aplastic anemia)
8. Microcytic Hypochromic Anemia: abnormal low MCV and low MCHC, most common of all forms of anemia