Hemodynamic Disorders
Thrombosis and Shock

I. Body water, where is it and what keeps it there?

A. Intracellular

B. Extracellular (intercellular)

1. Interstitial, between the cells, but "outside" of the vascular system

- lymph fluid while it is between the cells, but not yet collected by the lymphatic vessels

2. Intravascular

- water making up the blood and

- lymphatic fluid when it is within the lymph vessels

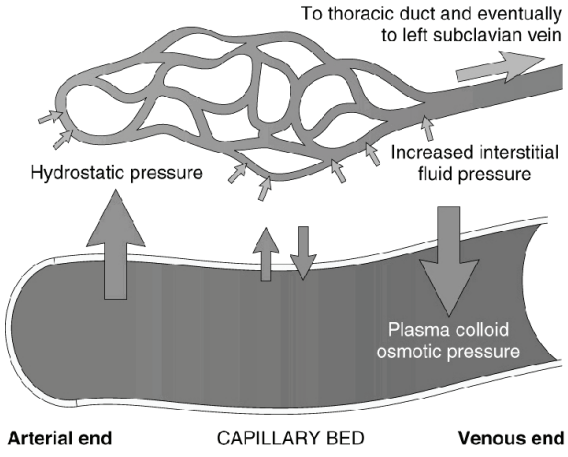
C. Oncotic pressure

D. Hydrostatic pressure

- local

- generalized -CHF

- lymphatic obstruction



Ref: Robbins, Pathologic Basis of Disease, 6th Ed.

E. Constant leakage and retrieval by lymph vessels. As long as everything is working right, the balance is maintained and the water goes merrily around and around.

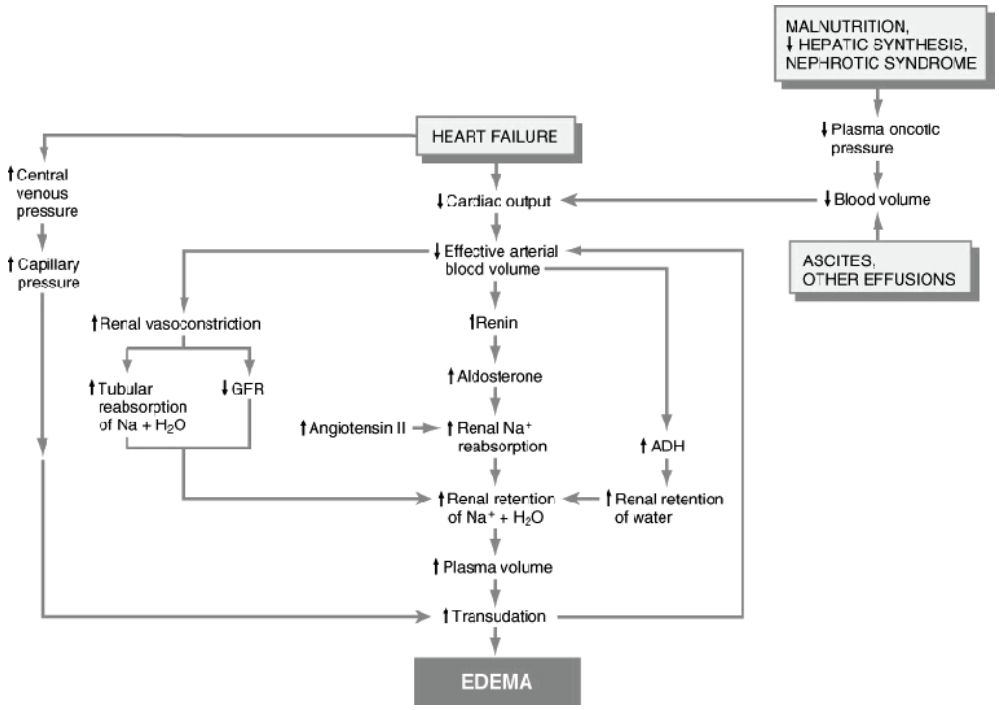
But things don't always go as planned. **Disorders described here are a daily events in every hospital. The leading causes of death in the "civilized" world revolves around excessive and/or inappropriate blood clotting.**

-Myocardial infarction (MI)

- Pulmonary embolus (PE)

- "Stroke" (CVA)

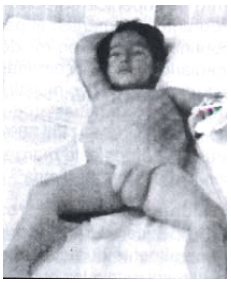
II. Too much water in the intercellular (between the cells) space. **EDEMA**



A. **TRANSUDATE vs EXUDATE** (How do you tell which is which)

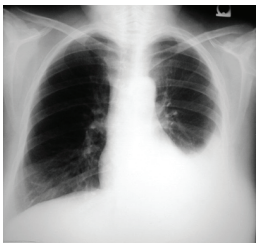
B. Inflammatory edema “injury water” “Tumor of inflammation”

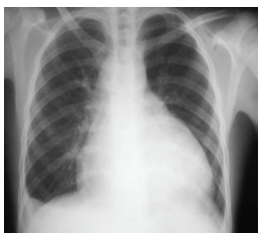
C. Non-inflammatory edema



1. Water in the tissues themselves
   - Anasarca
   - “Pre-sacral” edema
   - periorbital edema

2. Water filling up hollow (or potential) spaces
   - hydrothorax
   - hydropericardium
   - hydroperitoneum



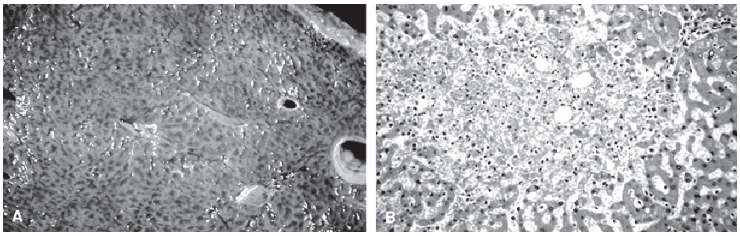


3. “Third space” concept

4. Cerebral edema, a special situation

III. Hyperemia and congestion

A. Active



B. Passive

- acute

- chronic (nutmeg liver for example)

IV. Hemorrhage, hemostasis and thrombosis

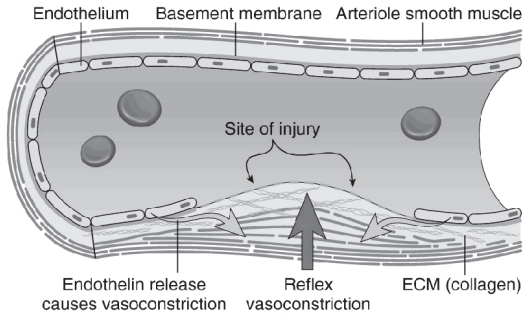
A. There are three essential elements for blood clotting to work as it should.

1. Platelets

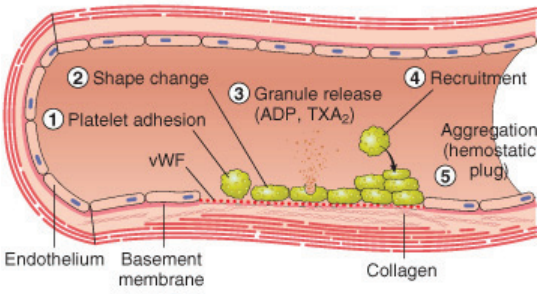
2. Vessels

3. Proteins (no I am not going to ask you to produce the clotting sequence)

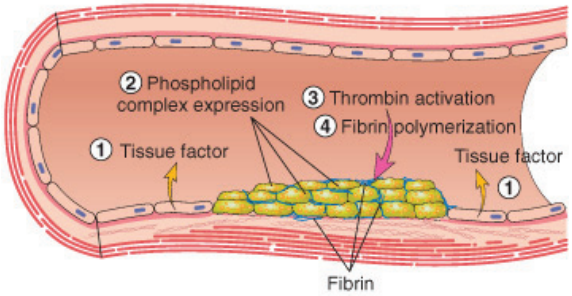
A. VASOCONSTRICTION



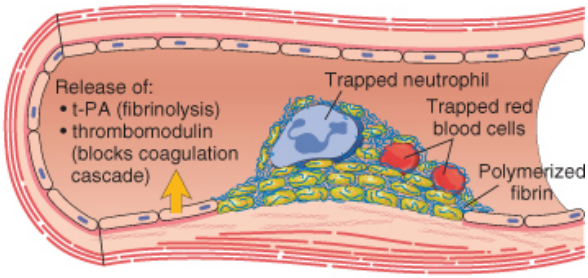
B. PRIMARY HEMOSTASIS

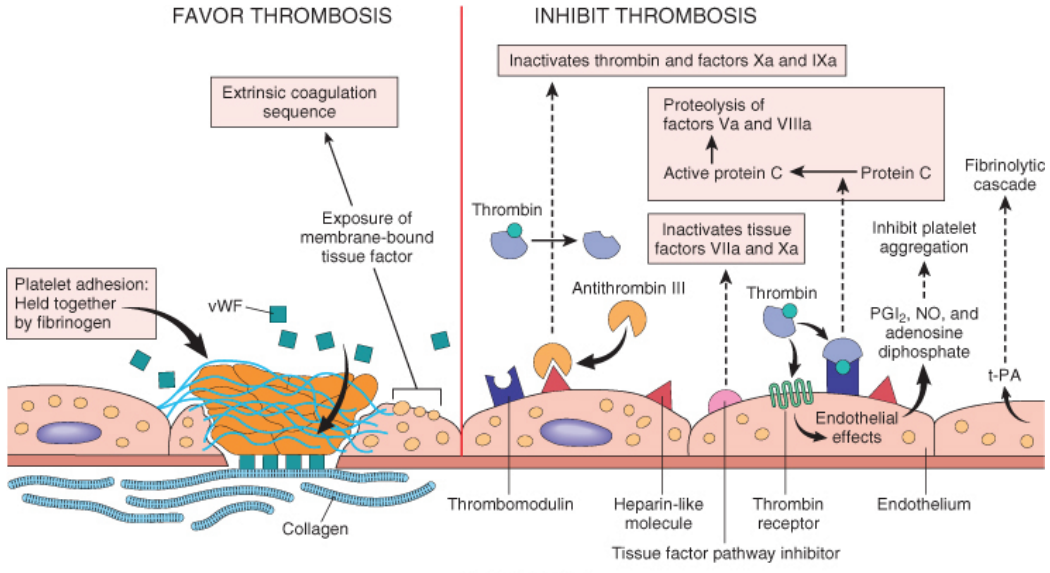


C. SECONDARY HEMOSTASIS



D. THROMBUS AND ANTITHROMBOTIC EVENTS





B. Hemorrhage

- what does it look like?

- volume?

- location?

- duration (in other words did you have time to accommodate)

1. What does it look like? Skin

- petechiae, little bitsy specks, often called a "rash" (platelet abnormality)

- purpura

- ecchymosis

2. Accumulation = hematoma (a blood "body" or "tumor")

3. In a hollow or potential space

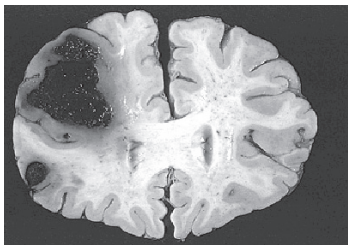
- hemopericardium

- hemothorax

- hemarthrosis

- hemoperitoneum

4. Location



- vital structures, CNS

- can you even see it, GI loss

- expected, but is too much, menstrual

5. Volume, much harder to assess than you might think.

6. Duration

- The longer it takes, the lower it can go.

- Sudden massive loss is a disaster.

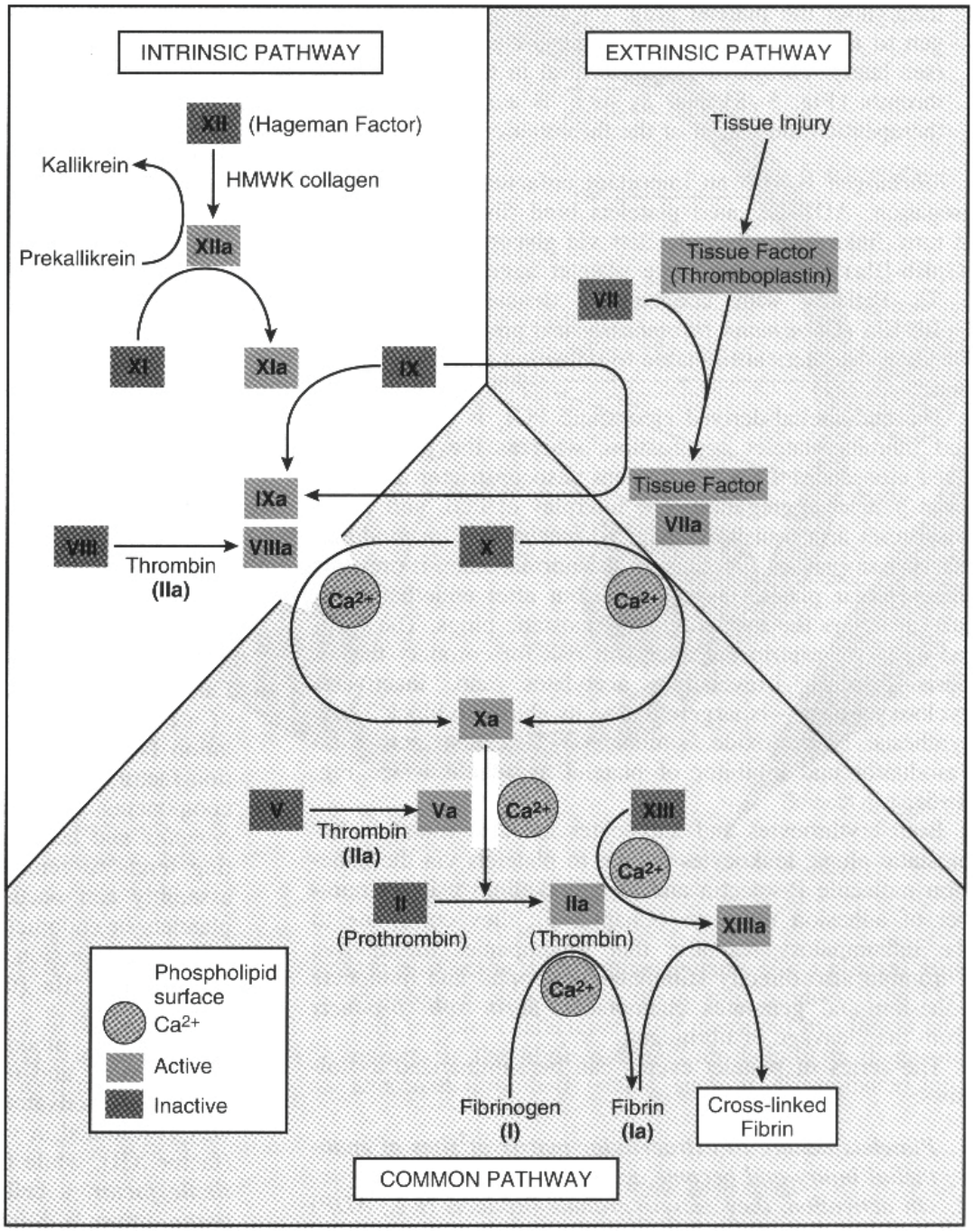
7. Genetic causes

- family history

8. Acquired deficiency (can this be for real?)

- habits and coexisting diseases

- medications (Do really mean aspirin is a medication?)



C. Thrombosis

1. How does a thrombus differ from a blood clot? You know, philosophically.

2. **Three major factors in increased coagulability**

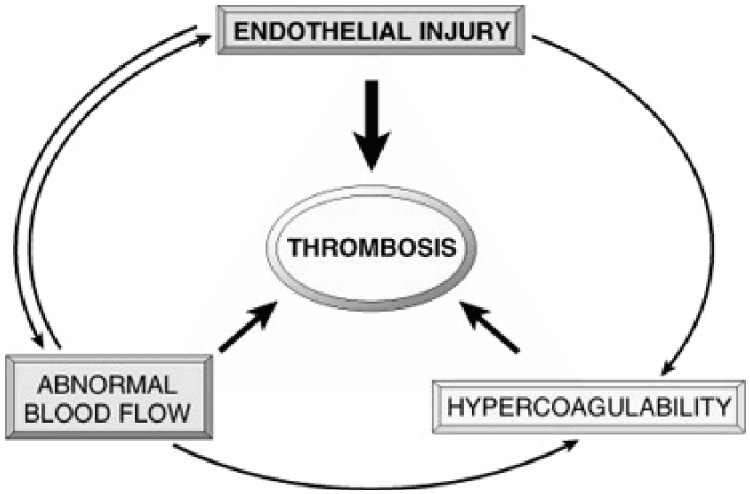
- endothelial damage

- stasis (alterations in normal blood flow, ie slowing and pooling)

- abnormalities of proteins

- too many clotting factors

- too little inhibition (AT III, Protein C and S)



3. Venous thrombosis

- location

- stasis (get ‘um up)

- factor activation with slowed flow

4. Arterial thrombosis

- endothelial damage

- platelet adherence

Primary (Genetic)
Common
- Mutation in factor V gene (factor V Leiden)
- Mutation in prothrombin gene
- Mutation in methyltetrahydrofolate gene

Rare
- Antithrombin III deficiency
- Protein C deficiency
- Protein S deficiency

Very rare
- Fibrinolysis defects

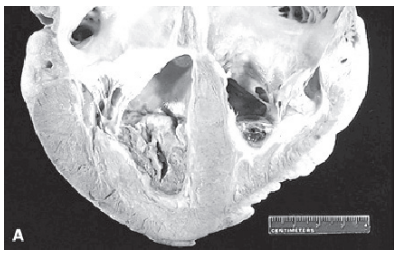
Secondary (Acquired)
High risk for thrombosis
- Prolonged bed rest or immobilization
- Myocardial infarction
- Tissue damage (surgery, fracture, burns)
- Cancer
- Prosthetic cardiac valves
- Disseminated intravascular coagulation
- Heparin-induced thrombocytopenia
- Antiphospholipid antibody syndrome (lupus anticoagulant syndrome)

Lower risk for thrombosis
- Atrial fibrillation
- Cardiomyopathy
- Nephrotic syndrome
- Hyperestrogenic states (pregnancy)
- Oral contraceptive use
- Sickle cell anemia
- Smoking

5. Morphologic characteristics

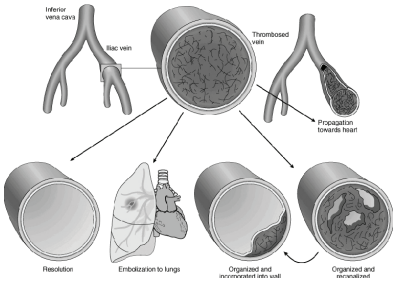
- laminar assembly

- lines of Zahn



- mural thrombus

6. What happens now?



- resolution

- propagation

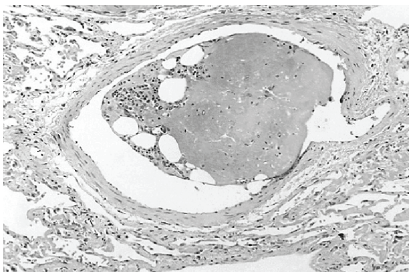
- organization and “recanalization”

- embolization (actually, lots of things can be emboli)

    - pulmonary embolization (PE)

        - “saddle embolus” a really big one

    - air



    - bone chips and marrow

    - bullets

    - amniotic fluid

    - These are all space occupying substances, moving along in the more or less intact circulatory system.

7. The disastrous “melt down” - Disseminated Intravascular Coagulation (DIC)

1. Never on it’s own

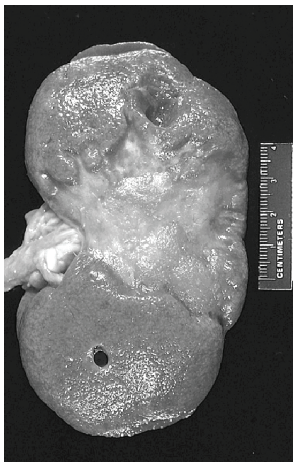
2. Remove or correct the offending circumstance

3. Obstetrical disaster most commonly

4. Sepsis

5. Shock

V. Infarction: an area of ***ischemic*** necrosis ***within*** a tissue or an organ, resulting from occlusion of the arterial supply or venous drainage. (Please know this definition.)



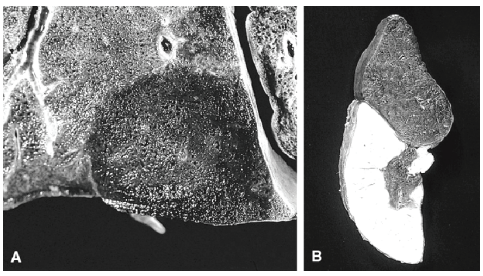
A. Most are arterial thrombosis, but not all.

B. Anemic (white)

1. “End artery” supply system.

2. No blood, the tissue dies and there is no hemorrhage into the dead meat.

- organs?



C. Hemorrhagic (red)

1. Venous occlusion generally

- often previous congestion

2. “Looser” tissues

3. Overlapping or “dual circulation systems

- lung

- gastrointestinal

4. Bleeding into the area of ischemic necrosis

D. Other complicating factors

1. “bland” (sterile)

2. “septic” (infected area of necrosis)

E. What features make an infarction likely to develop?

1. What kind of vascular supply does this organ have?

2. Rate of development of the occlusion. Again, is there time to develop collateral channels?

3. Vulnerability of the organ

- Skeletal muscle

- CNS

- Heart

4. Outcome or significance?

VI. Shock: widespread hypoperfusion of tissues due to reduction in blood volume or cardiac output, or even redistribution of blood, resulting in an inadequate effective circulatory volume.

A. **TISSUE HYPOXIA AND ACIDOSIS**

B. Downward spiral that may not turn around

C. Basic patterns

1. Hypovolemic or hemorrhagic

2. Cardiogenic

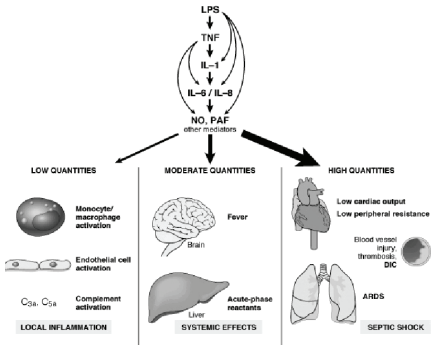
3. Septic, more common than many people think.

    - Intensive care units, catheters, really sick folks

    - gram negative bugs

        - endotoxins

    - rarely gram positive or fungi



4. Anaphylactic - type 1 hypersensitivity

5. Neurogenic

    - anaesthetic disasters

    - spinal cord injuries

D. Stages

1. Non-progressive; compensatory mechanisms activated and vital organs spared

2. Progressive: going down the spiral with acidosis and further deterioration

3. Irreversible: enough ischemic injury so that survival is not possible

E. Vulnerable organs

1. CNS

2. Kidneys - tubular damage - no urine output

3. GI - hemorrhagic enteropathy

4. Adrenals - they get wrung dry

5. Heart - subendocardial hemorrhages - generalized dysfunction of pump

F. Clinical course and outcome

1. Depends on age, general state of health and type of precipitating condition

- cardiogenic and septic very poor prognosis in elderly

- young people with hypovolemia actually do pretty well

2. Clinical appearance

- cardiogenic or hypovolemic

    - ashen grey with weak and thready pulse

    - cold and clammy

    - mottled areas of skin coloration

- septic

    - with septic may be flushed and even seem warm because of peripheral vascular dilation