Hemodynamic disorders
Objectives

• What are the disorders that affect the fluid status of circulation?
• What are the causes and important pathological features of each one?
• Mentioning some clinical examples of these disorders
Hemodynamic disorders

• Disorders in the normal fluid status
• Abnormality in maintenance of the blood vessel wall integrity as well as intravascular pressure and osmolarity.
Hemodynamic disorders include:

• Fluid extravasation to the interstitial spaces=..?
• Locally increased volume of blood in a particular tissue=..?
• Inappropriate clotting =...?
• Migration of clots or other objects=..?
• Inability to clot after injury=..?
• Extensive reduction in the intravascular volume=..?
Factors that control different fluid compartments
Pitting edema

Ankle region
Ascites (severe form) complicating liver cirrhosis
Rt sided heart failure secondary to lung disease (note cyanosis)
The alveolar spaces are filled with pale pink amorphous fluid. Note the congested septal
The surfaces of the gyri are flattened as a result of compression of the expanding brain by the dura mater and inner surface of the skull. The sulci are very narrow. Such changes are associated with Cerebral edema.
CONGESTION
There is engorgement of capillaries within alveolar septa, pinkish edema fluid within alveolar spaces. The latter also contains red cells and macrophages.

Chronic venous congestion (CVC) lung
A: Central areas are red and slightly depressed compared with the surrounding tan viable parenchyma.

B: Centrilobular
Numerous raised 3- to 5-mm palpable hemorrhages of the skin. In this case purpura is due to small-vessel vasculitis.
Punctate petechial hemorrhages of the colonic mucosa, a consequence of thrombocytopenia.
The blotchy areas of hemorrhage in the skin are called ecchymoses (singular ecchymosis). Ecchymoses are larger than petechiae.
Even relatively inconsequential volumes of hemorrhage in a critical location, or into a closed space (such as the cranium), can have fatal outcomes.
THROMBOSIS

• inappropriate (pathologic) activation of normal hemostatic processes that eventuate in the formation of a solid mass of blood constituents (thrombus). (Virchow’s triad)
  ➢ 1. Endothelial injury
  ➢ 2. Stasis or turbulence of blood flow
  ➢ 3. Blood hypercoagulability
Virchow's triad in thrombosis

- Endothelial Injury
- Abnormal Blood Flow
- Hypercoagulability

Thrombosis
Endothelial injury

• Endothelial cell loss:
  - Ulcerated atherosclerotic arterial plaques.
  - Traumatic vascular injury
  - Vasculitis

• Endothelial dysfunction:
  - Hypertension
  - Hypercholesterolemia
  - Products absorbed from cigarette smoke
  - Turbulent flow over deformed cardiac valves
  - Bacterial toxins
Stasis and turbulence

1) Disrupting the laminar flow =...??
2) Preventing dilution of activated clotting factors by fresh-flowing blood
3) Retarding the inflow of clotting factor inhibitors
4) Permitting the buildup of thrombi
5) Promoting endothelial cell activation
Examples of thrombosis resulting from stasis and turbulence:

- Ulcerated atherosclerotic plaque.
- Aneurysm.
- Myocardial infarction.
- Mitral valve stenosis.
- Hyperviscosity syndromes.
- Sickle cell anemia.
Hypercoagulability:
any alteration of the coagulation pathways that predisposes to thrombosis

• **Primary**: e.g., mutations in *factor V* & *prothrombin* genes.

• **Secondary**:
  • 1. Cardiac failure
  • 2. Trauma
  • 3. Oral contraceptive use & pregnancy
  • 4. Disseminated cancers.
  • 5. Advancing age
  • 6. Smoking
  • 7. Obesity
Thrombi: general features:

- can develop anywhere in the cardiovascular system.
- focally attached to the underlying vascular surface.
- propagating part of a thrombus tends to be poorly attached and may form emboli.
- can have grossly (and microscopically) obvious laminations called **lines of Zahn**...?
Cardiac auricle thrombus showing lines of Zahn
Thrombi can have grossly (and microscopically) apparent laminations called lines of Zahn; these represent pale platelet and fibrin layers alternating with darker
Pathological features of arterial thrombi:

- begin at sites of endothelial injury
- propagate in a retrograde direction.
- Prominent lines of Zahn.
- are paler in color.
- are frequently occlusive.
- superimposed on an atherosclerotic plaque, and vascular injury (vasculitis, trauma) can be involved.
An occlusive thrombus within the anterior descending branch of the Lt. coronary artery.

Coronary atherosclerosis + superadded thrombosis
The narrowed lumen is occupied by an occlusive thrombus (arrow). The narrowing is due to atherosclerosis. Note that the intimal atheroma display cholesterol clefts (whitish needle-like spaces). There is atrophy of the media.
Extensive involvement of the aortic intima by coalescent yellowish plaques of atherosclerosis with foci of ulceration that harbor brownish thrombi.
Pathological features of venous thrombi:

- begin at sites of venous stasis.
- Propagate in the direction of blood flow.
- Less Prominent lines of Zahn.
- contain more enmeshed RBCs and are therefore called red, or stasis thrombi.
- almost invariably occlusive,
- May create long cast by propagation.
A 35-year-old woman has a massively swollen Rt. leg.

Deep vein thrombosis (DVT)
Red thrombus (femoral vein)
Clinical examples of thrombi:

- Arterial: coronary thrombi
- Venous: DVT(?) 90% in lower extremities.... Other...?
- Vegetation=..?
- Mural thrombi=...?
- Intracranial??
- DIC=..??
Mural thrombi

Thrombus in the left and right ventricular apices, overlying white fibrous scar.
Note the relatively bulky vegetation causing destruction of the aortic valve cusps.
FATE OF THE THROMBUS

1) Propagation.
2) Embolization.
3) Dissolution.
4) Organization and recanalization.
Potential outcomes of venous thrombosis
Embolism
Embolism

- An embolus is a detached intravascular, solid, liquid, or gaseous mass that is carried by the blood to a site distant from its point of origin.
  - 1. Thromboemboli: 99% of all emboli.
  - 2. Fat emboli.
  - 3. Air emboli: nitrogen?
  - 4. Atherosclerotic emboli: atheromatous debris
  - 5. Tumor emboli
  - 6. Bone marrow emboli:
  - 7. Foreign body emboli as bullets or shrapnel.
Pulmonary thromboembolism

• preventable cause of death in hospitalized patients.
• majority of cases the emboli originate from deep veins of the lower limbs.
• May settle within the pulmonary trunk, bifurcation, or smaller arteries causing different clinical presentations that depend also on the cardiopulmonary status.. Discuss?
Embolus derived from a lower extremity deep venous thrombosis and now impacted astride the bifurcation of the main pulmonary artery.
The rounded holes that appear in the vascular spaces here in the lung are fat emboli. Fat embolization syndrome occurs most often following trauma with fracture of long bones that releases fat globules into the circulation which are trapped in pulmonary capillaries.
This is a rare finding that may complicate a term pregnancy at delivery. Seen here in a pulmonary artery branch is an amniotic fluid embolus that has layers of fetal squames. Amniotic fluid embolization can have the same outcome as a large saddle pulmonary embolus.
SYSTEMIC (arterial) THROMBOEMBOLISM

• Sources include:
  • Intracardiac mural emboli
  • Atherosclerotic plugs
  • Aneurysm.
  • Valvular vegetation.
  • Paradoxical ?

• Clinical consequences depend on artery affected.
INFARCTION

• a localized area of ischemic cell necrosis in a living organ or tissue resulting from:
  • cessation of arterial blood supply (mainly)
  • Defect in venous drainage (occasionally)

• Causes of vascular obstruction:
  • Thromboembolism
  • Atherosclerosis.. How?
  • Spasm
  • Pressure from outside
  • Twisting of vascular bed.. Examples?
Gross features of infarction

• All infarctions are wedge shape...Why?
• Can be pale or red...Why?
• Line of demarcation appear with the time.,Why?
• Wedge shape of the infarct
Red infarction - intestine

Pale infarction - spleen
Gross features of infarction

**Red infarcts**
- With venous occlusion.
- Loose tissues.
- Double blood supply.
- Tissues rich in collaterals.
- Previously congested organ.
- Re-establishment of arterial supply

**White (pale) infarcts**
- With arterial occlusion.
- In solid organ
Renal pale infarction – note the line of demarcation
Cerebral infarction from an arterial embolus, which often leads to a hemorrhagic appearance. There is edema which obscures the structures. The acutely edematous infarcted tissue may produce a mass effect. Note the decrease in size of the ventricle on the left with shift of the midline.
Healed infarctions
Microscopic features of infarction:

- Ischemic coagulative necrosis (dominant)
- In the brain..?
- Inflammatory response starts within 1-2 days.
- Reparative response starts.. Where?
- Ultimately it transformed into fibrous scar within a period depends on the size.
- Septic infarcts convert to abscess
Early infarction

hyperemia

Inflammatory response
Coagulative necrosis-infarction
Factors influence infarct development

• 1. The nature of the vascular supply
• 2. The rate of development of the occlusion
• 3. Vulnerability of the tissue to hypoxia
• 4. The oxygen content of blood.
SHOCK

is a state of systemic hypoperfusion that is caused either by reduced cardiac output or by reduced effective circulating blood volume:

• A. Cardiogenic
• B. Hypovolemic
• C. Septic
• Less common are
• D. Neurogenic
• E. Anaphylactic
summary

• Hemodynamic disorders affect the normal circulation fluid status and include edema, congestion, thrombosis, embolism, and shock.
• Edema denote extravasation of fluid to the interstitial compartment.
• Thrombosis is an inappropriate clotting that results from endothelial disruption, stasis, or hypercogulability.
Summary (cont.)

• Emboli are circulating solid material that may lodge in the systemic or venous circulation.
• Infarction is a common consequence of many hemodynamic disorders including thrombosis and embolism.
• Shock is a clinical manifestation of significant fluid volume loss (hypovolemia).