DISORDERS OF CIRCULATION

HYPEREMIA AND CONGESTION

A. DEFINITION: Hyperemia and congestion refer to increased intravascular blood volume in tissue, or an organ, or part of the body, such as an extremity.

1. Hyperemia (active hyperemia) occurs with dilatation of an artery or arteriole and increased blood flow into the capillaries. Hyperemia is caused by sympathetic neurogenic discharge or chemical mediators.
   a. Clinical examples: Acute inflammation of tissue, blushing, body’s need to dissipate heat. Hyperemia of the skin appears red (oxygenated blood).

2. Congestion (passive hyperemia) occurs because of impaired venous drainage. The veins do not dilate because of the active influence of a sympathetic discharge or chemical mediator.
   a. Chronic passive congestion is a form of long standing congestion which produces characteristic changes in the lungs, liver and spleen. The congestion occurs because of right heart failure. (Refer to Figure 4-1 in “Robbins Basic Pathology”)
   Clinical examples: Congestive heart failure, poor venous drainage (blue – red deoxygenated blood).

EDEMA

A. DEFINITION: Accumulation of abnormal amounts of fluid in interstitial spaces or body cavities.

B. MECHANISMS: How does edema form? Why does your patient have edema?

1. Normal physiology: Distribution of fluid between the intravascular and interstitial compartment.
   The forces which influence the movement of fluid across the capillary wall are capillary hydrostatic pressure, interstitial hydrostatic pressure, plasma colloid osmotic pressure and interstitial colloid osmotic pressure. The near equilibrium created by these forces allows for most of the fluid to remain within the vascular compartment; nevertheless, even under normal conditions, some fluid accumulates in the interstitium. This (slight) excess fluid is removed and returned to the circulation by lymphatics.
   Therefore, if you are going to try to understand why your patient has edema, you must consider the various factors which determine the volume of fluid in the interstitium. (Refer to Table 4-1 and Figure 4-2 in “Robbins Basic Pathology”)

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2. Noninflammatory vs inflammatory edema

a. Non-inflammatory: Fluid accumulates in the interstitium as a consequence of changes in hydrostatic pressure and colloid osmotic pressure. Referred to as TRANSUDATE

b. Inflammatory: Tissue injury alters blood flow and vascular permeability as a result of increased interendothelial space. This process is modulated by a variety of chemical mediators. Referred to as EXUDATE

HEMORRHAGE

A. DEFINITION: Hemorrhage is a flow of blood from a ruptured blood vessel. The blood may flow into tissue, into a body cavity or outside the body.

B. MECHANISM OF HEMORRHAGE: Bleeding occurs when a large or small blood vessel is disrupted by a mechanical force (trauma) or a pathologic process (inflammation, neoplasm). The process of hemostasis, which you will study later in the course, maintains blood in a liquid state and prevents uncontrolled bleeding. Abnormal hemostasis causes a predisposition to bleeding. (bleeding diatheses).

C. CLINICAL CORRELATIONS

1. Other than menstruation, hemorrhage of any kind should be considered abnormal. When developing differential diagnoses for hemorrhage, consider abnormalities in the blood vessel wall or with platelets or with clotting factors.

2. Define the following terms: Hematoma, hemothorax, hemopericardium, hemoperitoneum, hemarthrosis, petechiae, purpura, ecchymosis. (refer Robbins Basic Pathology p. 100,101)

THROMBOSIS

A. DEFINITION: Formation of thrombus (blood clot) within an uninterrupted vascular system. It is considered a pathologic process.

B. MECHANISMS (Refer to Figure 4-12 in “Robbins Basic Pathology”)

1. Endothelial injury
   Clinical examples: Inflammation; advanced atherosclerosis
2. **Altered blood flow:** turbulence vs. stasis  
   Clinical example: atrial fibrillation, bed rest

3. **Hypercoagulable state:** Predisposition to easy clot formation  
   Clinical examples: Protein C deficiency; woman who smokes and uses oral contraceptives.

C. **Fate of a Thrombus**

1. Propagation – Thrombus enlarges. Results in increased odds of vascular occlusion, or embolization

2. Dissolution – Activation of the body’s fibrinolytic system may lead to rapid shrinkage and even complete dissolution of a newly formed thrombus. With time, extensive fibrin polymerization renders the thrombus increasingly resistant to lysis/dissolution.

3. Embolization – Part or all of the thrombus is dislodged and travels elsewhere in the vasculature.

4. Organization and recanalization- Older thrombi become “organized” with the ingrowth of endothelial cells, smooth muscle cells, and fibroblasts into the thrombus. With time small vascular (capillary) channels are formed re-establishing a degree of flow within the vascular lumen.

**EMBOLISM**

A. **DEFINITION:** An embolus is an intravascular substance (solid, liquid, gas) which is carried by blood from a point of origin to a distant site.
B. TYPES OF EMBOLI
1. Fragments of thrombi (thromboembolism)
2. Amniotic fluid.
   Uncommon, but serious complication of active labor. Amniotic fluid enters placental membranes and/or uterine vein rupture.
3. Air (gas).
   Gas bubbles within circulation obstruct vascular flow.
   Can develop during medical procedures (air trapped in coronary artery during bypass surgery, in cerebral artery during neurosurgery).
   Decompression sickness is a form of gas embolism encountered by scuba divers caused by sudden changes in atmospheric pressure.
4. Fat and marrow embolism
   s/p soft tissue crush injury or long bone injury where marrow vascular sinusoids release microscopic fat globules into the circulation.
   “Fat Embolism Syndrome” – systemic, fatal in up to 10+% patients

(Refer to Figures 4-15, 4-16 in “Robbins Basic Pathology”).

INFARCTION

A. DEFINITION: An infarct is an area of ischemic necrosis within tissue or an organ, produced by occlusion of either its arterial supply or venous drainage. The majority of infarcts are associated with thromboembolism and involve arterial occlusions.

B. TYPES OF INFARCT
1. White: ischemic, usually arterial occlusion
   Cite 2 classic examples:

2. Red (Hemorrhagic): may be venous or arterial occlusion
   Cite 2 classic examples:

Note: Not always a clear distinction. Think about ischemic and hemorrhagic infarctions as part of a continuum.

C. FACTORS WHICH INFLUENCE THE DEVELOPMENT OF AN INFARCT
1. Nature of the vascular supply

2. Rate of development of the occlusion

3. Vulnerability of tissue to hypoxia

4. Oxygen content of blood

D. WHAT HAPPENS WHEN A THROMBUS FORMS IN AN ARTERY OR VEIN? The clinical outcome of thrombosis depends on many factors which include:

1. Degree of occlusion of vessel lumen: Partial occlusion of a vessel, even a coronary artery, may allow enough blood to flow to vital tissue. However, complete occlusion will usually result in infarction, unless there is enough collateral circulation to support oxygen requirements of the myocardium. Survival of the patient will be determined by the size and location of the infarct as well as collateral circulation.

2. Size and location of the blood vessel: Occlusion of the carotid artery may result in death. Occlusion of a small vessel in the cerebrum may cause an incapacitating stroke or death. Occlusion of secondary branches of a renal or pulmonary artery may cause an asymptomatic infarct.

SHOCK

A. DEFINITION: Final common pathway for a number of possible events which result in systemic hypoperfusion of tissues. Can be caused by diminished cardiac output or by reduced effective circulating blood volume.

B. THREE MOST COMMON FORMS of SHOCK

1. Cardiogenic Shock
   Results from low cardiac output due to myocardial pump failure

2. Hypovolemic Shock
   Results from low cardiac output due to loss of blood or plasma volume

3. Septic shock
   Results from arteriolar vasodilatation and venous blood pooling that stems from systemic immune response to microbial infection. (Refer to Figure 4-19, Robbins Basic Pathology). A future MHD lecture will be devoted to the topic Septic Shock