Hemodynamic Disorders
Normal fluid homeostasis is maintained by vessel wall integrity, intravascular pressure and osmolarity within certain physiologic ranges.

Changes in intravascular volume, pressure, or protein content, or alterations in endothelial function will affect the movement of water across the vascular wall.
1- Edema

Increased fluid in the interstitial tissue spaces.
Patho-physiologic Causes of Edema

- **Increased Hydrostatic Pressure**: Impaired venous return

- **Reduced plasma osmotic pressure (Hypo-proteinemia)**: Liver cirrhosis, nephrotic syndrome

- **Lymphatic Obstruction**: Neoplastic, or postsurgical

- **Sodium Retention**: Excessive salt intake with renal insufficiency

- **Inflammation**: Acute inflammation, Chronic inflammation
Types of edema

- **Anasarca**: Generalized edema

- **Dependent edema**: Prominent feature of congestive heart failure, particularly of the right ventricle.

- **Renal edema**: Edema as a result of renal dysfunction or nephrotic syndrome is generally more severe than cardiac edema and affects all parts of the body equally.
• Peri-orbital edema: is a characteristic finding in severe renal disease.

• Pitting edema: finger pressure over substantially edematous subcutaneous tissue displaces the interstitial fluid and leaves a finger-shaped depression.

• Pulmonary edema: most typically seen in the setting of left ventricular failure.
Fetal Anasarca
2- Hyperemia and Congestion

- Both indicate a local increased volume of blood in a particular tissue.
Hyperemia versus congestion.

In both cases there is an increased volume and pressure of blood in a given tissue with associated capillary dilatation and a potential for fluid extravasation.
In hyperemia, increased inflow leads to engorgement with oxygenated blood, resulting in *erythema*.

In congestion, diminished outflow leads to a capillary bed swollen with deoxygenated venous blood and resulting in *cyanosis*. 
Hyperemia
Varicose Veins

Congestion
3- Hemorrhage

Extravasation of blood due to vessel rupture
Types

- **Hematoma**: accumulation of blood within tissue.

- **Petechiae**: minute 1 to 2mm hemorrhages into skin, mucous membranes, or serosal surfaces.

- **Purpura**: slightly larger (≥3 mm) hemorrhages.
• **Ecchymoses**: larger (>1 to 2 cm) subcutaneous hematomas (i.e., bruises)

• **Hemothorax, hemopericardium, hemoperitoneum, or hemarthrosis (in joints)**: Large accumulations of blood in one of the body cavities
Petechial hemorrhages of the colonic mucosa

Intracerebral bleeding
Subarachnoid Haemorrhage:
Petechiae & Ecchymoses
Conjunctival Petechiae
Hemorrhage: Epidural hematoma
Hemothorax
4- Thrombosis
Hemostasis and Thrombosis

Normal hemostasis result of a set of well-regulated processes that accomplish two important functions:

1. They maintain blood in a fluid, clot-free state in normal vessels.
2. They are aimed to induce a rapid and localized hemostatic plug at a site of vascular injury.
Thrombosis: an inappropriate activation of normal hemostatic processes, such as the formation of a blood clot (thrombus) in uninjured vasculature or thrombotic occlusion of a vessel after relatively minor injury.
Both hemostasis and thrombosis are regulated by three general components:

– the vascular wall

– platelets

– the coagulation factors.
• Three primary causes for thrombus formation, the so-called Virchow triad:

  (1) Endothelial injury

  (2) Stasis or slowing of blood flow

  (3) Blood hyper-coagulability
Virchow triad in thrombosis. Endothelial integrity is the single most important factor. Note that injury to endothelial cells can affect local blood flow and/or coagulability; abnormal blood flow (stasis or turbulence) can, in turn, cause endothelial injury. The elements of the triad may act independently or may combine to cause thrombus formation.
• Thrombi may develop anywhere in the cardiovascular system, but stasis is a major factor in the development of venous thrombi.

• An area of attachment to the underlying vessel or heart wall, frequently firmest at the point of origin, is characteristic of all thromboses.
• The propagating tail may not be well attached and, particularly in veins, is prone to fragmentation, creating an embolus.

• Mural thrombi - arterial thrombi that arise in heart chambers or in the aortic lumen, that usually adhere to the wall of the underlying structure
• Mural thrombi. Thrombus in the left and right ventricular apices, overlying a white fibrous scar.
Thrombosis

Deep Vein Thrombosis (DVT)

- Blood flow
- Detached blood clot
- Valve
- Blood clots

Normal vs. DVT
• Fate of the Thrombus.

1. Propagation.

2. Embolization.

3. Dissolution.

4. Organization and recanalization.
Potential outcomes of venous thrombosis.
Laminated thrombus in a dilated abdominal aortic aneurysm.
Lines of Zahn: alternating layers of platelets and fibrin in the thrombus
5- 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.

• Emboli lodge in vessels too small to permit further passage, resulting in partial or complete vascular occlusion
Pulmonary Thrombo-embolism

- 95% of venous emboli originate from deep leg vein thrombi
• Large embolus derived from a lower extremity deep venous thrombosis and now impacted in a pulmonary artery branch.
Systemic Thromboembolism

- Emboli traveling within the arterial circulation.
- Most (80%) arise from intra-cardiac mural thrombi,
- Two thirds of which are associated with left ventricular wall infarcts
- The major sites for arteriolar embolization are:
  1. Lower extremities (75%)
  2. Brain (10%)
A- Fat Embolism

- Microscopic fat globules may be found in the circulation after fractures of long bones (which have fatty marrow) or, rarely, in the setting of soft tissue trauma and burns.
• Bone marrow embolus in the pulmonary circulation. The cleared vacuoles represent marrow fat that is now impacted in a distal vessel along with the cellular hematopoietic precursors.
Fat embolus in a glomerulus (kidney)
B- Air Embolism

- Gas bubbles within the circulation can obstruct vascular flow.
- Enter the circulation during obstetric procedures or as a consequence of chest wall injury.
- In excess of 100 cc is required to have a clinical effect
C- Amniotic Fluid Embolism

- Underlying cause is the infusion of amniotic fluid or fetal tissue into the maternal circulation via a tear in the placental membranes or rupture of uterine veins.
- Characterized by sudden severe dyspnea, cyanosis, and hypotensive shock, followed by seizures and coma.
6- Infarction
• An infarct is an area of ischemic necrosis caused by occlusion of either the arterial supply or the venous drainage in a particular tissue.

• Nearly 99% of all infarcts result from thrombotic or embolic events, and almost all result from arterial occlusion.
• Infarcts are classified on the basis of their color (reflecting the amount of hemorrhage) and the presence or absence of microbial infection
• Red (hemorrhagic) infarcts occur

(1) with venous occlusions (such as in ovarian torsion);

(2) in loose tissues (such as lung)

(3) in tissues with dual circulations (e.g., lung and small intestine).
- White (anemic) infarcts occur

1. with arterial occlusions in solid organs with end-arterial circulation (such as heart, spleen, and kidney)

2. Solid tissues
Examples of infarcts. (A) Hemorrhagic, roughly wedge-shaped pulmonary infarct. (B) Sharply demarcated white infarct in the spleen.
• The dominant histologic characteristic of infarction is ischemic coagulative necrosis.

• most infarcts are ultimately replaced by scar tissue.

• The brain is an exception to these generalizations; ischemic injury in the central nervous system results in liquefactive necrosis.
• Remote kidney infarct, now replaced by a large fibrotic cortical scar.
• Septic infarctions may develop when embolization occurs by fragmentation of a bacterial vegetation from a heart valve or when microbes seed an area of necrotic tissue.
Intimal Proliferation (Atherosclerosis), Lumen Greatly Reduced

Medial Calcification (Mönckeberg's Type of Sclerosis) Plus Some Intimal Thickening and Thrombosis

Dependent Rubor, Absence of Dorsalis Pedis Pulsation

Diabetic Ulcer

Gangrene of Toe

Extensive Gangrene

Aortogram: Obstruction of Left Iliac Artery in a Diabetic

Calcified Femoral Artery in a Diabetic Demonstrated by X-Ray

Atherosclerosis in Diabetes

Vascular Insufficiency in Diabetes
7- Shock
• Shock, or cardiovascular collapse, is the final common pathway for a number of potentially lethal clinical events, including severe hemorrhage, extensive trauma or burns, large myocardial infarction, massive pulmonary embolism, and microbial sepsis.
• gives rise to systemic hypo-perfusion caused by reduction in:

1. cardiac output

2. the effective circulating blood volume.

• The end results are hypotension, followed by impaired tissue perfusion and cellular hypoxia.
<table>
<thead>
<tr>
<th>Type of Shock</th>
<th>Clinical Examples</th>
<th>Principal Mechanism</th>
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<tbody>
<tr>
<td><strong>Cardiogenic</strong></td>
<td>- Ventricular rupture</td>
<td>Failure of myocardial pump owing to intrinsic myocardial damage, extrinsic pressure, or obstruction to outflow</td>
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<td>- Arrhythmia</td>
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<td>- Cardiac tamponade</td>
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<td></td>
<td>- Pulmonary embolism</td>
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<td></td>
<td>- Myocardial infarction</td>
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<td><strong>Hypo-volemic</strong></td>
<td>- Hemorrhage</td>
<td>Inadequate blood or plasma volume</td>
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<td>- Fluid loss, e.g., vomiting, diarrhea, burns, or trauma</td>
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<td><strong>Septic</strong></td>
<td>- Overwhelming microbial infections</td>
<td>Peripheral vasodilation and pooling of blood; endothelial activation/injury; leukocyte-induced damage; disseminated intravascular coagulation; activation of cytokine cascades</td>
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<td>- Endotoxic shock</td>
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<td>- Gram-positive septicemia</td>
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<td>- Fungal sepsis</td>
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Less commonly:

1. **Neurogenic shock** - in the setting of anesthetic accident or spinal cord injury, owing to loss of vascular tone and peripheral pooling of blood.

2. **Anaphylactic shock**, initiated by a generalized IgE-mediated hypersensitivity response, is associated with systemic vasodilatation and increased vascular permeability
Clinical Course

• The clinical manifestations depend on the precipitating insult.

• In hypovolemic and cardiogenic shock, the patient presents with hypotension; a weak, rapid pulse; tachypnea; and cool, clammy, cyanotic skin.

• In septic shock, the skin may initially be warm and flushed because of peripheral vasodilation.
END