# Hemodynamic Disorders, Thromboembolism and Shock

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## Infarction

- **Infarction**: are area of ischemic necrosis caused by occlusion of either arterial supply or venous drainage in particular tissue.
- **Causes of vascular obstruction:**

1. Vast majority of infarctions results from thrombotic or embolic events & almost all result from arterial occlusion.
2. Uncommon causes include:

a. Local vasospasm
b. Expansion of an atheroma secondary to intraplaque hemorrhage
c. Extrinsic compression of vessels e.g. by tumor, or edema within a confined space (e.g., in anterior tibial compartment syndrome). and entrapment in a hernia sac.
d. Vessel twisting (e.g., in testicular torsion or bowel volvulus),
e. Traumatic vascular rupture.

### Morphology of infarction

- Infarcts may be either red (hemorrhagic) or white (pale, anemic) and may be either septic or sterile.

### 1. Red Infarct:

**occur with:**

**1- Venous occlusion** (e.g. ovarian torsion).
**2- Loose tissues** (e.g. lung that allow blood to diffuse through and collect in infarcted zone).
**3- Tissues with dual blood supply** (e.g. lung & small intestines). It allows blood flow from an unobstructed parallel blood supply into infarcted zone.
**4- Tissues that previously congested** because of sluggish venous outflow.
**5-** When **blood flow is re-established to a site of previous arterial occlusion** and necrosis, e.g. following coronary angioplasty of an obstructed coronary artery.

### 2. White Infarct:

**Occur with**:

- **arterial occlusions**
- In **solid organs**
- With **end-arterial circulation without a dual blood supply** (e.g. heart, spleen, and kidney)
- **Tissue with increased density** which prevents the diffusion of RBCs from adjoining capillary beds into the necrotic area.

### ❖ Histologically:

- In most tissues, the main histologic finding associated with infarcts is **ischemic coagulative necrosis**.

- **Inflammatory response** begin within few hours along margin & becomes well defined in 1-2 days caused by necrotic tissues then gradual degradation of dead tissues with phagocytosis by inflammatory cells.
- Reparative response begins in margin & most infarction replaced by **scar tissues**.
- Infarction in the central nervous system **(CNS)** results in **liquefactive necrosis.**
- **Septic infarctions** occur when infected heart valve vegetations embolize or when microbes seed an area of necrosis; the infarct converted in to an abscess.

## Factors that Influence Development of an Infarct

- The outcomes of vascular occlusion can range from no effect to death of a tissue or person.
- **Major determinants of outcome include:**

1. ***Anatomy of the vascular supply.*** (The presence or absence of an alternative blood supply): Dual supply (i.e., lung, liver) or anastomosing circulations (i.e., radial and ulnar arteries, circle of Willis, small intestine) protect against infarction. By contrast, obstruction of end-arterial vessels generally causes infarction (i.e., spleen, kidneys).
2. ***Rate of occlusion:*** Slowly developing occlusions less often cause infarction by allow time for the development of collateral blood supplies. (e.g., collateral coronary circulation).
3. ***Tissue vulnerability to hypoxia*****:** Neurons undergo irreversible damage after 3 to 4 minutes of ischemia; myocardial cells die after only 20 to 30 minutes. In contrast, fibroblasts within ischemic myocardium are viable even after many hours.
4. ***Oxygen content of blood:*** Anemia, cyanosis, or CHF (with hypoxia) can cause infarction in an otherwise insignificant blockage.

# Shock

- **Shock:** is a state of systemic hypo-perfusion caused by reduction either in cardiac output or in the effective circulating blood volume, that lead to; hypotension, impairs tissue perfusion and cellular hypoxia.
- **Initially,** the cellular injury is reversible; however, **prolonged shock** eventually leads to irreversible tissue injury and is often fatal.
- **Shock is categorized in to:**

### 1- Hypovolemic shock:

- Result from low cardiac output due to loss of blood or plasma volume. Examples include:
  - Hemorrhage (external or internal)
  - Fluid loss as in severe vomiting, diarrhea & extensive burns.

### 2- Cardiogenic shock:

- Results from low cardiac output due to myocardial pump failure. It may be caused by:
  - Myocardial damage (infarction),
  - Ventricular arrhythmias,
  - Extrinsic compression (cardiac tamponade)
  - Outflow obstruction (e.g., pulmonary embolism)

### 3- Septic shock:

- Results from vasodilation and peripheral blood pooling caused by microbial infections associated with severe systemic inflammatory response syndrome.
- **Causes:**

- Overwhelming bacterial infection (Gram-positive bacteria, followed by Gram-negative bacteria and fungi. (Hence, an older synonym, "endotoxic shock," is no longer appropriate).

## ● Pathogenic mechanism:

- Bacterial toxin or microbial cell wall components **cause;**
  1. **Activation of leucocytes and endothelial cells** and trigger release of inflammatory cytokines that cause peripheral vasodilatation & increase vascular permeability.
  2. **Endothelial cell injury and direct activation of coagulation and complement cascades** lead to increased thrombotic tendencies with DIC (disseminated intravascular coagulopathy).
- These end with multi-organ failure; since hypotension, edema, and small vessel thrombosis all reduce oxygen and nutrient delivery to tissues.

❖ **Less commonly shock:**

**4- Neurogenic shock:**

- Result from a loss of vascular tone associated with anesthesia or secondary to a spinal cord injury.

**5- Anaphylactic shock:**

- Results from systemic vasodilation and increased vascular permeability that is triggered by an immunoglobulin E–mediated hypersensitivity reaction.

## ❖ Stages of shock

- Shock is a progressive disorder often culminating in death.
- Shock tend to evolve through three phases:

**1. Nonprogressive phase:** during which reflex neurohmoral compensatory mechanisms are activated (catecholamines, sympathetic stimulation, ADH, renin-angiotensin axis, etc.) to maintain cardiac output, blood pressure and perfusion of vital organs.

**The compensatory mechanisms include:**

a- Arteriolar constriction leading to increase peripheral vascular resistance and blood pressure.
b- Increase heart rate leading to increase cardiac output.
c- Retention of fluid through increase secretion of ADH & activation of rennin angiotensin aldosterone axis to retain fluid.

- **If the underlying causes are not corrected, shock passes imperceptibly to the progressive phase.**

**2. Progressive phase**: characterized by tissue hypoperfusion and worsening circulatory and metabolic abnormalities including lactic acidosis due to anaerobic glycolysis. The acidosis also blunts the vasomotor response, causing vasodilation.

- In the absence of appropriate intervention, or in severe cases, the process eventually enters an irreversible stage.

**3. Irreversible Phase:** In which cellular and tissue injury is so severe that even if the hemodynamic defects are corrected, survival is not possible.

- ➢ Widespread cell injury is reflected in lysosomal enzyme leakage, further aggravating the shock state.
- ➢ Myocardial contractile function worsens, in part because of increased Nitric oxide synthesis.
- ➢ The ischemic bowel may allow intestinal flora to enter the circulation, and thus bacteremic shock may be superimposed.

- Commonly, further progression to renal failure occurs as a consequence of ischemic injury of the kidney, and despite the best therapeutic interventions, the downward spiral frequently culminates in death.

## Pathological Changes

- The cellular and tissue changes induced by shock are essentially those of hypoxic injury due to a combination of **hypoperfusion** and **microvascular thrombosis.**
- Although any organ can be affected, the brain, heart, kidneys, adrenals, and gastrointestinal tract are most commonly involved.
- Brain: Ischemic encephalopathy.
- Heart: coagulation necrosis
- Adrenal: cortical cell lipid depletion is akin to that seen in all forms of stress and reflects increased use of stored lipids for steroid synthesis.
- Kidneys: acute tubular necrosis which lead to oliguria or anuria &electrolytes disturbances.
- Lungs: diffuse alveolar damage.
- GIT: focal mucosal hemorrhage &necrosis.
- Except of neuron and myocyte loss, virtually all affected tissues can recover completely if the patient survives.
- **Fibrin thrombi** can form in any tissue but typically are most readily visualized in kidney glomeruli.

## Clinical Features

- The clinical manifestations of shock **depend on the cause.**
- **In hypovolemic and cardiogenic shock,** patients present with hypotension, a weak rapid pulse, tachypnea, and cool, clammy, cyanotic skin.
- **In septic shock,** the skin may be warm and flushed owing to peripheral vasodilation.
- The initial underlying cause that precipitated the shock may be life-threatening (e.g. myocardial infarct, severe hemorrhage, or sepsis). Later, the organ dysfunction involving **cardiac, cerebral, and pulmonary** function worsen the situation.
- **If patients survive** the initial complications may develop **renal insufficiency** characterized by a progressive decrease in urine output and severe fluid and electrolyte imbalances.

## Prognosis

- The prognosis depends on the **cause** and **duration** of shock.
- Patients with **hypovolemic shock** may survive with appropriate management (more than 90% of young, otherwise healthy patients survive with appropriate management)
- **Septic shock, or cardiogenic shock** associated with worse outcomes, even with state-of-the-art care.

## References


- Chapter 4: Hemodynamic Disorders, Thromboembolism, and Shock