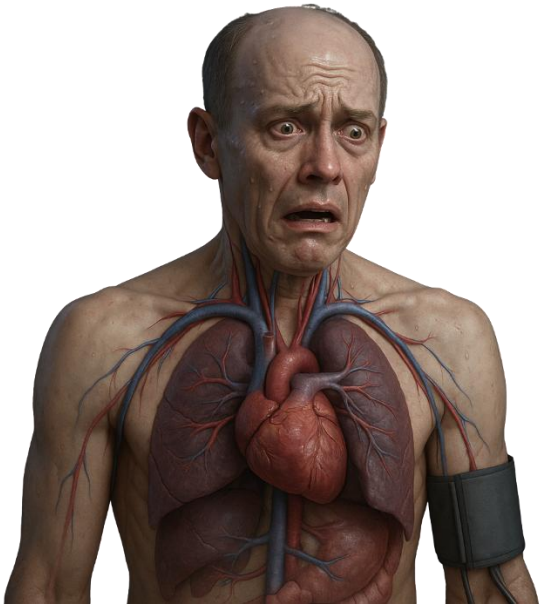
Diseases of Immunity

Types of Shock

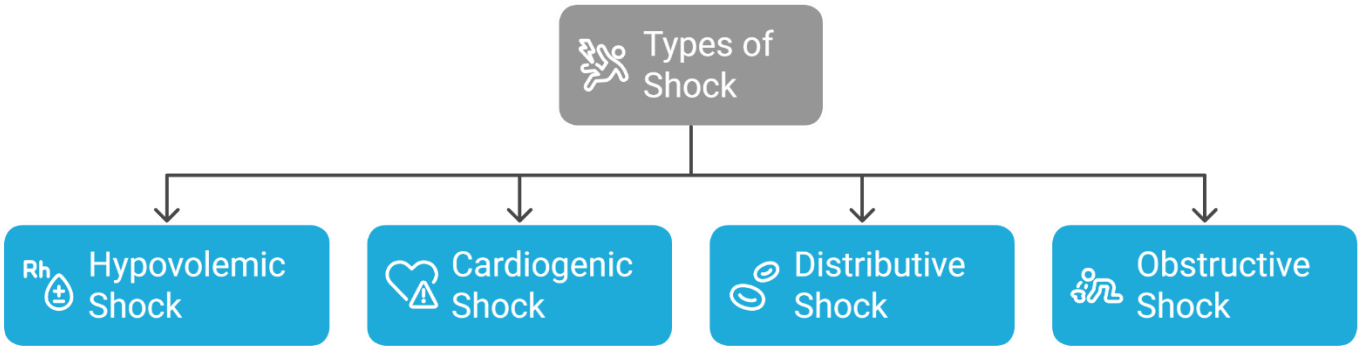
What is shock?

- Shock is a life-threatening medical condition where **tissue perfusion is inadequate**, leading to **cellular hypoxia** and **organ dysfunction**.
- It represents a state of circulatory failure that impairs oxygen delivery and nutrient supply.



Types of Shock

Shock is classified based on **underlying mechanisms**, each with distinct causes and pathophysiological features:



A. Hypovolemic Shock

- **Definition**: Caused by **reduced intravascular volume**, leading to decreased preload and cardiac output.
- **Mechanism**: Decreased preload → reduced cardiac output → inadequate tissue perfusion.
- **Causes**:
  - Hemorrhage (trauma, GI bleeding, ruptured aneurysm)

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- Dehydration (vomiting, diarrhea, burns)
- Third spacing (ascites, pancreatitis)

➢ **Key Features**:

- Low BP, tachycardia, cold clammy skin
- Narrow pulse pressure
- Low central venous pressure (CVP)

B. Cardiogenic Shock

➢ **Definition**: Failure of the heart to pump effectively despite adequate volume.

➢ **Mechanism**: Impaired contractility → decreased cardiac output despite normal or increased blood volume.

➢ **Causes**:

- Myocardial infarction (most common)
- Arrhythmias
- Cardiomyopathy
- Valve diseases

➢ **Key Features**:

- Hypotension, pulmonary edema (crackles), raised JVP
- Low cardiac output
- High filling pressures (PCWP, CVP)

C. Distributive Shock

➢ **Definition**: Abnormal distribution of blood due to vasodilation.

➢ **Mechanism**: Blood vessels dilate, leading to relative hypovolemia even with normal/increased blood volume.

➢ **Subtypes**:

- **Septic shock**: due to infection (most common distributive type)
- **Anaphylactic shock**: IgE-mediated hypersensitivity
- **Neurogenic shock**: spinal cord injury causing sympathetic loss

➢ **Key Features**:

- **Warm extremities** (early septic shock)
- Low systemic vascular resistance (SVR)
- High cardiac output (initially in sepsis)

D. Obstructive Shock

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- **Definition**: Physical obstruction to circulation.
- **Mechanism**: Reduced cardiac output due to mechanical blockage.
- **Causes**:
  - Pulmonary embolism
  - Cardiac tamponade
  - Tension pneumothorax
  - Constrictive pericarditis
- **Key Features**:
  - Hypotension with signs of obstruction
  - Raised JVP (tamponade/PE)
  - Pulsus paradoxus (in tamponade)

Mechanisms of Shock

- Shock leads to tissue hypoxia through multiple interconnected mechanisms that can escalate to **Multiple Organ Dysfunction Syndrome (MODS)** if not corrected.
- These mechanisms can be grouped into pathological processes and compensatory responses:

Pathophysiological Mechanisms

1. Inadequate Perfusion

- ↓ Oxygen and nutrient delivery to tissues impairs cellular metabolism.

2. Anaerobic Metabolism

- Oxygen deficiency forces cells into anaerobic glycolysis → **lactic acid accumulation** → **metabolic acidosis**.

3. Cell Membrane Dysfunction

- Failure of ATP-dependent ion pumps
- Sodium and water influx → **cellular edema**
- Calcium influx → **cell death**

4. Systemic Inflammatory Response *(particularly prominent in septic shock)*

- Cytokine release
- Endothelial dysfunction
- Capillary leak and vasodilation
- Coagulopathy (may contribute to DIC)

Compensatory Mechanisms

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- These short-term responses aim to maintain perfusion and blood pressure:

A. Sympathetic Nervous System Activation

➢ → Vasoconstriction, tachycardia, ↑ cardiac output.

B. Renin-Angiotensin-Aldosterone System (RAAS) Activation

➢ → Salt and water retention to expand intravascular volume.

C. Antidiuretic Hormone (ADH) Release

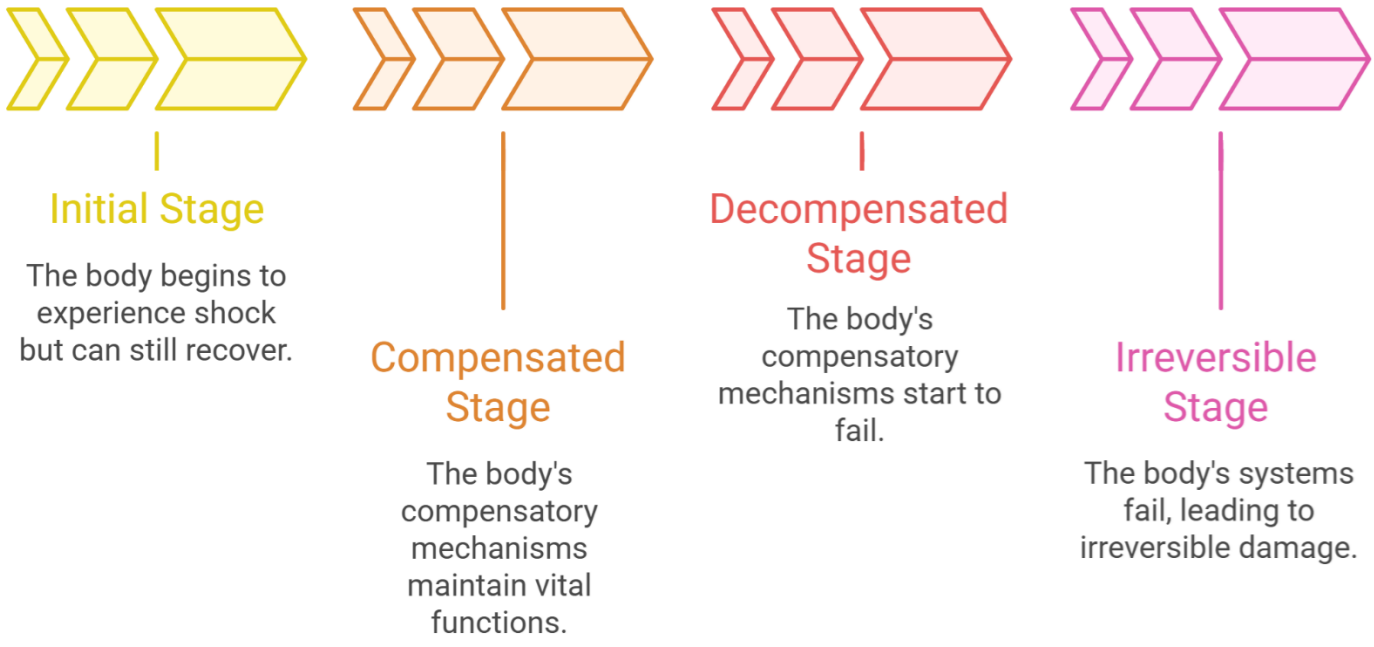
➢ → Promotes water reabsorption in the kidneys → fluid retention.

Progression to MODS (Multiple Organ Dysfunction Syndrome)

- If shock remains uncorrected:
  - Initial compensatory mechanisms fail
  - Hypoperfusion worsens
  - Inflammatory and cellular injury becomes systemic
  - **MODS develops**, involving failure of vital organs such as lungs, kidneys, liver, and heart.

Stages of Shock

Shock progresses through several stages:



A. Initial Stage (Pre-shock or Early Shock)

Physiological Changes:

➢ Reduced tissue perfusion

➢ No visible clinical symptoms

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