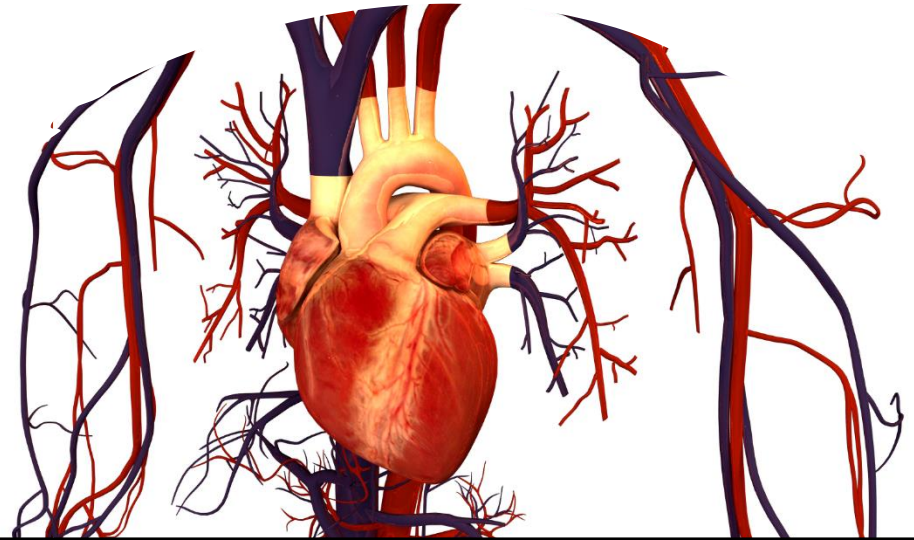
Shock



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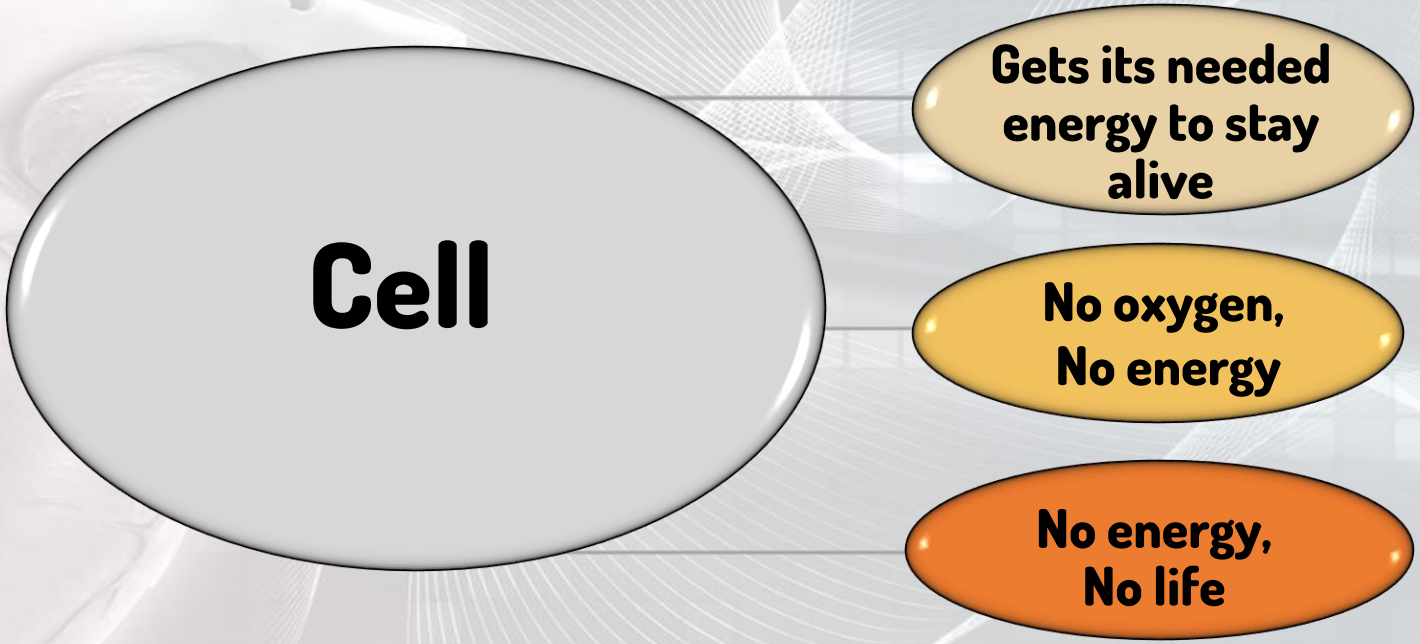
Shock

Lecture Outcomes

- To define shock & to recognize its different stages.
- To identify etiology & pathophysiology of different types of shock.
- To understand compensatory reflex mechanisms of shock.
- To understand complications & causes of irreversible shock.

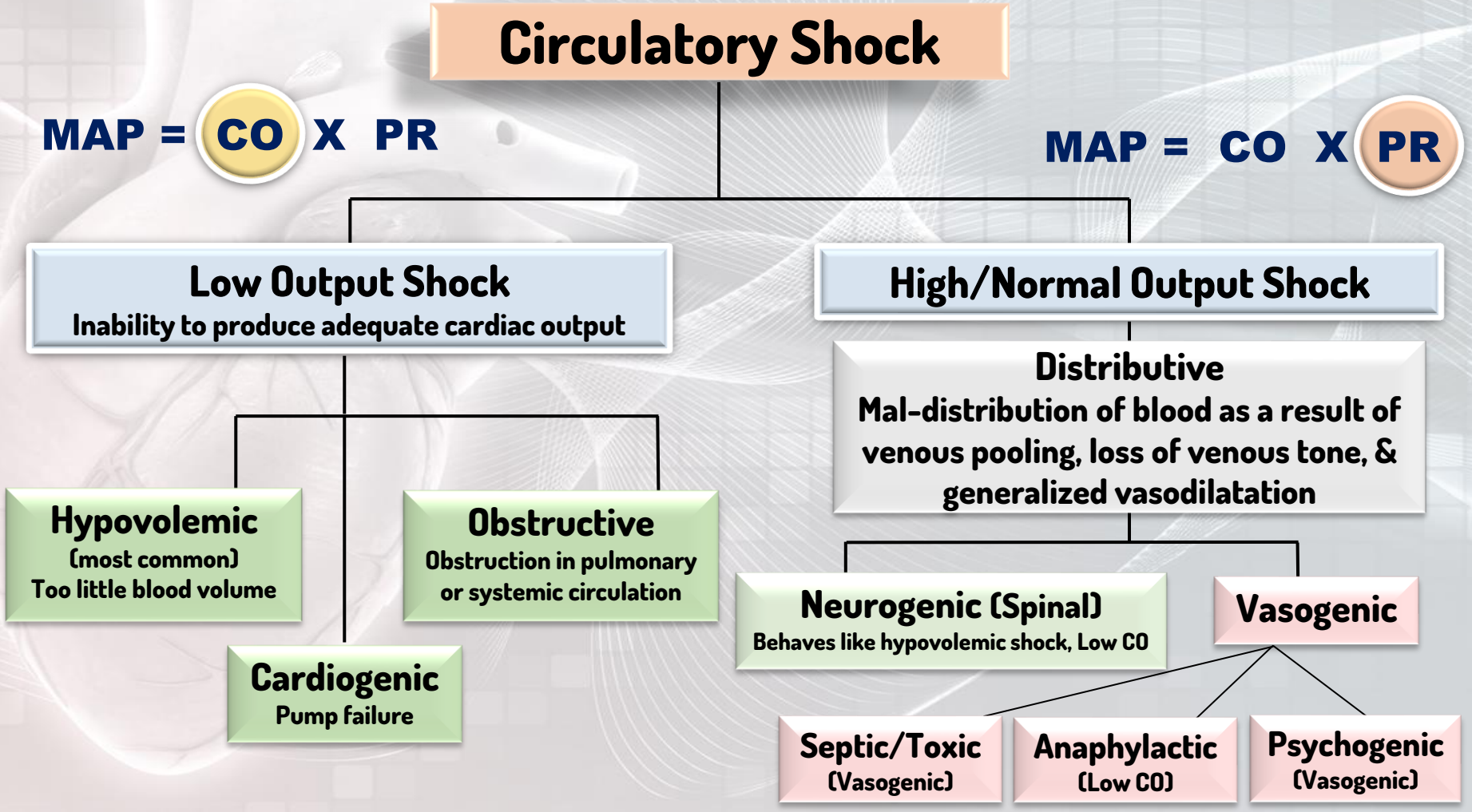
Basic Unit of Life



What is Shock?

- **A state of acute emergency** in which **acute circulatory failure** occur as a result of diminished cardiac output (CO) or reduced effective circulating blood volume, leading to inadequate tissue perfusion severe enough to induce derangements in normal cellular metabolic function.
- **Consequences?**
  - Cellular hypoxia due to failure to deliver oxygen to the tissues, leading to cellular damage.
  - End organ injury/dysfunction (vital organs) relative to their impaired metabolic requirements.
- Shock is a **progressive**, rather than a static condition.
- **If not controlled** & corrected quickly, it may lead to **irreversible shock** & **death**.
- **Note:** Medical & not electric shock.

Types of Shock



MAP=Mean arterial pressure; CO=Cardiac output; PR=Peripheral resistance

MAP = CO X PR

Hypovolemic Shock

Low Cardiac Output

- **Most common type of shock.**
- **A life- threatening condition, due to inadequate blood or plasma volume.**
- **Etiology (Causes):**
  - Excessive/severe/massive **volume loss** of body fluid (blood/plasma).
  - Volume **loss of almost15%** (one-fifth) of body fluid (blood/plasma).
  - **Blood loss/hemorrhage: Commonest**. Any source of bleeding (internal or external).
  - **Fluid/plasma loss:** Vomiting, diarrhea, burn, excess sweating, dehydration, trauma.
- **Physiology:**
  - **Hemodynamic changes:**
    - ✓ ↓ VR (preload): leading to ↓ in EDV, & stroke volume.
    - ✓ ↓ CO: The heart is unable to pump sufficient amounts to the body parts.
    - ✓ End organ hypoperfusion.
- Insufficient perfusion can lead to organ failure.
- Requires immediate emergency & medical attention.

MAP=Mean arterial pressure; CO=Cardiac output; PR=Peripheral resistance; EDV=End-diastolic volume

Classification of Hypovolemic Shock by the amount of blood loss

	Class I	Class II	Class III	Class IV
Blood Loss	< 750	750-1500	1500-2000	> 2000
% Blood Vol.	< 15%	15 – 30%	30 – 40%	> 40%
Pulse	< 100	**> 100**	> 120	> 140
Blood Pressure	Normal	Normal	**Decreased**	**Decreased**
Pulse Pressure	Normal	**Decreased**	**Decreased**	**Decreased**
Resp. Rate	14 – 20	**20 – 30**	**30 – 40**	**> 40**
UOP	> 30	**20 – 30**	**5 – 15**	**negligible**
Mental Status	sl. Anxious	mildly anx	**confused**	**lethargic**
Fluid	crystalloid	crystalloid	**blood**	**blood**

Amount of blood loss= (ml); Vol=Volume; Resp=Respiratory; UOP= Urine output (ml/hour)

Signs and Symptoms: Hypovolemic Shock

- **Sustained Hypotension** (? ≤ 80-85/40 mmHg for 30 min.)
- **Tachycardia**, sensed by Baroreceptors in compensation to the ↓ MAP.
- **Weak, rapid, & thready pulse** (? 140/min).
- **Tachypnea** (rapid respiration), sensed by Chemoreceptors in compensation to hypoxia.
- **Restlessness**, due to hypo-perfusion.
- **Cold, pale clammy skin**, due to hypo-perfusion.
- **Intense thirst**.
- **Oliguria** (low urine output)/ Anuria (no urine output).
- **Blood test:** Lactic acidosis & increase in anion gap.
- **Treatment:** fluid replacement or blood transfusion & treat the underlying cause.

MAP=Mean arterial pressure; min=Minute(s)

MAP = CO X PR

Cardiogenic Shock

Low Cardiac Output

- **Pump Failure:** Cardiac muscle is unable to pump adequate blood flow to the vital organs & body parts in presence of a normal blood volume.
- Is associated with **loss of > 40%** of Left ventricular myocardial function.
- **Etiology (Causes): Deterioration or failure of cardiac function.**
  - **Myocardial:** Either Acute **intrinsic** myocardial damage: Myocardial Infarction (Most common.), Myocarditis, Cardiomyopathy. Or **extrinsic** compression.
  - **Mechanical:** Acute valvular dysfunction, e.g., rupture of papillary muscle post MI.
  - **Arrhythmogenic:** Sustained Arrhythmias, e.g., heart block, ventricular tachycardia.
  - **? Obstructive:** Pulmonary embolism, Cardiac tamponade.
- **Physiology:**
  - **Hemodynamic changes:**
    - ✓ Severe ↓ CO with ↓ stroke volume (SV).
    - ✓ ↑ Left ventricular end diastolic filling pressure LVEDP (right/left/or Both).
    - ✓ ↓ Coronary perfusion: leading to ischemia & further myocardial dysfunction.
    - ✓ Persistent hypotension (Systolic pressure < 90 mmHg / MAP 30 mmHg below baseline).
    - ✓ End organ hypoperfusion.
- **Mortality rate is high, 60-90%.**

MAP=Mean arterial pressure; CO=Cardiac output; PR=Peripheral resistance; LV=Left ventricle; MI=Myocardial infarction

Signs and Symptoms: Cardiogenic Shock

- **Similar signs & symptoms to that of hypovolemic shock.**
- **Laboratory findings:** Increase troponin I & T.
- **Congestion of lungs & viscera:** (Chest X-Ray -CXR)
  - Interstitial pulmonary edema.
  - Alveolar edema.
  - Cardiomegaly.
- **Prognosis:** 70% mortality.

MAP = CO X PR

Obstructive Shock

Low Cardiac Output

- **Etiology (Causes):**
  - Causative factors may be located within the pulmonary or systemic circulation, or associated with the heart itself, or caused by trauma surgery.
  - Extra-cardiac obstructive shock results from an obstruction to the flow in the cardiovascular circuit.
- **Examples:**
  - **Obstruction of venous return:** e.g., Vena Cava Syndrome (usually neoplasms).
  - **Compression of the heart:** e.g., hemorrhagic pericarditis → cardiac tamponade.
  - **Obstruction of the outflow of the heart:**
    - Aortic coarctation or dissection.
    - Pulmonary or systemic hypertension.
    - Massive pulmonary embolism.
    - Tension pneumothorax.
    - Congenital or acquired outflow obstructions.
- **Physiology: Hemodynamic changes:**
  - ↓ CO despite normal intravascular volume & myocardial function.
  - ↓ Stroke volume (SV).
  - End organ hypoperfusion.

MAP=Mean arterial pressure; CO=Cardiac output; PR=Peripheral resistance

MAP = CO X PR

Distributive Shock

High/Normal Cardiac Output

- Distributive shock is also known as **vasodilatory shock**.
- There will be an **inappropriate distribution of blood** flow which will lead to a decrease in blood flow to the vital organs resulting in their damage.
- **Types:** either,
  - **Neurogenic** (Spinal)
  - **Vasogenic:** Septic, or non-septic (anaphylactic or psychogenic or adrenal insufficiency)
- **Etiology (Causes):**
  - It occurs as a result of systemic vasodilatation which is caused by loss of vascular resistance (tone).
  - It can be also caused by leakage of fluid from capillaries into the surrounding tissues (capillary leak syndrome).
  - It can be caused by sepsis, allergic reactions, adrenal insufficiency, trauma, drug overdose & toxicity.
- **Physiology: Hemodynamic changes:**
  - Cardiac output (CO) is mostly normal or elevated.
  - ↑ Venous capacitance due to vasodilatation.
  - ↓ Peripheral resistance (PR) & venous return (VR).

Distributive Shock

I: Neurogenic Shock

- **Neurogenic/Spinal Shock (venous pooling):**
  - Can be caused by trauma/injury involving the brain/spinal cord (devastating cervical or head injury), or by anesthetic accident.
  - **Loss of disruption of autonomic nervous system innervation** below the level of injury.
  - **Sympathetic nervous system is damaged** resulting in a decreased adrenergic input to the blood vessels & heart, causing **loss or drop in vasomotor (vascular) tone**.
  - **Consequences?**
    - ✓ **Generalized peripheral vasodilation**.
    - ✓ **Orthostatic (postural) hypotension.**
    - ✓ **CO is severely reduced** as blood is pooled in the peripheral veins (Capacity of blood ↑ & venous return ↓).
    - ✓ **bradycardia, & low body temperature.**
    - ✓ **Behaves like hypovolemic shock.**
    - ✓ Blood volume remains normal.

MAP=Mean arterial pressure; CO=Cardiac output; PR=Peripheral resistance

MAP = CO X PR

Distributive Shock

II: Vasogenic Shock

Septic/Toxic/Endotoxic Shock

- **Most common in emergency**.
- Dysregulation of the immune response to infection that leads to **activation of systemic cytokine cascades release.**
- **Results in:**

✓ Peripheral vasodilatation, pooling of blood & fluid leak from capillaries.
✓ Endothelial activation/injury.
✓ Leukocyte-induced damage
✓ Bacterial endotoxin triggers peripheral vasodilatation & endothelial injury.
✓ Inflammatory cytokines may also cause some cardiac dysfunction.
✓ Disseminated intravascular coagulation (coagulopathy).
✓ Hyper-dynamic state.

Anaphylactic Shock

- **Most common in emergency**.
- Caused by exposure to an antigen resulting in a massive & generalized allergic reaction.
- **Systemic release of inflammatory mediators from mast cells & basophils.**
- Histamine triggers **systemic& generalized peripheral vasodilation** & ↑ capillary permeability leakage.
- Can lead to **low CO** distributive shock.
- Clinical example: IgE- Mediated hypersensitivity reactions.

Psychogenic Shock

- Simple fainting (syncope) as a result of stress, pain, or fright.
- **Dilatation of blood vessels.**
- **Results in:**

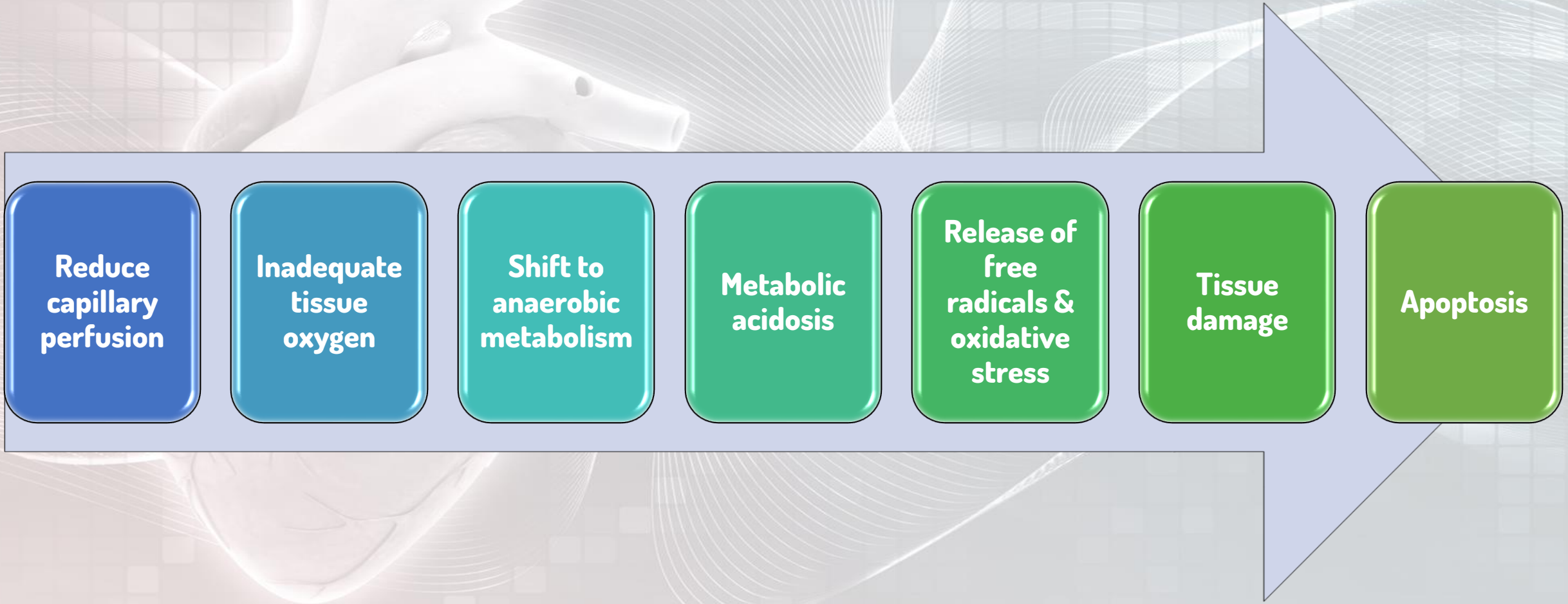
✓ Blood pressure falls.
✓ ↑ HR (pulse).
✓ Brain becomes hypo-perfused.
✓ Loss of consciousness.

MAP=Mean arterial pressure; CO=Cardiac output; PR=Peripheral resistance; HR=Heart rate

Signs and Symptoms: Septic Shock

- **Patient flushed & warm:** due to his/her hyper-dynamic state.

Pathophysiology of Shock



Metabolic Changes & Cellular Response to Shock

1. Reduce capillary perfusion:

→ Spasm of pre/post capillary sphincters.

→ Hypoxic tissue damage (**oxidative stress**.)

→ Anaerobic metabolism (anaerobic glycolysis.)

→ Lactic acid production.

→ Metabolic acidosis **(intracellular acidosis)**.

→ **Failure of Na^+/K^+ pump** (↑ $[Na^+]$ & $[Ca^{2+}]$).

→ **Breakdown of Lysosomes, nuclear membranes & mitochondria**.

2. After 3 - 5 hours of shock:

→ Precapillary sphincters dilate, venules are still constricted.

→ Blood stagnation in capillaries.

→ Hypoxia continue & fluid leaves to extra vascular compartment.

→ Further reduction in circulating blood volume.

3. Granulocytes accumulation:

→ Granulocytes will accumulate at the injured vessels.

→ **Free radicals release.**

→ Further tissue damage.

Na+=Sodium ion; K+=Potassium ion; Ca+2=Calcium ion

Metabolic Changes & Organ Response to Shock

1. **Damage in GIT mucosa** → Allows bacteria into circulation.

2. **Cerebral ischemia** → Depression of VMC → vasodilation + ↓ HR
   further decrease in blood pressure.

3. **Myocardial ischemia** → Myocardial damage + depressed contractility
   more shock & acidosis.

4. **Respiratory distress syndrome** occurs due to damage of capillary endothelial cells & alveolar epithelial cells, with release of cytokines.

5. **Multiple organ failure & death.**

GIT=Gastro-Intestinal Tract; VMC=Vasomotor center (Sympathetic); HR=Heart rate.

Compensatory Mechanisms to Shock

1. **↑ HR, ↑ Myocardial contractility, ↑ CO, ↑ Preload filling pressure, & Vasoconstriction in order to ↑ BP:**
   - **By stimulation of Sympathetic Nervous System through,**
     - **Baroreceptors reflex** mechanism, which is stimulated by low blood pressure.
     - **Chemoreceptors reflex** mechanism, which is stimulated by hypoxia & acidosis.
2. **↑ Blood volume: through,**
   - **Activation of Renin-Angiotensin Aldosterone System:**
     - Angiotensin II & III: Are powerful vasoconstrictors.
     - Aldosterone: will lead to Na+ & water retention.
   - **Stimulation of ADH (vasopressin):**
     - Water retention, vasoconstriction, with thirst & drinking stimulation.
3. **Synthesis of Plasma Proteins: (after 3-4 days)**
4. **Fluid- shift mechanism.**

HR=Heart rate; BP=Blood pressure; Na+=Sodium ion; CO=Cardiac output

In Summary: Reflex Compensation to Shock

Physiological reflex compensatory reaction in response to ↓ BP will result in:

- **↑ HR & myocardial contractility → ↑ CO .**
- **Vasoconstriction → ↑ Preload & filling pressure.**
- **↑ Blood volume.**
- **↑ Vascular compliance.**
- **Restoration of tissue perfusion.**

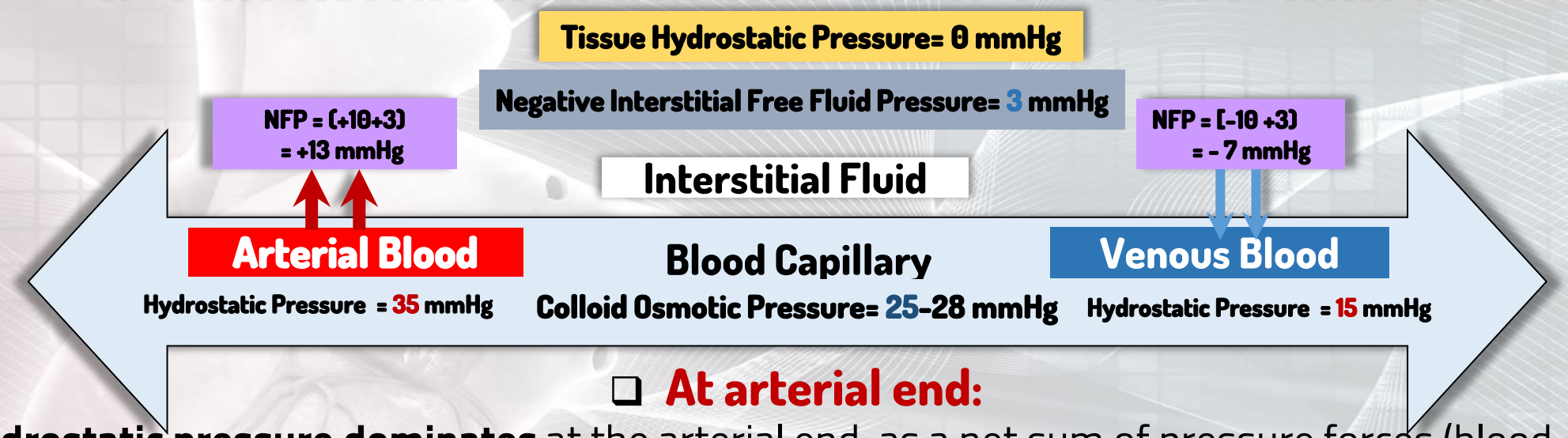
BP=Blood pressure; HR=Heart rate; CO=Cardiac output

Fluid- Shift Mechanism in Shock

- **In most cases of shock, the hydrostatic pressure decreases while the oncotic pressure remains constant, as a result:**
  - The fluid transfer from the capillary to the extracellular space decreases.
  - The fluid return from the extracellular space to the capillary increases.
- **In compensating to shock situation, this will help to increase the blood volume in order to restore the BP.**

BP=Blood pressure

Normal Forces at The Arterial & Venous Ends of The Capillary



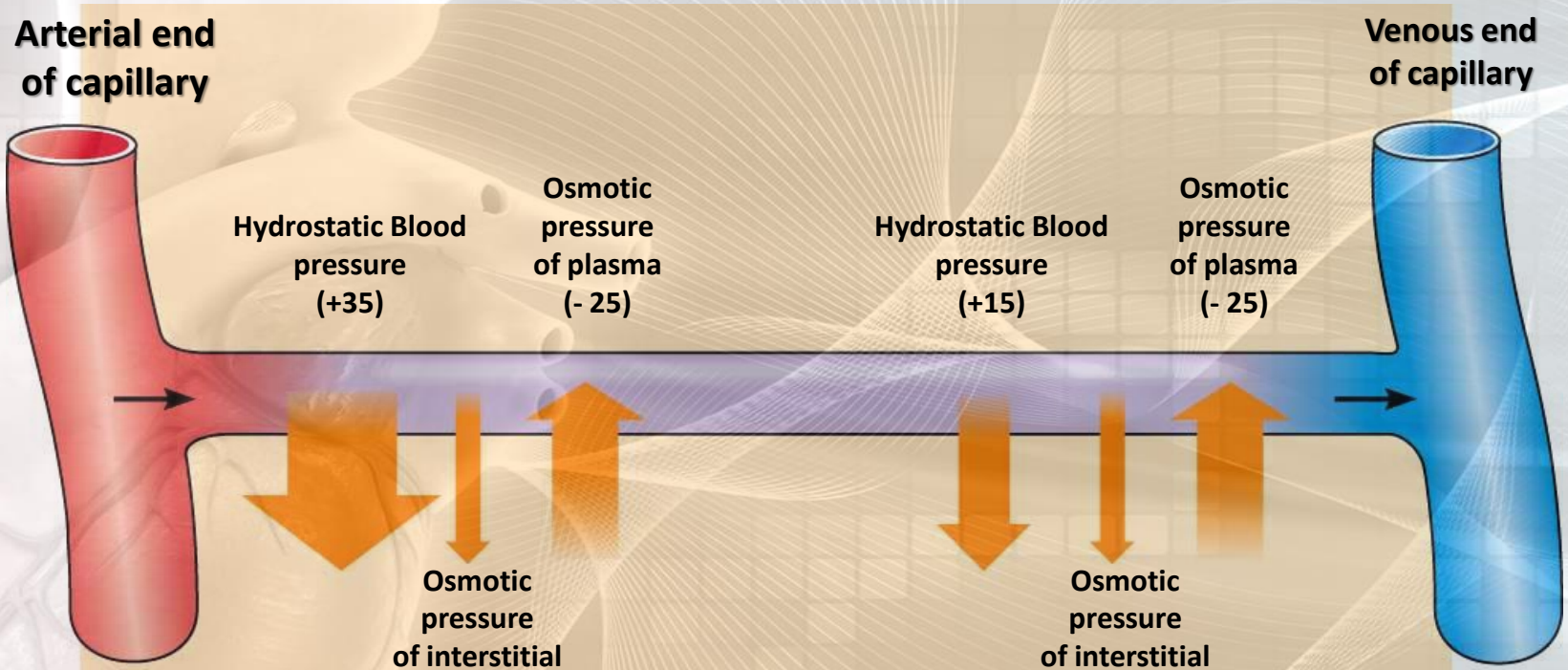
❑ At arterial end:

- **Hydrostatic pressure dominates** at the arterial end, as a net sum of pressure forces (blood hydrostatic pressure + Interstitial fluid (IF) osmotic pressure) flow fluid out of the circulation.
- **Water moves out** of the capillary with a net filtration pressure (**NFP**) of **+13 mmHg**.
- 13 mmHg NFP causes an average of 1/200 of plasma in flowing blood to filter **out** of arterial end of the capillary **into the intestinal space**.

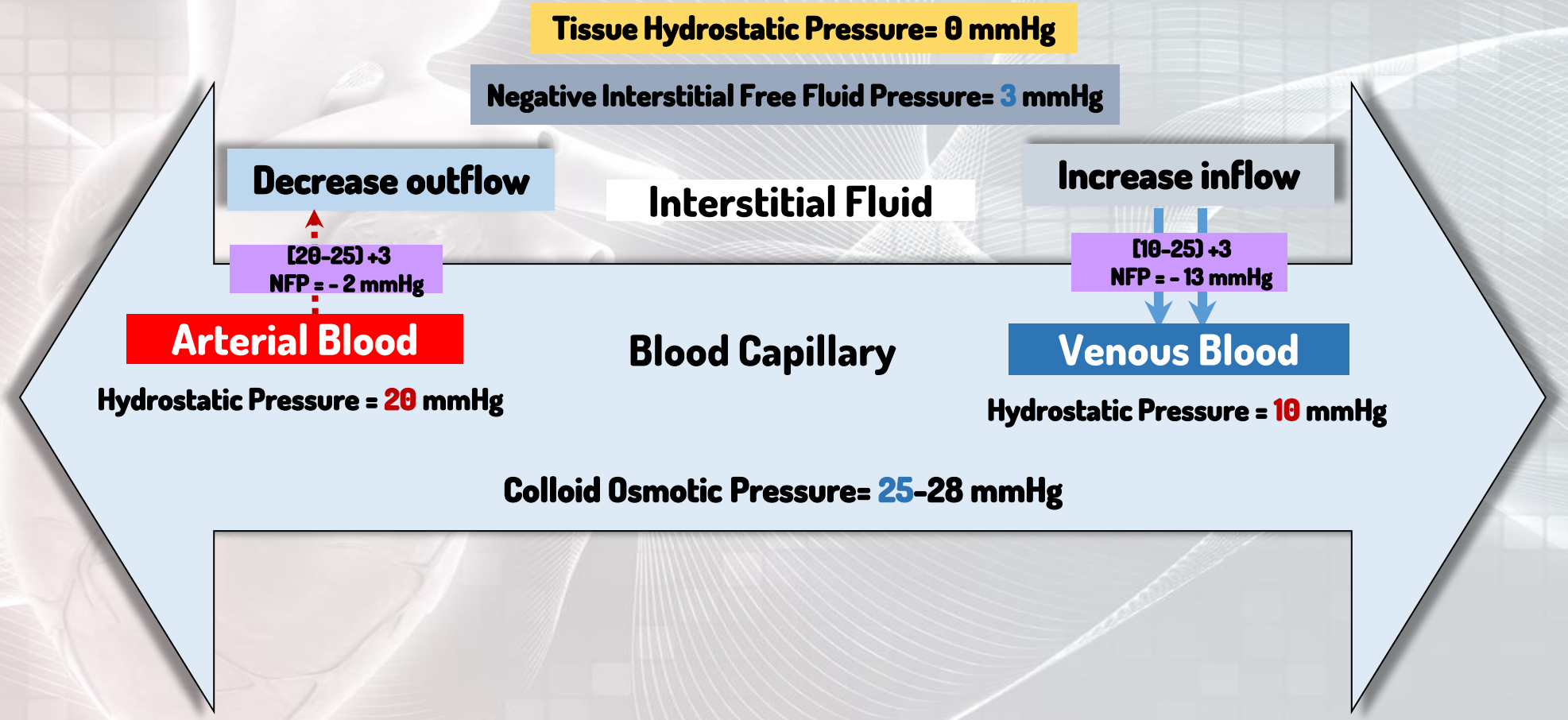
❑ At venous end:

- **Oncotic pressure dominates** at the venous end, as a net sum of pressure forces (blood osmotic pressure + Interstitial fluid (IF) hydrostatic pressure) flow fluid into the bloodstream.
- **Water moves into** the capillary with a **NFP** of **-7 mmHg**.

Normal Forces at The Arterial & Venous Ends of The Capillary



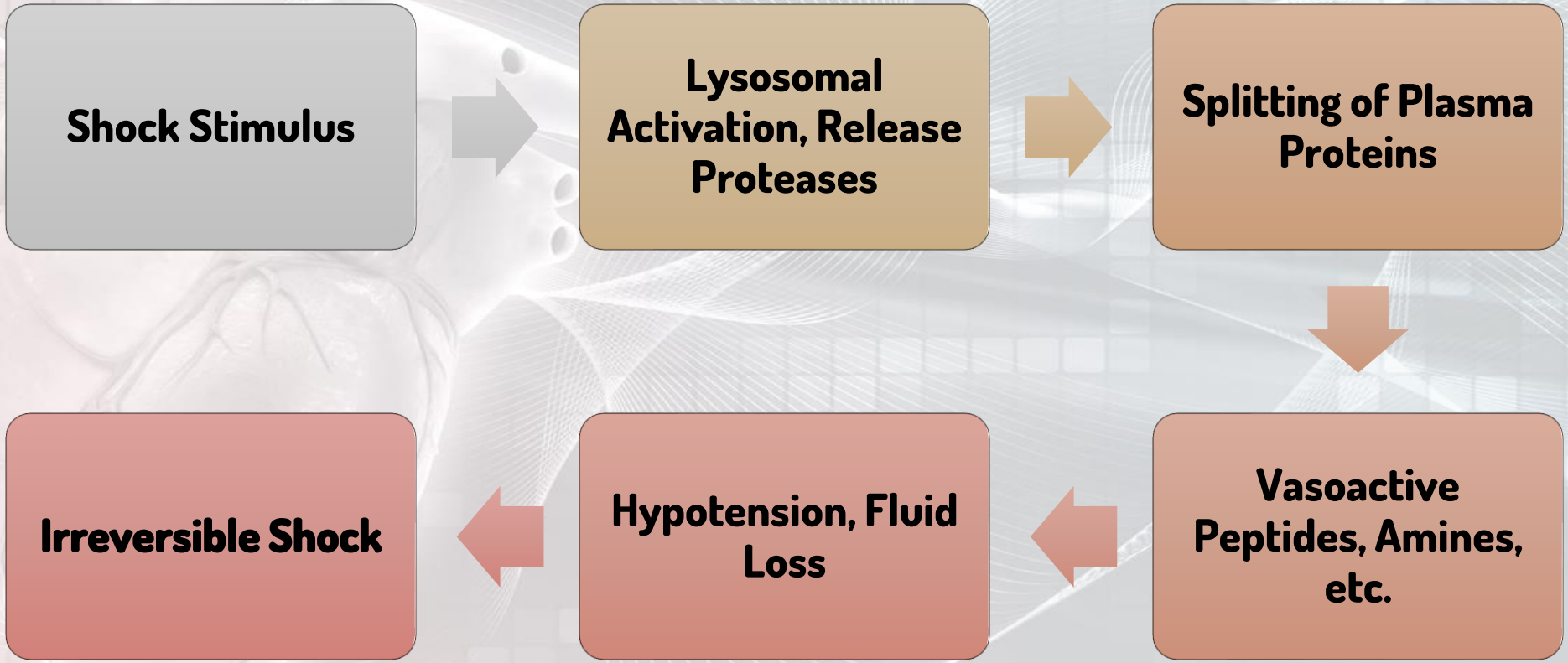
Fluid- Shift Mechanism In Shock



Stages of Shock

- **Non-progressive (Reversible shock): (Compensated)**
  - Reflex compensatory mechanisms are activated (neuro-hormonal activation).
  - Changes can be reversed by compensatory mechanisms or by treatment.
  - Defense mechanisms are successful in maintaining perfusion.
- **Progressive:**
  - Defense mechanisms begin to fall.
  - Tissue hypoperfusion with worsening of circulatory & metabolic imbalance including lactic acidosis.
  - Multi-organ failure.
- **Irreversible shock:**
  - Complete failure of compensatory mechanisms.
  - Severe cell & tissue injury.
  - Can lead to death.

Possible Mechanisms Leading to Development of Irreversible Shock




Thank You