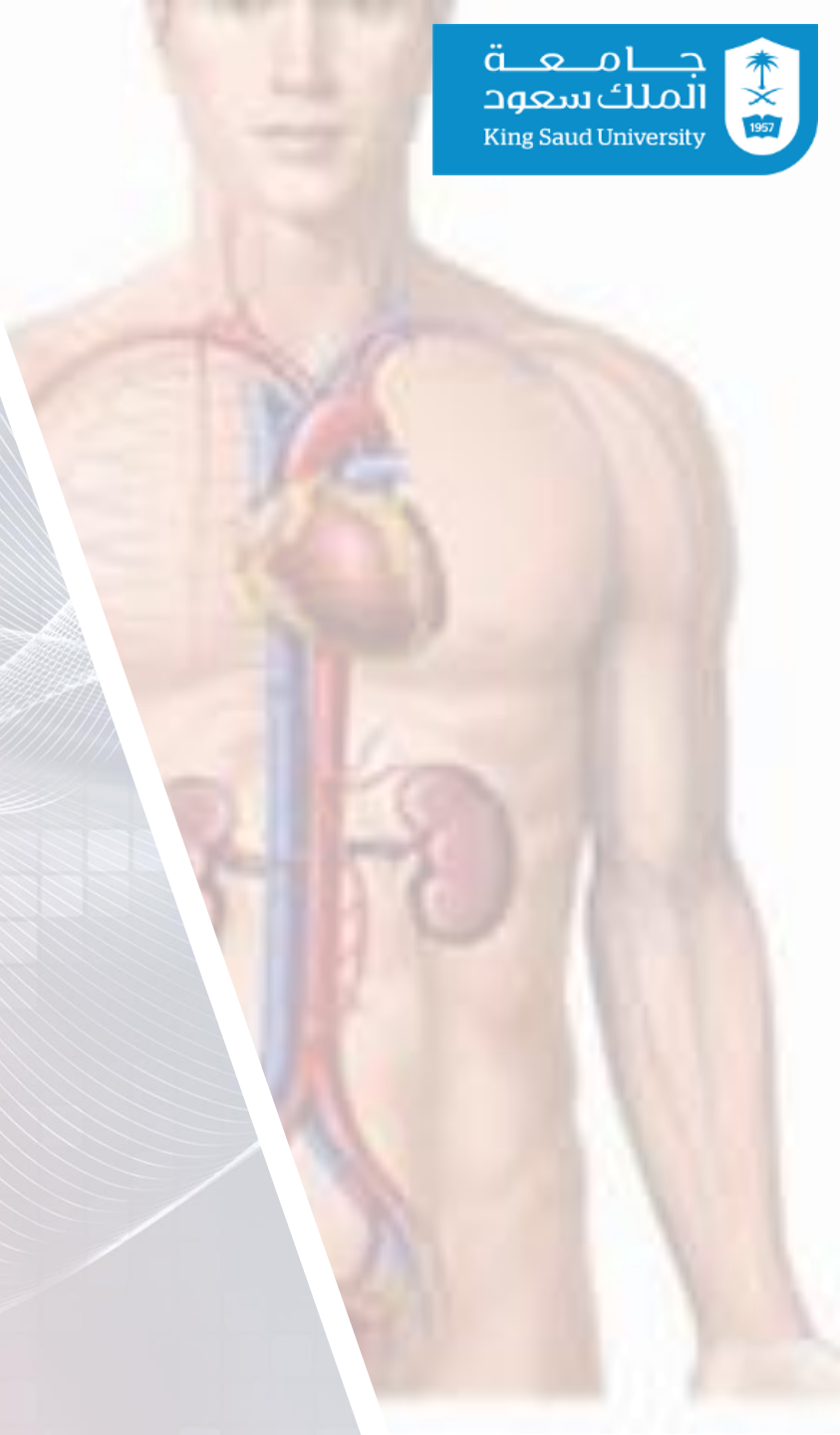
Cardiovascular Physiology

Shock



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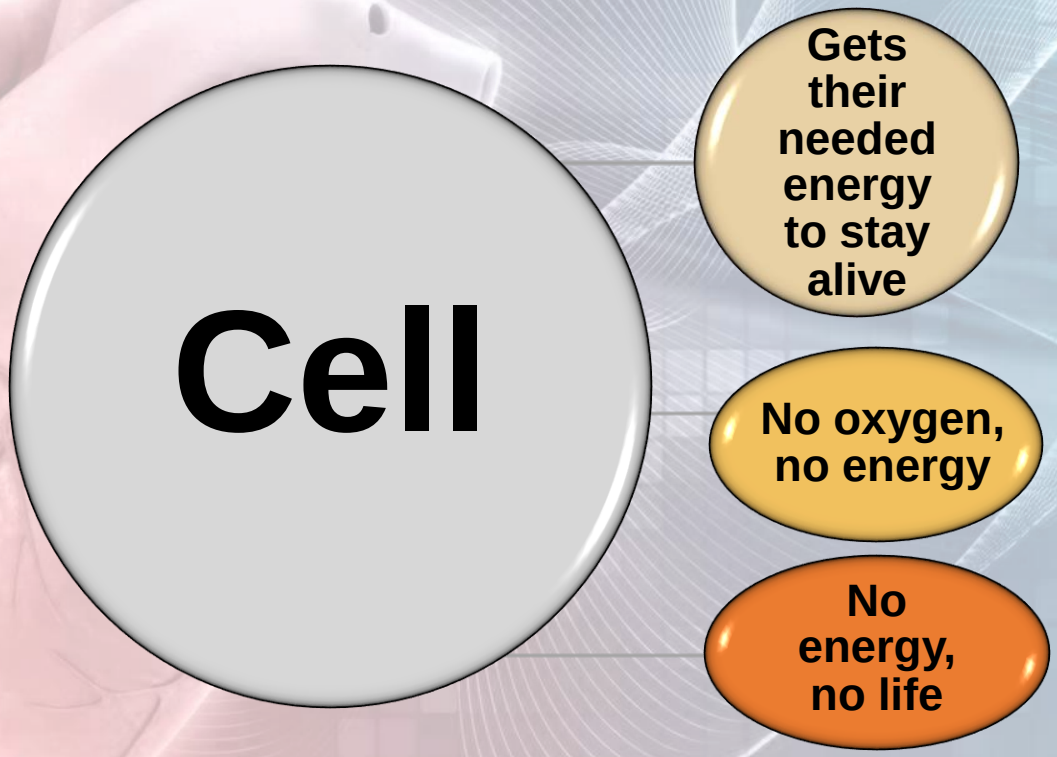
Shock

Lecture Outlines

- To define Shock.
- To describe different types of Shock.
- To understand the pathophysiology of Shock.
- To define different stages of Shock.
- To understand different compensatory mechanisms in response to Shock.
- To define different mechanisms responsible for Irreversible Shock.

Basic Unit of Life



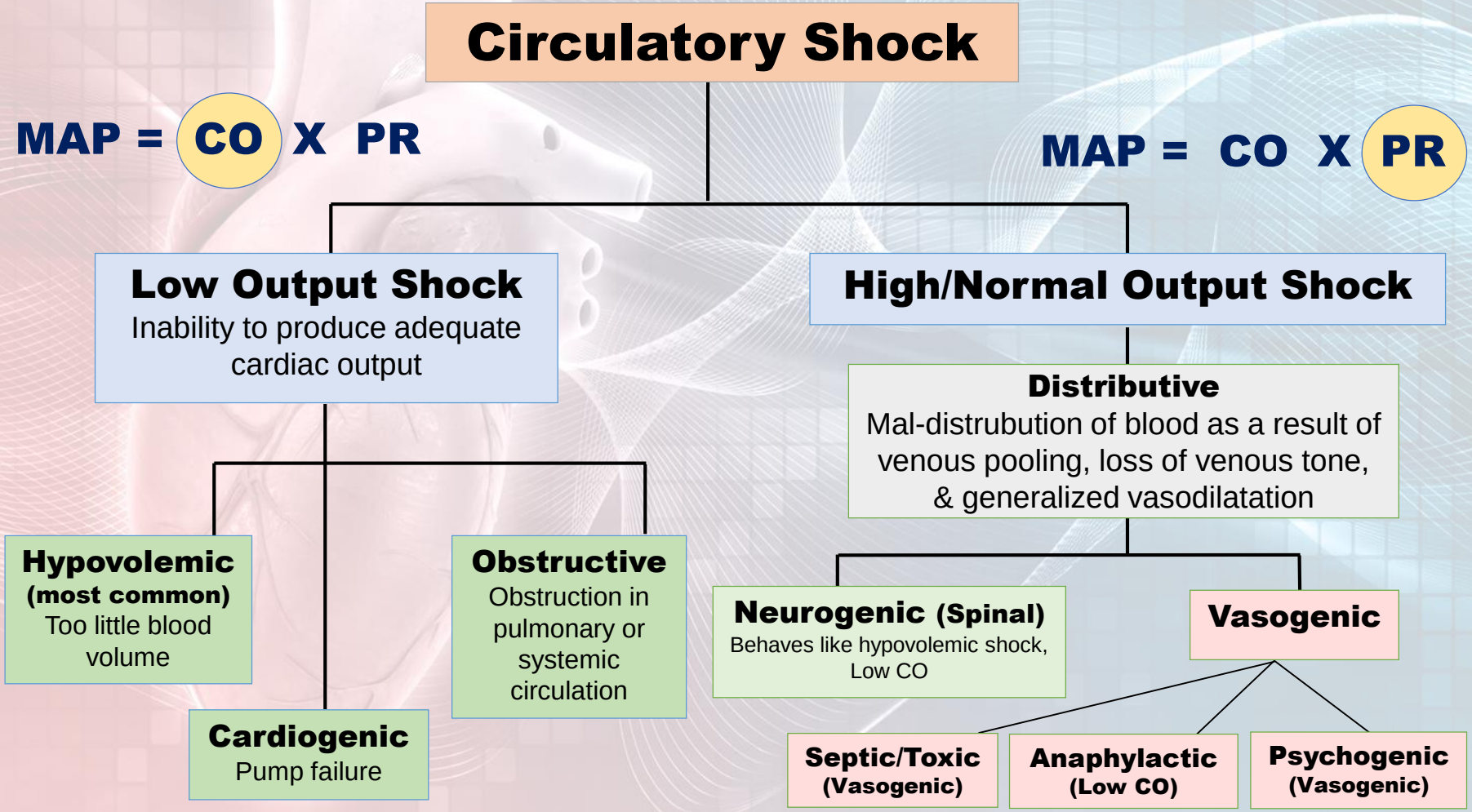
What is Shock?

- An acute circulatory failure leading to inadequate tissue perfusion & end organ injury (Medical & not electrical shock).
- Defined as **Circulatory Shock**.
- The main feature of circulatory shock is loss of fluid from circulating blood volume, so that the circulatory system is unable to provide adequate circulation & tissue perfusion.
- Results in failure to deliver oxygen to the tissues & vital organs relative to their metabolic requirements, leading to **organ dysfunction** & **cellular damage**.

What is Shock?

- Shock is an acute emergency situation.
- Shock is a progressive, rather than a static condition.
- If not controlled & corrected quickly, it may lead to irreversible shock & death.

Types of Circulatory 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.
- **Causes:**
  - Caused by severe loss of body fluid (blood/plasma).
  - Loss of more than or equal to 15% (one-fifth) volume 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.
- **Hemodynamic changes:**
  - The heart is unable to pump sufficient amounts to the body parts.
  - **Low cardiac output (CO).**
  - Reduced venous return (preload), leading to reduction in EDV, & in stroke volume.
  - 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

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 LV myocardial function.
- **Causes: Deterioration of cardiac function**
  - **Myocardial:** Acute Myocardial Infarction (Most common.), Myocarditis, Cardiomyopathy.
  - **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.
- **Hemodynamic changes:**
  - **Low cardiac output (CO) with reduced stroke volume (SV).**
  - Elevated Left ventricular end diastolic filling pressure LVEDP (right/left/or Both).
  - Decreased coronary perfusion, leading to ischemia & further myocardial dysfunction.
  - Persistent hypotension (systolic pressure < 80 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

MAP = CO X PR

Obstructive Shock

Low Cardiac Output

- **Cardiac output (CO) is reduced** despite normal intravascular volume & myocardial function.
- Decrease stroke volume.
- End organ hypoperfusion.
- **Causes:**
  - Causative factors may be located within the pulmonary or systemic circulation or associated with the heart itself or caused by trauma suegery.
  - Extracardiac obstructive shock results from an obstruction to 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.

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.
- **Cardiac output (CO) is mostly normal or elevated.**
- **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.
- **Types:** either,
  - **Neurogenic** (Spinal)
  - **Vasogenic:** Septic, or non-septic (anaphylactic or phsycogenic)

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

Distributive Shock

I: Neurogenic Shock

- **Neurogenic/ Spinal Shock (venous pooling):**
  - Can be caused by trauma involving the cervical spinal cord.
  - Sympathetic nervous system is damaged resulting in a decreased adrenergic input to the blood vessels & heart, causing loss or drop in vasomotor (vascular) tone.
  - Generalized peripheral vasodilation & hypotension.
  - Blood volume remains normal.
  - **Cardiac output (CO) is severely reduced** as blood is pooled in the peripheral veins.. (Capacity of blood ↑, & venous return ↓.)
  - **Behaves like hypovolemic shock.**

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 systemic cytokine release & resultant vasodilatation & fluid leak from capillaries.
- Bacterial endotoxin triggers peripheral vasodilatation & endothelial injury.
- Inflammatory cytokines may also cause some cardiac dysfunction.
- Hyper-dynamic state.

Anaphylactic Shock

- Most common in emergency.
- Exposure to an antigen resulting in a massive & generalized allergic reaction.
- IgE- Mediated hypersensitivity.
- Histamine triggers systemic peripheral vasodilation & ↑ capillary permeability leakage.
- Can lead to **low cardiac output (CO)** distributive shock.

Psychogenic Shock

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

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+}]).

→ **Lysosomes, nuclear membranes & mitochondrial breakdown**.

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.

2. Granulocytes accumulation at injured vessels:

→ **Free radicals release.**

→ Further tissue damage.

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

Metabolic Changes & Organ Response to Shock

4. **Damage in GIT mucosa** → allows bacteria into circulation.

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

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

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

8. **Multiple organ failure & death.**

VMC=Vasomotor center (Sympathetic); HR=Heart rate; Ca+2=Calcium ion

Compensatory Mechanisms to Shock

Physiological reaction in response to ↓ BP will lead to the following in order to ↑ BP:

- ↑ 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

Compensatory Mechanisms to Shock

1. Stimulation of Sympathetic Nervous System through:

- **Baroreceptors reflex** mechanism.
- Acidosis stimulates **chemoreceptors reflex mechanism** → sympathetic stimulation.

❑ **Leads to ↑ HR, ↑ Myocardial contractility, Vasoconstricion, & ↑ BP.**

2. Activation of Renin-Angiotensin System:

- Angiotensin II & III: Powerful vasoconstrictors.
- Aldosterone: Na+ retention.

3. Stimulation of ADH (vasopressin):

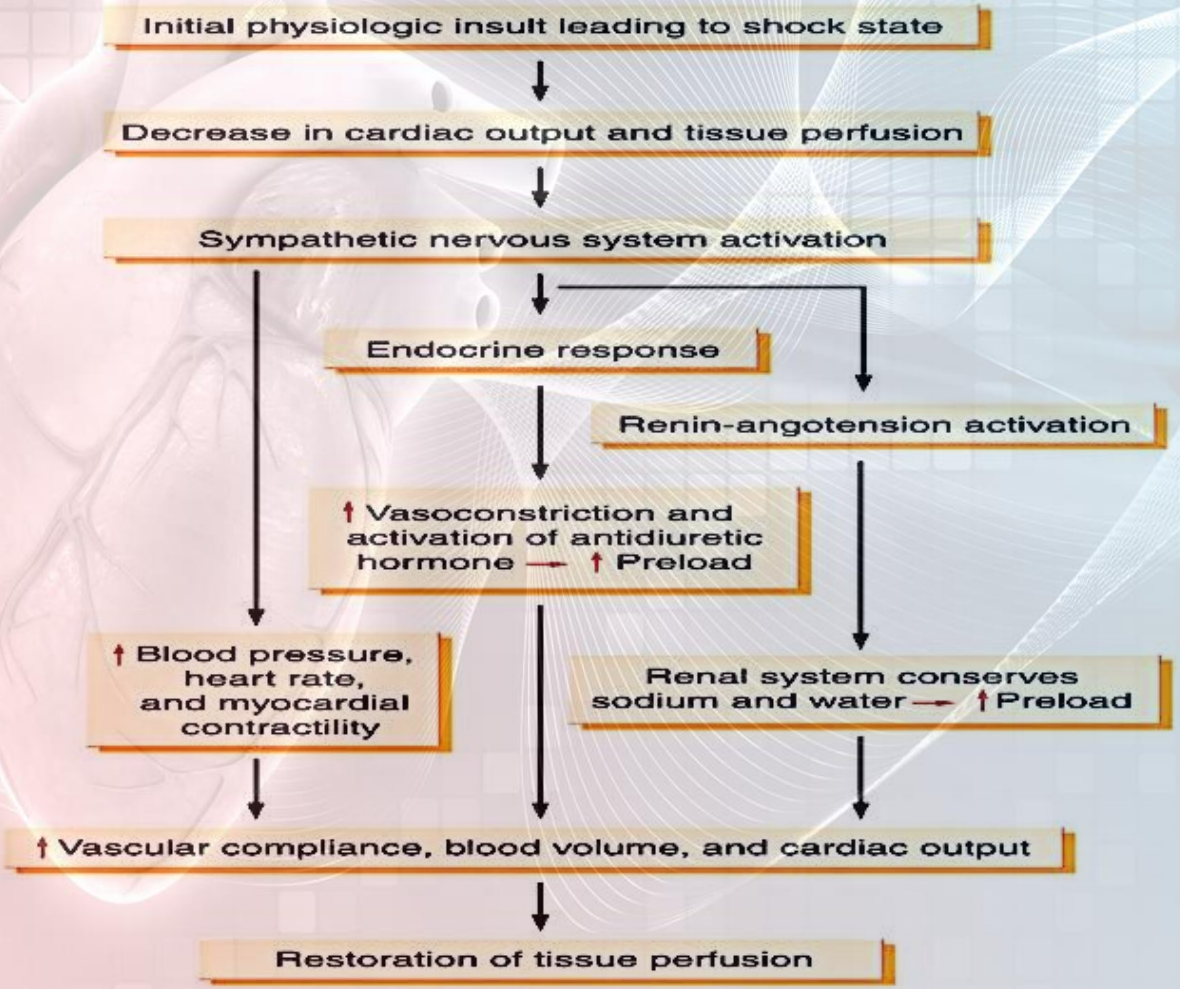
- Water retention, vasoconstriction & thirst stimulation.

4. Synthesis of Plasma Proteins: (3-4 days)

5. Fluid- shift mechanism.

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

Compensatory Mechanisms

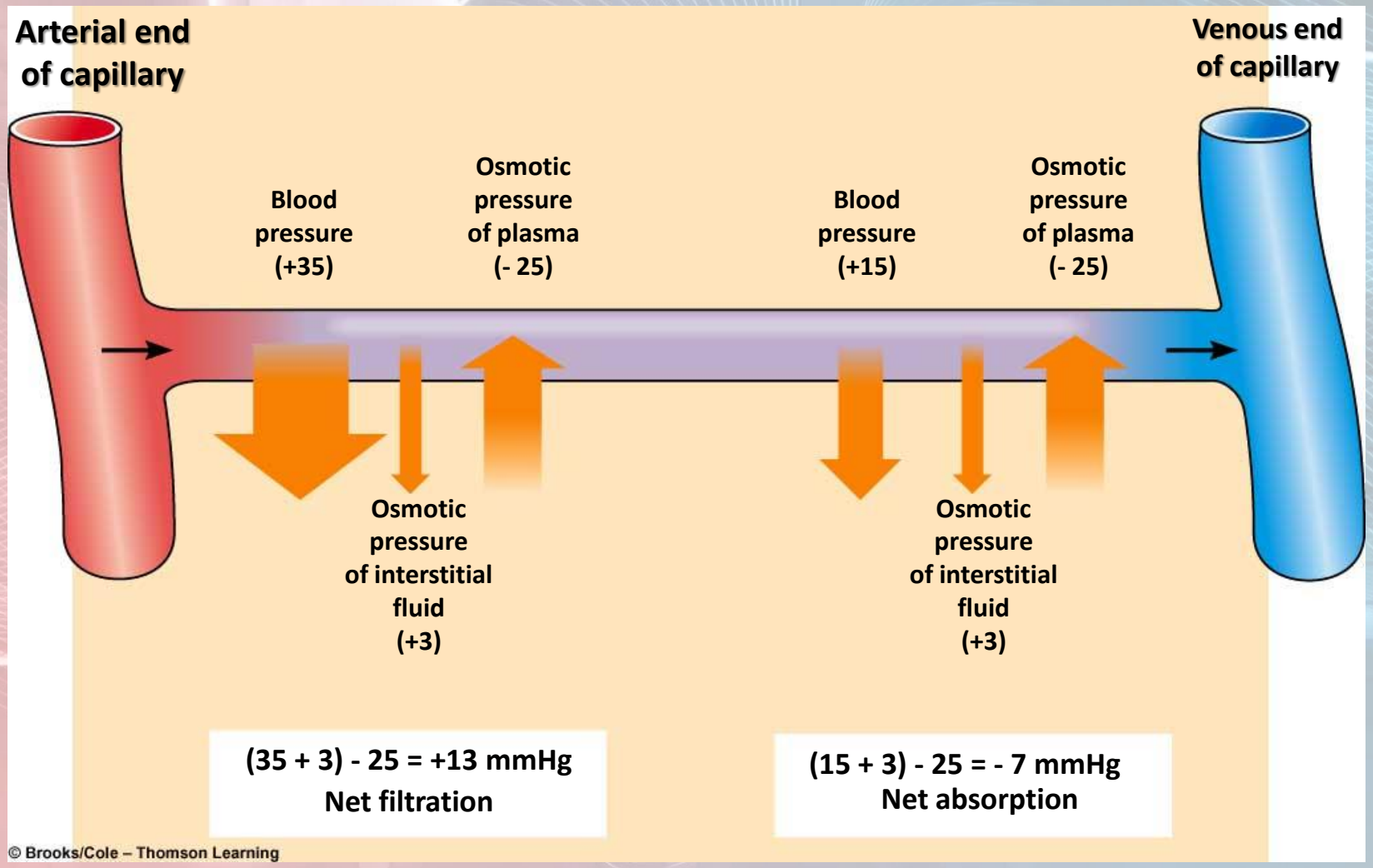


Fluid- Shift Mechanism in Shock

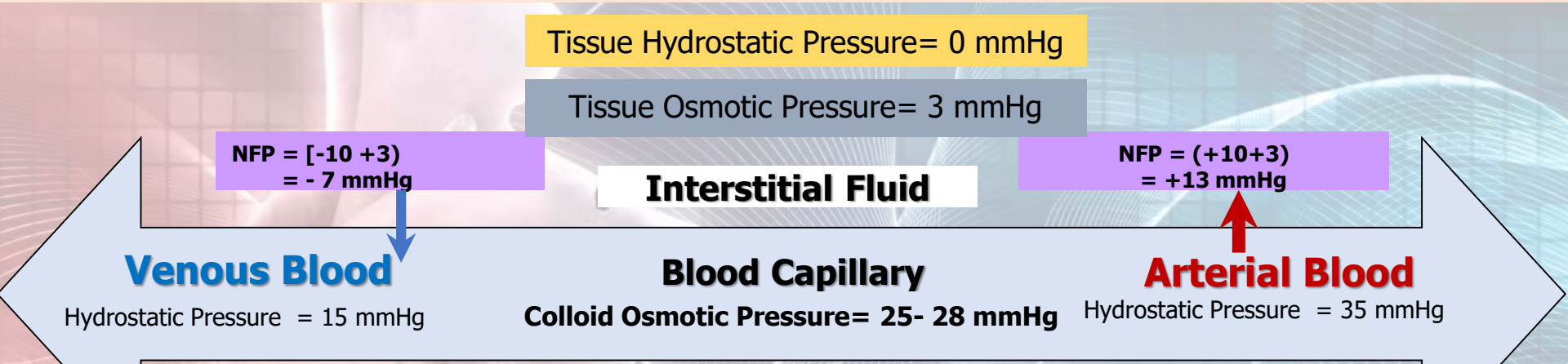
- In shock, the hydrostatic pressure decreases & oncotic pressure is constant, as a result:
  - The fluid exchange from the capillary to the extracellular space decreases.
  - The fluid return from the extracellular space to the capillary increases.
- This will lead to an increase in the blood volume & the BP in order to help in compensating shock situation.

BP=Blood pressure

Normal Forces at The Arterial & Venous Ends of The Capillary



Normal Forces at The Arterial & Venous Ends of The Capillary



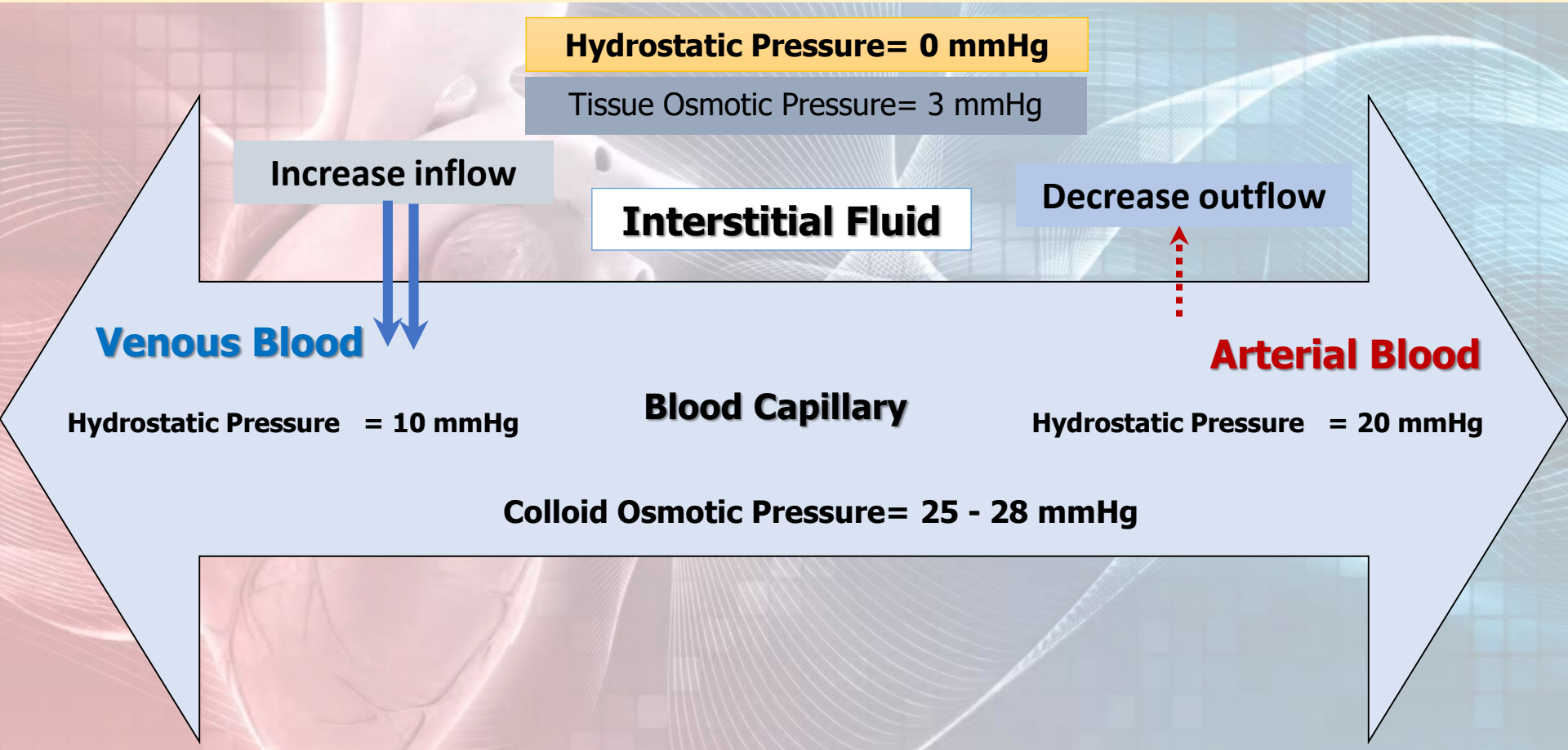
❑ At arterial end:

- 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.

Fluid- Shift Mechanism In Shock



Stages of Shock

- **Reversible shock: (Compensated)**
  - Changes can be reversed by compensatory mechanism (neurohormonal activation) or by treatment.
  - Defense mechanisms are successful in maintaining perfusion.
  - Non-progressive.
- **Progressive:**
  - Defense mechanisms begin to fall.
  - Multi-organ failure.
- **Irreversible shock:**
  - Complete failure of compensatory mechanisms.
  - Can lead to death.

Possible Mechanisms That Lead to Developing Irreversible Shock

Shock Stimulus

↓

Lysosomal Activation, Release Proteases

↓

Splitting of Plasma Proteins

↓

Vasoactive Peptides, Amines, etc.

↓

Hypotension, Fluid Loss

↓

Irreversible Shock

Signs and Symptoms: Hypovolemic Shock

- **Sustained Hypotension**… (? ≤ 80-85/40 mmHg for 30 min.)
- Tachycardia, sensed by Baroreceptors in compensation to the ↓ MAP.
- Rapid, weak, & thready pulse… (? 140/min).
- Intense thirst.
- Tachypnea (rapid respiration), sensed by Chemoreceptors in compensation to hypoxia.
- Restlessness, due to hypo-perfusion.
- Cold, pale skin, due to hypo-perfusion.
- Oliguria (low urine output)/ Anuria (no urine output).
- Blood test: Lactic acidosis.

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

Signs, Symptoms & Treatment:
Classes of Hypovolemic Shock

	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**

Signs and Symptoms:
Cardiogenic Shock

- Similar signs & symptoms to that of hypovolemic shock.
- Congestion of lungs & viscera: (Chest X-Ray -CXR)
  - Interstitial pulmonary edema.
  - Alveolar edema.
  - Cardiomegaly.

Signs and Symptoms:
Septic Shock

- Patient flushed & warm: due to hyper-dynamic state.

Thank You