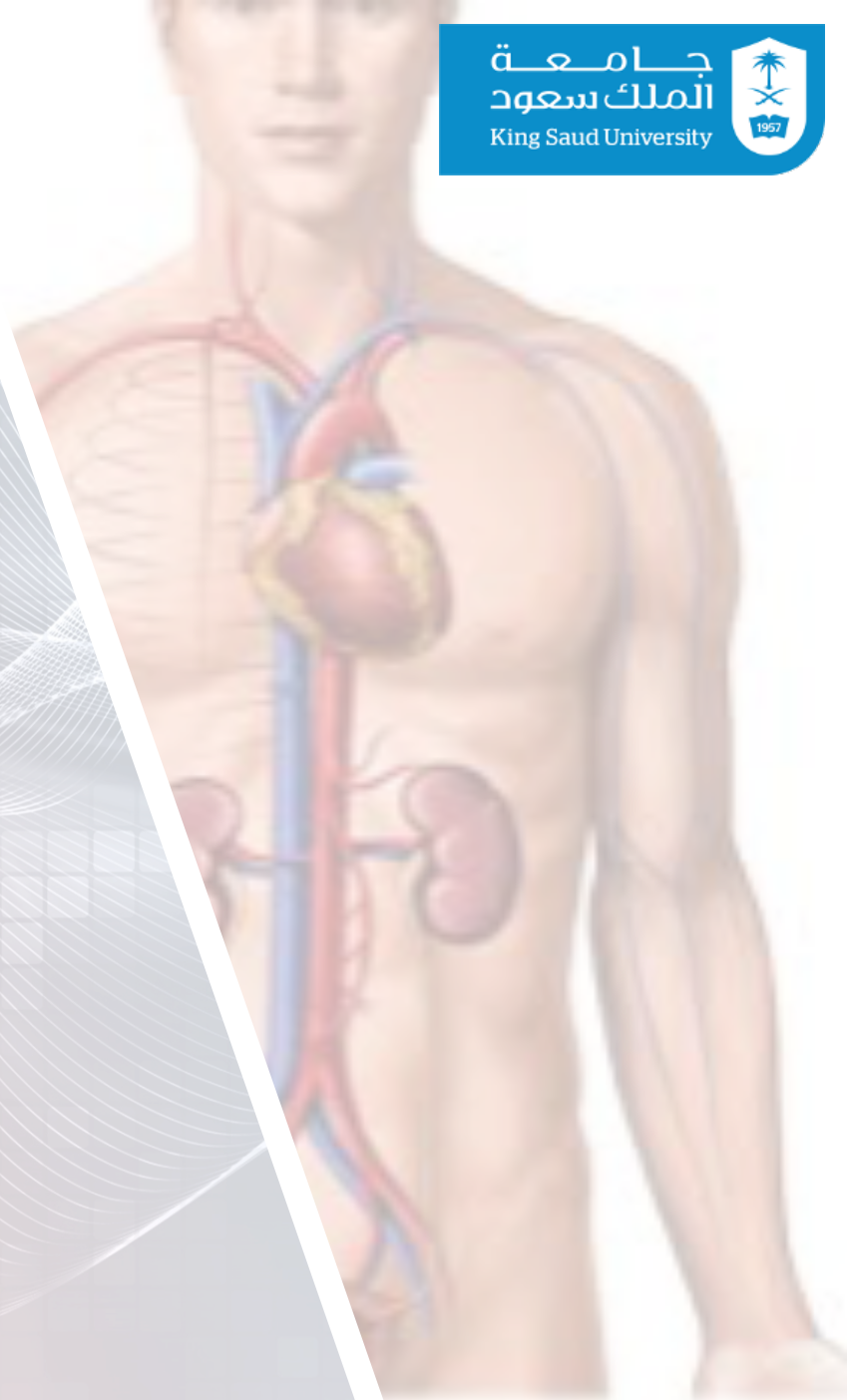


Cardiovascular Physiology

Shock

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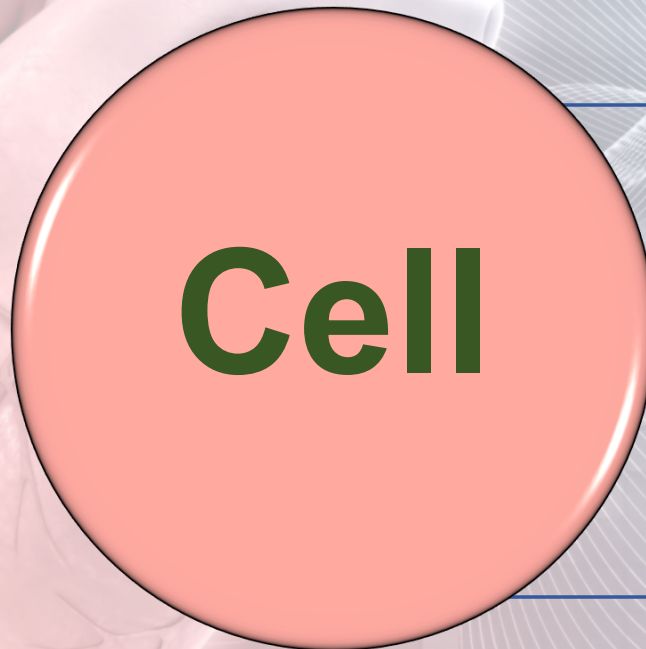


Shock

Lecture Outcomes

- 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



Gets their needed energy to stay alive.

No oxygen, no energy.

No energy, no life.

What is Shock?

- Any condition in which circulatory system is unable to provide adequate circulation & tissue perfusion.
- Results in failure to deliver oxygen to the tissues & vital body organs relative to its metabolic requirements, leading to **organ dysfunction & cellular damage**.
- Defined as **Circulatory Shock**.
- If not corrected quickly, it may lead to irreversible shock & death.

Types of Circulatory Shock

Circulatory Shock

$$\text{MAP} = \text{CO} \times \text{PR}$$

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Low Output Shock

High/N/Low Output Shock

Hypovolemic
(most common)
Too little blood
volume

Obstructive

Cardiogenic
Pump failure

Distributive
Venous pooling,
loss of venous
tone, & general
vasodilatation

Anaphylactic
(Vasogenic)

Psychogenic
(Vasogenic)

Neurogenic
(Spinal)

Septic/Toxic
(Vasogenic)

$$\text{MAP} = \text{CO} \times \text{PR}$$

Hypovolemic Shock

Low Cardiac Output

□ Low CO due to:

- Inadequate blood/plasma volume (loss of <15 - >40% or <750mL - >2L).
- Reduced venous return (preload.)

□ Causes:

- Blood loss/ Hemorrhage: (commonest)
 - Internal or external.
- Fluid/plasma loss:
 - Vomiting, diarrhea, burn, excess sweating, dehydration, trauma.



$$\text{MAP} = \text{CO} \times \text{PR}$$

Cardiogenic Shock

Low Cardiac Output

- ❑ **Low CO due to:**
Inability of cardiac muscle to pump adequate blood in presence of a normal blood volume.
Almost associated with elevated ventricular filling pressure (Rt/Lt/or Both).
- ❑ **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.
- ❑ Is associated with **loss of > 40%** of LV myocardial function.
- ❑ **Mortality rate is high, 60-90%.**

$$\text{MAP} = \text{CO} \times \text{PR}$$

Obstructive Shock

Low Cardiac Output

- ❑ **CO is reduced by vascular obstruction:**
 - **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 dissection.
Massive pulmonary embolism.
Pneumothorax.

$$\text{MAP} = \text{CO} \times \text{PR}$$

Distributive Shock

High/Normal Cardiac Output

- ❑ CO is mostly normal or elevated.
- ❑ Distribution of blood is inappropriate.
- ❑ Shock is due to loss of vascular resistance.

$$\text{MAP} = \text{CO} \times \text{PR}$$

Distributive Shock

High/Normal Cardiac Output

❑ Septic/ Toxic/ Endotoxic Shock:

- Bacterial endotoxin triggers peripheral vasodilatation & endothelial injury.
- Hyper-dynamic state.

❑ Psychogenic shock:

- Simple fainting (syncope.)
- Caused by stress, pain, or fright.
- ↓ HR & vessels dilate.
- Brain becomes hypo-perfused.
- Loss of consciousness.

$$\text{MAP} = \text{CO} \times \text{PR}$$

Distributive Shock

Low Cardiac Output

□ Anaphylactic shock:

- Massive & generalized allergic reaction.
- IgE- Mediated hypersensitivity.
- Histamine triggers peripheral vasodilation & ↑ capillary permeability.
- Can lead to low cardiac output distributive shock.



□ Neurogenic/ Spinal Shock (venous pooling):

- Loss or drop in vasomotor (vascular) tone/ spinal cord injury.
- Generalized peripheral vasodilation.
- Blood volume remains normal.
- CO is severely reduced as blood is pooled in peripheral veins.. (Capacity of blood ↑, & venous return ↓.)
- Behaves like hypovolemic shock.

Pathophysiology of Shock

Reduce
capillary
perfusion

Inadequate
tissue
oxygen

Shift to
anaerobic
metabolism

Metabolic
acidosis

Release of
free
radicals &
oxidative
stress

Tissue
damage

Apoptosis

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⁺] & [Ca²⁺]).**
- **Lysosomes, nuclear membranes & mitochondrial breakdown.**

Metabolic Changes & Cellular Response to Shock

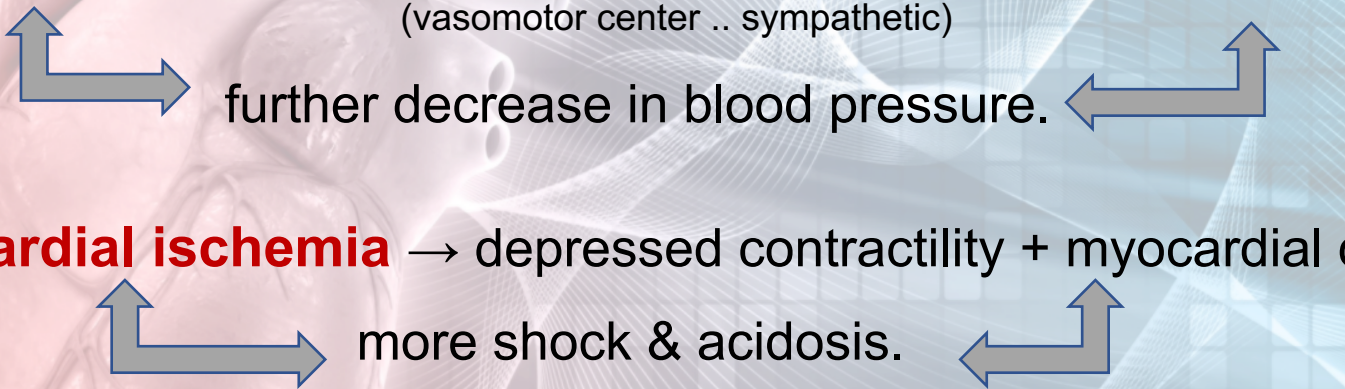
2. **After 3 - 5 hrs of shock:**

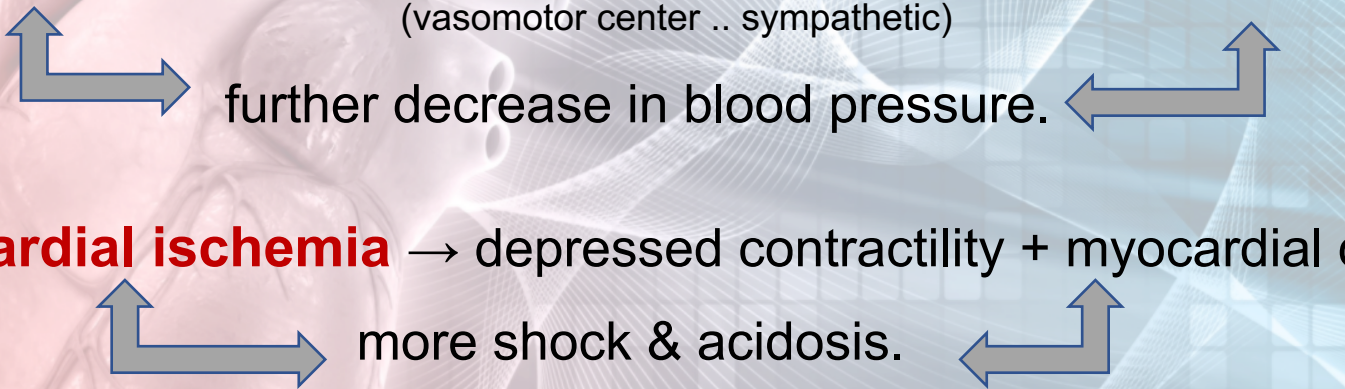
- precapillary sphincters dilate, venules are still constricted.
- blood stagnation in capillaries.
- hypoxia continue + fluid leaves to extra vascular compartment.
- further reduction in circulating blood volume.

3. **Granulocytes accumulation at injured vessels:**

- **free radicals release.**
- further tissue damage.

Metabolic Changes & Cellular Response to Shock

4. **Damage in GIT mucosa** → allows bacteria into circulation.
5. **Cerebral ischemia** → depression of VMC → vasodilation + ↓ HR
(vasomotor center .. sympathetic)


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.**

Compensatory Mechanisms to Shock

Physiological reaction in response to \downarrow BP will lead to the following in order to \uparrow BP:

- ▶ \uparrow HR & myocardial contractility \rightarrow \uparrow CO .
- ▶ Vasoconstriction \rightarrow \uparrow Preload & filling pressure.
- ▶ \uparrow Blood volume.
- ▶ \uparrow Vascular compliance.
- ▶ Restoration of tissue perfusion.

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, Vasoconstriction, & ↑ BP.**

2. Activation of Renin-Angiotensin System:

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

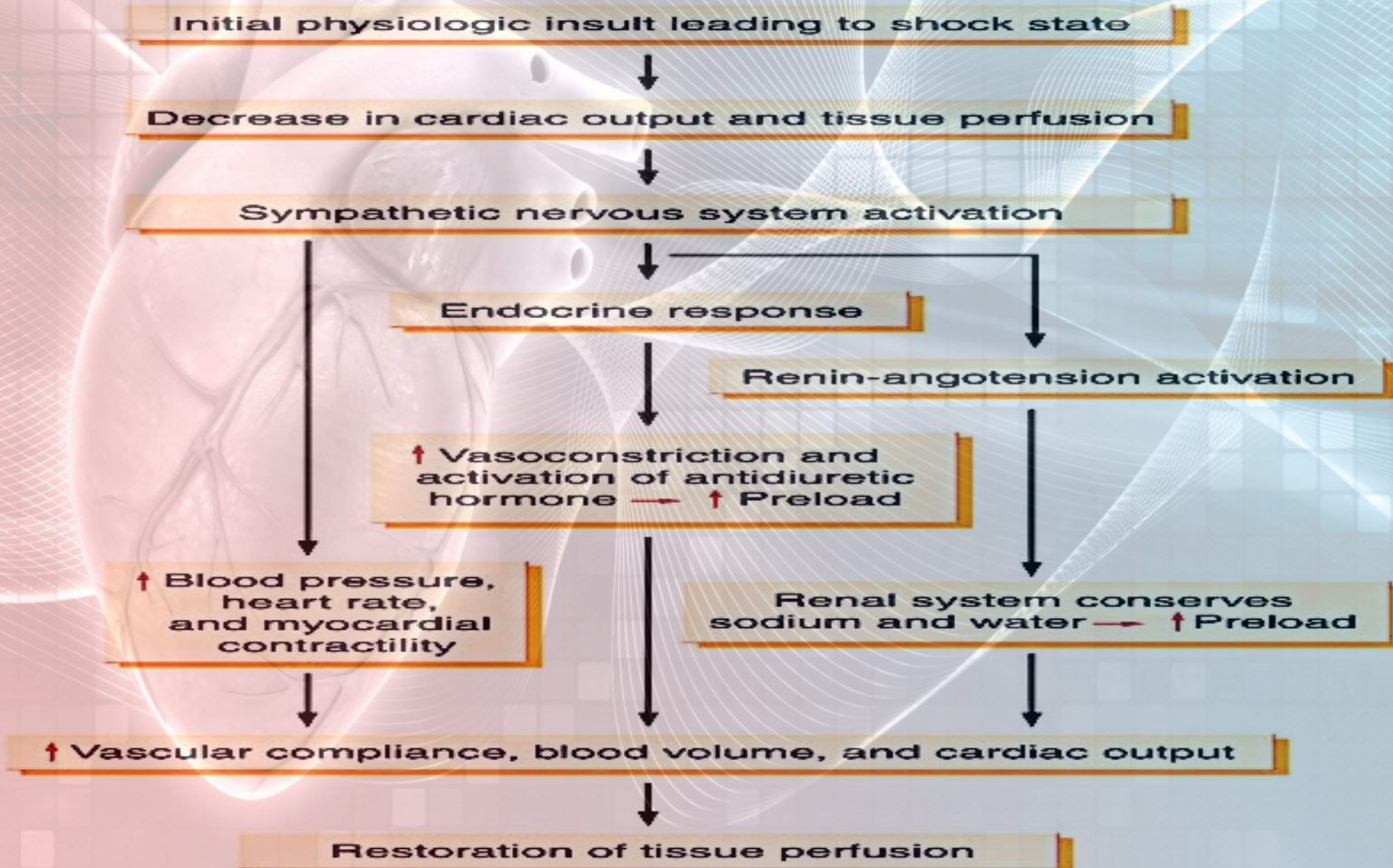
3. Stimulation of ADH (vasopressin):

- Water retention, vasoconstriction & thirst stimulation.

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

5. Fluid- shift mechanism:

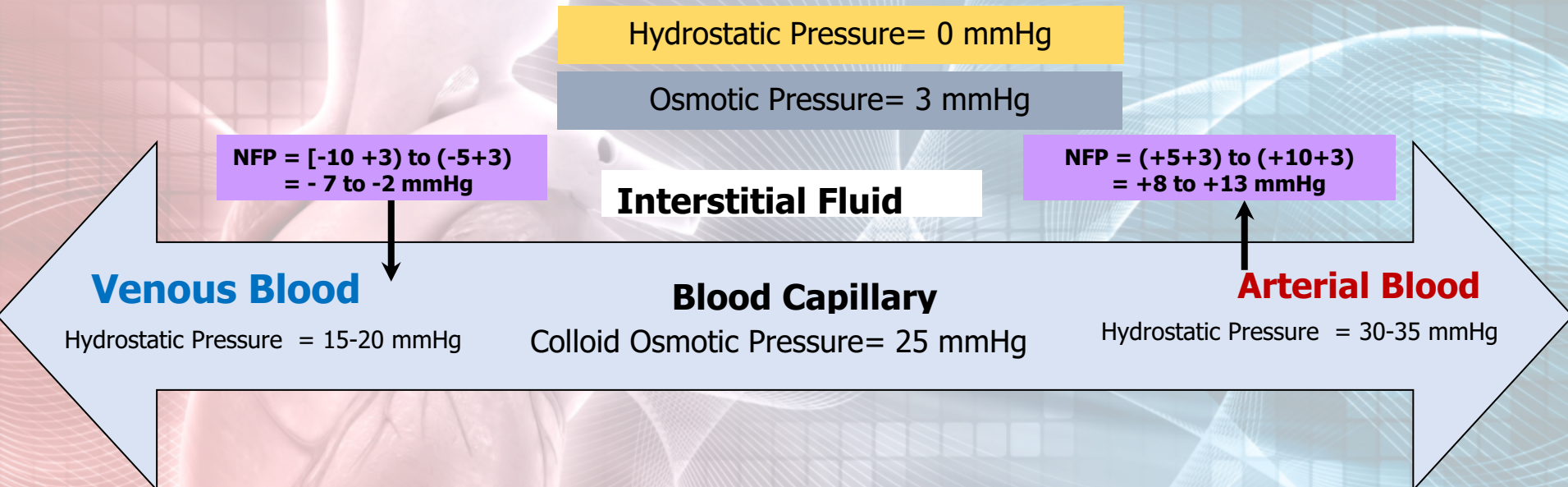
Compensatory Mechanisms



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.

In Normal Microcirculation



At arterial end:

- Water moves **out** of the capillary with a NFP of +8 to +13 mmHg.
- Hydrostatic pressure dominates at the arterial end & a net sum of pressure forces (blood hydrostatic + IF osmotic pressures) flow fluid out of the circulation.

At venous end:

- Water moves **into** the capillary with a NFP of -7 to -2 mmHg.
- Oncotic pressure dominates at the venous end & a net sum of pressure forces (blood osmotic + IF hydrostatic pressures) flow fluid into the bloodstream.

Fluid- Shift Mechanism In Shock

Hydrostatic Pressure= 0 mmHg

Increase inflow

Interstitial Fluid

Decrease outflow

Venous Blood

Hydrostatic Pressure = 10 mmHg

Blood Capillary

Hydrostatic Pressure = 20 mmHg

Arterial Blood

Colloid Osmotic Pressure= 25 mmHg

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... (? $\leq 85/40$ mmHg for 30 min.)
- Tachycardia... sensed by Baroreceptors in compensation to the \downarrow MAP.
- Rapid, weak, & thready pulse... (? 140/min).
- Intense thirst.
- Tachypnea (rapid respiration)... sensed by Chemoreceptors in compensation for 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.

Signs, Symptoms & Treatment: Hypovolemic Shock

	<u>Class I</u>	<u>Class II</u>	<u>Class III</u>	<u>Class IV</u>
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 his hyper-dynamic state.



Thank You