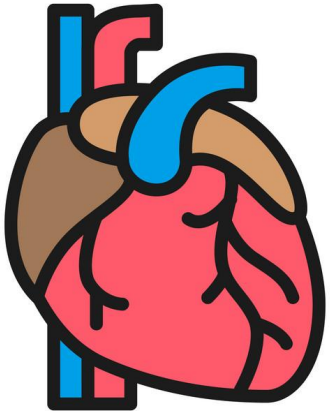


Shock

MED441
KING SAUD UNIVERSITY

Color Index:

- Main text
- Important
- Girls Slides
- Boys Slides
- Notes
- Extra

Editing File

Objectives

- 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.

Introduction

Cells are the basic unit of life,and should get their needed **energy** to stay **alive.**

No **oxygen** → No **energy** → No **life**

What is Meant by Shock?

*(Medical & not electrical shock)

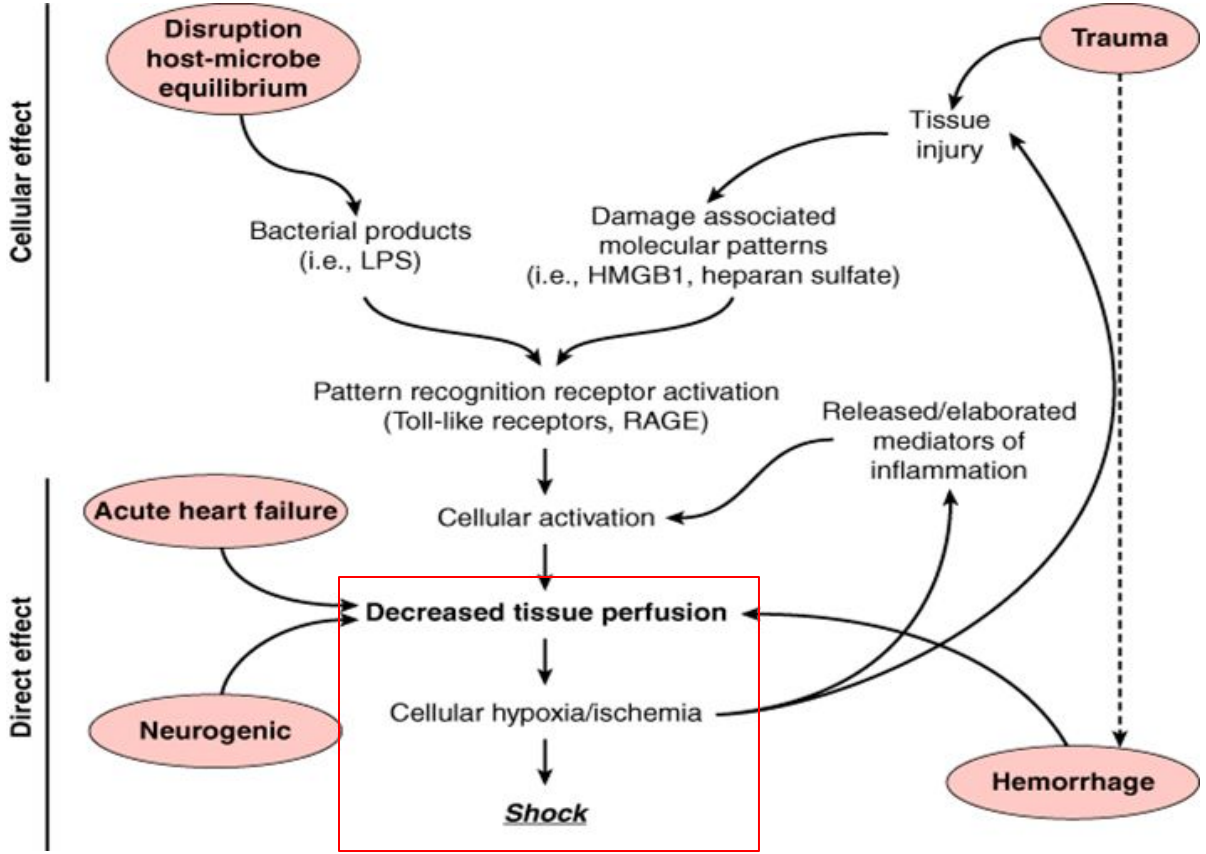
An acute emergency situation, where an acute circulatory failure occur as a result of diminished Cardiac Output or reduced effective circulating blood volume, leading to : 1- inadequate tissue perfusion 2- cellular hypoxia 3- end organ injury.

-Defined as Circulatory Shock.

-The main feature of circulatory shock is **loss of fluid from the circulating blood volume**, so that adequate circulation to all parts of body cannot be maintained

WHAT IS SHOCK?

Boys slide



Features of Circulatory Shock

- **The main feature** of circulatory shock is **acute circulatory failure**, 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.
- Shock is a progressive, rather than a static condition.
- If not controlled & corrected quickly, it may lead to irreversible shock & death.

General mechanism

In Adequate pump:

- inadequate preload
- Excessive after load
- -Poor contractility
- -Inadequate heart rate

In Adequate fluid volume :

- inadequate preload

In Adequate fluid container :

- Excessive dilatation
- Inadequate systemic vascular resistance.

Types of Circulatory Shock

MAP = CO X PR

Low Output Shock
Inability to produce adequate cardiac output

- **Obstructive**
  Obstruction in pulmonary or systemic circulation
- **Hypovolemic**
  (most common)
  Too little blood volume
- **Cardiogenic**
  Pump failure

MAP = CO X PR

High/Normal Output Shock

- **Distributive**

  Mal-distribution of blood as a result of *venous pooling, loss of venous tone, & generalized vasodilatation

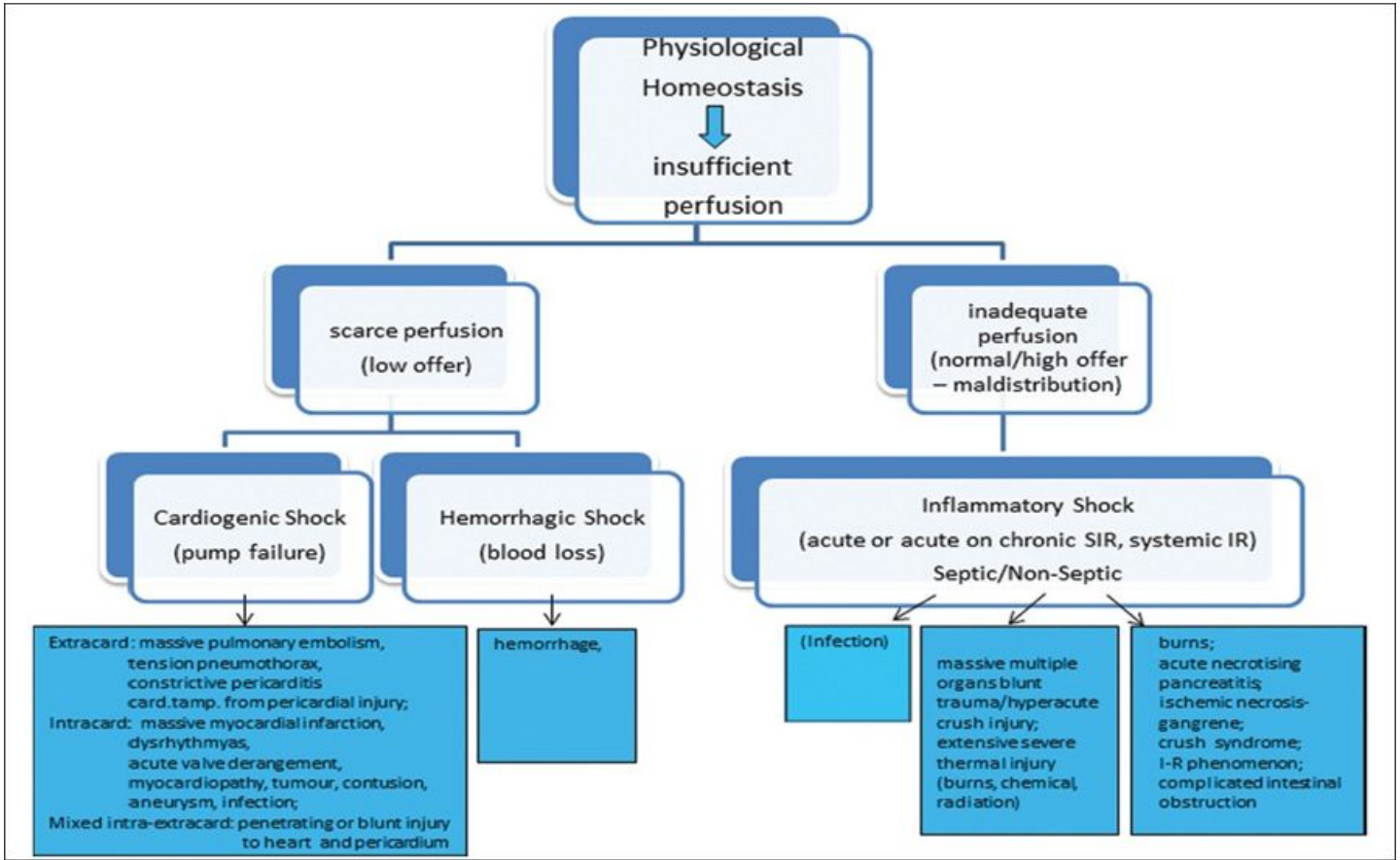
  - **Neurogenic (Spinal)**
    Behaves like hypovolemic shock, Low CO
  - **Vasogenic**
    - **Septic/Toxic**(infection)
      (Vasogenic)
    - **Anaphylactic**
      (Low CO)
    - ***Psychogenic**
      (Vasogenic)

لما يسمع الشخص خبر مفرح او محزن زيادة يستثار ال sympathetic system بشكل كبير في يتعب ويتوقف عن العمل ويشتغل بداله ال parasympathetic system واللي يسبب excessive vasodilation and shock.

*venous pooling (blood collecting in veins due to vasodilatation.

*MAP=Mean arterial Pressure
*CO=Cardiac output
* PR=Peripheral resistance

CLASSIFICATION OF SHOCK: Boys slide



Hypovolemic Shock (Low Cardiac Output)

- **Features:**

-Most common type of shock.
-A life- threatening condition, due to inadequate blood or plasma volume.
-Insufficient perfusion can lead to organ failure.
-Requires immediate emergency medical attention.

MAP = CO X PR

Caused by:

- excessive/severe/massive 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.

Hypovolemic Shock (Low Cardiac Output)

- **Response**

Boys slide

By activating major physiological system:
I- haematologic ii. Cardiovascular
iii- Renal iv- Neuroendocrine

MAP = CO X PR

Hematologic

- activating of coagulation cascade
- Contracting the bleeding vessels (via thromboxane A2 release)
- Platelet activated which form an immature clot on the bleeding source
- The damaged vessel exposes collagen, which subsequently causes fibrin deposition and stabilization of the clot.

Cardiovascular

- Increase : heart rate, myocardial contractility. And constrict peripheral blood vessels
- This response occurs secondary to an increase secretion of NE and decrease in vagal tone (regulated by baroreceptors)
- Redistributing of blood to brain, heart, kidneys and away from skin, Muscles, GIT

Hypovolemic Shock (Low Cardiac Output)

- **Response**

Boys slide

By activating major physiological system:
I- haematologic ii. Cardiovascular
iii- Renal iv- Neuroendocrine

MAP = CO X PR

Renal

- Stimulating renin secretion from juxtaglomerular apparatus

Ang ii has to main effects:

- Reverse hypovolemic shock, vasoconstriction of arteriolar smooth muscle
- Stimulate aldosterone secretion by adrenal cortex

Neuroendocrinal

- Causes an increase in circulating ADH
- ADH released in response to a decrease in blood pressure and decrease in Na concentrations
- ADH increases the reaspsorbtion of water and salt (Na, Cl) by distal tubule and collecting ducts

Classes of Hypovolemic Shock

	Class 1	Class 2	Class 3	Class 4
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	Nromal	Decreased	Decreased	Decreased
Resp.rate	14-20	20-30	30-40	>40
UOP	>30	20-30	5-15	negligible
Mental status	Sl. Anxious	Mildly Anxious	Confused	Lethargic
Fluid	Crystalloid	Crystalloid	Blood	Blood

Signs and Symptoms:
Hypovolemic Shock

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

Cardiogenic Shock
Low Cardiac Output

Features: **MAP = CO X PR**

-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.

-Mortality rate is high, 60-90%.

Causes:

Deterioration of cardiac function

-**Myocardia**l: 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.

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.

LV=Left ventricle; MI=Myocardial infarction

Signs and Symptoms:
Cardiogenic Shock

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

Obstructive Shock
Low Cardiac Output

Features: **MAP = CO X PR**

-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 surgery.

-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.

Distributive Shock

High/Normal Cardiac Output

MAP = CO X PR

Neurogenic (Spinal)

Vasogenic: -Septic or non-septic

(anaphylactic or phsycogenic)

Features:

-**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)**(compliance).

-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.

I: Neurogenic Shock

Neurogenic/ Spinal Shock (venous pooling):

-Can be caused by trauma or injury involving the spinal cord, or by devastating cervical or head injury, or by anesthetic accident.
-Loss of disruption of autonomic nervous system innervation below the level of injury, causing orthostatic hypotension, bradycardia, low body temperature.
-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.

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 & resultant peripheral vasodilatation, pooling of blood & fluid leak from capillaries.
-Bacterial endotoxin triggers peripheral vasodilatation & endothelial injury.
-Inflammatory cytokines may also cause some cardiac dysfunction.
-Leukocytes- induced damage.
-Disseminated intravascular coagulopathy.
-**Hyper-dynamic state.**

-Signs and Symptoms Septic Shock;
Patient flushed & warm: due to hyper-dynamic state.

Non- Septic

Anaphylactic Shock

-**Most common in emergency.**
-Exposure to an antigen resulting in a massive & generalized allergic reaction.
-Systemic release of inflammatory mediators from mast cells & basophils.
-Clinical example: IgE- Mediated hypersensitivity reaction.
-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.

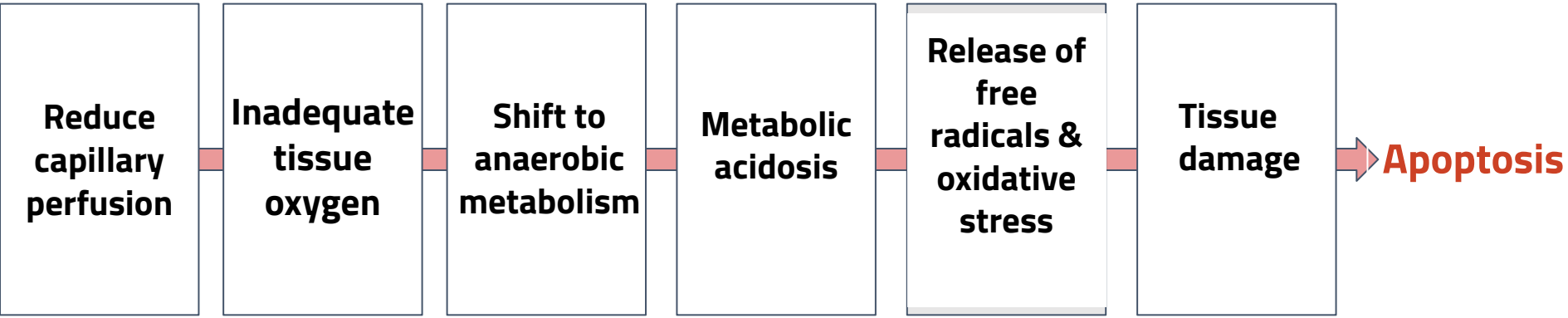
-Blood pressure falls.

-↑ HR (pulse).

-Brain becomes hypo-perfused.

-Loss of consciousness.

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+] & [Ca2+]).**
→ **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.

3 **Damage in GIT mucosa:**

→ allows bacteria into circulation.

4 **Cerebral ischemia:**

→ depression of VMC → vasodilation + ↓ HR

-further decrease in blood pressure.

5 **Myocardial ischemia**

→ depressed contractility + myocardial damage

-more shock & acidosis.

6 **Respiratory distress syndrome**

occurs, due to damage of capillary endothelial cells &
alveolar epithelial cells, with release of cytokines.

7 **Multiple organ failure & death.**

Compensatory mechanism to shock

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

1. ↑ HR & myocardial contractility ⟶ ↑ CO.
2. Vasoconstriction ⟶ ↑ Preload & filling pressure.
3. ↑ Blood volume.
4. ↑ Vascular compliance.
5. Restoration of tissue perfusion.

HR= heart rate CO=cardiac output BP=blood pressure

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.

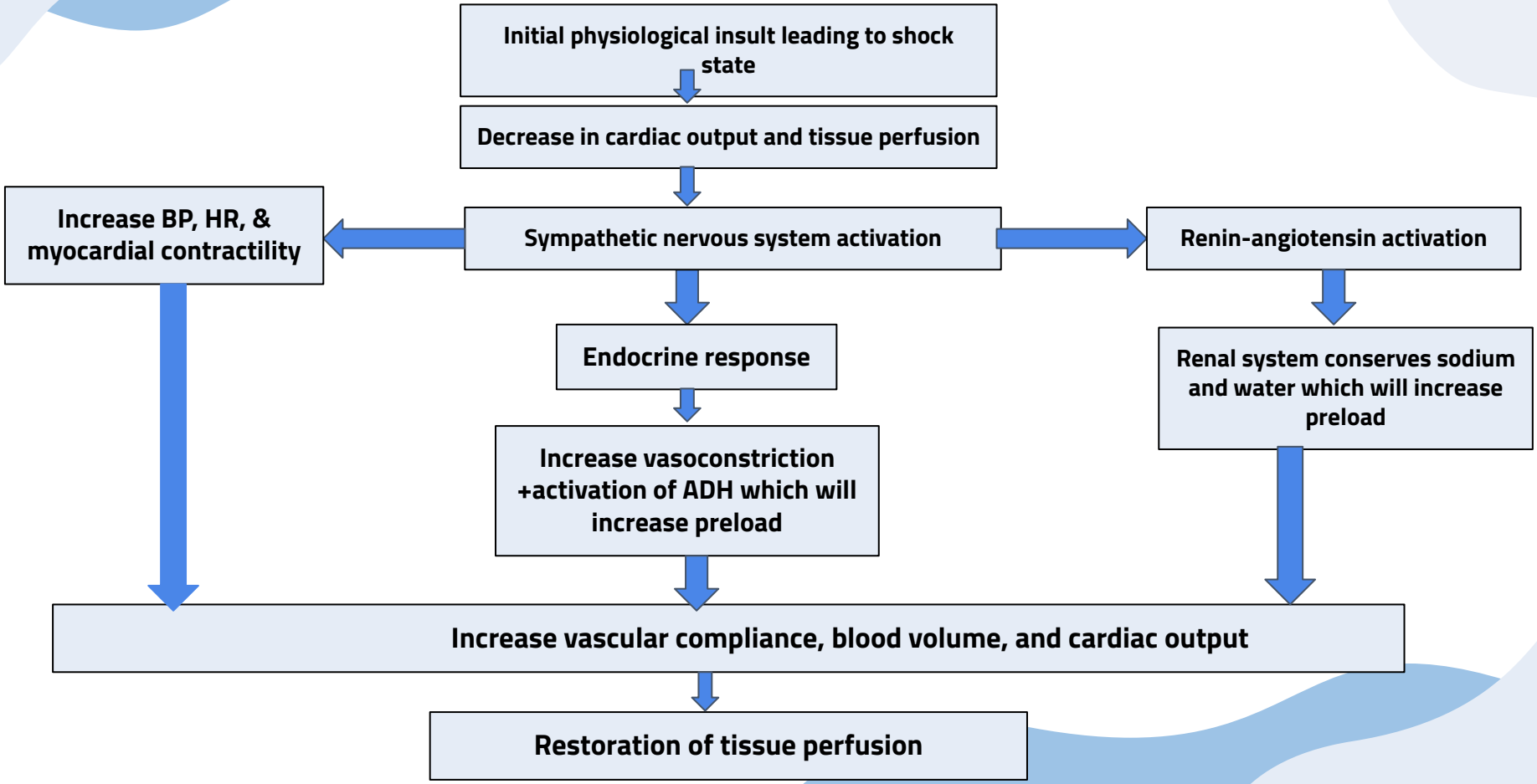
3. **Stimulation of ADH (vasopressin):**

▪ Water retention, vasoconstriction & thirst stimulation.

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

5. Fluid- shift mechanism.

In summary: compensatory mechanism to shock



Fluid-Shift Mechanism in Shock

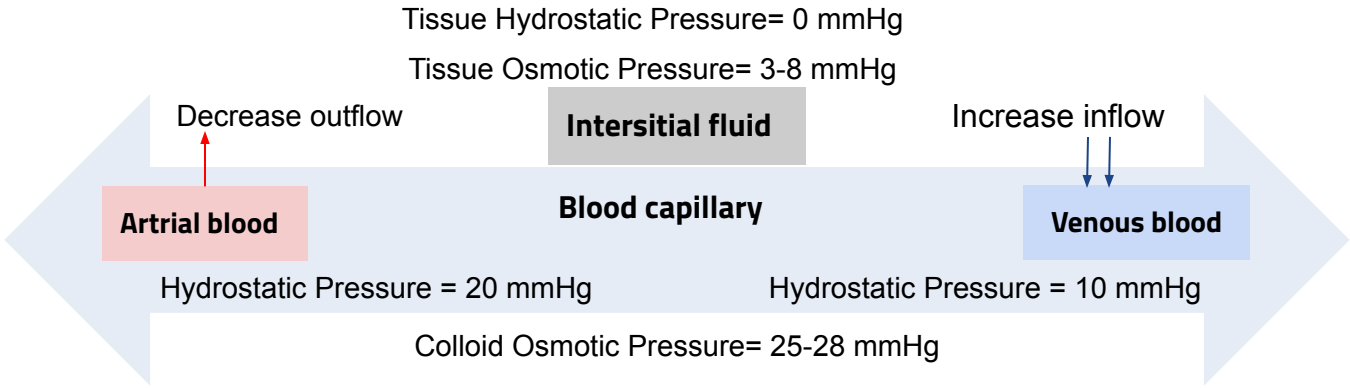
-In shock, both the hydrostatic pressure(in capillary) decreases & oncotic pressure(osmotic pressure) are constant, as a result:

- The fluid exchange from the capillary to the extracellular space **decreases**.
- The fluid return from the extracellular space to the capillary **increases**.

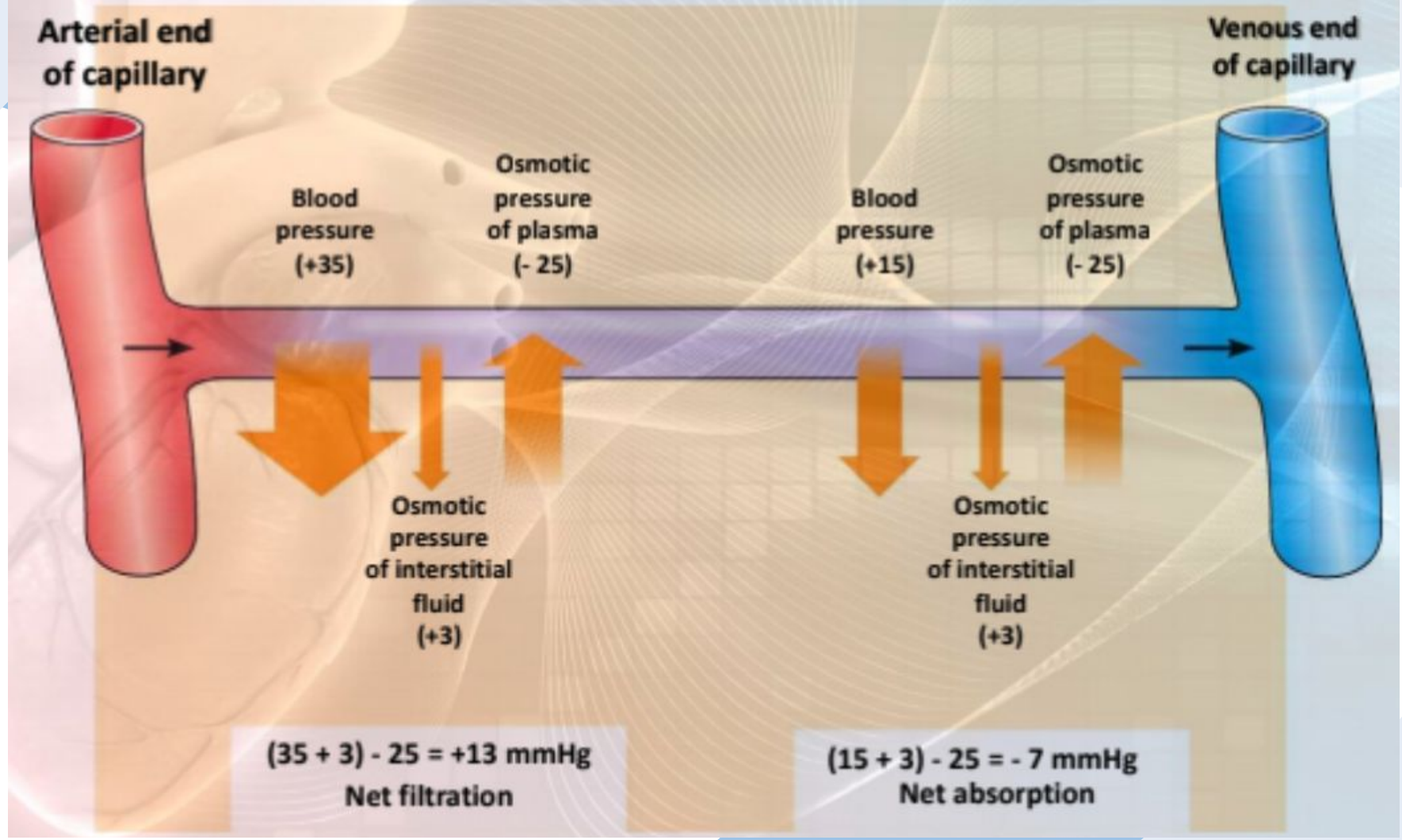
-This will lead to an increase in the **blood volume** & the **BP** in order to help in compensating the shock situation.

Hydrostatic pressure is the force of the fluid volume against a membrane, while **osmotic pressure** is related to the protein concentration on either side of a membrane pulling water toward the region of greater concentration

Normal Forces at The Arterial & Venous Ends of The Capillary



At arterial end:	At venous 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.	-**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.


Arterial end of capillary
Venous end of capillary
Blood pressure (+35)
Osmotic pressure of plasma (- 25)
Blood pressure (+15)
Osmotic pressure of plasma (- 25)
Osmotic pressure of interstitial fluid (+3)
Osmotic pressure of interstitial fluid (+3)
(35 + 3) - 25 = +13 mmHg
Net filtration
(15 + 3) - 25 = - 7 mmHg
Net absorption

Stages of Shock

1-Reversible shock: (Compensated)

- Changes can be reversed by compensatory mechanism (neurohormonal activation) or by treatment.
- Defense mechanisms are successful in maintaining perfusion.
- Non-progressive.

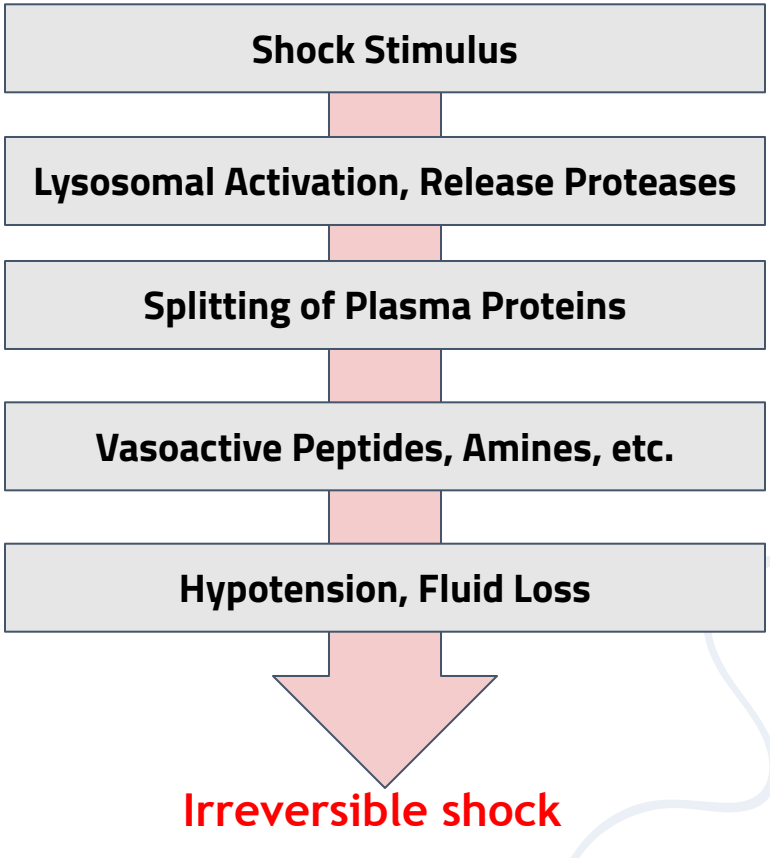
2-Progressive:

- Defense mechanisms begin to fall.
- Multi-organ failure.

3-Irreversible shock:

- Complete failure of compensatory mechanisms.
- Can lead to death.

Possible Mechanisms That Lead to Developing Irreversible Shock



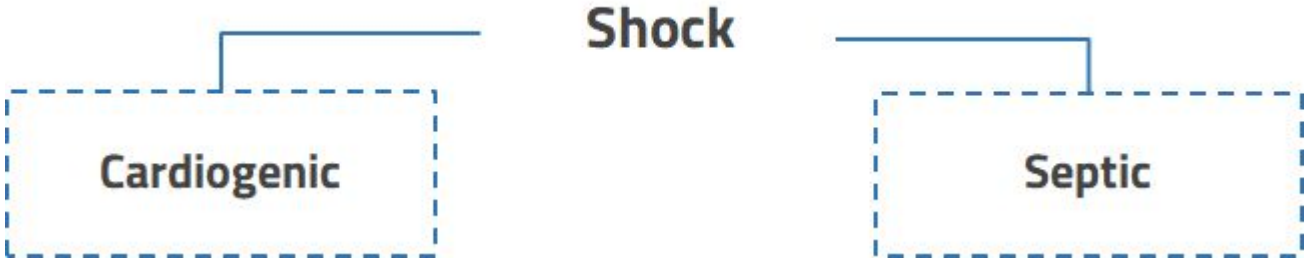
Signs & Symptoms of Hypovolemic Shock

Symptoms relating to heart		Systemic symptoms
Sustained hypotension (≤80-85/40 mmHg for 30 min)	Sustained: مستمر	Intense thirst
Tachycardia (sensed by baroreceptors in compensation to ↓ MAP)		Tachypnea (rapid respiration) (sensed by chemoreceptors in compensation to hypoxia)
Rapid, weak, & thready pulse (140/min)		Oliguria (low urine output)/ Anuria (no urine output)
1-Restlessness, 2-Cold, pale skin Both due to hypo-perfusion		Blood test: Lactic acidosis

Classes of Hypovolemic Shock

	Class I	Class II	Class III	Class IV
Blood Loss (% blood volume)	<750ml (<15%)	700-1500ml (15-30%)	1500-2000ml (30-40%)	>2000ml (>40%)
Pulse	<100 (no tachycardia)	**>100** (compensatory begins)	>120	>140
Blood Pressure	Normal	Almost Normal	**Decreased**	**Decreased**
Pulse Pressure	Normal	Slightly **Decreased**	**Decreased**	**Decreased**
Resp.Rate	14-20 (normal)	**20-30**	**30-40**	**>40**
UOP (urine output)	>30	**20-30**	**5-15**	**Negligible**
Mental state	Sl. anxious	Mildly anxious	**Confused**	**Legarthic**
Fluid Treatment	crystalloid	crystalloid	**Blood**	**Blood**

Signs & Symptoms of Other Shocks



Similar S&S to hypovolemic shock

Congestion of lungs and viscera:
(chest x-ray: CXR)

- Interstitial pulmonary edema
- Alveolar edema
- cardiomegaly

Patient flushed and warm
Due to hyperdynamic state

- Opposite of hypovolemic which is cold and pale extremities

Shock manage And treatment

1 Management:

- Control airway and breathing
- Maximize oxygen delivery
- Place lines, tubes, monitors
- Get and run :
  -IVF on a pressure bag
  - blood if required
  - hang pressors
- Call your senior/ attending

2 Definitive management

Goal : restore normal tissue perfusion:

- blood pressure, pulse, respiration, Skin apperance
- Urine output (30-50 cc) per minute
- Hemoglobin 8-10gm / hematocrit 24-30

3 Treatment:

- draw blood for lab test and blood typing while inserting IV
- Relieve pain using IV narcotics
- Reassess
- Blood transfusion : think twice
- Vasopressor
- may use antibiotic
- Maintain IV fluids

Team Leaders

Rand aldajani

Nawaf Alshehri

Sub Leader

Samiah AlQutub

Team Members

Raneem Alanazi

Mohammed Alsaqabi