Lecture 12
Shock

- Red: important
- Black: in male / female slides
- Pink: in female slides only
- Blue: in male slides only
- Gray: extra information

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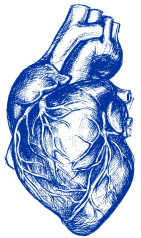
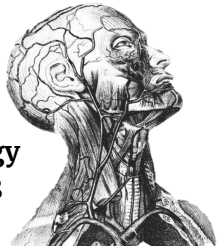


Physiology
MED438



Objectives:

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

Shock
“circulatory shock”

Definition

Shock is profound hemodynamic and metabolic disturbance characterized by failure of the circulatory system to deliver oxygen & to maintain adequate perfusion of vital organs relative to metabolic requirement.

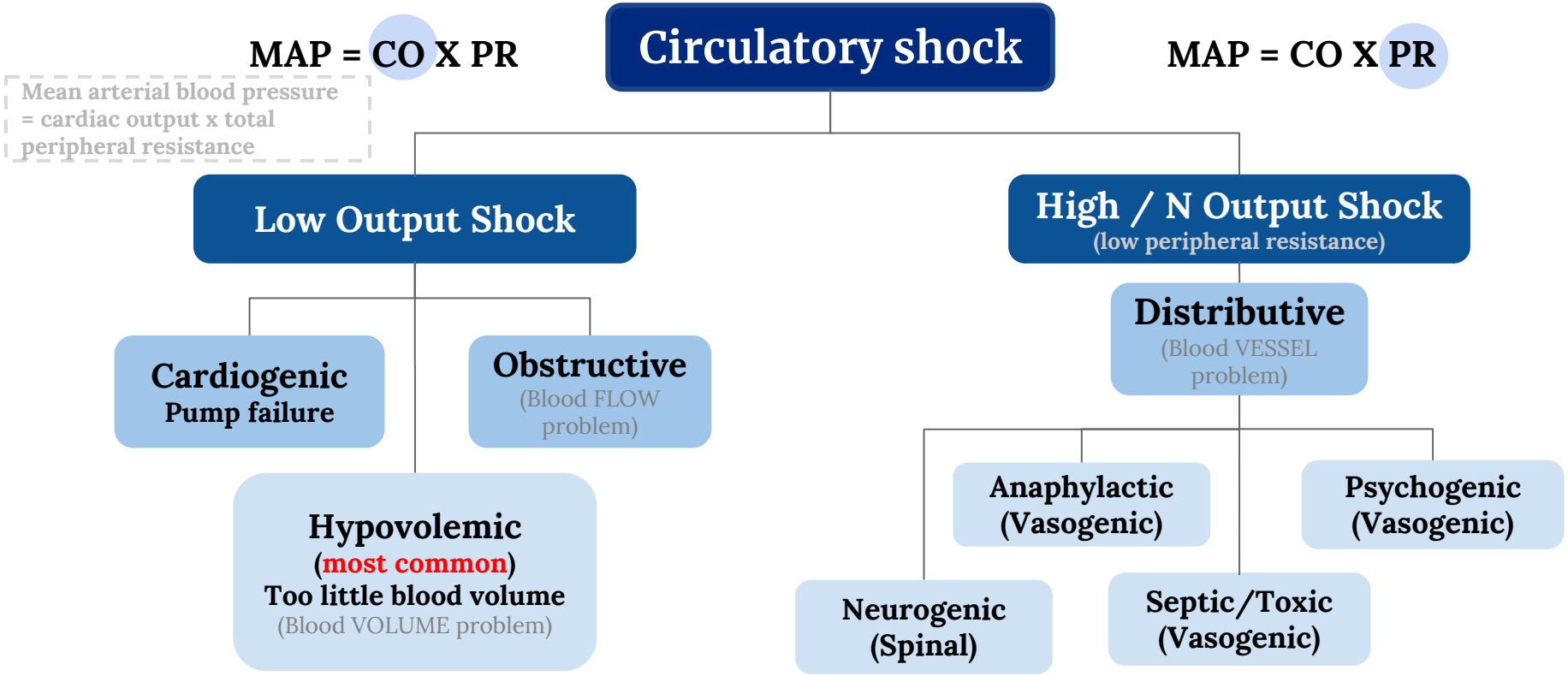
The Blood reaching the organs is less than the amount needed.

A normal cardiac pump, circulatory system and/or volume are important to maintain blood flow to tissues. (1- Pump, 2-Fluid, 3-Tubing)

General mechanism

Inadequate pump due to	Inadequate fluid volume	Inadequate container
1)Inadequate preload 2)Inadequate heart rate 3)Excessive after load 4)poor contractility	1)Hypovolemia	1)Excessive dilation 2)Inadequate systemic vascular resistance

Types of Circulatory Shock



Hypovolemic Shock

MAP = CO X PR

Low CO due to	Causes	Clinical Presentation
o Inadequate blood/plasma volume (loss of 15-25%,/ 1-2 L). **o Reduced venous return (preload).**	**o Internal fluid loss:** • Increased capillary membrane permeability. • Decreased plasma colloidal osmotic pressure (Inward force). **o External fluid loss:** • Hemorrhage (most common). • Plasma loss as in extensive burns. • Severe vomiting, excess diarrhea, excess sweating, or massive diuresis (increased or excessive production of urine.) **o Surgery**	**o Tachycardia** Compensation for ↓ MAP sensed by Baroreceptors. **o Tachypnea** (rapid respiration) Compensation for hypoxia sensed by Chemoreceptors. **o Rapid, weak, & thready pulse** (140/min). **o Hypotension** (85/40 mmHg) (because low volume). **o Cold, pale skin due to hypoperfusion.** **o Intense thirst.** **o Oliguria** (low urine output) **/ Anuria** (no urine output): dark & concentrated urine) due to poor tissue perfusion **o Mental status changes.** (drowsiness- confusion) **o Restlessness due to hypoperfusion.** **o Blood test: Lactic acidosis.**

Hypovolemic shock

Human body responds to acute hemorrhage by activating **four major physiological systems:**

01

Hematologic

- Activation of coagulation cascade
- Vasoconstriction (thromboxane A2)
- Platelets form immature clot
- Exposed collagen caused fibrin deposition

02

Cardiovascular

- ↑ HR + contractility
- Constricted peripheral vessels (↑ norepinephrine + ↓ vagal activity)
- Blood redistributed to brain, kidneys and away from skin, G.I and muscles.

03

Renal

↑ renin from juxtaglomerular apparatus

04

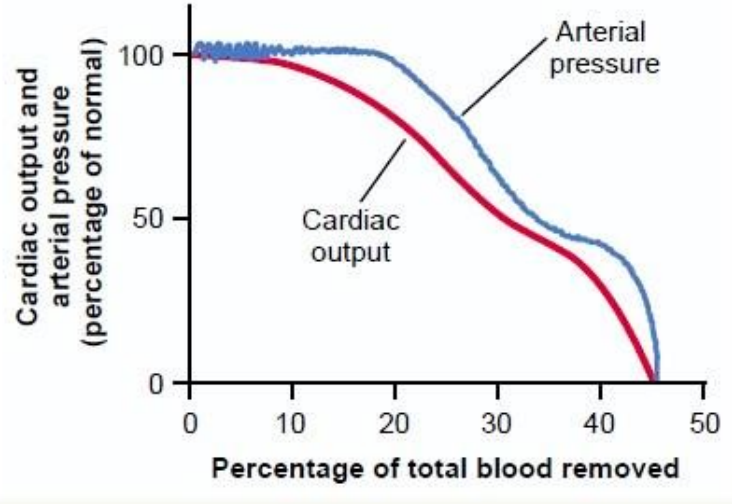
Endocrine

- ↑ ADH due to ↓ BP
- ↑ water retention from ADH by distal tubules

Types of Hypovolemic Shock

parameters	Type I	Type II	Type III	Type IV
Blood loss (ml)	<750	750-1500	1500-2000	>2000
Blood loss (%)	<15	15-30	30-40	>40
Pulse rate (bpm)	<100	>100	>120	>140
BP	normal	decreased	decreased	decreased
Urine output (ml/hr)	>30	20-30	5-15	negligible
Respiratory rate	14-20	20-30	30-40	>35
CNS symptoms	normal	anxious	confused	lethargic

Effect of hemorrhage on cardiac output and arterial pressure



As the figure shows the body can still maintain the atrial pressure till the loss of blood exceeded 20% after that the pressure starts to decrease at relatively constant rate, until 40% blood loss is reached the pressure goes at free fall eventually causing death.

Cardiogenic shock

- Characterized by low CO due to failure of myocardial pump.
- Has a high mortality rate
- Is associated with loss of > 40% of LV myocardial function.

Causes:

- Decreased contractility
- Myocardial infarction (most common)
- Sustained arrhythmia (Heart block, ventricular tachycardia, supraventricular tachycardia, atrial fibrillation) (conductive
- Mechanical dysfunction (acute valvular dysfunction, e.g. papillary muscle rupture post-MI, severe aortic stenosis, rupture of ventricular aneurysms)
- Cardiotoxicity (B blocker and **calcium channel blocker overdose**)

Clinical signs:

- Same as hypovolemic + distended jugular veins and pulse may be absent
- May not show tachycardia if patient is on B blockers or he has a heart block
- MAP ↓ 70 mmHg compromises coronary perfusion
- Congestion of lungs and viscera can **be seen on x-ray as:**

- Interstitial pulmonary edema - Alveolar edema - Cardiomegaly

MAP = CO X PR

Obstructive Shock

Low CO due to	Causes	Clinical Presentation
obstruction to the flow of the blood, but the heart pumping capacity is well. (circulatory obstruction)	**o Obstruction of venous return:** • e.g. Vena Cava Syndrome (usually neoplasms). **o Compression of the heart:** • e.g. hemorrhagic pericarditis → cardiac tamponade. (build up of blood or other fluid in pericardial) **o Obstruction of the outflow of the heart:** • Aortic dissection[1]. • Massive pulmonary embolism. • Pneumothorax.	o Jugular venous distension (congestion) [2]. o Distant heart sound in cardiac tamponade[3].(weak) o Tracheal deviation & decreased or absent unilateral breath sounds in tension pneumothorax. o Chest pain, dyspnea and hemoptysis in pulmonary embolism.

[1] Tear in the wall of aorta. (emergency, could be fatal).
[2] Due to increased atrial pressure.
[3] Accumulation of fluid in the pericardium.

Female slides

Distributive Shock (vasogenic)

(low resistance shock)

MAP = CO X PR

- Shock is due to inadequate perfusion of tissues through maldistribution of blood flow.
- Intravascular volume is maldistributed because of alterations in blood vessels, i.e. loss of vascular resistance.
- **Cardiac pump & blood volume are normal** but blood is not reaching the tissues (there is peripheral vasodilation due to loss of vessel tone).

Neurogenic (spinal) Shock (less common) (venous pooling)

Anaphylactic shock

Septic (Toxic, Endotoxic) **shock (most common)**

Psychogenic shock

1- Septic shock

MAP = CO X PR

Causes:

Bacterial endotoxins activate NOS in macrophages, releasing NO and causing an increase in capillary permeability & peripheral vasodilation (hyperdynamic response). **E.g:**

- Peritonitis
- Generalized bodily infections
- Generalized gangrenous infection
- Infection spreading into blood from kidney or urinary tract.

Clinical picture:

- Flushed and warm, sweaty skin
- Increased HR
- Tachypnea
- Severe vasodilation

2- Anaphylactic shock:

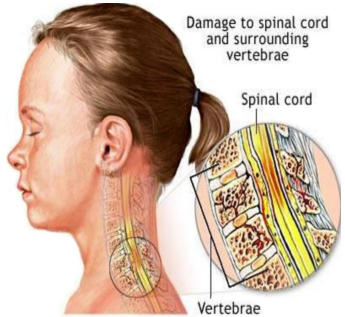
- Type I hypersensitivity
- Basophils + mast cells release histamine causing vasodilation and increased permeability
- It can lead to **low output** distributive shock, which can be fatal.

Clinical picture:

- **Circulatory collapse**
  - Tachycardia + hypotension, weak & rapid pulse
- **Cutaneous manifestations**
  - Urticaria, erythema, angioedema, pruritis
- **Respiratory issues**
  - Stridor, wheezing, respiratory distress

3- Neurogenic (Spinal) Shock

- Loss of autonomic motor reflexes, leading to a **drop in vasomotor tone.**
- This causes vasodilation and a drop in vascular resistance, leading to hypotension.
- CO is severely reduced as vascular capacity increases with venous blood pooling in peripheral veins (↓venous return) **(Behaves like hypovolemic shock)**.
- Neurogenic is the **rarest** form of shock, **Caused by:**
- **Spinal cord injury** (above C7, thoracic segment).
- Spinal anesthesia
- Deep general anesthesia.
- Brain damage.
- Prolonged brain ischemia that cause total inactivation of the vasomotor neurons.



4- Psychogenic shock:

- Simple fainting (syncope.)
- Caused by stress, pain, fright or emotional crisis.
- ↓ HR.
- Sudden temporary, generalized dilation of blood vessels.
- Brain becomes hypoperfused → Loss of consciousness.

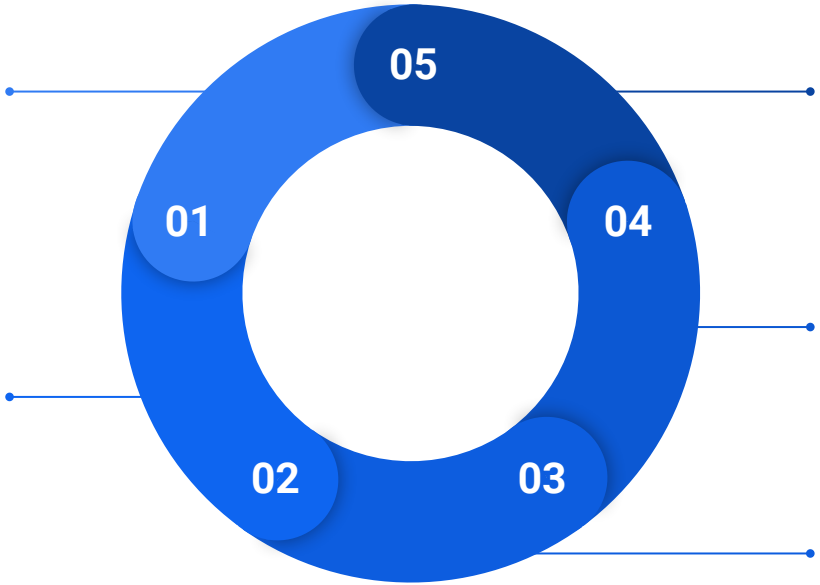
Pathophysiology of shock

Impaired tissue perfusion occurs when an imbalance develops between cellular oxygen supply and demand.

Cell switches from aerobic to anaerobic metabolism

This leads to lactic acid production

Cell function stops and it starts swelling



Apoptosis

Na K pump is impaired
(accumulation of Na, Cl and water)

Membrane becomes more permeable

This allows electrolytes and fluids to seep in and out of the cell

Metabolic Changes & Cellular Response to Shock

1- Reduced Capillary perfusion:

- Spasm of pre/post capillary sphincters.
- hypoxic tissue damage, (oxidative stress.)
- anaerobic metabolism (anaerobic glycolysis.)
- lactic acid production. (the cause of acidosis)
- metabolic acidosis (intracellular acidosis).
- Failure of Na+/K+ pump (↑ [Na+] & [Ca2+]).
- Lysosomes, nuclear membranes & mitochondrial breakdown.

2- After 3-5 hrs of shock:

- precapillary sphincters dilate, venules are still constricted.
- blood stagnation in capillaries.
- hypoxia continue + fluid leaves to extravascular 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 cont.

4- Damage in GIT mucosa:

- allows bacteria into circulation.

5- Cerebral ischemia:

- depression of VMC → vasodilation + ↓ HR (vasomotor center .. sympathetic) leading to 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.

Stages of shock

Reversible stage
(Non Progressive, Compensated Shock)

- In which compensatory mechanism (neurohormonal activation) & appropriate treatment help restoration of blood pressure & blood loss.
- Defense mechanisms are successful in maintaining perfusion.

Progressive

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

Irreversible stage
(Decompensated Shock)

- There is complete failure of compensatory mechanisms.
- Series of positive feedback mechanisms take place leading to further deterioration & tissue hypoxia.
- This stage is reached and patient may die, when blood loss is excess and not immediately replaced and proper treatment is delayed.

Compensatory Mechanisms to Shock

1. Stimulation of Sympathetic Nervous System:

- Baroreceptors reflex mechanism due to ↓BP
- Acidosis stimulates chemoreceptors reflex mechanism → sympathetic stimulation **Leading to:** Vasoconstriction & tachycardia →increases TPR ,ABP

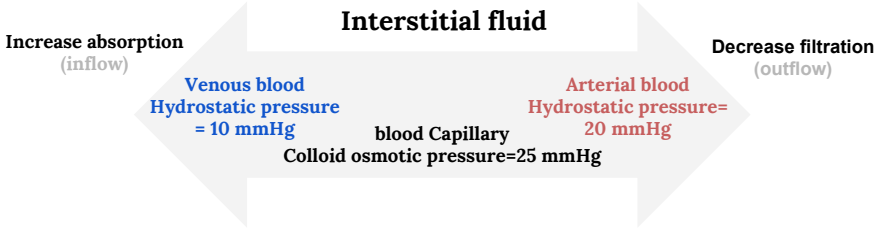
2. Release of vasoconstrictor factors/hormones as :

- **Catecholamines**
- **Vasopressin** →vasoconstriction, increase BP & acts on renal tubules to restore fluid volume & thirst stimulation.
- **Glucocorticoids** to ↑ blood sugar to meet increased metabolic needs.
- **Renin-angiotensin-aldosterone** → angiotensin II → potent vasoconstriction & releases aldosterone adrenal cortex → Na+ & water retention (↑ intravascular volume)

3. Tachypnea

Caused by activation of chemoreceptor reflex and sympathetic overactivity

4. Increased movement of interstitial fluid into capillaries (capillary fluid shift) as a result of decreased capillary hydrostatic pressure while oncotic pressure is constant→↑blood volume & BP



5. Increased 2,3 DPG concentration in RBCs:

Important to help Hb deliver more O2 to the tissues (**shift O2 dissociation curve to the right**)

6. Restoration of circulatory plasma volume, plasma proteins and RBCs mass.

Causes of irreversible stage of shock

Cardiac depression

The drop in APB leads to drop in coronary flow → (-) heart → drop CO

Vasomotor failure

Results from depression of vasomotor center → the heart becomes depressed (same) and CO drops.

Generalized cellular deterioration

↓ in ATP, lysosomes rupture, ↓Na+ and K+ pump (Na retention), cell swelling, depressed cellular metabolism of nutrients

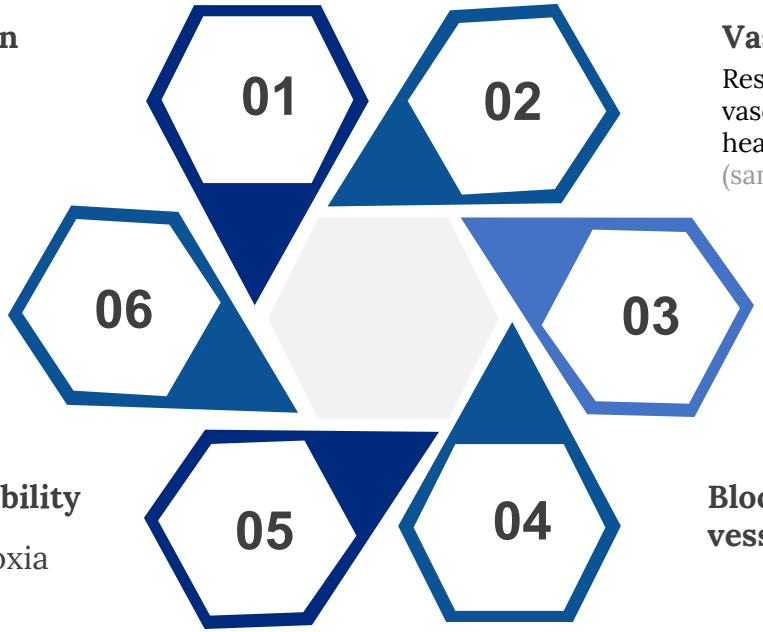
Release of toxins and endotoxin

Leads to cardiac depression

↑ Capillary permeability

Capillary hypoxia

Blockage of small vessels “Sludged Blood.”



male slides

Management

Emergency Management:

1. Control airway and breathing
2. Maximize oxygen delivery
3. Place lines, tubes, and monitors
4. Get and run IVF on a pressure bag
5. Get and run blood (if appropriate)
6. Get and hang pressors
7. Call your senior/fellow/attending

Definitive Management:

1. Hypovolemic – Fluid resuscitate (blood or crystalloid) and control ongoing loss
2. Cardiogenic - Restore blood pressure (chemical and mechanical) and prevent ongoing cardiac death
3. Distributive – Fluid resuscitate, immediate surgical control for infection

Treatment

Goal:Restore normal tissue perfusion by restoring

1. Blood pressure, Pulse, Respirations
2. Skin Appearance
3. Urine output (30-50 cc per hour)
4. Hemoglobin 8-10 gm or Hematocrit 24-30

1. While inserting IVs, draw blood for laboratories and for blood typing
2. Relieve pain with IV narcotics
3. Reassess
4. Blood transfusion: think twice Vasopressors
5. Antibiotics
6. Maintain IV fluids

Quiz

1. Which of the shock stages is caused by sludged blood?
   A. non-progressive
   B. progressive
   C. irreversible
   D. Non-reversible

2. Which of the following causes obstructive shock?
   A. Bleeding
   B. Dehydration
   C. tension pneumothorax
   D. Heart failure

3.which of the following is the goal for the treatment of the shock?
   A. Restore normal tissue perfusion
   B. increase rate of respiration
   C. decrease peripheral resistance
   D. decrease heart rate

4. In almost all patients who have severe burns, with so much plasma lost, the resulting condition is?
   A. Neurogenic shock
   B. Hypovolemic shock
   C. Septic shock
   D. Histamine shock

5. Which of these factors will not cause circulatory shock?
   A. Cardiac abnormality
   B. Decrease venous return
   C. Increase venous return
   D. Diminished blood volume

SAQ:

1.Explain the pathophysiology of shocks.

Slide 14

Answers: C - C - A - B - C

Leaders

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- Abdullah Aldawood
- Abdullah Shadid
- Meshari Alzeer
- Mohammed Alhamad
- Abdullah Alassaf
- Khalid Alkhani
- Amjad Albaroudi
- Mohammed Alhuqbani