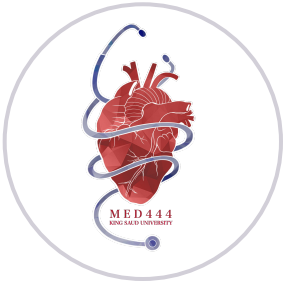
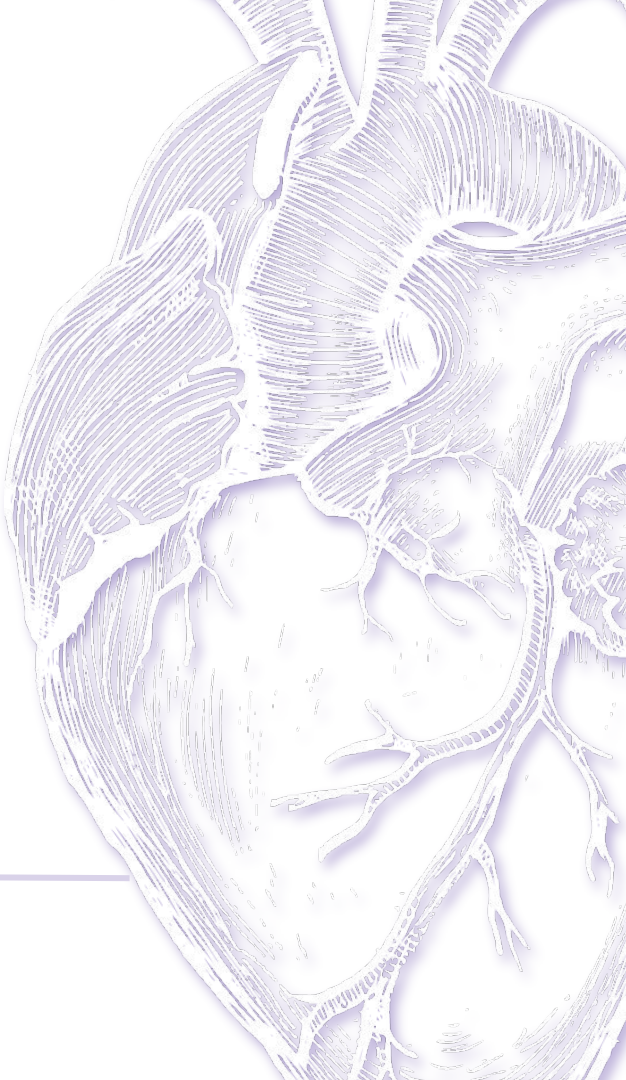




Shock

Editing File

Main text Female's slide Male's slide Important text Doctor's note Extra

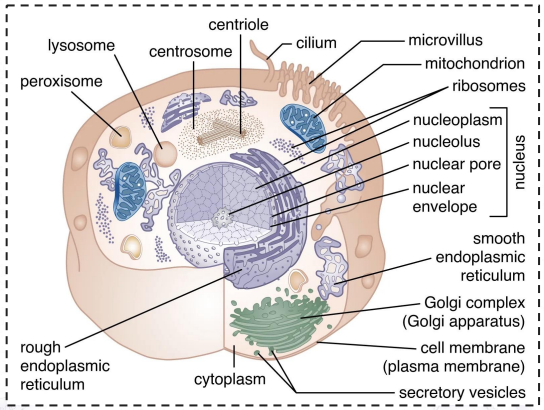
Objectives:

- To define shock & to recognize its different stages.
- To identify etiology & pathophysiology of different types of shock.
- Describe the pathways leading to shock.
- Discuss the stages of a hypovolemic shock.
- To understand compensatory reflex mechanisms of shock during the hemorrhagic shock.
- To understand complications & causes of irreversible shock.
- Clinical features and management.

Recommended video by osmosis
Recommended video by ninja nerd (First), (second)

Introduction

The cell Gets its needed energy to stay alive, If there is no oxygen → No energy → No life



What is meant by shock ?

- A state of acute emergency / life-threatening condition that occurs when the body is not getting enough blood flow. in which acute circulatory failure occur as a result of diminished
cardiac output (CO) or reduced effective circulating blood volume, leading to altered and
inadequate tissue perfusion, severe enough to induce derangements in normal cellular metabolic function.
- Consequences ?

1 (Lack of blood flow means the cells and organs do not get enough oxygen and nutrients to function properly). The effects of tissue hypoperfusion are initially reversible but can lead to cellular hypoxia which causes: cellular damage. Cell membrane and ion pump dysfunction, Intracellular edema, Leakage of intracellular contents into extracellular space, Inadequate regulation of intracellular pH (due to shifting to anaerobic cellular respiration)

2. Many organs can be damaged as a result. (End organ injury/dysfunction (vital organs)) relative to their impaired metabolic requirements.

Shock requires immediate treatment and can get worse very rapidly , as the Shock is a progressive, rather than a static condition. If not controlled & corrected quickly, it may lead to irreversible shock & death.
- Note: We study medical & not electric shock.

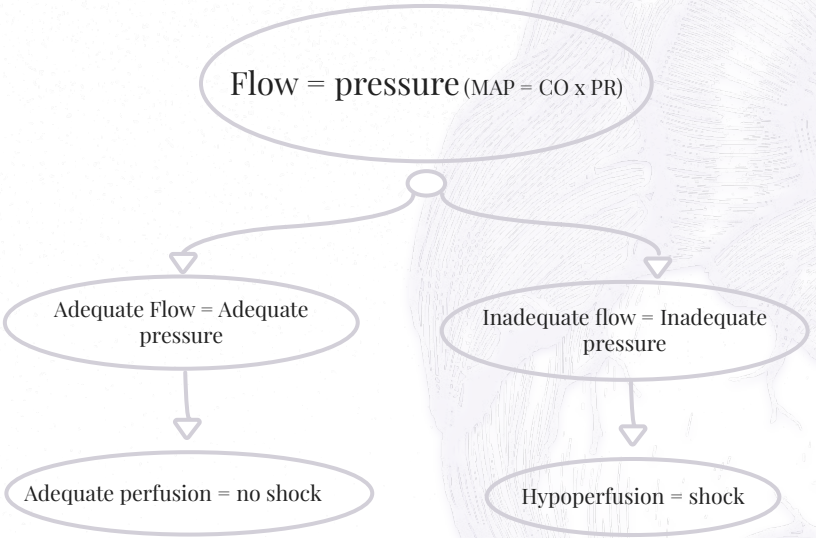
Physiological Causes of Shock

Circulatory shock caused by decreased cardiac output, two types of factors can severely reduce CO :

1. Cardiac abnormalities that decrease the heart to pump blood. These includes MI, toxic heart, severe heart valve dysfunction, heart arrhythmias. Circulatory shock results from diminished cardiac pumping ability is called cardiogenic shock. 85% people die who develop cardiogenic shock.

2. Factors decrease venous return also decrease cardiac output because the heart cannot pump blood that does not flow into it. The common cause of decreased venous return is diminished blood volume, decreased vascular tone (The driving force of pressure toward heart).

General mechanism



Circulatory shock means generalized inadequate blood flow through the body to the extent that the body tissues are damaged, especially because too little oxygen and other nutrients are delivered to the tissue cells. Even the cardiovascular system itself—the heart musculature, walls of the blood vessels, vasomotor system, and other circulatory parts—begins to deteriorate, so the shock, once begun, is prone to become progressively worse.

Male's slide

Important slide

General Mechanism Causing Shock

Inadequate Pump	Inadequate Fluid volume	Inadequate Container
1. Inadequate preload	1. Hypovolemia (decreased body fluid)	1. Excessive dilation
2. Poor contractility	-	2. Inadequate systemic vascular resistance
3. Excessive afterload	-	-
4. Inadequate heart rate	-	-

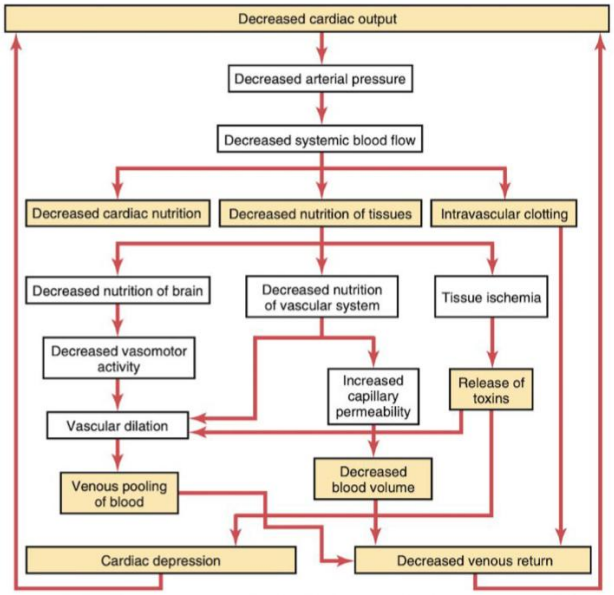


Figure 24-3. Different types of positive feedback that can lead to progression of shock.

(Figure 24-3): shows some of the positive feedbacks that further depress cardiac output in shock, thus causing the shock to become progressive

Shock Pathophysiology

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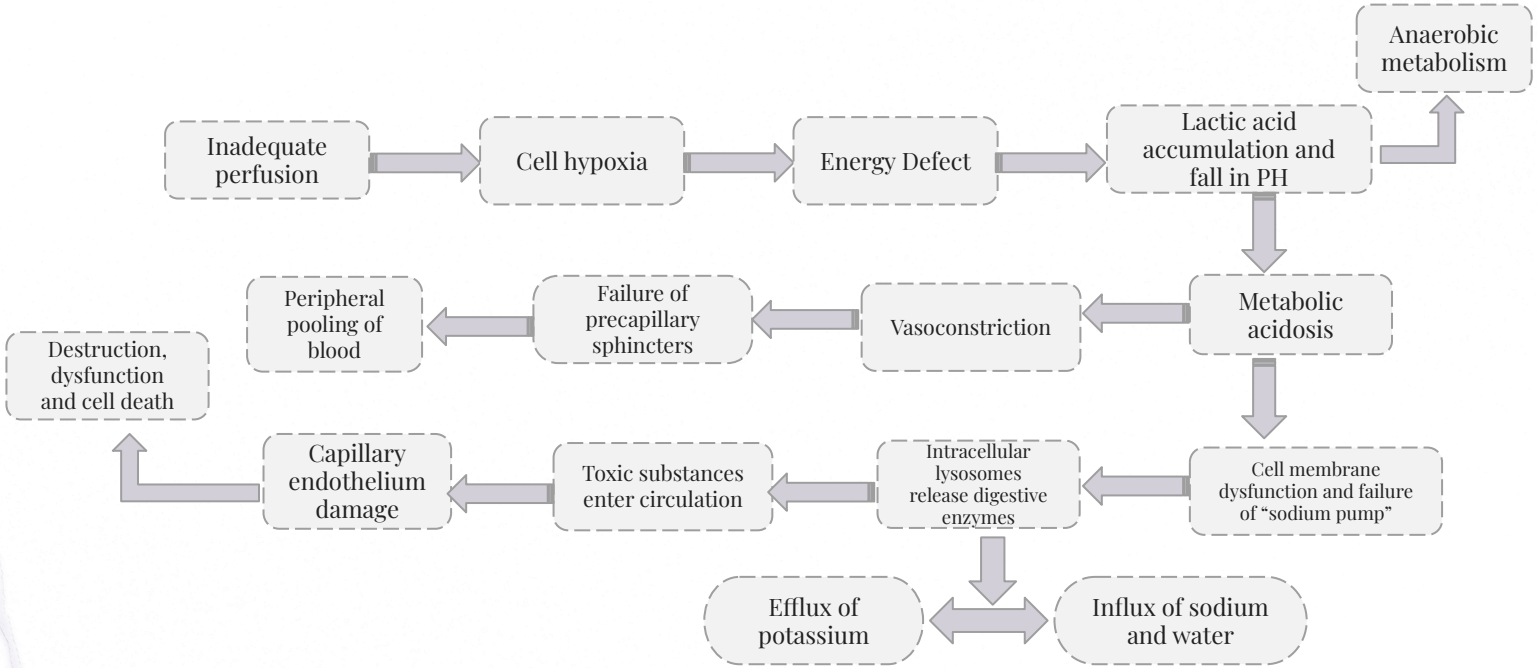


1. Reduce capillary perfusion
2. Inadequate tissue oxygen
3. Shift to anaerobic metabolism
4. Metabolic acidosis
5. Release of free radicals & oxidative stress
6. Tissue damage
7. Apoptosis

Important slide

Types / Classifications of Shock

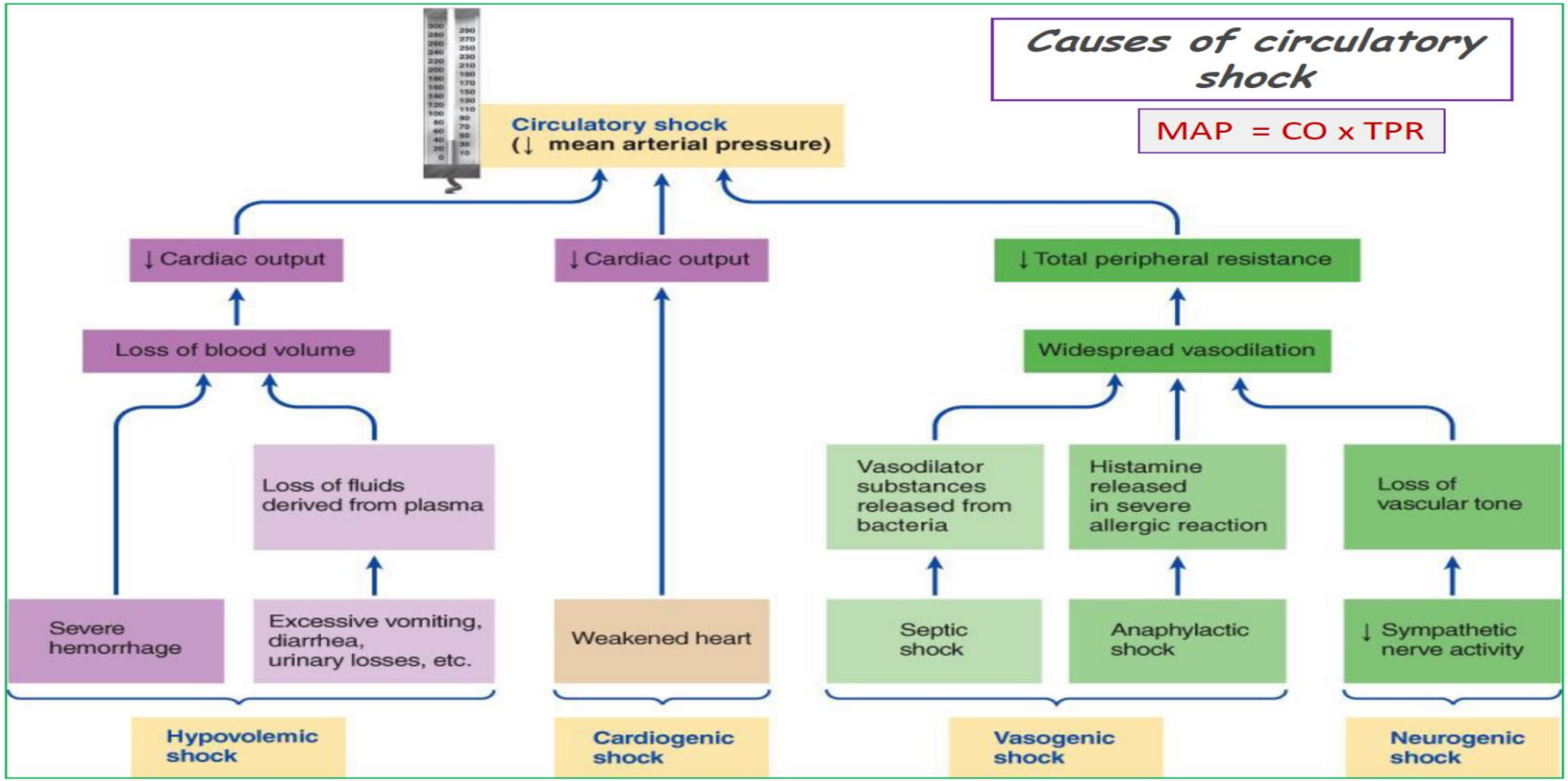
Each one of these will be discussed later on..

Circulatory shock
(↓ mean arterial pressure)

Low output shock (MAP= CO x PR) Inability to produce adequate CO. PR = SVR = TPR			High/Normal output shock (MAP= CO x PR) Distributive Mal-distribution of blood as a result of venous pooling, loss of venous tone, & generalized (widespread) vasodilatation (Reduced total peripheral resistance).	
Hypovolemic (most common) too little blood volume	Cardiogenic (heart produced)	Obstructive	Neurogenic (spinal) (nerve produced)	Vasogenic (vessels produced)
- severe hemorrhage - Excessive vomiting - diarrhea - loss of fluids derived from plasma - loss blood volume - Urinary loss	- Pump failure - weakened heart	- Obstruction in pulmonary or systemic circulation	Behaves like hypovolemic shock, low CO, reduce sympathetic nerve activity -> loss vascular tone	- Septic/toxic/endotoxic massive infection releases vasodilators “cytokines” - Psychogenic - Anaphylactic (Low CO) extensive histamine release in allergy

⅛ of fluid should be lost in order to be considered hypovolemic

Types / Classifications of Shock



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Circulatory Shock

Low Cardiac Output Shock (MAP= CO x PR)

Hypovolemic

- Most common type of shock.
- A life- threatening condition, due to inadequate blood or plasma volume.
- **Etiology (Clinical causes):**
● Excessive/severe/massive volume loss of body fluid (blood/plasma).

● Volume loss of almost 15% (one-fifth) of body fluid (blood/plasma).

● **Blood loss/hemorrhage**: Commonest. Any source of bleeding (internal or external), e.g. Trauma, GI bleeding, ruptured aneurysm, surgery.

● Fluid/plasma loss: Vomiting, diarrhea, burn, excess sweating, dehydration, trauma.

- Physiology, Hemodynamic changes:

● **Less VR (preload)**: leading to decrease in EDV, & stroke Volume.

● **Less 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.

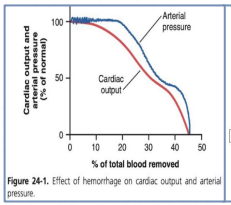
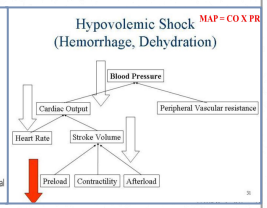


Figure 24-1. Effect of hemorrhage on cardiac output and arterial pressure.



Cardiogenic

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 (Clinical causes):

● Pump failure of the heart, or Deterioration of cardiac function.

● **Myocardial**: Either Acute intrinsic myocardial damage(من نفس العضلة): 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.

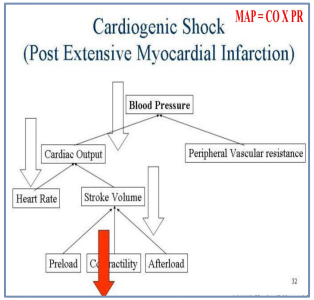
- Physiology, Hemodynamic changes:

● 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%..
- Heart becomes incapable of contracting with sufficient force to pump enough blood into the peripheral arterial tree. Cardiac shock occurs when more than 40% of the left ventricle is infarcted and death occurs in about 85% of patients once they develop cardiac shock.

Obstructive

Female's slide only

- Obstructive shock is a type of shock that occurs when there is a physical obstruction to blood flow, resulting in inadequate perfusion of organs and tissues.

- Etiology (Clinical causes):

● Extracardiac obstructive shock results from an obstruction to the flow in the cardiovascular circuit.

● Causative factors may be located within the pulmonary or systemic circulation, or associated with the heart itself, or caused by trauma surgery.

- 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.
- Cardiac tamponade.
- Congenital or acquired outflow obstructions.

- Physiology: Hemodynamic changes:

● low CO despite normal intravascular volume & myocardial Function.

● low Stroke volume (SV).

● End organ hypoperfusion.

Important slide
It is mix between male and female slide

Classification of Hypovolemic Shock by the Amount of Blood Loss

Parameter	Class I	Class II	Class III	Class IV
			من هنا تعطيه blood	Most dangerous
Blood Loss (ml)	<750	750-1500	1500-2000	>2000
Blood Volume/ Loss %	<15%	15-30%	30-40%	>40%
Pulse Rate (bpm)	<100	>100	>120	>140
Blood Pressure	Normal	Normal	Decreased	Decreased
Pulse Pressure	Normal	Decreased	Decreased	Decreased
Respiratory Rate (bpm)	14 -20	20-30	30-40	>35 / >40
Urine Output (ml/hrs)	>30	20-30	5-15	Negligible
Metal Status	sl.Anxious	Mildly anx	Confused	Lethargic
Fluid	Crystalloid	Crystalloid	Blood	Blood
CNS Symptoms	Normal	Anxious	Confused	Lethargic

سُبْحَانَ اللَّهِ
رَبِّ الْعَالَمِينَ

Human Body Responds to Acute Hemorrhage

Activating four major physiological systems

وَفِي أَنْفُسِكُمْ أَفَلا تُبْصِرُونَ

Hematologic system

1. Injury to the blood vessel causes blood to leak out of the vessel.

2. Constriction of the blood vessels (via local thromboxane A2 release)

3. platelets aggregation and adherence form an immature clot on the bleeding source (platelet plug).

4. The platelet plug provides a temporary seal for the damaged vessel, but it is not strong enough to withstand the force of blood flow for an extended period of time.

5. Activation of coagulation cascade.

6. Fibrin plug, (The damage vessel expose collagen, which subsequently causes fibrin deposition and stabilization of the clot).

Cardiovascular system

1. Increase in heart rate, myocardial contractility and constrict peripheral blood vessels (sympathetic activation).

2. This response occurs secondary to an increase secretion of norepinephrine and a decrease vagal tone (regulated by baroreceptor in the carotid arch, aortic arch, left atrium, and pulmonary vessels).

3. The CVS also respond by redistributing blood to the brain, heart, kidneys and away from skin, muscles, GIT.

Renal system

1. The kidneys respond to hemorrhagic shock by stimulating an increase in renin secretion from juxtaglomerular Apparatus.

2. Renin is an enzyme that is released to the blood to act on a protein called angiotensinogen that was made by the liver to convert it into angiotensin I.

3. Angiotensin I is then converted into angiotensin II in the lungs by ACE.

- Ang II has two main effects:

- Vasoconstriction which increases blood pressure.

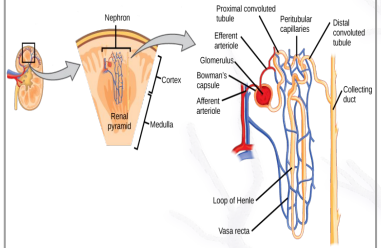
- Stimulate aldosterone secretion which retain salt and water. Thus, increases thirst and the desire to salt.

Neuroendocrine system

1. Decreased blood pressure causes an increase in circulating ADH (antidiuretic hormone).

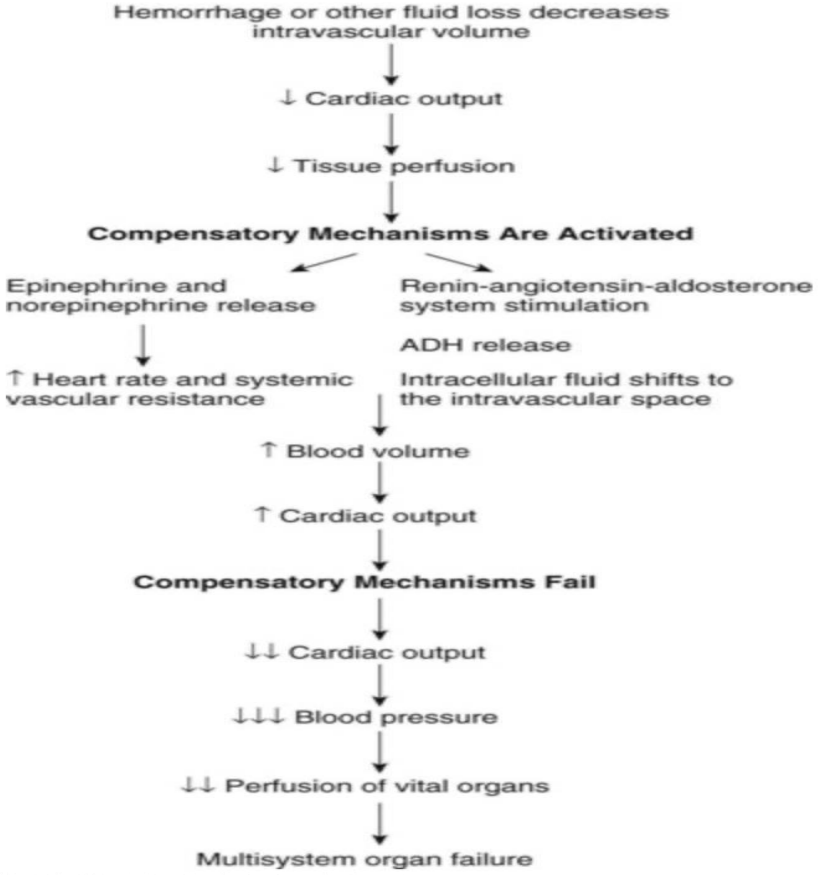
2. ADH is released in response to a decrease in blood pressure (detected by baroreceptor) and a decrease in Na+ concentrations.

3. ADH increases the reabsorption of water and salt (NaCl) by the distal tubule and collecting ducts.



Extra Photo

Human Body Responds to Acute Hemorrhage

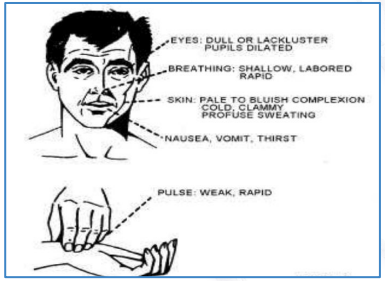


Signs and Symptoms/clinical features

Hypovolemic shock	Cardiogenic shock Female slides only
Tachypnea (rapid respiration/increased resp.rate) (sensed by chemoreceptors in compensation to hypoxia)	Similar signs & symptoms to that of hypovolemic shock.
-Tachycardia (increased HR) (sensed by baroreceptors in compensation to decrease MAP) - Rapid, weak, & thready pulse (140/min)	Laboratory findings: Increase troponin I & T.
Cold clammy pale skin, decreased capillary refill (caused by vasoconstriction due to potent sympathetic stimulation / due to hypoperfusion), pupillary dilation	Congestion of lungs & viscera (Chest X-Ray -CXR) : • Interstitial pulmonary edema. • Alveolar edema. • Cardiomegaly
Sweating, oliguria (low urine output)/ Anuria (no urine output)	Prognosis: 70% mortality
Intense/Increased thirst	-
- Blood test: lactic acidosis , increased anion gap. - Metabolic acidosis symptoms (confusion, vomiting, loss of appetite	-
Sustained/generalized hypotension (<= 80-85/40 mmHg for 30 min)	-
Restlessness due to hypoperfusion	-
Loss of consciousness	-
Bradycardia and death (end stage)	-

Treatment of hypovolemic shock:
Early, adequate hemodynamic support is critical to prevent worsening as organ dysfunction and failure. By fluid replacement or blood transfusion & treat the underlying cause.

For Management of hypovolemic shock there is The VIP rule:
- Ventilate (oxygen administration)
- Infuse (fluid resuscitation)
- Pump (administration of vasoactive agents)



Hypovolemic shock

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 are either Neurogenic (Spinal) or Vasogenic {Septic or non-septic (anaphylactic or psychogenic or adrenal insufficiency)}

Etiology (Clinical causes): Increase venous capacitance.

- It occurs as a result of systemic vasodilation 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.
- Low Peripheral resistance (PR) & venous return (VR).

Distributive Shock

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

Vasogenic shock

Septic/Toxic/Endotoxic Shock

- Most common in emergency.

- Dysregulation of the immune response 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).
- Hyperdynamic state.

- **Signs and Symptoms:**

- Patient flushed & Warm due to his hyperdynamic state.

Anaphylactic Shock

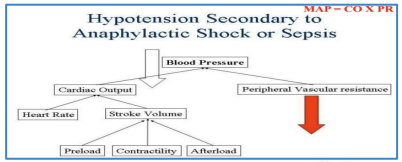
- Most common in emergency.

- 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 hypoperfused.
- Loss of consciousness.

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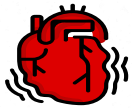
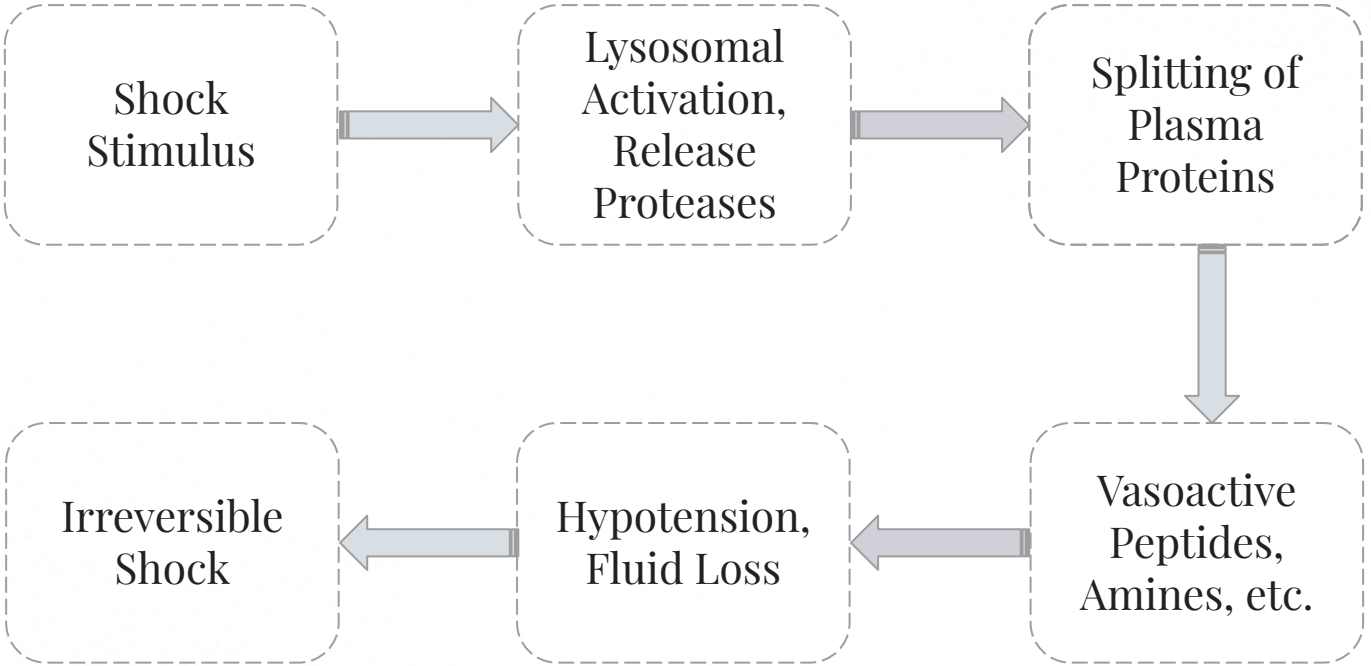
Stages of shock



Circulatory shock change with different degrees of severity, shock is divided into		
Reversible shock / Non-progressive (Compensated)	**Progressive**	**Irreversible shock (uncompensated)**
-Reflex compensatory mechanisms are activated (neurohormonal activation) - Defense mechanisms are successful in maintaining perfusion. -Full recovery can be caused/changes can be reversed by normal circulatory compensatory mechanisms (without help from outside therapy) or by treatment	- Defense mechanisms begin to fall/Body begins to loss its ability to compensate “needs urgent Therapy” - Inadequate perfusion begins/tissue hypoperfusion with worsening of circulatory & metabolic imbalance including lactic acidosis - Multi-organ failure.	- Complete failure of compensatory mechanisms. - Shock progressed to an extent that all forms of known therapy are inadequate to save the life, even though, for the moment, the person is still alive. -Severe cell and tissue injury result in multi-system organ failure and may end with death.

Female's slide

Possible Mechanisms Leading to Development of Irreversible Shock



Metabolic Changes & Cellular Response to Shock

1. Reduce capillary perfusion	2. After 3-5 hours of shock	3. Granulocytes Accumulation
- 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). - Breakdown of Lysosomes, nuclear membranes & mitochondria.	- Precapillary sphincters dilate, venules still constricted. - Blood stagnation in capillaries. - Hypoxia continue & fluid leaves to extravascular compartment. - Further reduction in circulating blood volume.	- Granulocytes will accumulate at the Injured vessels. -Free radical release. - Further tissue damage.

Metabolic Changes & Cellular Response to Shock

- Damage in GIT mucosa → Failure of interstitial barrier allowing bacteria into circulation..
- Damage in Kidneys → Oliguric renal failure (↑ Tubular reabsorption & ↓Secretion).
- Cerebral ischemia → Depression of VMC → vasodilation + ↓ HR & further decrease in BP.
- Myocardial ischemia → Myocardial damage + depressed contractility, leading to more shock & acidosis.
- Respiratory distress syndrome occurs due to capillary endothelial cells leak & damage to alveolar epithelial cells, with release of cytokines.
- Multiple organ failure & death.

Compensatory Mechanisms to Shock

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

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:**

   1. Activation of Renin-Angiotensin Aldosterone System:
   - Angiotensin II & III: Are powerful vasoconstrictors.
   - Aldosterone: will lead to Na+ & water retention.

   2. Stimulation of ADH (vasopressin):
   - Water retention, vasoconstriction, with thirst and drinking stimulation.

3. **Synthesis of Plasma Proteins: after 3-4 days**

4. **Fluid- shift mechanism.**

Summary :

1. ↑HR & myocardial contractility → ↑CO
2. V.C → ↑Preload & filling pressure.
3. ↑Blood volume.
4. ↑Vascular compliance and restoration of tissue perfusion

Normal Forces at The Arterial & Venous Ends of The capillary

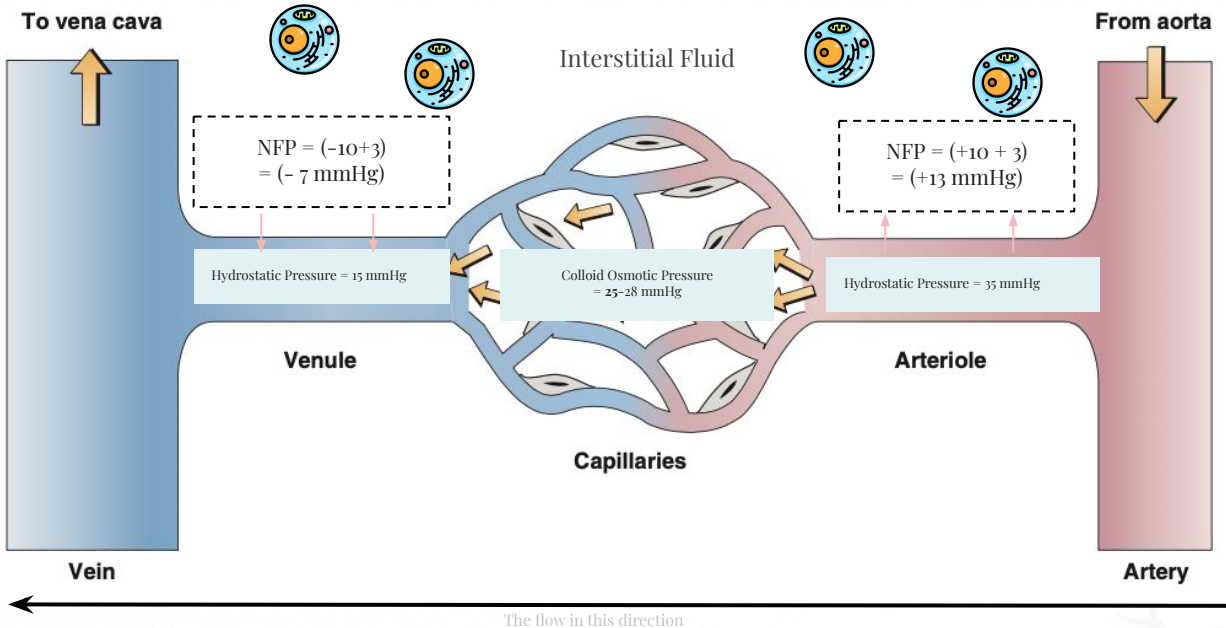
Note that this is the normal situation

Tissue Hydrostatic Pressure= 0 mmHg

Negative Interstitial Free Fluid Pressure = 3 mmHg

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.



The flow in this direction

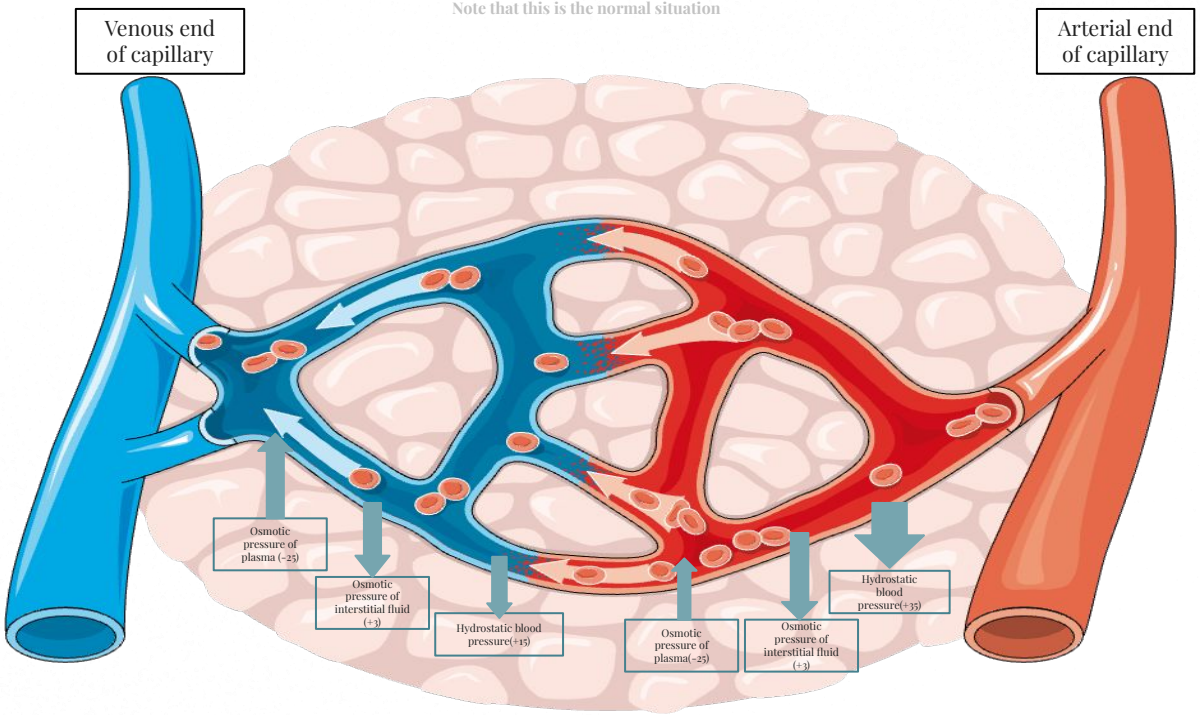
Arterial end

- 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 interstitial space.

Female's slide

Normal Forces at The Arterial & Venous Ends of The capillary

Note that this is the normal situation



(15+3)-25=-7 mmHg
Net absorption pressure

(35+3)-25=+13 mmHg
Net filtration pressure

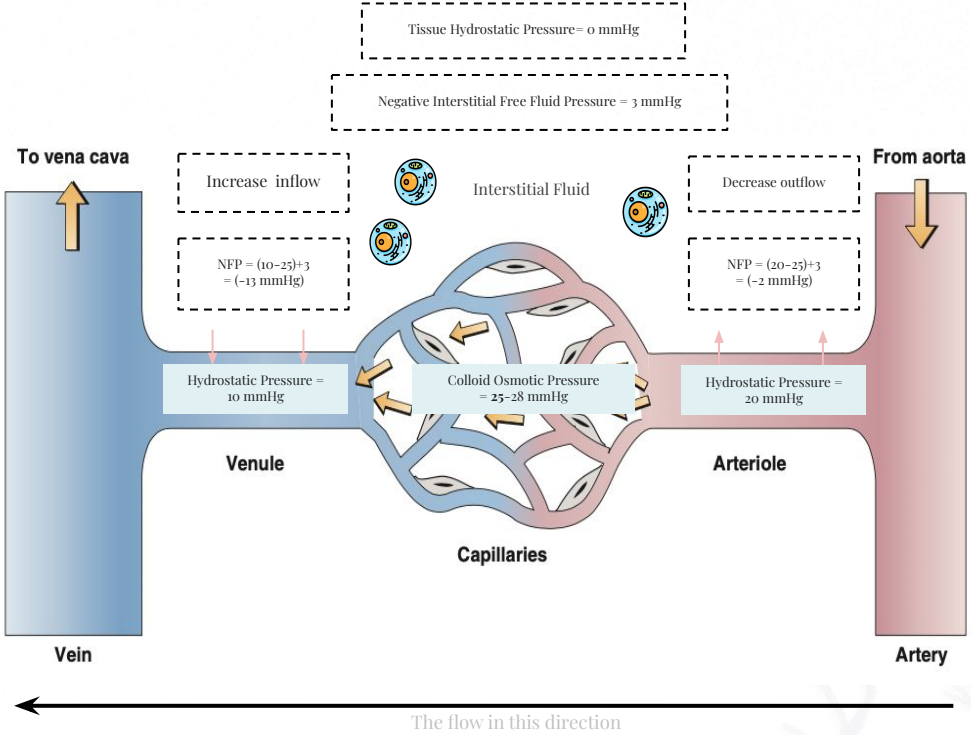
Fluid- Shift Mechanism In Shock

This is the abnormal situation

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. (filtration)
- The fluid return from the extracellular space to the capillary increases. (Reabsorption)

In compensating to shock situation, this will help to increase the blood volume in order to restore the BP.



Cause of shock summary

Type	Causes	Symptoms and Signs
Hypovolemic shock	Bleeding (internal/external), dehydration (severe vomiting, severe diarrhea), plasma loss (as in burns) → low blood volume → decreased cardiac output → hypotension	hypotension; weak but rapid pulse; cool,clammy skin; rapid, shallow breathing;anxiety, altered mental state
Cardiogenic shock	Heart problems (e.g., myocardial infarction, heart failure; cardiac dysrhythmias) → decreased contractility → decrease in stroke volume → decreased cardiac output → hypotension	as for hypovolemic shock + distended jugular veins & may be absent pulse
Obstructive shock	Circulatory obstruction (e.g., constrictive pericarditis, cardiac tamponade, tension pneumothorax, pulmonary embolism → reduced blood flow to lungs → decreased cardiac output → hypotension	As for hypovolemic shock + distended jugular veins & pulsus paradoxus (in cardiac tamponade).
Distributive shock Vasogenic Low-resistance shock	Septic shock: infection → release of bacterial toxins → activation of NOS in macrophages → production of NO→ vasodilation → decreased vascular resistance → hypotension	hypotension; fever; warm, sweaty skin
	Anaphylactic shock: allergy (release of histamine↠ V.D → decreased vascular resistance→ hypotension)	skin eruptions; breathlessness, coughing; localized edema; weak, rapid pulse
	Neurogenic shock: spinal injury→ loss of autonomic and motor reflexes → reduction of peripheral vasomotor tone → vasodilation→ decrease in peripheral vascular resistance→ hypotension	as for hypovolemic except dry skin

Check here for our summary
Highly recommended !!!!!

Sorry but if you will not cheack it راحت عليك المليون

MCQs:

Answers

For more question check our summary file!

1/C
2/B
3/D

1 **Which stage of shock associated with complete failure of compensatory mechanism?**

A	Progressive stage	B	Reversible stage	C	Irreversible stage	D	Initial non progressive stage

2 **During shock, what happens to hydrostatic & oncotic pressure respectively?**

A	Increases, increases	B	Decreases, constant	C	Constant, decreases	D	Decreases, increases

3 **Which of the following clinical signs is not typical for classic presentation of shock?**

A	Cool extremities	B	Weak pulse	C	Tachypnea	D	Systemic hypertension

MCQs:

Answers

For more question check our summary file!

4/A
5/D
6/A

4 **Which of the following leads to the progression of a shock ?**

A	Cardiotoxins	B	Negative feedback mechanisms	C	Increase VR	D	Increased CO

5 **Which of the following is an indicator of poor tissue perfusion in hypovolemic shock?**

A	Hyperthermia	B	Decreased respiratory rate	C	Bradycardia	D	Oliguria

6 **Which of the following raises arterial pressure by causing vasoconstriction ?**

A	Ang II	B	Aldosterone	C	ADH	D	Renin

SAQ

What are the consequences of shock?

-Cellular hypoxia due to failure to deliver oxygen to the tissues, leading to cellular damage.
-End organ injury/ dysfunction relative to their impaired metabolic requirements.

Explain pathophysiology of shock

Slide 5

Treatment of hypovolemic shock is

fluid replacement or blood transfusion & treat the underlying cause and VIP rule

What is the cardiac abnormalities that decrease the heart to pump blood.

includes MI, toxic heart, severe heart valve dysfunction, heart arrhythmias.

Finally you have arrived , we have been waiting for you !!

Meet our team !

Team leaders

Rimaz Alhammad

Noreen Almaraba

Rayan Alshehri

Omar Albaqami

Aljoharah Alyahya

Heroes of the lecture :

Nasser Alabdulsalam

Haya Alateeq

Did you like the lecture ? we mean our work :)

Contact with us! physiology.444ksu@gmail.com