Shock

Physiology Team 439

MED439
KING SAUD UNIVERSITY

Black: in male / female slides

Red : important

Pink: in female slides only

Blue: in male slides only

Green: notes

Gray: extra information

Introductory video (recommended before reading)

Editing File

Objectives

- To define Shock.
- To describe different types and causes 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.
- To understand the Clinical features and management of shock.

Introduction

O2 delivery < O2 demand

Shock (Circulatory shock)

It's an acute medical emergency situation in which there is acute circulatory **failure** leading to inadequate tissue perfusion (blood flow) & end organ injury (medical and not electrical shock). Inadequate flow = inadequate pressure = hypoperfusion= shock

In other words: it's a life-threatening clinical state categorized by body wide deficiency of blood supply causing oxygen deprivation, build up of waste product, and eventual organ failure if untreated.

Features:

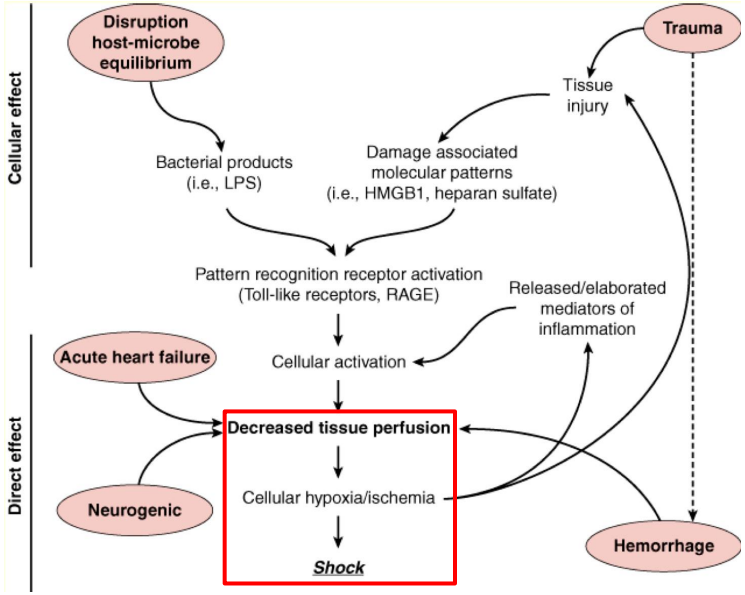
- Main feature is **loss of fluid** from circulating blood volume (hypovolemic shock, only one type of shock), so that the circulatory system is unable to provide adequate circulation & tissue perfusion
- Once the shock has begun, its prone to be progressively worse. Its not a static condition مايقعد الانسان على وضعه
- If not controlled and corrected quickly it may lead to **irreversible** shock and death

Male slide:

Shock usually results from inadequate cardiac output. Two types of factors can severely reduce cardiac output:

1. **Cardiac abnormalities** that decrease the heart to pump blood: These includes MI, toxic heart, severe heart valve dysfunction, heart arrhythmias.
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

Circulatory shock results from *diminished cardiac pumping ability* is called cardiogenic shock. 85% people who develop cardiogenic shock unfortunately die.

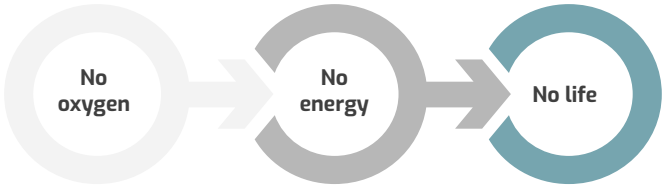


Introduction

O2 delivery < O2 demand

- Any type of shock that leads inadequate tissue perfusion will result in failure to deliver oxygen to the tissues and vital organs relative to their metabolic requirements, leading to organ dysfunction and cellular damage

بحكم ان الخلايا تموت اذا ما تواجد الاوكسجين

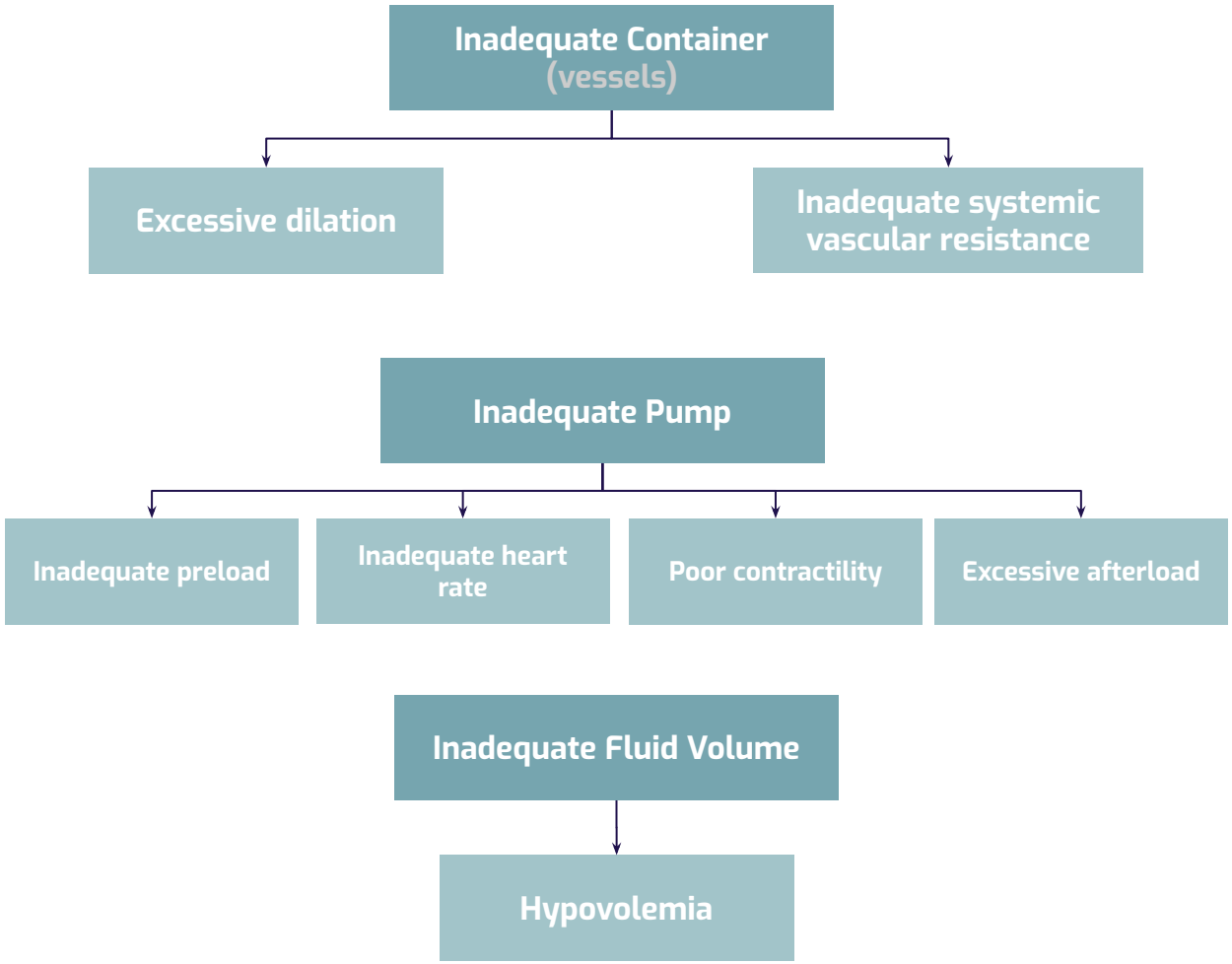


Cells must get their needed energy to stay alive.

Mechanism of tissue destruction:

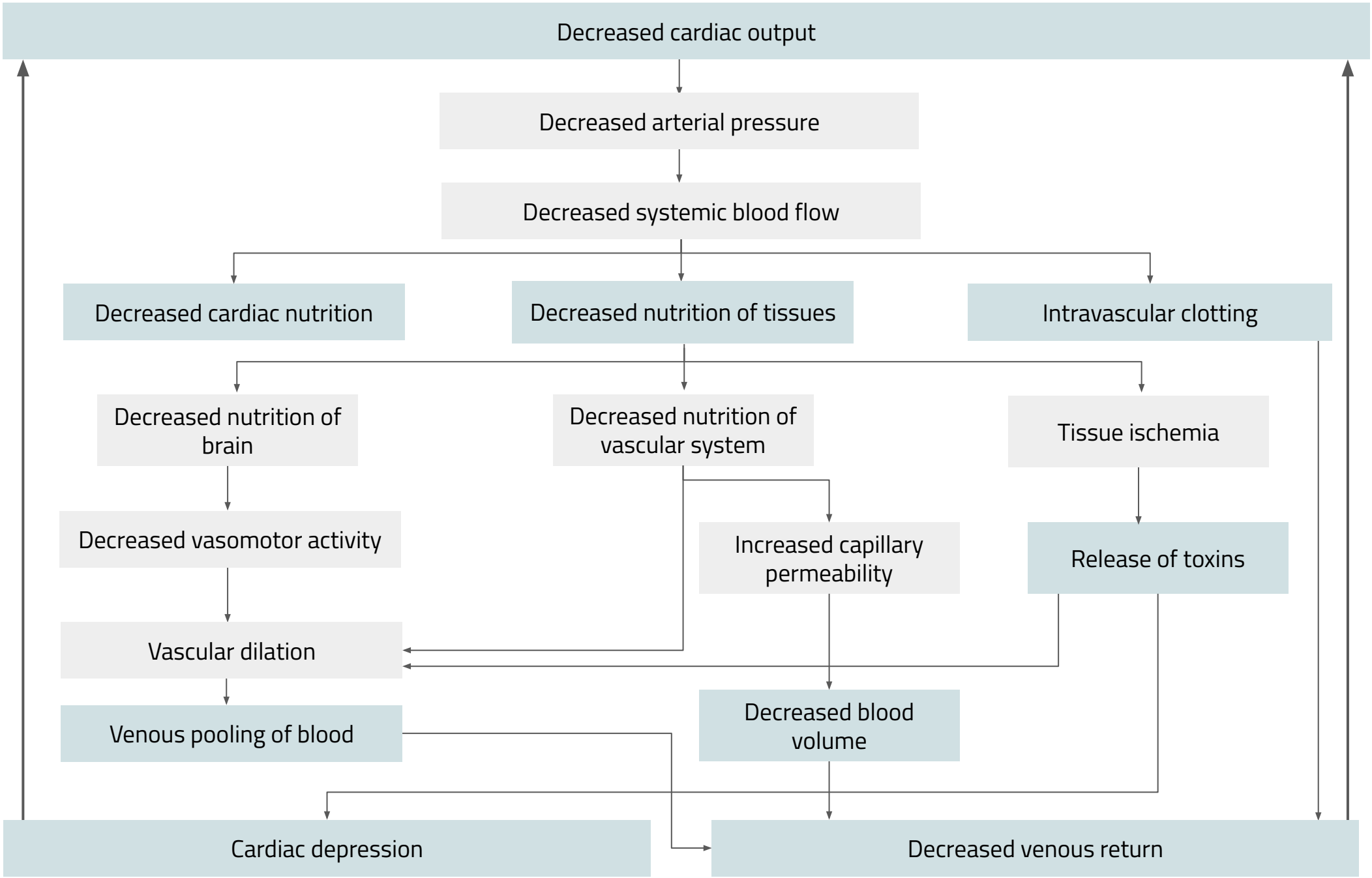
Inadequate perfusion → **cell hypoxia** → **energy deficit** → lactic acid accumulation & fall in pH (Anaerobic metabolism) → metabolic acidosis (peripheral pooling of blood) → cell membrane dysfunction & failure of sodium pump → intracellular lysosomes release digestive enzymes → toxic substances enter circulation → capillary endothelium damage → **further destruction, dysfunction & cell death**

Male slides



- **Preload** is the end-diastolic volume (EDV) at the beginning of systole. Directly allied to degree of stretch It is related to ventricular filling.
- **Afterload** is the ventricular pressure at the end of systole. Force against which heart contract to eject the blood.

GENERAL MECHANISM



- **MAP:** Mean arterial pressure.
- **CO:** Cardiac output.
- **PR:** Peripheral resistance.

Overview: Types of Shock

Circulatory shock

MAP = CO X PR — **Low output shock (Scarce perfusion)**

MAP = CO X PR — **High/Normal output shock (inadequate perfusion)**

1

Hypovolemic
(Most common)
Too little blood **volume**

Note: in Hypovolemic
Lost fluid might be:

1) Blood, as in hemorrhage

2)Plasma, as seen in burns
علشان كذا فالاماكن المحترقه القفاعات تكون صفراء وهذا دليل انها طلعت من البلازما

3)Serum, loss water as in dehydration occur in patients with diarrhea & vomiting

2

Cardiogenic
(Most dangerous)
Pump failure (Problem in heart function)

Extracardiac: massive pulmonary embolism, tension pneumothorax, constrictive pericarditis, cardiac tamponade from pericardial injury.

Intracardiac: massive myocardial infarction, dysrhythmias, acute valve derangement, myocardiopathy, tumor, contusion, aneurysm, infection.

Mixed intra-extracardiac: penetrating or blunt injury to heart & pericardium

Male slides

3

Obstructive
Obstruction in pulmonary / systemic circulation

4

Distributive
Mal-distribution of blood as a result of venous pooling, loss of venous tone, & generalized vasodilation

(1)Neurogenic (spinal)
Behaves like hypovolemic shock, Low CO
(Severe fall in both CO & PR)

Vasogenic (Inflammatory Shock)

Nonseptic

(1) Psychogenic

Anaphylactic (Allergen)
severe fall in both CO & PR

Examples of Non-septic shock
Massive multiple organs blunt trauma, Hyperacute crush injury, extensive thermal injury (burns, chemical, radiation), acute necrotising pancreatitis, ischemic necrosis gangrene, crush syndrome, I-R phenomenon, complicated intestinal obstruction.

Male slides

Septic/Toxic (Infection)

Bacterial substance cause severe vasodilation > severe hypotension > shock

(1)مثل اللي يغمى عليهم بعد تلقي الأخبار المؤلمة، يغمى عليهم ليه؟ لأن السمباثاتك طفى بعد ما اشتغل بقوة، فمسك المهمة البارا سمباثتك لحاله
Vagus nerve (which affect the heart + peripheral vasculature mainly) which cause:
Vasodilation > blood pooling in legs > مايروح الدم للقلب من الأساس this condition is called "Vasovagal syncope" Learn more about it: click here

Hypovolemic Shock

Low CO

MAP = CO X PR

- Most common type of shock.
- A life threatening condition.
- Causes insufficient perfusion (End organ hypoperfusion) which can leads to **organ failure**.
- Requires immediate emergency medical attention.

Inadequate pressure (low bp)= hypoperfusion = ↓CO = ↓Stroke volume = ↓**Preload**

Hemodynamic Changes

- The heart is unable to pump sufficient amounts to body parts.
- Reduced venous Return (Preload), leading to reduction in EDV and Stroke volume.
- End organ hypoperfusion.
- Low CO.

Causes

Severe loss of body fluid (blood/plasma), more than 15% due to either:

1. **Hemorrhage (most common):**
   Could be internal or external bleeding. e.g. Trauma, GI bleeding, Ruptured aneurysm
2. **Fluid/plasma loss:** Through vomiting, Diarrhea, Burn, Excess sweating, **Dehydration**, Trauma.
3. **Surgery**

Clinical presentation

- **Tachycardia:** (is a reflex mechanism of hypotension > tachycardia means **more pulses** > means more blood need to oxygenate by the lung > increase **respiratory rate)** Sensed by Baroreceptors in compensation for the↓MAP.
- **Rapid, weak, thready pulse (140/min)**
- **Tachypnea** (rapid respiration):دايم معدل التنفس يقلد معدل pulse في الزيادة و النقص
  Sensed by Chemoreceptors (respiration = O2, CO2 =chemoreceptor) in compensation to hypoxia.
- **Sustained hypotension** (85/40 mmHg for 30 minutes) (due to low volume).
- **Cold, pale skin due to hypoperfusion** to the skin**/vasoconstriction due to increased sympathetic stimulation.**
- **Sweating, dull and dilated pupils.**
- **Mental status changes** (confusion - nausea).
- **Oliguria** (low urine output)**/ Anuria** (no urine output).
- **Restlessness** due to hypoperfusion of the brain.
- **Intense thirst.**
- **Blood test: Lactic acidosis.** (due to absence of oxygen to tissues and the switching to anaerobic metabolism)

Classes of Hypovolemic Shock

لأي درجة الشخص قاعد يخسر فلودز؟ صنفوها لاربع اقسام

Parameter	Class I	Class II	Class III	Class IV
Blood loss (ml)	<750	750-1500	1500-2000	>2000
Blood loss (%)	<15 Least blood loss	15-30	30-40	>40 Greatest blood loss
Pulse rate (bpm)	<100	>100	>120	>140
Blood pressure	Normal	Normal/decreased	**Decreased**	**Decreased**
Pulse pressure	Normal	**Decreased**	**Decreased**	**Decreased**
Respiratory rate	14-20	20-30	30-40	>40 >35
Urine output (ml/hr)	>30	20-30	5-15	Negligible
Mental status	Normal/Sl. Anxious	Mildly Anxious	**Confused**	**Lethargic**
Fluid اللي نعطيه المريض	Crystalloid A crystalloid fluid is an aqueous solution of mineral salts and other small, water-soluble molecules, isotonic to human plasma.		**Blood**	

Hypovolemic Shock

The human body responds to acute hemorrhage by activating four major physiological systems:

Hematologic

- Activating the coagulation cascade.
- Contracting the bleeding vessels (via local thromboxane A2 release).
- Platelets formation of immature clot.
- Damaged vessel exposes collagen, which causes fibrin deposition (and stabilization of clot)

Cardiovascular

- ↑HR, ↑Contractility
- Constricted peripheral blood vessels (↑Norepinephrine + ↓Vagal activity) Which are regulated by the Baroreceptors in the carotid arch, aortic arch, left atrium, and pulmonary vessels)
- Redistribution of blood **to** the brain, heart, kidneys and **away from** skin, muscle, and GIT.

Renal

- ↑Renin secretion from juxtaglomerular apparatus. which then leads to the formation of **angiotensin II**. And it has two main effects:

- Vasoconstriction of arteriolar smooth muscle.
- Secretion of aldosterone by the adrenal cortex (causes water and salt retention).

(both of which help reverse hypovolemic shock).

Renin is released when a fall in arterial blood pressure is found.

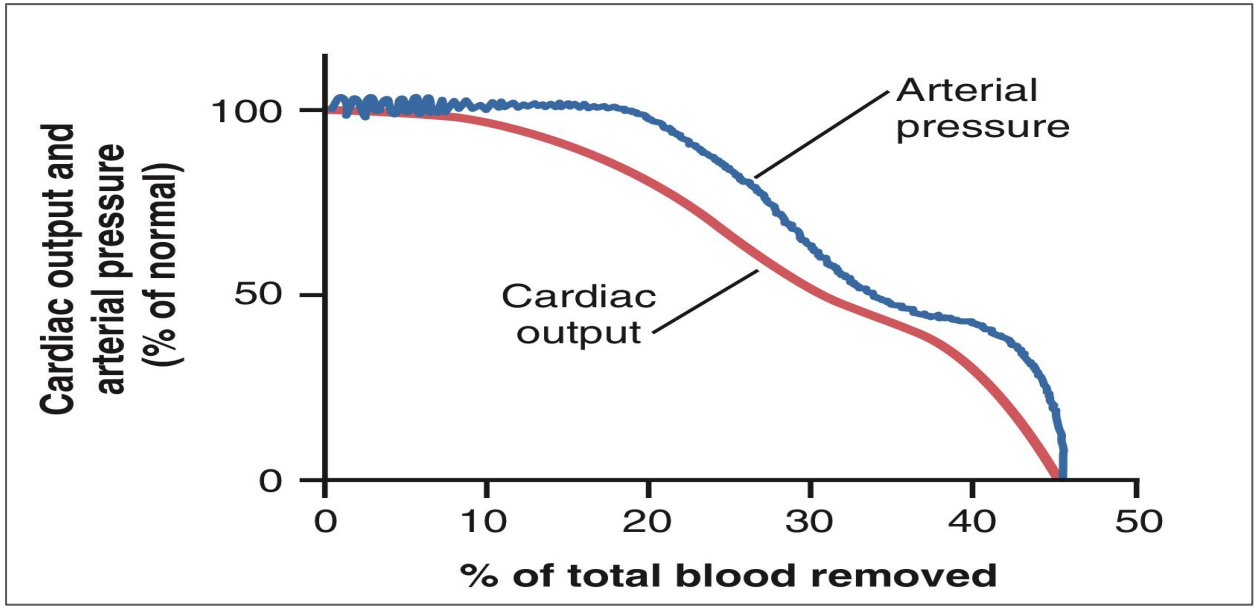
[1] Neuroendocrine

- Antidiuretic hormone (ADH) due to decrease in BP (as detected by Baroreceptors) and a decrease in sodium concentration.
- ADH causes retention of water and salt by the distal tubule and the collecting ducts.

➔ **If the compensatory mechanisms fails, a decrease CO and blood pressure occurs which causes a decrease in perfusion of vital organs and eventually multisystem organ failure.**

(1) Extra: There are a receptor called atrial receptor, which stimulated by increase pressure , make these receptors send impulses, so when the pressure is low like in shock condition, its impulses decrease > stimulate ADH to release,Impulses يعني ADH يتحفز اذا اختفت

Effect of hemorrhage on cardiac output and arterial pressure



-Guyton

The figure shows, the effects on **cardiac output** and **arterial pressure** of removing blood from circulatory system over a period of about 30 minutes. About 10% of the total blood volume can be removed with almost no effect on either arterial pressure of cardiac output. But greater blood loss usually diminishes the cardiac output first and later the arterial pressure, both of which fall to zero when about 40-45% if the total blood volume has been removed.

-Dr notes

Up to 10% loss of blood, **both** arterial pressure and cardiac output experience **no major change**. But around **20% arterial pressure** starts to fall, and around **10% cardiac output** starts to fall and **both** start to drop to zero around 40%.

Cardiogenic Shock Low CO

MAP = CO X PR

- Pump Failure: cardiac muscle is **unable to pump (contract) with sufficient force to pump adequate blood flow** to the vital organs & body parts in presence of normal blood volume.
- Has a high mortality rate (60-90%).
- Is associated with loss of 40%> of LV myocardial function = pump failure.

Inadequate pressure (low bp)= hypoperfusion = ↓CO = ↓Stroke volume = ↓Contractility

Hemodynamic Changes

- Low CO with reduced Stroke volume.
- Elevated Left Ventricular End Diastolic filling Pressure LVEDP (right/left/or both).
- Decreased coronary perfusion(supplyاذا القلب مب قادر يضخ فحتى الكورونواري ماراح يجيها دم فبيأدي ان القلب ما يجيه,) leading to ischemia & further myocardial dysfunction.
- Persistent hypotension (systolic pressure <80mmHg / MAP 30mmHg below baseline)(either/or).
- End organ hypoperfusion.

Causes (deterioration of cardiac function):

- **Myocardial:**
  Acute myocardial infarction (most common),
  Myocarditis, Cardiomyopathy, heart failure.
- **Mechanical:**
  Acute valvular dysfunction, e.g. Papillary muscle rupture post-MI.
- **Arrhythmogenic:**
  Sustained arrhythmias, e.g. Heart block (impulses that originate from the upper chamber cannot get down to lower chambers), Ventricular tachycardia. (إذا كانت severe
  فما فيه وقت يعبي الدم، فبالتالي وش يضخ اذا مافيه شيء؟)
- **Obstructive: (obstructive shock could also lead to cardiogenic shock)**
  Pulmonary embolism, Cardiac tamponade.(accumulation of fluid in the pericardial space, resulting in reduced ventricular filling يعني القلب ما تعبى لأنه مضغوط بالسوائل حوله)

Myocarditis affect the heart muscle and heart's electrical system, reducing the heart's ability to pump and causing rapid or abnormal heart rhythms (arrhythmias).

Clinical Presentations

- **Similar signs and symptoms to that of hypovolemic shock**
- **Congestion of lungs & viscera can be seen on chest X-ray as:**
  - Interstitial pulmonary edema
  - Alveolar edema
  - Cardiomegaly
- **Distended jugular veins & may be absent pulse.**

Obstructive Shock

Low CO

MAP = CO X PR

Hemodynamic Changes

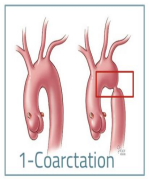
- Cardiac output is reduced despite normal intravascular volume & myocardial function.
- Decreased Stroke volume.
- End organ hypoperfusion.

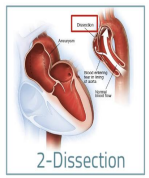
Causes

- Causative factors may be located within the pulmonary or systemic circulation or associated with the heart itself or caused by trauma.
- Extracardiac obstructive shock results from an obstruction to flow in the cardiovascular circuit

Examples:

1. **Obstruction of venous return:**
   - e.g. Vena Cava Syndrome (usually neoplasms).
2. **Compression of the heart:**
   - e.g. hemorrhagic pericarditis → cardiac tamponade (blood/fluid accumulation in the pericardial)
3. **Obstruction of outflow of the heart:**
   - Aortic coarctation[1] ,dissection[2]
   - Pulmonary or systemic hypertension
   - Massive pulmonary embolism
   - congenital or acquired outflow obstructions
   - Tension pneumothorax, why?


1-Coarctation


2-Dissection

معد يرقى الدم من الجسم للقلب< Pressure graident الضغط يكون موجب فما فيه < Pleural cavity يدخل هواء لل

Clinical Presentations

- **Similar to hypovolemic shock.**
- **Jugular venous distension** (congestion due to increased atrial pressure).
- **Pulsus paradoxus (in cardiac tamponade).**

Pulsus paradoxus:
is an abnormally **large decrease in stroke volume**, systolic blood pressure and pulse wave amplitude during inspiration.
The normal fall in pressure is less than 10 mmHg. When the drop is more than 10 mmHg, it is referred to as pulsus paradoxus.

Distributive Shock

High/normal CO

MAP = CO X PR

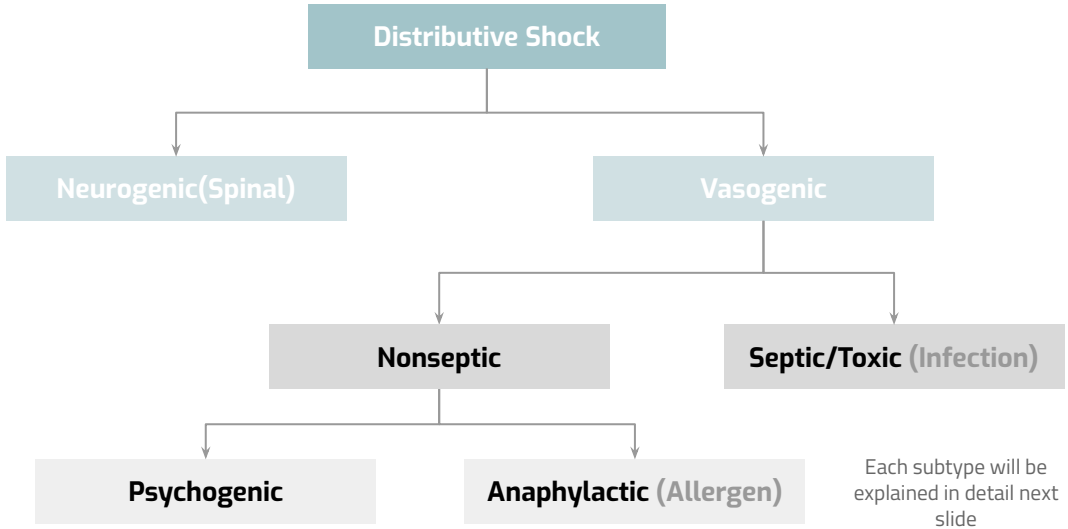
- Also known as **Vasodilatory shock** or **Low-resistance shock.**
- There will be an inappropriate distribution of blood flow which will lead to a decrease in blood flow to the vital organs resulting in their damage.
- Cardiac output is mostly normal or elevated

Causes:

1. It occurs as a result of systemic vasodilation which is caused by loss of vascular resistance (tone).
2. It can be also caused by leakage of fluid from capillaries into the surrounding tissues (capillary leak syndrome)
3. It can be caused by Sepsis, Allergic reactions, Adrenal insufficiency, Trauma, Drug overdose & toxicity.

Extra: "why adrenal insufficiency can cause distributive shock?"
The adrenal gland produces **cortisol (which increase peripheral resistance>**
يعني مفروض تسوي هايبر تنشن؟فإذا الغدة وقفت انتاجهم بيصير هايبوتنشن
and aldosterone and others, **glucocorticoid** deficiency may also contribute to hypotension by decreasing vascular responsiveness to angiotensin II + vasoconstrictive hormones + reducing the synthesis of renin substrate + increase the production and effect of prostacyclin and other "vasodilatory hormones

Inadequate pressure (low bp)= hypoperfusion = ↓**Peripheral Vascular Resistance**



Each subtype will be explained in detail next slide

Neurogenic Shock

MAP = CO X PR

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 (↓CO+↓PR⟶severe drop in MAP).
- Blood volume remains normal.
- Cardiac output is severely reduced as blood is pooled in the peripheral veins (Capacity of blood ↑, & venous return ↓).
- Behaves like **Hypovolemic shock.**

Vasogenic Shock

MAP = CO X PR

1

Septic / Toxic / Endotoxic Shock
(Infection)

1. Most common in emergency.
2. Dysregulation of the immune response to infection that leads to systemic cytokine release & resultant vasodilation & fluid leak from capillaries.
3. **Bacterial Endotoxin** triggers peripheral vasodilation & endothelial injury.
4. Inflammatory cytokines may also cause some cardiac dysfunction.
5. Hyperdynamic state.
6. Clinical Presentation: causes patient to become hypotension, fever, sweaty skin and **Flushed** & **Warm** due to hyper-dynamic state.

2

Nonseptic Shock

Anaphylactic Shock

1. Most common in emergency.
2. Exposure to an antigen resulting in a massive & generalized allergic reaction.
3. IgE- Mediated hypersensitivity (type I hypersensitivity).
4. Histamine triggers systemic peripheral vasodilation & increase capillary permeability leakage.
5. Can lead to low cardiac output distributive Shock. (both CO+PR are affected)
6. Clinical Presentation: Skin eruptions, Breathlessness, Coughing, Localized edema, rapid pulse.

Psychogenic Shock

1. Simple fainting (syncope) as a result of **stress, pain, or fright.**
2. Dilatation of blood vessels.
3. Blood pressure falls.
4. Increase heart rate.
5. Brain becomes hypoperfused .
6. Loss of consciousness.
7. Clinical Presentation: Similar to hypovolemic shock except for dry skin.

Pathophysiology of Shock

Reduce capillary perfusion → Inadequate tissue oxygen → Shift to anaerobic metabolism → Metabolic acidosis (lactic acidosis) → Release of free radicals & oxidative stress → Tissue damage → Apoptosis

إنه يخرج Anaerobic مشكلة
Lactic acid > cause acidosis

Oxidative stress:
imbalance between free radicals and antioxidants in your body. Free radicals are oxygen-containing molecules with an uneven number of electrons. The uneven number allows them to easily react with other molecules.

Metabolic Changes & Cellular Response to Shock

01

Reduce capillary perfusion:

Post sphincter: جهة venules

- Spasm of pre/post capillary sphincters (both sphincter and venule spasm > no blood flow
- Hypoxic tissue damage, (**oxidative stress**)
- Anaerobic metabolism (anaerobic glycolysis)
- Lactic acid production.
- **Metabolic acidosis (intracellular acidosis).**
- **Failure of Na+/K+ pump (** ↑$[Na^+]$ & $[Ca^{2+}]$ intracellularly **)** failure of normal cellular functions
- **Lysosomes, nuclear membranes & mitochondrial breakdown.**

After 3 - 5 hours of shock:

- Precapillary sphincters dilate, venules are still constricted.
- Blood stagnation in capillaries. (pre-sphincter opens > blood flow into the capillaries but post-sphincter is closed > blood stagnation)
- Hypoxia continue + fluid leaves to extravascular compartment.
- Further reduction in circulating blood volume.

Continued in the next slide

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

Metabolic Changes & Cellular Response to Shock:Cont...

02

Granulocytes accumulation at injured vessels

- Free radicals release.
- Further tissue damage.

03

Damage in GIT mucosa

- Allows bacteria into circulation.

04

Cerebral ischemia

- Depression of VMC → vasodilation (leading to further decrease in blood pressure) + ↓ HR

05

Myocardial ischemia

- Depressed contractility + myocardial damage (more shock & acidosis) > cycle repeats

06

Respiratory distress syndrome

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

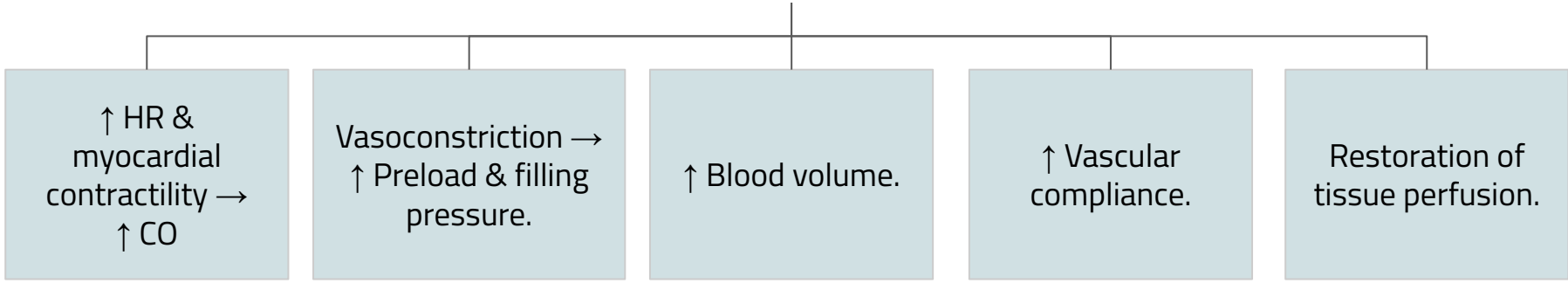
07

Multiple organ failure & death

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

Compensatory Mechanisms to Shock

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



1. **Stimulation of Sympathetic Nervous System through:** 1,2,3 are BP regulatory mechanisms

- 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.

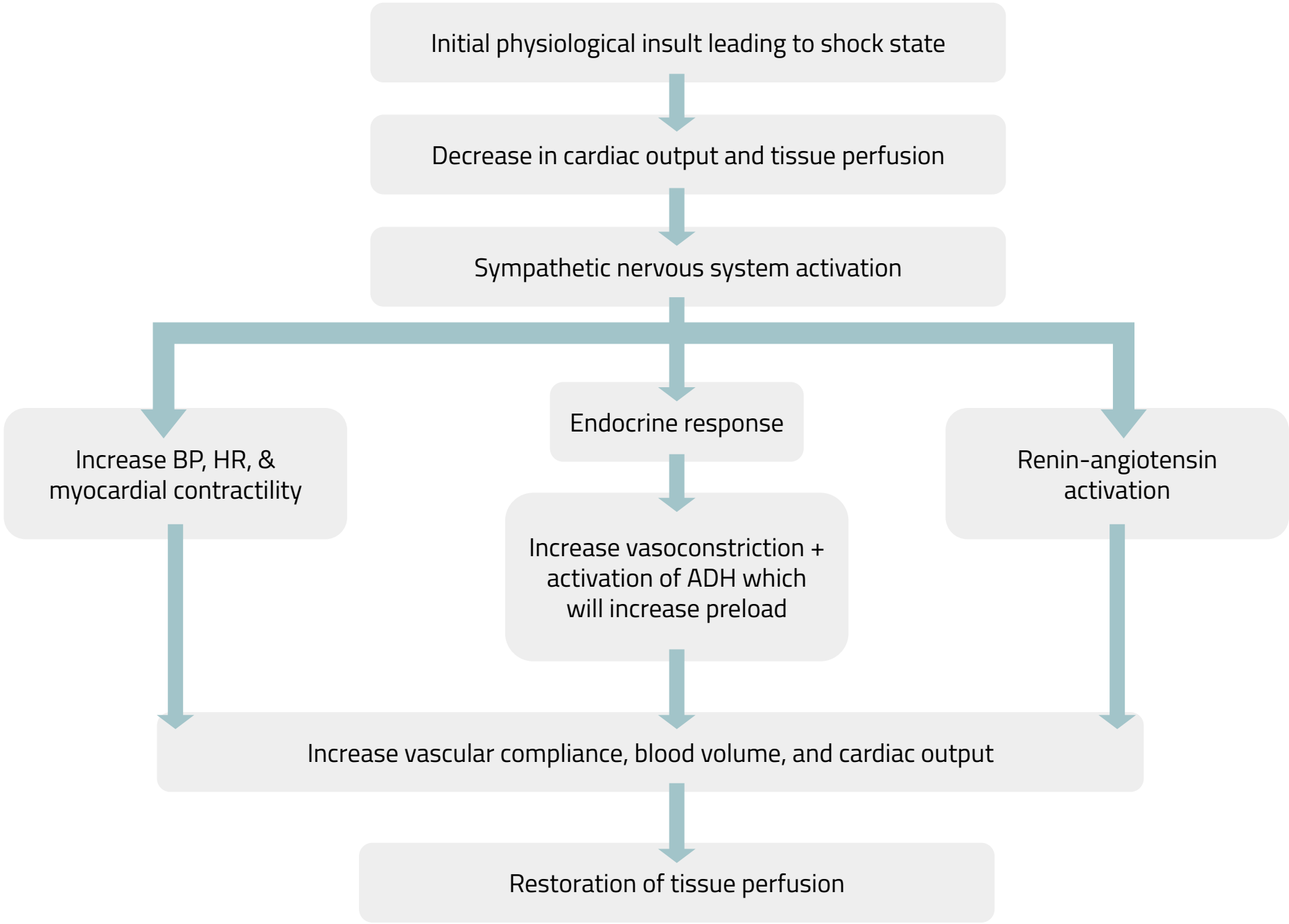
3. **Stimulation of ADH (vasopressin):**
   Water retention, vasoconstriction & thirst stimulation.

4. **Synthesis of Plasma Proteins:** to increase oncotic pressure of plasma (pull fluid into the capillaries) (3-4 days)

5. **Fluid- shift mechanism**

BP=Blood pressure; HR=Heart rate; CO=Cardiac output

Compensatory mechanisms to shock



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.

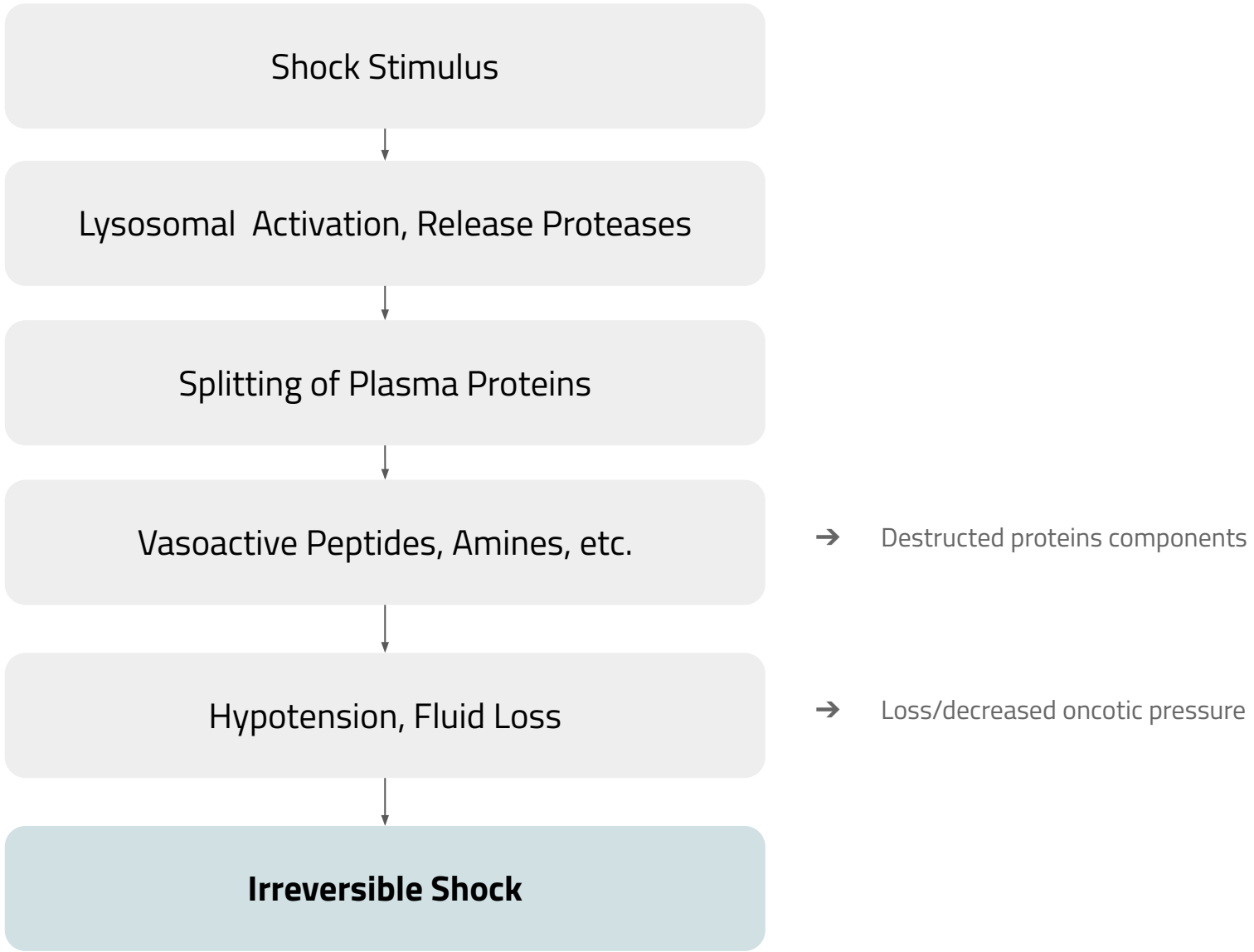
team 436: The capillary fluid shift mechanism means simply that anytime capillary pressure falls too low, fluid is absorbed from the tissues through the capillary membranes and into the circulation, thus building up blood volume and increasing the pressure in the circulation.

Stages of Shock

Circulatory shock change with different degrees of severity, shock is divided into following major stages:

Reversible shock: (Compensated)	➢ Changes can be reversed by compensatory mechanism (neurohormonal activation) which could cause full recovery without outside therapy or by treatment. ➢ Defense mechanisms are successful in maintaining perfusion. ➢ **Non-progressive**.
Progressive	➢ Defense mechanisms begin to fall. ➢ Multi-organ failure. ➢ Without therapy, shock worsens until death, progresses to irreversible shock.
Irreversible shock	➢ Complete failure of compensatory mechanisms. ➢ All forms of known therapy are inadequate to save the life ➢ Can lead to death (for the moment the person is still alive)

Possible Mechanisms That Lead to Developing Irreversible Shock



Summary

Type	Causes	Signs & Symptoms
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 **Low resistance shock**	**Septic shock**: infection → release of bacterial toxins → activation of NOS in macrophages → production of NO → vasodilation → decreased vascular resistance → **hypotension** **Anaphylatic shock**: allergy (release of histamine) → vasodilation → decreased vascular resistance → **hypotension** **Neurogenic shock**: spinal injury → loss of autonomic & motor reflexes → reduction of peripheral vasomotor tone → vasodilation → decrease in peripheral vascular resistance → **hypotension**	**Septic shock:** hypotension; fever; warm, sweaty skin. **Anaphylatic shock:** skin eruptions; breathlessness, coughing; localized edema; weak, rapid pulse. **Neurogenic shock:** as for hypovolemic except dry skin.

Extra: If you recall in the Capillary circulation lecture

Movement of fluids across capillary walls depends on the balance of starling forces acting across the capillary wall.

Starling Forces: Forces that control movement of fluid in/out of a capillary

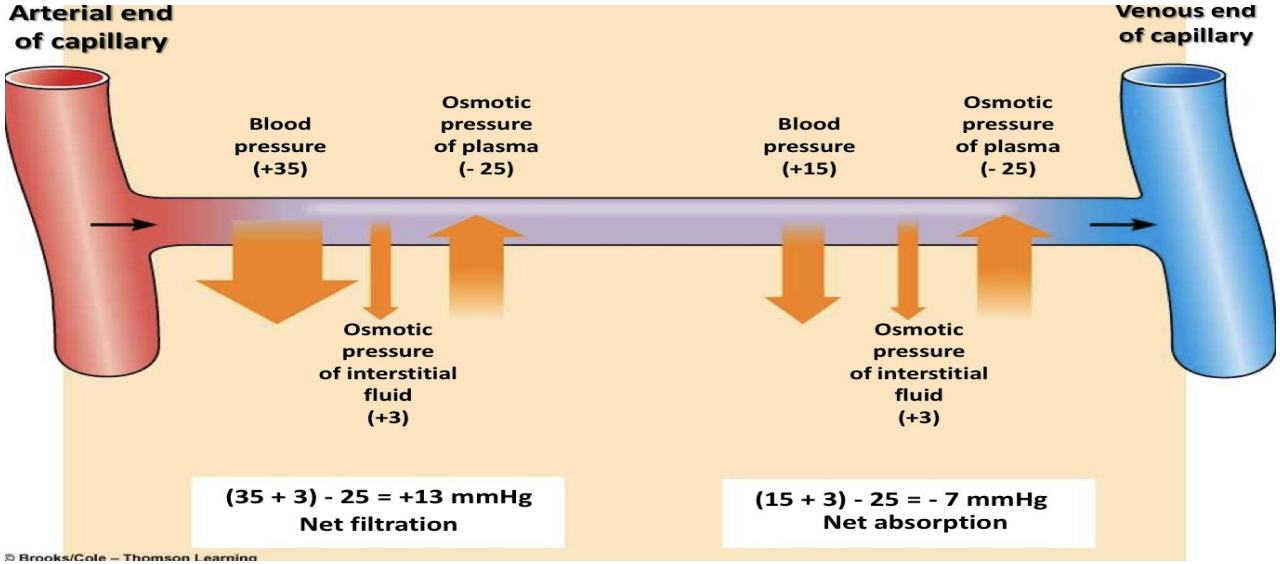
Hydrostatic pressure (P)		Colloid osmotic (oncotic) pressure	
Capillary Hydrostatic pressure (Pc)	Interstitial Fluid Hydrostatic pressure (Pif)	Plasma Colloid osmotic (oncotic) pressure	Interstitial fluid (tissue) Colloid osmotic (oncotic) pressure
The pressure exerted by blood (water) on the walls of the blood vessels Pushes the fluid **outside** Arterial end= +35mmHg Venous end= +15mmHg	= 0 mmHg	The osmotic pressure created of by the non-diffusible plasma proteins inside the blood vessel Pulls fluid **inside** = - (25-28) mmHg	Pulls fluid **outside** = +3mmHg

As blood passes through capillaries:

- Fluid **filters** from plasma to interstitial fluid.
- Fluid is **reabsorbed** from interstitial fluid to plasma

- ❖ - ⟶ inside (to the capillary)
- ❖ + ⟶ outside(to interstitial fluid)

Normal Forces at The Arterial & Venous Ends of The Capillary



Arterial end

Hydrostatic pressure dominates at the arterial end, as a net sum of pressure forces that flow fluid **out** of the circulation:

- Hydrostatic pressure= 35 mmHg
- Osmotic pressure in plasma = -25 mmHg
- Osmotic pressure in tissue = +3mmHg

➔ net filtration pressure (NFP) =35+3+(-25) = +13 mmHg

- 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.

Venous end

Oncotic pressure dominates at the venous end, as a net sum of pressure forces that flow fluid **into** the bloodstream.

- Hydrostatic pressure= + 15 mmHg
- Osmotic pressure in plasma = -25 mmHg
- Osmotic pressure in tissue = +3mmHg

➔ net filtration pressure (NFP) = 15 + 3 +(-25) = -7 mmHg

- Water moves **into** the capillary with a NFP of -7 mmHg

Management and Treatment of Shock

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.
   (IVF: Intravenous Fluid)
5. Get and run blood (if appropriate).
6. Get an hand 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

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.
5. While inserting IVs, draw blood for laboratories and for blood typing.
6. Relieve pain with IV narcotics
7. Reassess.
8. Blood transfusion: Think twice.
9. Vasopressors.
10. Antibiotics.
11. Maintain IV Fluids.

Quiz:

1-In a patient who experienced severe burns, which type of shock is most likely to happen?
A) Cardiogenic shock
B) Neurogenic shock
C) Vasogenic shock
D) Hypovolemic shock

2-A patient lost 9% of total blood volume, which is expected to happen?
A) Decreased arterial pressure.
B) Increased arterial pressure.
C) Increased cardiac output.
D) No effect on both

3-Which of the following is NOT a cause of obstructive shock?
A) Tension pneumothorax
B) Hemorrhagic pericarditis
C) Sustained arrhythmias
D) Systemic hypertension

4-A patient lost 1.7 L of blood and is showing signs of tachypnea and confusion. Based on what's given, what class of hypovolemic shock does he have?
A) Hypovolemic Class I
B) Hypovolemic Class II
C) Hypovolemic Class III
D) Hypovolemic Class IV

5- Which of the following is correct in shock?
A) hydrostatic pressure increases & oncotic pressure is increases
B) hydrostatic pressure decreases & oncotic pressure is constant
C) hydrostatic pressure increase & oncotic pressure is constant
D) hydrostatic pressure increase & oncotic pressure is decreases

6-Compensatory mechanisms in shock aim to which of the following?
A) increase BP
B) decrease HR
C) increase blood volume
D) decrease vascular compliance

7- What allows bacteria into circulation?
A) Cerebral ischemia
B) Granulocytes accumulation at injured vessels
C) Myocardial ischemia
D) Damaged GIT mucosa

Answer Key: 1D- 2D -3C
4C- 5B -6A- 7D

Quiz:

1- What are the clinical findings expected of someone suffering from cardiogenic shock?

2- A 24 years old patient suffered from trauma involving the cervical spinal cord, what is expected of his blood volume and cardiac output?

3-Explain the normal forces at the arterial & venous ends of the capillary

4- List three compensatory mechanisms to shock

A1: Tachycardia, Tachypnea, Sustained hypotension, Congestion of the lungs and viscera seen on x-ray as:1- interstitial pulmonary edema 2- Alveolar edema 3- Cardiomegaly

A2: blood volume is normal, cardiac output is severely reduced as blood is pooled in the peripheral veins (capacity of blood is increased, venous return is decreased)

A3: Slide 19

A4: Slide 15

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