MEDICINE
KING SAUD UNIVERSITY

Physiology Team 436

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CVS BLOCK

Shock

Red: very important.
Green: Doctor's notes.
Pink: formulas.
Yellow: numbers.
Gray: notes and explanation.

Physiology Team 436 – Cardiovascular Block Lecture 11

Lecture: If work is intended for initial studying.
Review: If work is intended for revision.

Objectives

Study Smart: focus on mutual topics.

- Types and causes of shock.
- Define circulatory shock.
- Mechanisms responsible for the irreversible phase of hemorrhagic shock.
- Body compensatory mechanisms during reversible phases of hemorrhagic shock.
- Define shock and state the pathophysiological classification of shock.
- Describe the pathways leading to shock and decreased tissue perfusion.
- Discuss the stages of a hypovolemic shock.
- Explain how stage III hypovolemic shock might result in major organs failure.
- Discuss the different compensatory mechanisms during a hypovolemic shock.
- Describe the positive feedback mechanisms in the irreversible stage of a hypovolemic shock.

Shock

The cell is the basic unit of life and it needs oxygen to produce energy.
No oxygen → No energy → No life

Circulatory shock: When the circulatory system is unable to provide adequate circulation & tissue perfusion → decreased availability of oxygen and nutrients → failure to deliver oxygen to the tissues & vital body organs relative to its metabolic requirement → cellular hypoxia and energy deficit .
Result: organ dysfunction and cellular damage. (not death)

If not quickly corrected: whole body failure → leads to irreversible shock and death.

ONLY IN MALES' SLIDES

Shock may be defined as a pathophysiological state in which there is :
1-widespread
2- serious reduction of tissue perfusion

which if prolonged :leads to generalized impairment of cellular function.

Video of (Shock)
Duration: (9:10 mins)

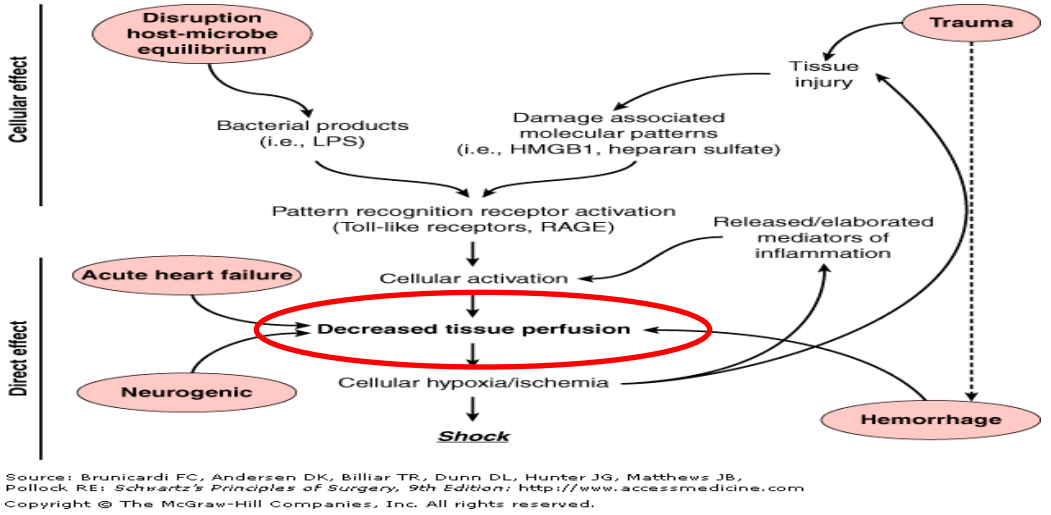
Video of (Shock)
Duration: (16:28 mins)

Pathways Leading to Decreased Tissue Perfusion and Shock

Decreased tissue perfusion

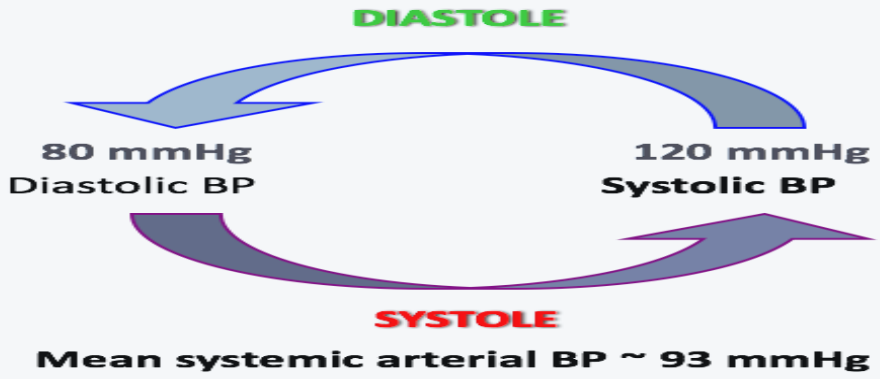
- **Causes:** hemorrhage / hypovolemia - cardiac failure - neurologic injury.
- **Result:** immune and inflammatory responses.

- Alternatively, elaboration of microbial products during infection or release of endogenous cellular products from tissue injury → cellular activation → influences tissue perfusion and the development of shock .

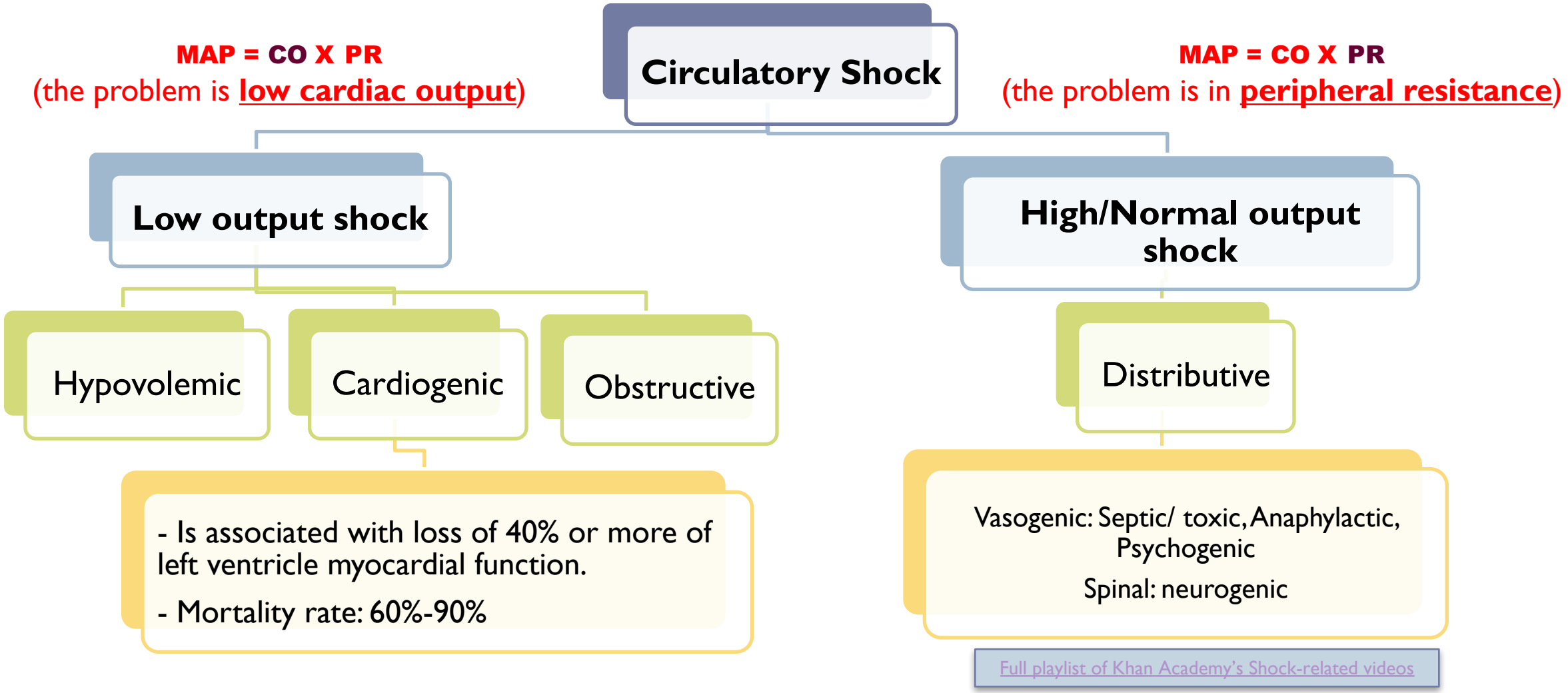


Systemic hypotension

- **NORMAL BP :** 120/80
- **LOW BP** < 90/60
- Arterial pressure = cardiac output x systemic vascular resistance
- Reduction in either component (cardiac output or vascular resistance) without a compensatory elevation of the other will result in hypotension.



Types of Shock



Causes and Classification of Shock

TYPE	CAUSES	SYMPTOMS AND SIGNS
Hypovolemic shock (most common)	• Bleeding/ Hemorrhage (internal/external) (Most Common) • dehydration (sever vomiting, sever diarrhea, excess sweating) • plasma loss (as in burns , trauma) → low blood volume Lead to: Reduced venous return (preload)→ loss of 15-25% of CO / 1-2 L→ hypotension	1. Hypotension (≤ 85/40mmHg) ; weak but rapid pulse . 2. Tachycardia (Compensation for↓ MAP sensed by Baroreceptors) Rapid, weak, & thready pulse (140/min). 3. Cool, clammy skin 4. Tachypnea "rapid respiration" (Compensation for hypoxia sensed by Chemoreceptors) shallow breathing , anxiety 5. Restlessness due to hypo-perfusion. 6. Altered mental state 7. Oliguria (low urine output)/ 8. Anuria (no urine output). 9. Blood test: Lactic acidosis.
Cardiogenic shock	Heart problems (e.g., myocardial infarction(Most common), heart failure , cardiac dysrhythmias) → despite adequate ventricular filling pressure →↓ contractility →↓in stroke volume →↓cardiac output → hypotension Myocarditis, cardiomyopathy, cardiac tamponade, congestive heart failure Acute valvular dysfunction (e.g., rapture of papillary muscles post MI) Sustained arrhythmias (e.g. heart block, ventricular tachycardia) pulmonary embolism Is associated with loss of > 40% of LV myocardial function, mortality rate is high 60-90%	1. Just like symptoms of a hypovolemic shock + 2. Distended jugular veins 3. Cardiomegaly 4. Congestion of lungs & viscera: Chest X-ray (CXR): 5. Interstitial pulmonary / Alveolar edema . 6. May be absent pulse
Obstructive shock	• Obstruction of venous return: like Vena Cava syndrome (usually neoplasms) • Compression of heart: like hemorrhagic pericarditis > cardiac tamponade • Obstruction of outflow: like aortic dissection*, massive pulmonary embolism, pneumothorax. • Circulatory obstruction (e.g., constrictive pericarditis) Lead to : reduced blood flow to lungs →↓ cardiac output → hypotension	1. Just like symptoms of a hypovolemic shock + 2. Distended jugular veins 3. Pulses paradoxes (in cardiac tamponade).

* Aortic dissection: aorta is weak, a cyst becomes a parallel vessel to aorta and makes it rupture > sudden death.

Causes and Classification of Shock

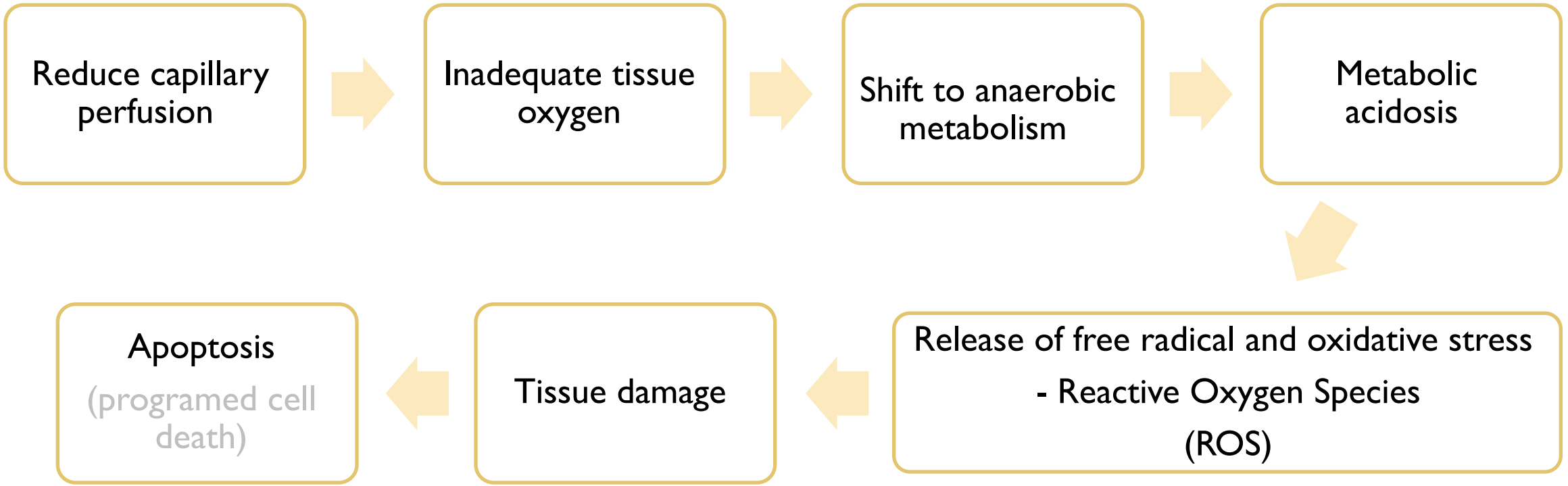
High/Normal Cardiac Output Shock (Distributive)

MAP = CO X PR

(the problem is in **vascular/peripheral resistance**)

TYPE	CAUSES	SYMPTOMS AND SIGNS
Distributive shock 1- Vasogenic (Valvular Obstruction) 2- Low-resistance shock	❑ Septic shock: infection → release of bacterial toxins → activation of NOS in macrophages → production of NO → vasodilation + endothelial injury → decreased vascular resistance→ hypotension ❑ Anaphylactic shock: allergy (release of histamine) → IgE Mediated hypersensitivity (histamine triggers peripheral vasodilation and an increase in capillary permeability → decreased vascular resistance→ hypotension ❑ Neurogenic shock: / Spinal Shock (venouspooling): spinal injury →↓ peripheral vasomotor tone → loss of autonomic and motor reflexes → vasodilation → Blood volume remains normal →↓in peripheral vascular resistance →↓ cardiac output as blood is pooled in peripheral veins → ↑ Capacity of blood →↓ Venous return → hypotension (Behaves like hypovolemic shock) ❑ Psychogenic shock : stress, pain, or fright → ↓ HR & vessels dilate → Brain becomes hypo perfused → Loss of consciousness.	❑ Septic shock: Patient flushed & warm due to his hyperdynamic state - Severe drop in BP or hemoglobin- fever – warm-sweaty skin and hypotension ❑ Anaphylatic shock: hypotension- skin eruptions- breathlessness- coughing - localized edema- weak - rapid pulse ❑ Neurogenic shock: as for hypovolemic except warm, dry skin ❑ Psychogenic shock : Simple fainting (syncope)

Pathophysiology of Shock



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 → Metabolic acidosis (intracellular acidosis) → Failure of Na/k pump (↑ Na & Ca inside the cell) → Lysosomes, nuclear membranes & mitochondrial breakdown

2- After 3-5 hours of shock:

Precapillary sphincters dilate
Venules are still constricted. → Blood stagnation in capillaries. → Hypoxia continue +Fluid leaves extra vascular compartment. (which will drain in lymphatic vessel) → Further reduction in circulating blood volume. بيقل الفوليوم لانه يروح للمفاتيك

3- granulocytes accumulation at injured vessels: (Free radical release and further tissue damage).

4- **damage in GIT mucosa :** allows bacteria into circulation.

Cont.

5- cerebral ischemia

depression of vasomotor center (sympathetic) → vasodilation+ ↓ HR → further decrease in blood pressure

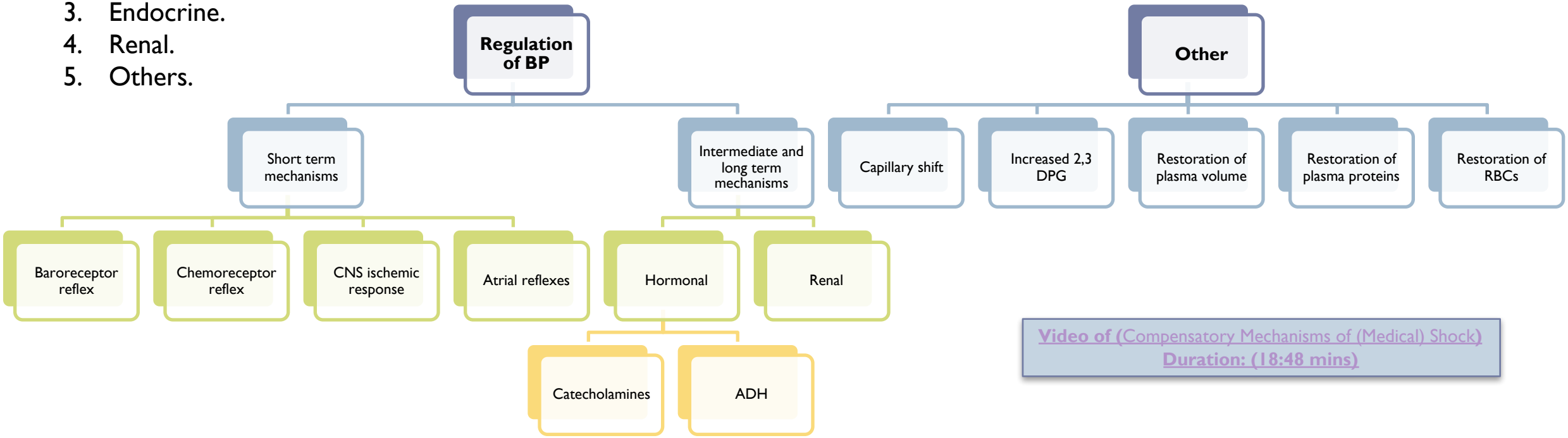
6- Myocardial ischemia → depressed contractility+ myocardial damage(more shock and acidosis).

7- respiratory distress syndrome occurs : due to damage of capillary endothelial cells & alveolar epithelial cells with release of cytokines**.**

8- multiple organ failure and death.

Compensatory Mechanisms During Hypovolemic Shock

1. Cardiovascular.
2. Respiratory.
3. Endocrine.
4. Renal.
5. Others.



Video of (Compensatory Mechanisms of (Medical) Shock)
Duration: (18:48 mins)

Short-term compensatory reactions:
are rapid and act within seconds to minutes

Intermediate-long term compensatory reactions:
are activated within hours or From hours to days

Other compensatory reactions:
are activated within hours From hours to days

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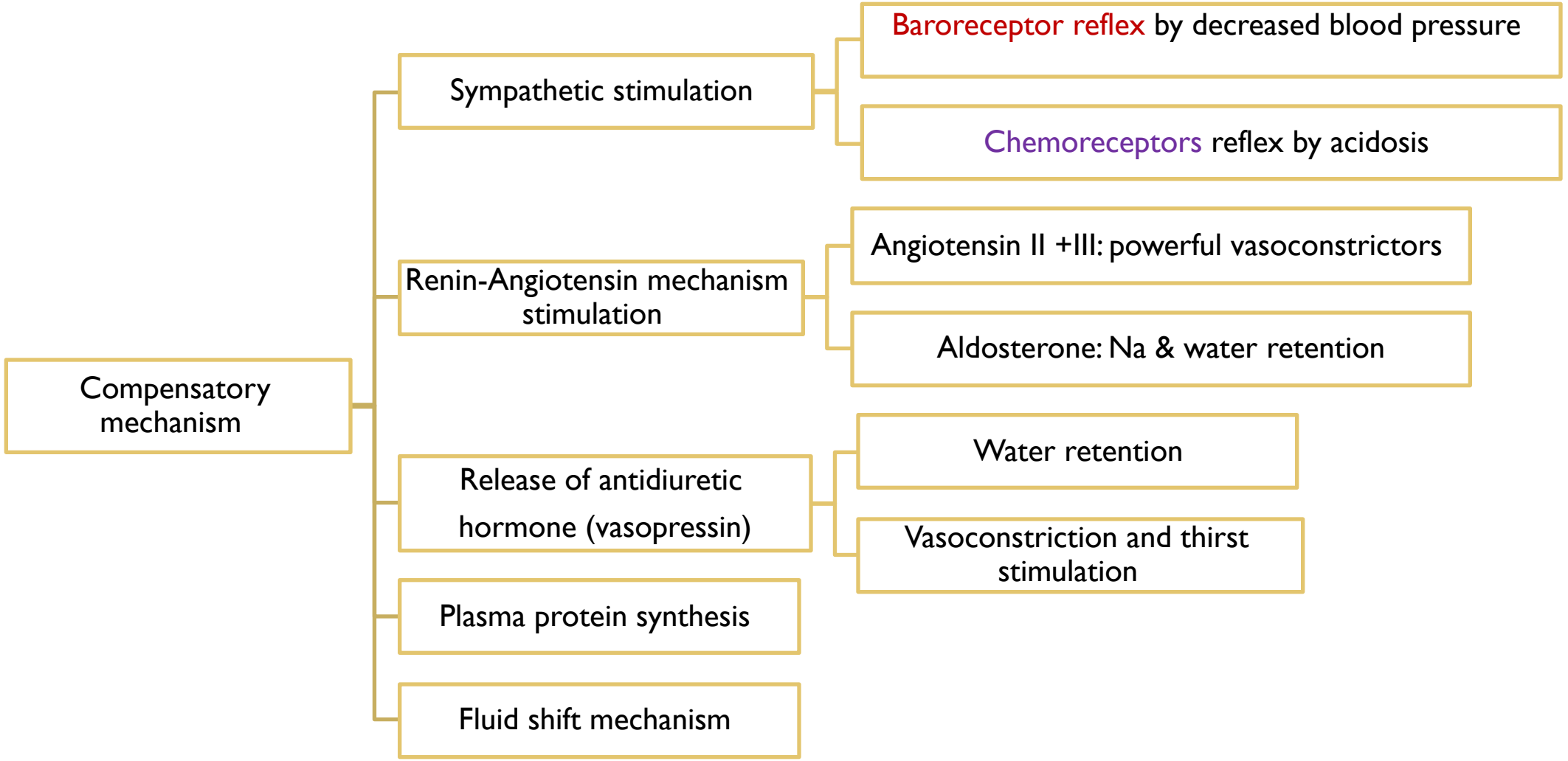
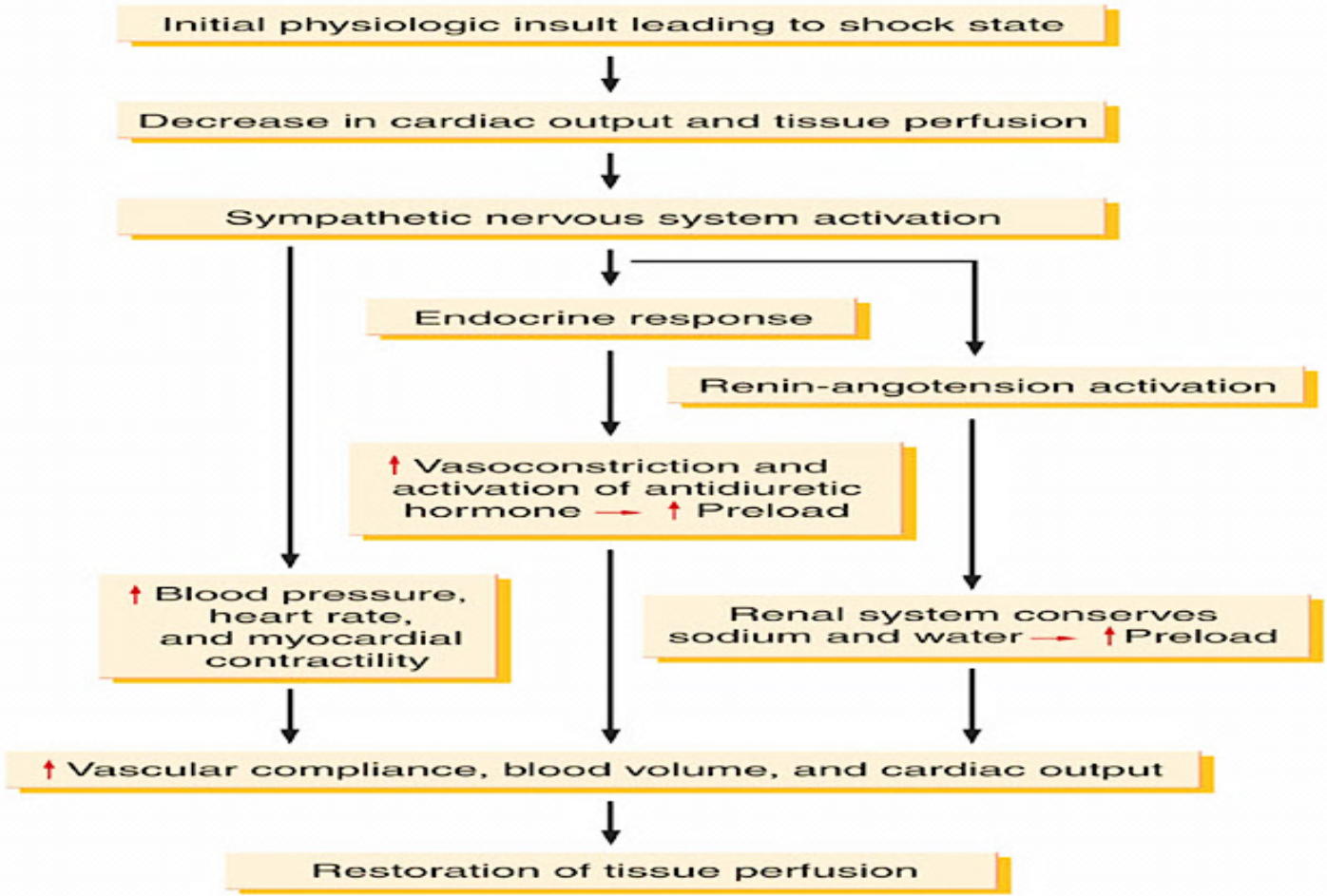
Compensatory Mechanisms (The Important Ones)



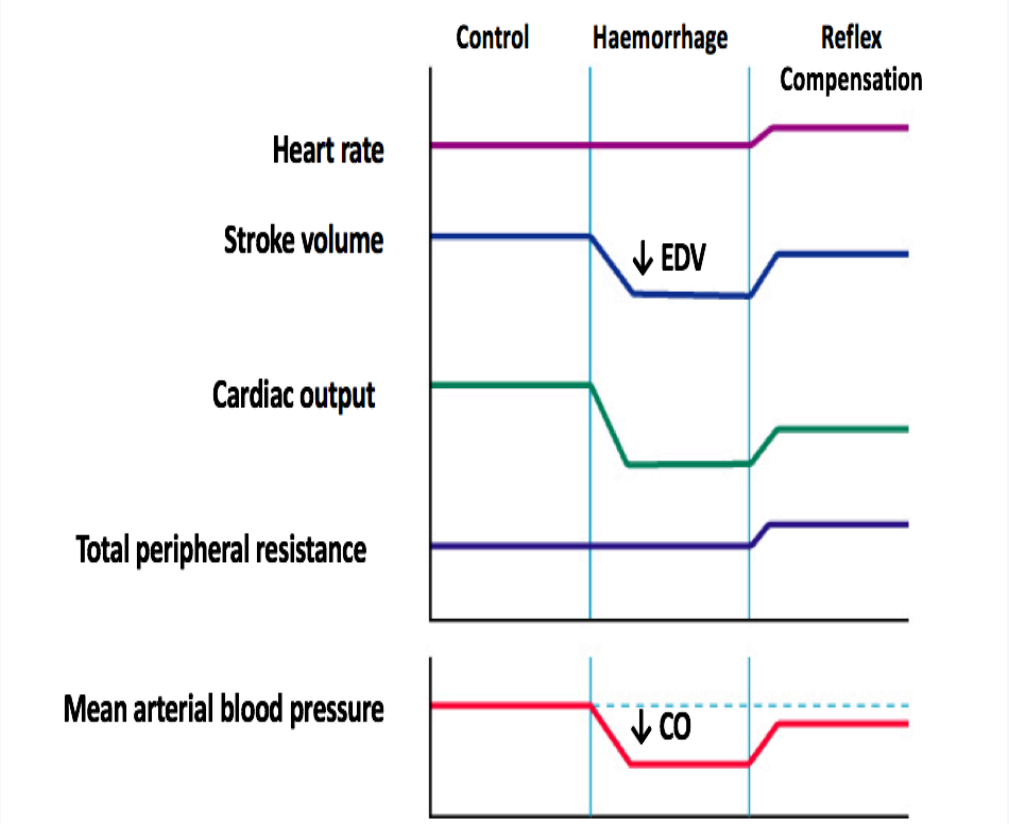
Compensatory Mechanisms

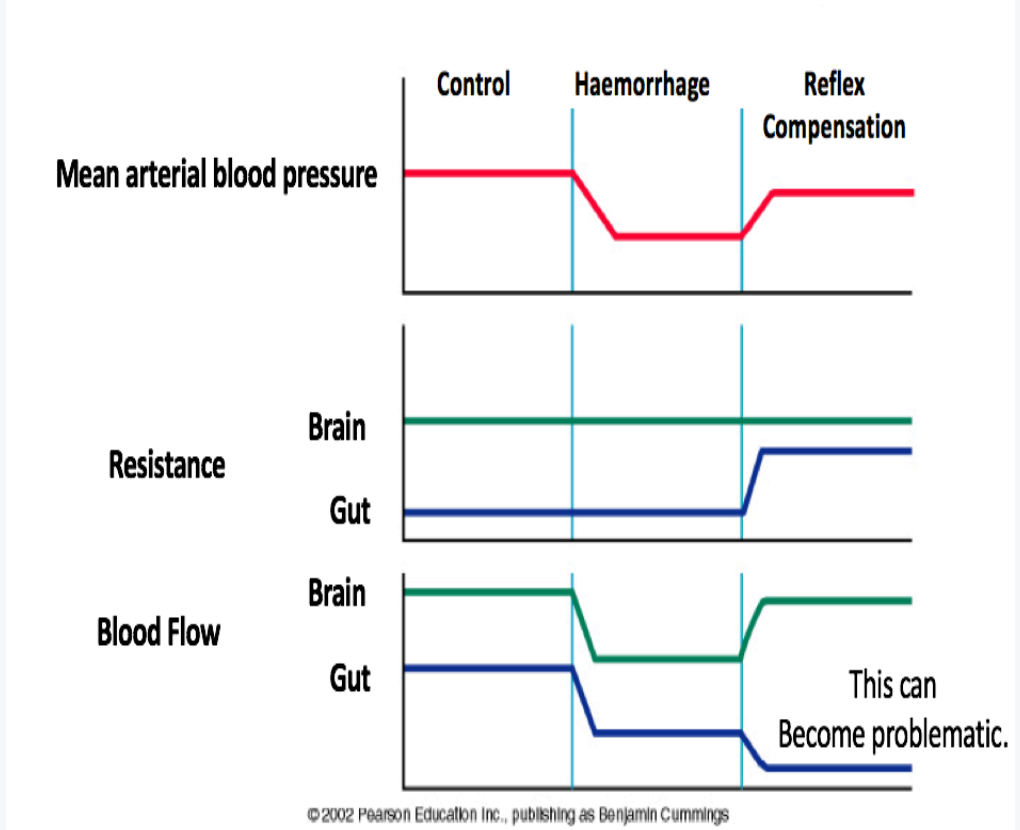


ONLY IN MALES' SLIDES

Short-Term Compensatory Mechanisms;
1- Arterial Baroreceptor Reflex

Flow to the heart and brain is maintained; reductions in flow to other organs.





Fully explained in arterial pressure regulation lecture.

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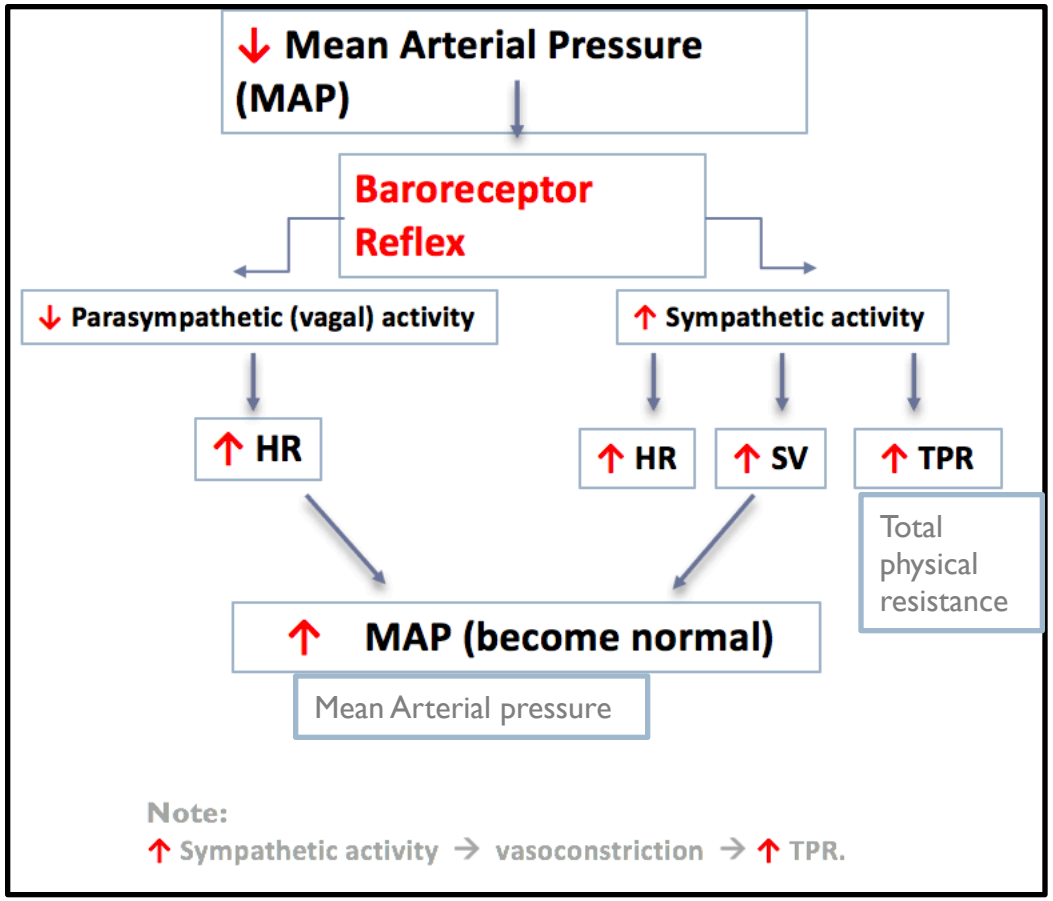
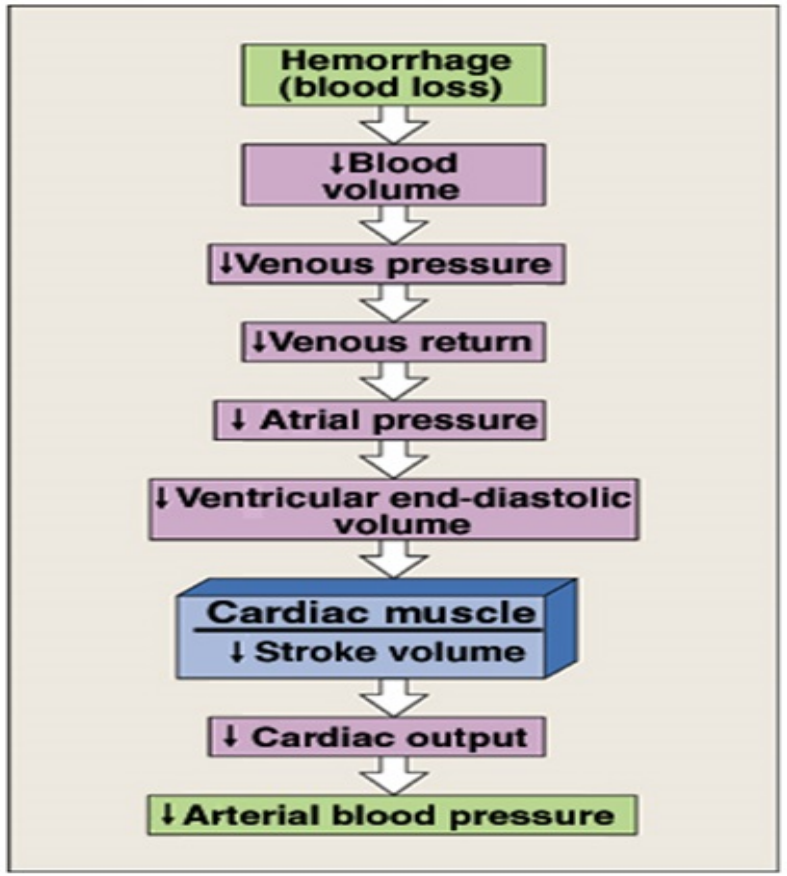




Fully explained in arterial pressure regulation lecture.

ONLY IN MALES' SLIDES

Short Term Compensatory Mechanisms;
2- Arterial Chemoreceptor Reflex

- Reductions in mean arterial blood pressure (MAP) below 60:
  1. do not evoke any additional responses through the baroreceptor reflex.
  2. stimulates peripheral chemoreceptors (aortic bodies, carotid bodies, heart) that sense changes in pO2, pCO2, and pH through tissue hypoxia and lactacidosis.

- This results in:
  - Enhancement of the existing tachycardia and vasoconstriction
  - Respiratory stimulation → tachypnea

Fully explained in arterial pressure regulation lecture.

Compensatory Mechanisms During Hypovolemic Shock
Arterial Baroreceptor and Chemoreceptor Reflexes

Vasoconstriction

(arteriolar constriction): this increases TPR and hence MAP.

It is produced by:

- Baroreceptor reflex.
- Chemoreceptor reflex.
- Noreadrenaline-adrenaline vasoconstrictor mechanism (due to activation of adrenal medulla).
- Vasoconstriction is also produced by vasopressin-vasoconstrictor mechanism (in subsequent slides).

Marked in:

- Skin: cold, pale.
- kidneys: drop in GFR & urine volume.
- Viscera.
- Flow to the heart and brain is maintained; reductions in flow to other organs.

Important in:

- Shifting blood from the venous reservoir into the circulation.
- Maintaining filling pressure of the heart.

Tachycardia

Produced by:

- Baroreceptor reflex.
- Chemoreceptor reflex.
- Increased sympathetic activity.

Tachypnea

Caused by : activation of chemoreceptor reflex and sympathetic over activity.

Importance:

- Increase O_2 delivery.
- Increase thoracic pump activity→ help venous return.

- The increased sympathetic activity also causes restlessness → ↑ skeletal muscle activity → help venous return.

Compensatory Mechanisms During Hypovolemic Shock; Endocrine and Renal

Mechanism:

1. Release of hormones that initiate vasoconstriction.
2. Renal conservation of salt and water.
3. Stimulation of hematopoeisis by erythropoietin

These compensatory reactions are activated:

Within hours

From hours to days

Endocrine and Renal Compensatory Mechanisms	
○ ↑ Circulating vasoconstrictors	○ Stimulation of sympathetic nervous system → release of epinephrine, norepinephrine from the adrenal medulla (sympatho-adrenal axis) . ○ Epinephrin is released almost exclusively from adrenal medulla where as norepinephrin is released from adrenal medulla and sympathetic nerve endings. ○ → Vasoconstriction.
	○ ADH is actively secreted by the posterior pituitary gland in response to hemorrhage. ○ Thus, hemorrhage → stimulates posterior pituitary → synthesis and release of vasopressin (ADH). ○ ADH is potent vasoconstrictor. ○ It also stimulates reabsorption of water.
	○ ↓ renal perfusion → secretion of renin from juxtaglomerular apparatus → generates angiotensin II → ○ Angiotensin II is a powerful vasoconstrictor

Cont.

Endocrine and Renal Compensatory Mechanisms	
○ ↑ secretion of glucocorticoids by adrenal cortex	○ This helps to maintain blood glucose
○ Stimulation of hematopoiesis	○ ↓ blood volume & hypoxia → synthesis of erythropoeitin (EPO) → hematopoiesis.

Endocrine and Renal Compensatory Mechanisms	
○ Renal conservation of salt and water	○ Vasopressin → ↑ water reabsorption → ↑ blood volume → ↑ arterial pressure. ○ Angiotensin II → release of aldosterone → ↑ salt reabsorption → ↑ blood volume → ↑ arterial pressure. ○ ↓ arterial pressure in kidney → ↓ decreases glomerular filtration rates and thereby decreases the excretion of water and electrolytes directly

Other Compensatory Mechanisms During Hypovolemic Shock

These compensatory reactions are activated within hours or from hours to days

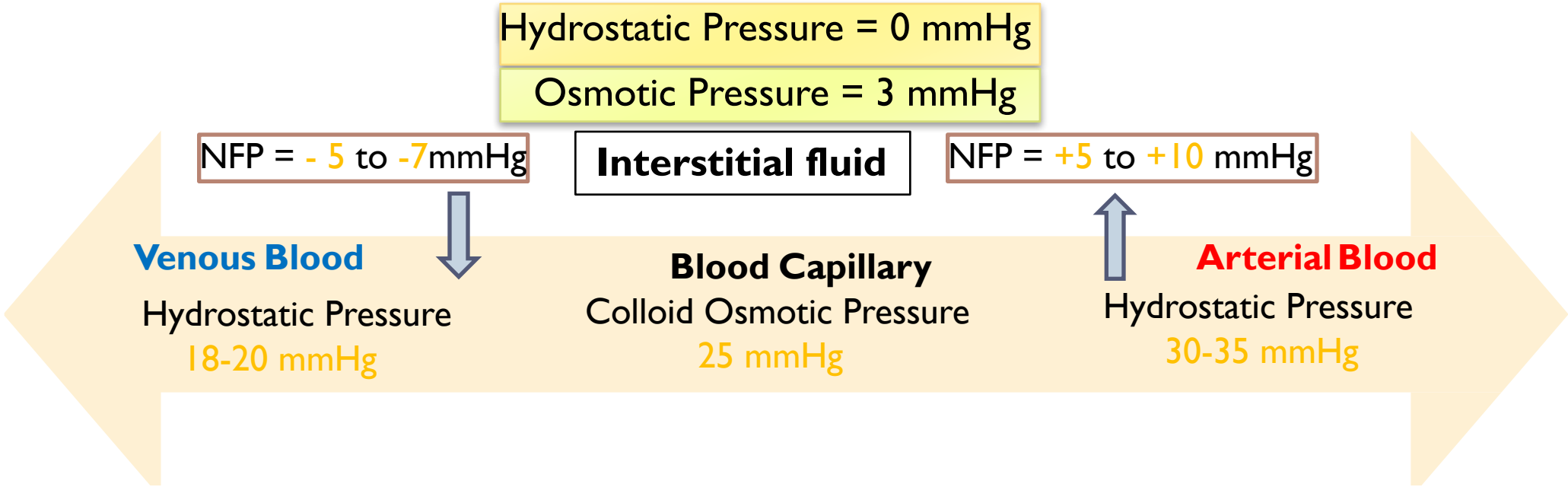
1. Responses activated: within hours

- Increased movement of interstitial fluid into capillaries (capillary fluid shift).
- Increased 2,3 DPG concentration in RBCs: This is important to help Hb deliver more O2 to the tissues (shift O2 dissociation curve to the right).

2. Responses activated: within hours-days

- Restoration of circulatory plasma volume: takes 12-72 hrs after moderate hemorrhage.
- Restoration of plasma proteins: this occurs in 2 stages:
  - Rapid entry of preformed albumin from extracellular stores.
  - Hepatic synthesis of proteins over 3-4 days.
- Restoration of RBCs: this occurs in 2 stages:
  - Increase RBCs count in response to erythropoietin within 10 days.
  - Restoration of red cell mass within 4-8 weeks.

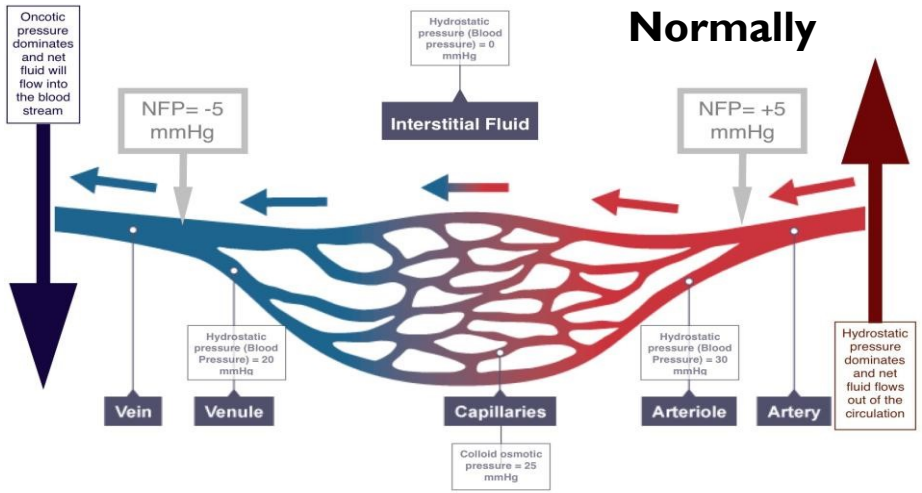
In Normal Microcirculation

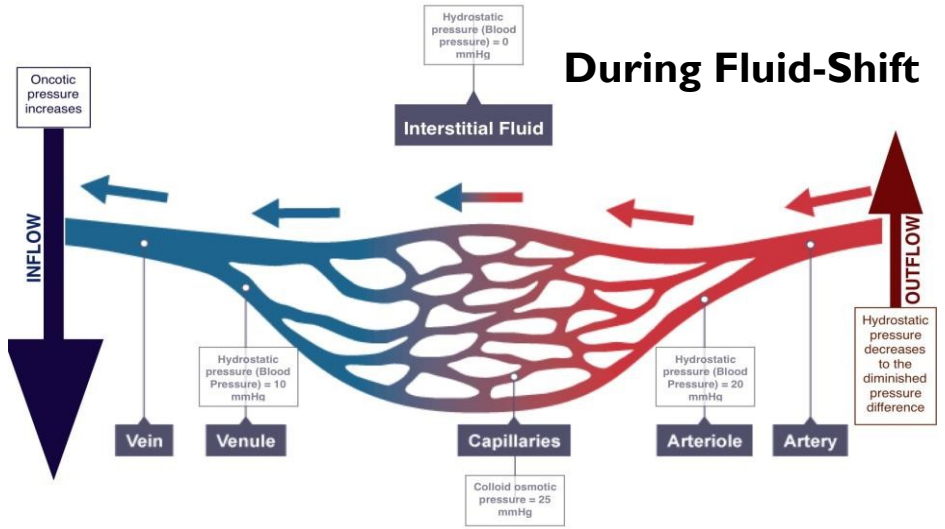


- At **arterial** end: Water moves out of the capillary with a NFP of +5 to +10 mmHg Hydrostatic pressure dominates at the arterial end & net fluid flows out of the circulation.
- (Arterial pressure - Colloid osmotic pressure = + NFP filtration to interstitial fluid)
- At **venous** end: Water moves into the capillary with a NFP of -5 to -7 mmHg. Oncotic pressure dominates at the venous end & net fluid will flow into the bloodstream.
- (Venous pressure - Colloid osmotic pressure = - NFP filtration to blood stream)

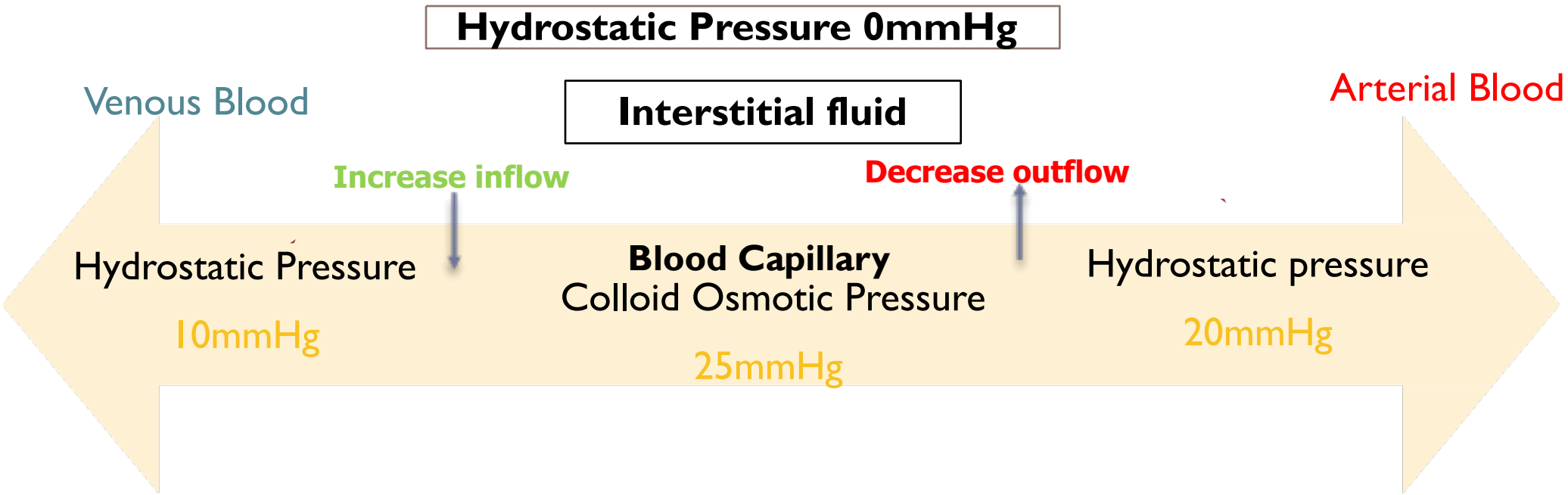
Fluid-Shift Mechanism

- 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.
- That will increase the blood volume & will increase BP helping to compensate shock.
- 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.





Fluid-Shift Mechanism in Shock



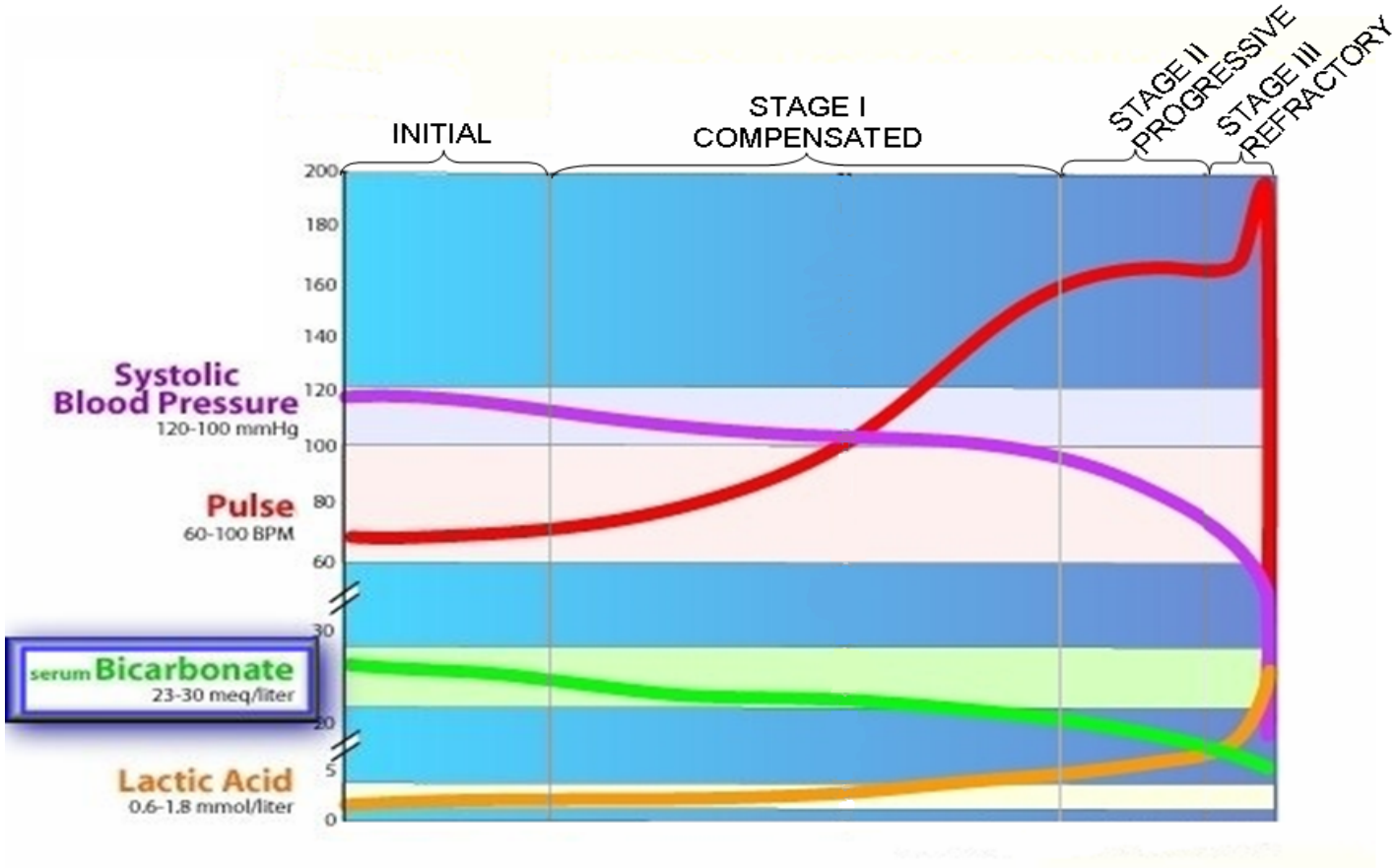
Stages of Shock

Stage 1 → pressure and cardiac output are maintained (in recumbent position)

Stage 2 → pressure and cardiac output are not maintained (10-20 mmHg drop)

Stage 3 → pressure and cardiac output decrease is life threatening

During Hypovolemic Shock :
Pressure decreases , HR increases
Bicarbonate decrease, Lactic acid increases



Stages of Shock

Reversible shock: (Compensated)

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

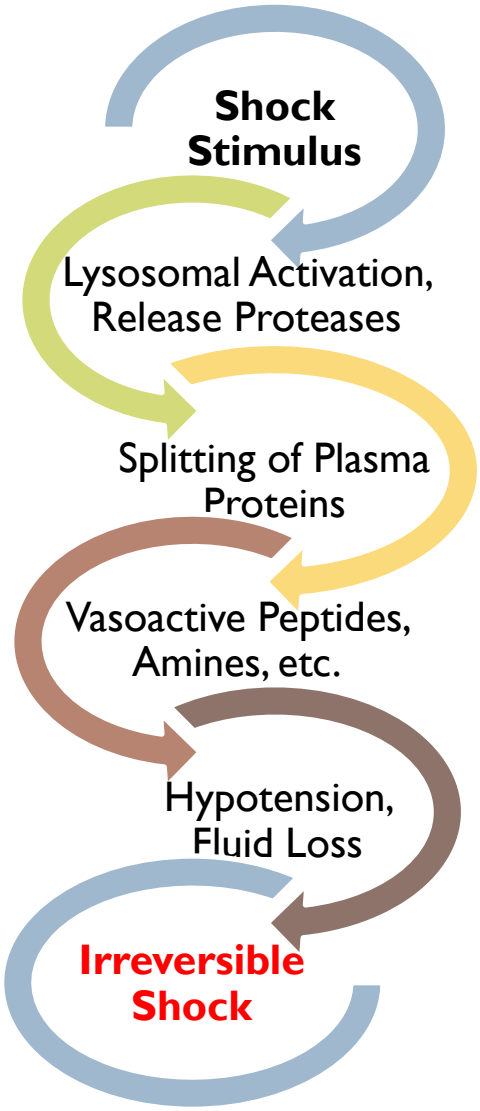
Progressive

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

Irreversible shock:

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

Possible Mechanism in the Development of Irreversible Shock



Stages of Shock

Stage 1 (Compensatory Stage)

Happen in: In healthy individuals, acute blood loss of 10 – 15 % of the normal blood volume (e.g., blood donation)
Results in : activation of the sympathetic nervous system.
Symptoms:

- Arterial pressure is maintained
- Pressure decline when the patient assumes the erect position
- Increase in heart rate, Paleness and Anxiety

Compensation is achieved acutely by:

- Increase in heart rate.
- Constriction of the arterioles.

➢ Thus, cardiac output is normal or slightly reduced especially when the patient assumes the erect position

Stage 2 (Progressive Stage)

Happen if: When 20-40% of blood volume are lost, cardiac output cannot be maintained and falls markedly, even in the recumbent position.

Result in:
1- Fluid also moves from the interstitial to the intravascular compartment (i.e., reabsorption of tissue fluids).
2- there will be massive activation of the sympathetic nervous system → Leading to:

→ intense arteriolar constriction in the vascular beds of the kidney, gut, and skin → redistribution of blood flow (i.e., centralization of blood flow) with larger fractions going to the vital organs (heart and brain) and smaller fractions going to the kidney, gut, and skin. Despite intense arteriolar constriction, systolic arterial BP declines by 10 to 20 mm Hg.

→ generalized vasoconstriction (increasing blood volume in the central circulation and tending to sustain venous return)

Cont.

Stage 2
(maximal mobilization of compensatory mechanisms)

Symptoms, signs and complications:

- Systolic arterial blood pressure declines by 10-20 mmHg
- Tachycardia (heart rate increases and pulse is thready and weak)
- Skin is pale and cool (peripheral pallor is common)
- Deterioration of the mental state because the brain is getting less oxygen. Patient looks weak, tired and drowsy. Patient may show anxiety, aggressiveness, and restlessness.
- Thirst.
- Oliguria (urine output is decreased).
- Angina may occur in patients who have intrinsic coronary vascular disease.
- pH drops and metabolic acidosis develops due to increased anaerobic glycolysis

Stage 3

The compensatory mechanisms are maximally mobilized in stage II. Thus,

Happen during : small additional losses of blood (>40 %) can result in life-threatening reduction in cardiac output, BP and tissue perfusion.

- Blood flow to heart, brain and kidney is further reduced → severe ischemia and irreversible tissue damage → this may result in impaired organ function and death.
- The severe vasoconstriction may itself become a complicating factor and initiate a vicious cycle

The factors that determine the ultimate outcome include:

- Duration of stage III.
- Severity of tissue anoxia.
- Age and condition of the patient.

Irreversible Stage of Hypovolemic Shock (Stage 3)

Mechanism In the irreversible stage of hypovolemic shock, a series of positive feed back mechanisms take place, leading to →
1- further deterioration in cardiac output and MAP
2- more tissue hypoxia
Depending on: the amount of blood lost
How : When blood loss is excessive and not immediately replaced, and proper treatment is delayed, this stage is reached and the patient dies.
In this stage, there is also failure of compensatory mechanisms.

The severe vasoconstriction may itself become a complicating factor and initiate a vicious cycle. It could happen also during **irreversible stage of hypovolemic shock**

Mechanism:

1- Impaired coronary perfusion → cardiac ischemia → depression of cardiac function → further lowering of cardiac output.

2- Reduced blood flow to the vasomoter centers in the medulla depresses the activity of compensatory reflexes → vasomotor failure → depression of the activity of compensatory reflex → depression of cardiac function → further lowering of cardiac output.

3- Prolonged renal ischemia → acute tubular necrosis → may lead to prolonged post-shock renal insufficiency.

4- Bowel ischemia → breakdown of the mucosal barrier . This may lead to entry of bacteria and toxins into the circulation → depression of cardiac function → further lowering of cardiac output

Generalized cellular deterioration:

- ↓ Mitochondrial activity inside the cells →↓ in ATP.
- ↓ of cellular metabolism, especially glucose →↓ in ATP.
- ↓ in active transport of Na^+ and K^+ across the cell → Na^+ accumulation inside the cell.
- Rupture of many lysosomes.

Quiz

- https://www.onlineexambuilder.com/shock/exam-142187

Link to Editing File

(Please be sure to check this file frequently for any edits or updates on all of our lectures.)

References:

- Girls' and boys' slides.
- Guyton and Hall Textbook of Medical Physiology (Thirteenth Edition.)

Thank you!

اعمل لترسم بسمة، اعمل لتمسح دمعة، اعمل و أنت تعلم أن الله لا يضيع أجر من أحسن عملا.

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