PHYSIOLOGY TEAM 432

LECTURE : 14

Shock & hemorrhage

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OBJECTIVES

By the end of this lecture the students are expected to:

- Define circulatory shock.
- List types and causes of shock.
- Understand the body compensatory mechanisms during the reversible phase of hemorrhagic shock.
- Understands the mechanisms responsible for the irreversible phase of hemorrhagic shock

MIND MAP

Blood , plasma or water loss → **Hypovolemic shock**

Pathophysiology:
- Hypotension.
- Tachycardia.
- Cold, pale skin.
- thirst.
- Tachypnea.
- Restlessness.

2 stages:

reversible
- Rapid compensatory reactions. (act within seconds-minutes).
- Responses Activated within hours
- Responses activated from hours-days

irreversible

Positive feedback mechanisms lead to drop in cardiac output

Definition of shock

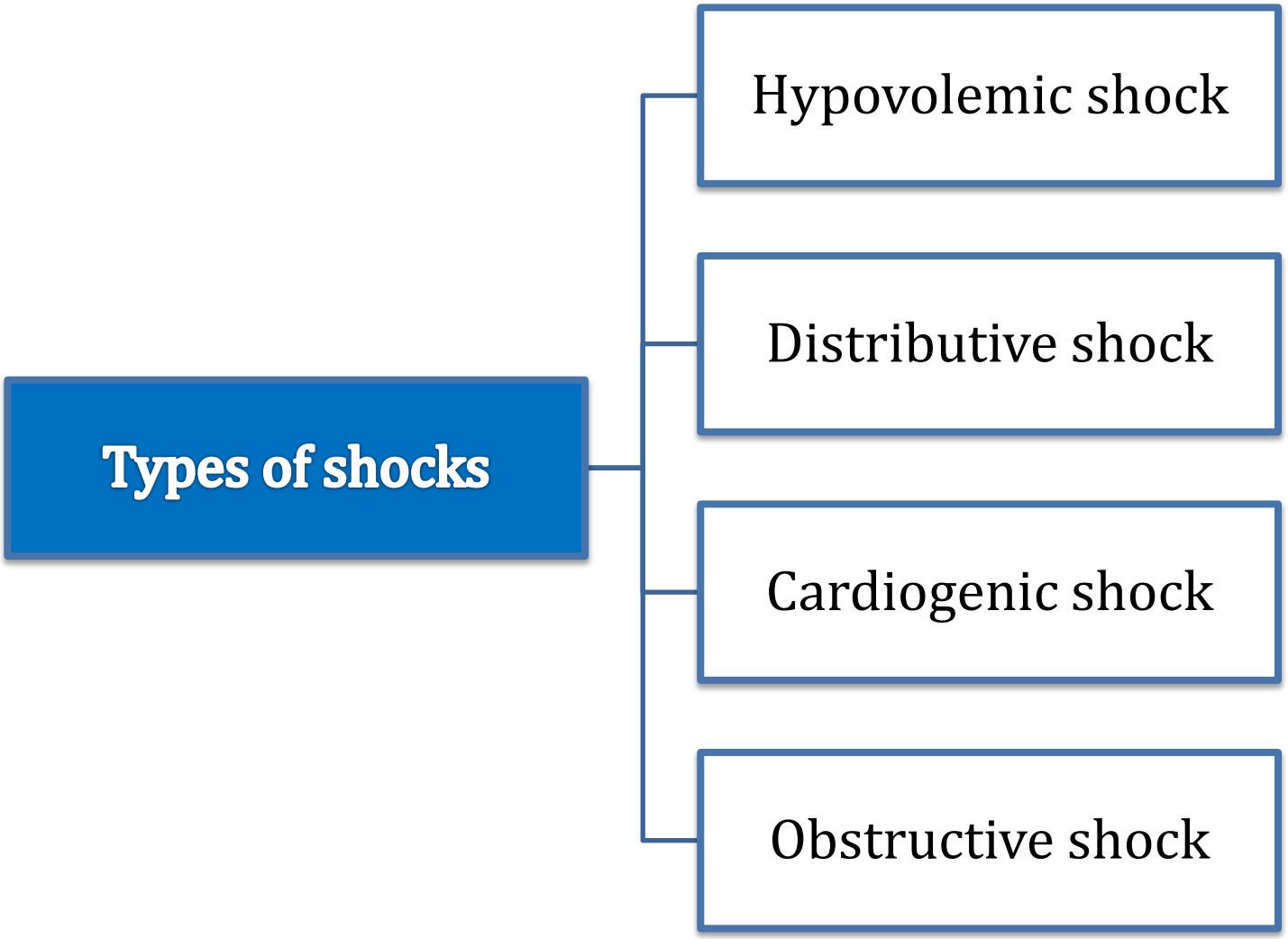
Inadequate tissue perfusion with relatively or absolutely inadequate cardiac output (CO).

- Relatively inadequate cardiac output:

-There is a drop in CO (from 5L to 3L).
-no good tissue perfusion.

- Relatively inadequate cardiac output:

-the body needs are more than the CO (CO can be high but still there is a shock because this high CO is not enough to match the body needs.)
-e.g: septic shock (bacteria in the blood)


Types of shocks
Hypovolemic shock
Distributive shock
Cardiogenic shock
Obstructive shock

PHYSIOLOGICAL CAUSES OF SHOCK:

Circulatory shock caused by decreased cardiac output.
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 include 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

Hypovolemic shock is due :

- Loss of blood volume
  - Due to:
    - •Hemorrhage.
    - •Trauma.
    - •Surgery.
- Loss of fluids(water)
  - Due to:
    - •Sever vomiting.
    - •Diarrhea.
- Loss of plasma
  - As in buns. (burns also can lead to septic shock which can be fatal.

Distributive shock

Blood go to the tissues & capillaries instead of vascular system & heart.

(also called vasogenic, low resistance shock) There is marked vasodilation caused by:

- Anaphylaxis (due to antigen-antibody reaction, e.g drug – induced..)
- .Sepsis.
- Neurogenic:

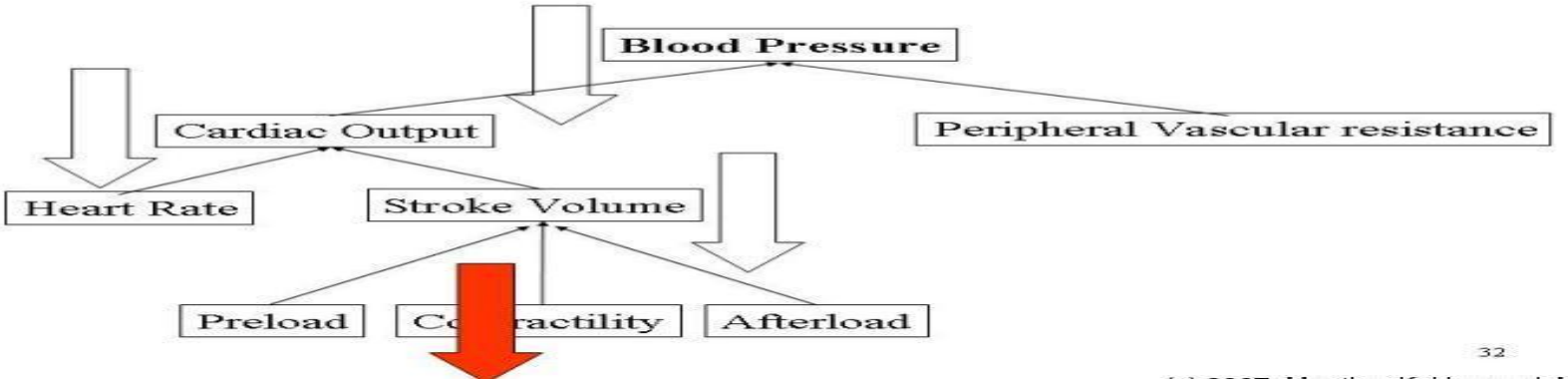
vasovagal, acute venous dilation, sever pain(during a surgery when the anesthesia is not good)…etc

Cardiogenic shock

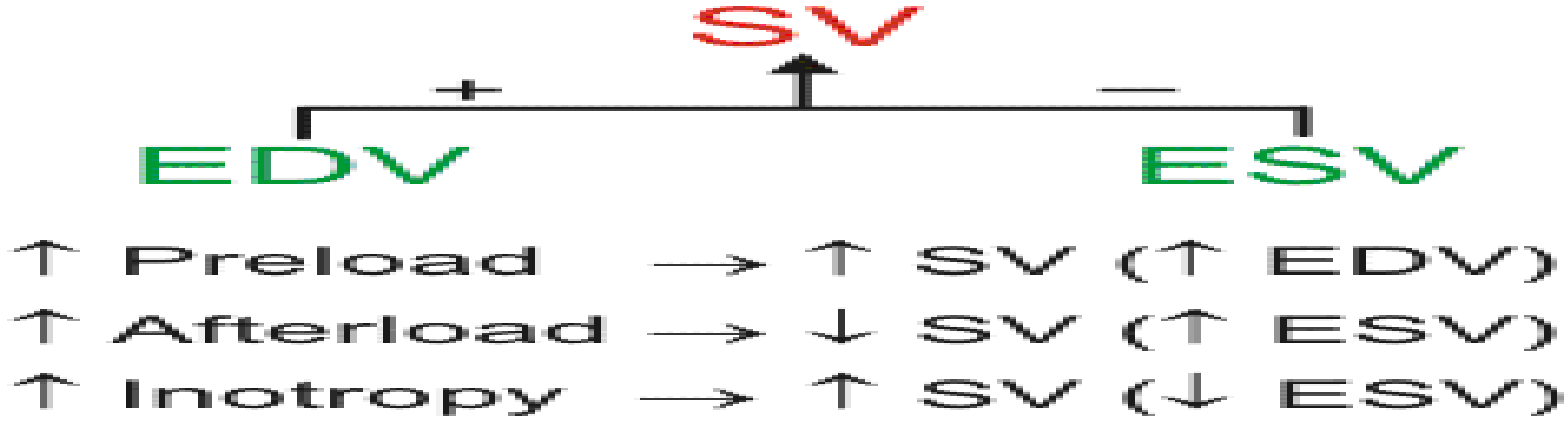
Results from inadequate output caused by diseased heart:

- Myocardial infarction.
- Congestive heart failure.
- Arrhythmias.

Cardiogenic Shock
(Post Extensive Myocardial Infarction)



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 shock

Due to obstruction to the flow of the blood by:

- Tension pneumothorax.

(if a person had an accident & he broke a rib this will allow the air to inter into his pleura & stay there –one way flow- this air will compress his heart which will make the heart unable to pump the blood).

- Pulmonary embolism.

(will prevent the blood flow to the heart).

Pathophysiology of hypovolemic shock

(signs & symptoms related to the blood loss)

Characterized by:

- Hypotension.(decrease in BP due to decrease in blood volume)
- Rapid thready pulse.(thready = weak)
- Cold, pale skin.(due to decrease in oxygen provided to the skin)
- Intense thirst.
- Rapid respiration. (increase in the respiratory pressure)
- Restlessness.

According to the cause hypovolemic shock is subdivided into; hemorrhagic, traumatic, surgical, burn shock.

Response to acute drop in ABP is tachycardia
Response to acute elevation in ABP is bradycardia

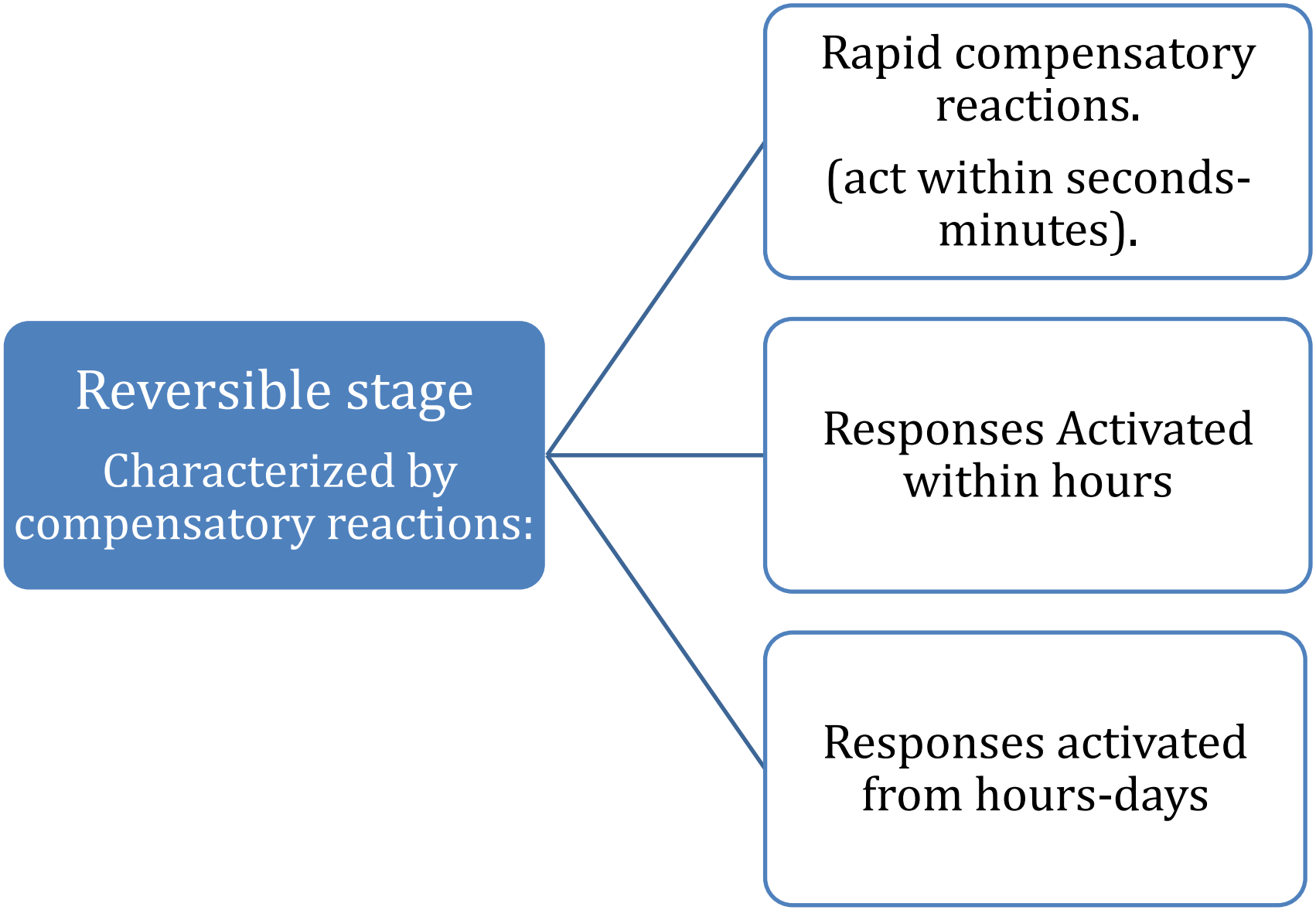
Stages of shock

Reversible

- In which compensatory reactions & appropriate treatment help restoration of blood pressure & blood loss.

Irreversible stage

- In which series of positive feed back mechanisms take place leading to further deterioration & tissue hypoxia. This depends on amount of blood lost. When blood loss is excess and not immediately replaced and proper treatment is delayed, this stage is reached and patient die. There is also failure of compensatory mechanisms.


Reversible stage
Characterized by compensatory reactions:
Rapid compensatory reactions.
(act within seconds-minutes).
Responses Activated within hours
Responses activated from hours-days

A. Rapid compensatory reactions:

I. Vasoconstriction: this increases TPR and hence ABP. It is produced by:

- Baroreceptor reflexes.
- Chemoreceptor reflex.
- Vasopressin-vasoconstrictor mechanism.
- Noradrenaline-adrenaline vasoconstrictor mechanism (due to activation of adrenal medulla).
- Renin- angiotensin - aldosterone.(by angiotensin II).
- Sympathetic over activity.

Vasoconstriction is marked in:

- Skin: cold, pale.
- kidneys: drop in GFR & urine volume.
- Viscera.

Heart and brain are spared.

II. Tachycardia: (due to drop in BP)

Produced by:

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

III. Venoconstriction:

Caused by sympathetic activity. It is important to:

-Maintain filling pressure of the heart.

-Shift blood from reservoirs into the circulation.

Vessels innervation :

- Systemic vessels: only innervated by sympathetic (no vagal).
- Vessels of the organs: innervated by sympathetic and parasympathetic

IV. Tachypnea:

Caused by activation of chemoreceptor reflex and sympathetic over activity.

Importance:

- Increase O2 delivery. (and wash out carbon dioxide).
- Increase thoracic pump activity….help VR.

V. Restlessness :

This increases skeletal Muscle Pump activity.

VI. Release of vasoconstrictor factors/hormones:

- Catechol amines (E & NE) by adrenal medulla.
- Vasopressin by posterior pituitary : besides vasoconstriction it restores fluid volume by reducing urine output.
- Renin-angiotensin-aldosterone. (preserve salt and water by aldosterone).

B. Responses Activated within hours:

- Increased movement of interstitial fluid into capillaries (capillary fluid shift) (4 hrs. needed).
(fluid will shift from ECF to the capillaries).
- Increased secretion of glucocorticoids by adrenal cortex.
(help to maintain blood sugar)
- Increased 2,3 DPG concentration in RBCs: important to help HB deliver more O2 to the tissues (shift O2 dissociation curve to the right because we want the oxygen to be delivered to the tissues)

C. Responses activated in hours-days:

- Restoration of circulatory plasma volume. Takes 12-72 hrs. after moderate hemorrhage.
- Restoration of plasma proteins: occur in 2 stages:

1. Rapid entry of preformed albumin from extracellular stores.
2. Hepatic synthesis of proteins over 3-4 days.

- Restoration of RBCs :

1. increase RBCs count in response to erythropoietin within 10 days.
2. Restoration of red cell mass within 4-8 weeks.

Irreversible stage

+ve feedback mechanisms lead to drop in CO:

I. Cardiac depression:

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

II. Vasomotor failure:

Results from depression of vasomotor center, the heart becomes depressed and CO drops.

III. Release of toxins by ischemic tissues:

e.g. histamine, tissue enzymes, potassium, ...

In this stage the patient will not respond due to the +ve feedback mechanism

IV. Endotoxin: (cardio toxins)

Released from gram +ve bacteria when blood flow to intestine decreases absorption of toxins Cardiac depression.

V. Generalized cellular deterioration:

-(-) of mitochondrial activity inside the cells lead to decrease in ATP.

-(-) of cellular metabolism, especially glucose.

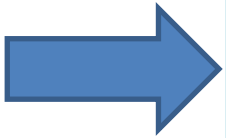
-Rupture of many lysosomes.

-Drop in active transport of Na+ and K+ across the cell Na+ accumulate inside the cell (NA+ will absorb water into the cell causing the swelling and rupture of the cell).

Signs of Hypovolemic shock

Patient become

- Pale
- Cold clamy skin



Vasoconstriction due to increased sympathetic stimulation

- Hypotension
- Rapid pulse
- Increased respiratory rate
- Sweating
- Increased thirst
- Decreased urinary output
- Metabolic Acidosis
- Restlessness

TREATMENT OF SHOCK:

Treatment of Shock: Goal [Restore Normal tissue perfusion]

- Blood pressure, Pulse, Respirations
- Skin Appearance
- Sensorium
- Urine output (30-50 cc per hour)
- Hemoglobin 8-10 gm or Hematocrit 24-30
- While inserting IVs, draw blood for laboratories and for blood typing
- Relieve pain with IV narcotics
- Reassess
- Blood transfusion: think twice
- Vasopressors
- Antibiotics?
- Maintenance IV fluids
- Inotropic support?
- Early removal of septic focus (i.e. dead bowel or large abscess) or other definitive surgery.

SUMMARY

Hypovolemic shock			Vasogenic shock	Neurogenic shock	Cardiogenic shock:
Loss of blood volume	Fluid loss	Plasma loss	There is marked vasodilation.	vasovagal, acute venous dilation.	Results from inadequate output caused by diseased heart
Hemorrhage. Trauma. Surgery.	Severe vomiting or diarrhea.	burns.	1. Anaphylaxis (due to antigen-antibody reaction, e.g drug – induced..) 2. Sepsis: (is the most dangerous) 3. Neurogenic: vasovagal, acute venous dilation.		1. Myocardial infarction. 2. Congestive heart failure. 3. Arrhythmias.

SUMMARY

Response to hypovolemic shock

Hematologic System	Cardiovascular System	Renal System	Neuroendocrine system
- Activating the coagulation cascade - Contracting the bleeding vessels (via local thromboxane A2 release) - Platelets activated which form an immature clot on the bleeding source - The damaged vessel exposes collagen, which subsequently causes fibrin deposition and stabilization of the clot	- Increases heart rate, increasing myocardial contractility, and constricting peripheral blood vessels. - This response occurs secondary to an increase secretion of norepinephrine and a decrease in vagal tone (regulated by the baroreceptors in the carotid arch, aortic arch, left atrium, and pulmonary vessels). - The CVS also responds by redistributing blood to the brain, heart, and kidneys and away from skin, muscle, and GI tract	- The kidneys respond to hemorrhagic. - shock by stimulating an increase in renin secretion from the juxtaglomerular apparatus. Renin → Angiotensinogen → Liver an lungs → Angiotensin I , Angiotensin II Angiotensin II has two main effects, both of which help to reverse hypovolemic shock, vasoconstriction of arteriolar smooth muscle and stimulation of aldosterone secretion by the adrenal cortex.	Causes an increase in circulating antidiuretic hormone (ADH) - ADH released in response to a decrease in blood pressure (as detected by baroreceptors) and a decrease in sodium concentration - ADH increase in reabsorption of water and salt (NaCl) by the distal tubule and the collecting ducts

SUMMARY

- Shock is defined as an acute circulatory failure leading to inadequate tissue perfusion and end organ injury.

- Shock usually results from inadequate cardiac output.

- In case of cardiogenic shock, heart becomes incapable of contracting with sufficient force to pump enough blood into the peripheral arterial tree.

- Stages of Shock:

1. A non-progressive stage : eventually cause full recovery without help from outside therapy.
2. A progressive stage: Without therapy, shock worse until death.
3. An irreversible stage: 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.

- Treatment of Shock : Goal [Restore Normal tissue perfusion]

SUMMARY

Acute bleeding (or other events leading to blood volume loss)

leads to

① Inadequate tissue perfusion → ↓ O_2 and nutrients to cells
② Cells begin to metabolize anaerobically (without O_2) → lactic acid accumulates
③ Water leaves tissue cells → moves into blood → cells dehydrate

Compensatory mechanisms activated

Brain

Chemoreceptors activated (by ↓ in blood pH)

Baroreceptors firing reduced (by ↓ in blood volume and blood pressure)

Hypothalamus activated (by ↓ pH and ↓ blood volume)

major effect *minor effect*

Activation of respiratory centers

Cardioacceleratory and vasomotor centers activated

Sympathetic nervous system activated

Thirst

Blood pressure maintained*

Neurons depressed by ↓ pH

↑ Rate and depth of breathing

↑ Heart rate

Intense vasoconstriction (only heart and brain spared)

ADH released

Restlessness (early sign)

CO_2 blown off; blood pH rises

Tachycardia, weak, thready pulse

↓ Renal blood flow

targets

Central nervous system depressed

Skin becomes cold, clammy, and cyanotic

Renin released

Kidney

Coma (late sign)

Adrenal cortex

Angiotensin II produced in the blood

Water retention

Aldosterone released

Kidneys retain salt and water

↓ Urine output

↓ Urine output

Increased blood volume

*If fluid volume continues to decrease, blood pressure ultimately drops. ↓ Blood pressure is a late sign.

THE END

If there are any problems or suggestions
Feel free to contact:

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THANK YOU

Actions speak louder than Words