Physiology Team 431

Team leaders

- Mohammed Asiri
- Nour Al-Khawaja

- Yafa Al-shamlan
- Sara Al-anazy
- Lama Mokhlis
- Tmader alaofi
- Hayfa alabdulkareem
- Dalal fatani
- Jomanah alshammari
- Shehanah alomair
- Alaa Al-anazi

- Abdullah Al-Towim
- Khalid Al Mohaimedi
- AbdulRahman Al-Bakr
- Fahad Al-showishi
- Saad Al-Mdemig
- Ahmad Al-Zuhair
- Mohammed Al Numeir
- Majid Al-Oriny
- Abdullrahman Alshahrani
- Tariq Al-Otaibi
- Abdulmalik Almufarrih
- Abdulaziz Al-hamad
- Ahmed Almarzuqi
- Nasser Al-moosa

Shock & Heamorrhage

Definition:

Inadequate tissue perfusion with relatively or absolutely inadequate cardiac output.

Types & causes:

1. Hypovolemic shock:
2. Distributive shock.
3. Cardiogenic shock.
4. Obstructive shock.

1. Hypovolemic shock:

-Loss of blood volume due to:

1. Hemorrhage.
2. Trauma.
3. Surgery.

-Fluid loss due to:

Severe vomiting or diarrhea.

-Plasma loss;

As in burns.

2.Distributive shock:

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

1. Anaphylaxis (due to antigen-antibody reaction, e.g drug – induced..)
2. Sepsis. **(septic shock is the most dangerous)**
3. Neurogenic:

vaspvagal, acute venous dilation,…

3.Cardiogenic shock:

- Results from inadequate output caused by diseased heart:

1. Myocardial infarction.
2. Congestive heart failure.
3. Arrhythmias.

4.Obstructive shock:

Due to obstruction to the flow of the blood:

1. Tension pneumothorax.
2. Pulmonary embolism.

Pathophysiology of hypovolemic shock

Characterized by:

- Hypotension
- Rapid thready pulse.
- Cold, pale skin.
- Intense thirst.
- Rapid respiration.
- Restlessness.
- According to the cause hypovolemic shock is subdivided into; hemorrhagic, traumatic, surgical, burn shock.

Stages of shock:

- **Reversible stage.**

  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:

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

A. Rapid compensatory reactions:

- **i. Vasoconstriction**: this increases TPR and hence ABP. It is produced by:
  * Baroreceptor refelexes.
  * Chemoreceptor reflex.
  * Vasopressin-vasoconstrictor mechanism.
  * Noreadrenaline-adrenaline vasoconstrictor mechanism (due to activation of adrenal medulla).

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

Heart and brain are spared.

ii. Tachycardia:

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.

iv. Tachypnea:

Caused by activation of chemoreceptor reflex and sympathetic over activity.

Imporatance:

Increase O2 delivery.

Increase thoracic pump activity….help VR.

V. Restlessness :

This increases sk. Ms. Pump activity.

vi. Release of vasoconstrictor factors/hormones:

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

B. Responses Activated within hours:

1. Increased movement of interstitial fluid into capillaries (capillary fluid shift) (4 hrs needed).
2. Increased secretion of glucocorticoids by adrenal cortex. (help to maintain blood sugar)
3. Increased 2,3 DPG concentration in RBCs: important to help HB deliver more O2 to the tissues (shift O2 dissociation curve to the right)

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 erythropoeitin within 10 days.
  2. restroration of red cell mass within 4-8 weeks.

Irreversible stage

+ve feedback mechanisms lead to drop in CO:

1. Cardiac depression.

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

2. Vasomotor failure.

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

3. Release of toxins by ischemic tissues.

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

4. Endotoxin:

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

5. 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