Shock and Hemorrhage

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ILOs

1. Describe circulatory shock (definition, classifications, mechanisms, and stages), types, mechanism, and stages of shock.
2. Describe hemorrhage and the compensatory reactions during haemorrhage (Immediate compensation, medium, long-term compensation).

Definition of shock:

- Tissue hypo perfusion as a result of relative or absolute inadequate cardiac output.

Types of shock

1- **hypovolemic shock**

2- **Distributive** (Vasogenic or low resistance shock)

❶ Anaphylactic shock

❷ Septic shock:

❸ Neurogenic shock:

3-**Cardiogenic shock**

4- **Obstructive shock:**

1- Hypovolemic shock: Caused by:

- Blood loss (Hemorrhage) → Hemorrhagic shock
- Plasma loss: as in burns
- Fluid loss: as in excessive diarrhea

2- Distributive shock (Vasogenic or low resistance)

❶ Anaphylactic shock	Widespread antigen-antibody reaction
❷ Septic shock:	Bacterial toxins produce VD
❸ Neurogenic shock:	pain produces sudden autonomic activity → VD & fainting

3- Cardiogenic shock

(heart disease → decrease CO)

- Myocardial Infarction (MI)
- Arrhythmias

4- Obstructive shock: Obstruction of blood flow: eg

- Tension pneumothorax (PTx)
- Pulmonary embolism (PE)
- Cardiac tamponade

Hemorrhagic shock:

- It is the major form of hypovolemic shock

- Manifestations:

❶ Drop of ABP (hypotension)

❷ Increase HR (rapid weak pulse)

❸ Increase respiratory rate

❹ Low skin temperature (cold & pale skin)

❺ Thirst

❻ Oliguria

❼ Acidosis (in severe cases lactate level increases from 1 to 9 mmol which depresses the myocardium)

❽ Restlessness (due to stimulation of reticular formation)

- Compensatory Reactions:

❶ Rapid compensatory reactions

- Aim: They are protective to maintain ABP for adequate perfusion of heart & brain
- Mechanisms: include 2 types:

① Nervous factors

There is: 1- Stimulation of PA (presser area)

2- Inhibition of DA (depressor area)

Causes:

1- Decrease inhibitory impulses from **arterial baroreceptors**

If ABP < 60 → arterial baroreceptors are not working. The pressure is maintained by chemoreceptors

2-Decrease Inhibitory impulses from atrial volume receptors

Decrease **atrial** discharge results also in increase release of ADH & aldosterone → salt & H_2O retention → increase blood volume

3- Increase excitatory impulses from **peripheral chemoreceptors**

Peripheral chemoreceptor stimulation: Results also in stimulation of respiration during shock

Results:

1- VC of arterioles →increase TPR →increase ABP

NB: - VC of all arterioles except brain & heart

- VC is marked in skin → pallor + cold skin

- VC of viscera & kidney [if prolonged → renal failure]

2- VC of veins →increase VR → increase CO → increase ABP

3- Increase HR→ increase CO → increase ABP

4- Increase SV → increase CO → increase ABP

② Humoral factors

Release of:

① Catecholamine

From adrenal medulla

1. VC: Little VC → Increase TPR → increase ABP
2. Stimulate reticular formation → restlessness

② Angiotensin II

1- VC 2- salt and water retention

3- It also causes thirst

③ Vasopressin (ADH "Antidiuretic Hormone")

1. VC effect
2. Antidiuretic effect (decrease H_2O in urine)

❷ Long term Compensatory Reactions

Aim: They are restorative

① Correction of plasma volume

In 12 - 72 hours by:

❶ Tissue fluid shift:

Decrease ABP ⟶ Decrease Hydrostatic Pressure of capillary ⟶ Increase fluid absorption into capillaries

⟶Increase volume of plasma ⟶ most of the retained fluid is protein free causing dilution of blood

❷ Thirst sensation: Due to:
1. Increased osmotic pressure
2. Decreased tissue fluid

❸ Aldosterone secretion:

Is increased due to decrease atrial volume receptors discharge

NB: Hematocrit drops: For hours because tissue fluid absorbed into capillaries is protein free & dilute plasma proteins & blood cells

② Correction of plasma proteins

In 3 - 4 days:

❶ Rapid release of stored albumin from liver into circulation

❷ Hepatic synthesis replaces the loss in 3-4 days

③ Correction of red cell mass

In 4 - 8 weeks:

❶ Erythropoietin

❷ 2, 3 DPG increases & shift to Rt of oxygen dissociation curve

❸ Squeezing of stored blood by contraction of splenic capsule

**** Outcome of shock state:**

- According to amount of blood loss & how fast we treat the patient:

1- If blood loss < 20%: Recovery by compensatory mechanisms & treatment

2- If blood loss > 20%: - Compensatory mechanisms are not sufficient, rapid blood transfusion should be done

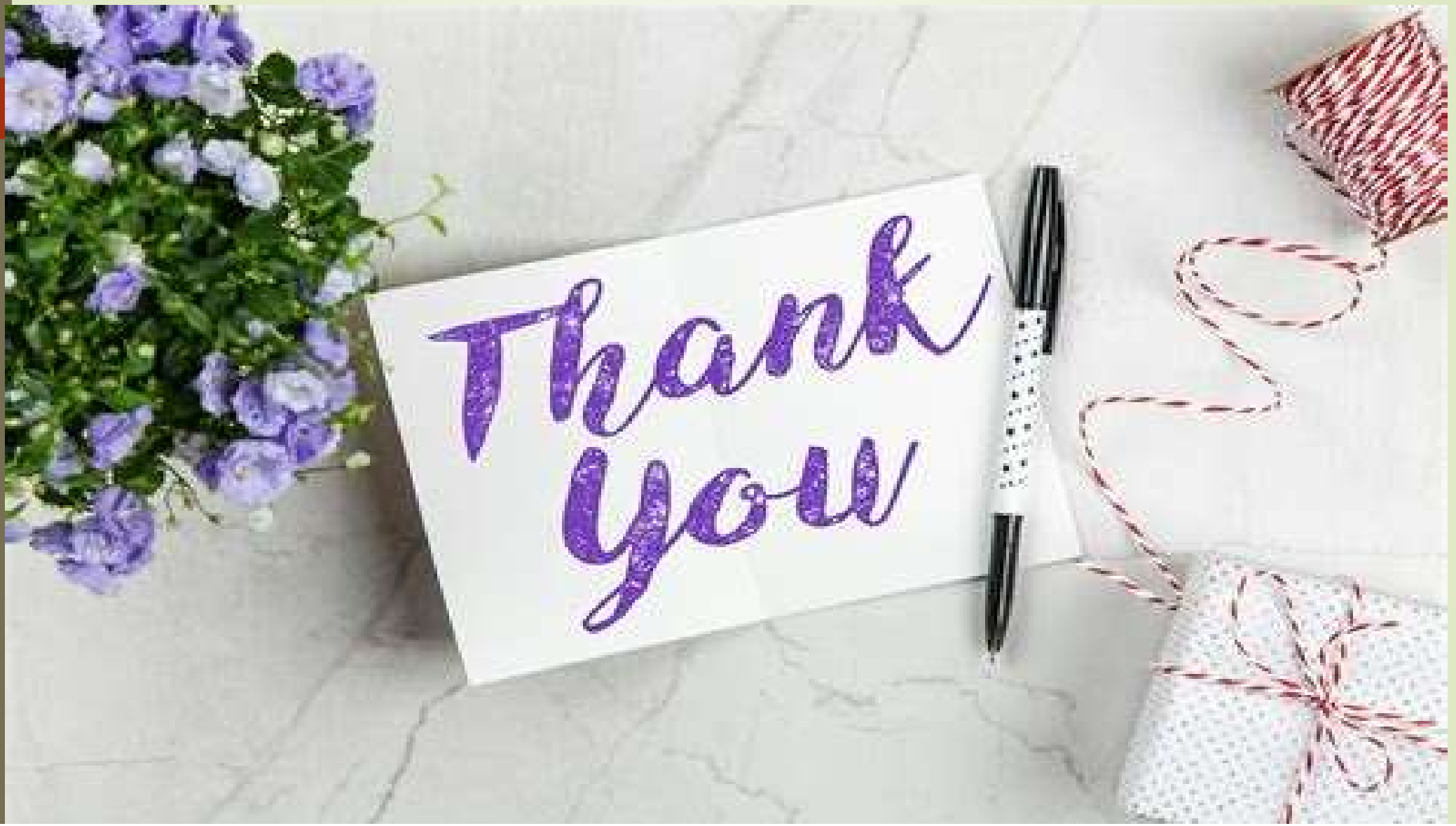
3- Irreversible shock: Progressive with no response to treatment, decrease CO "Cardiac Output" & death

Treatment:

1. Removal of the cause
2. Restoration of blood volume:
   - In hemorrhagic shock: Blood transfusion
   - In burn shock: Plasma expanders
3. Drugs:
   - Epinephrine (Adrenalin)
   - Norepinephrine (Nor Adrenalin)
   - Glucocorticoids
   - Sedatives
4. Supine position

Refractory shock (Irreversible shock)

Definition	Shock becomes resistant to treatment [cardiac output remains low even if blood volume is normal or above normal]
Mechanisms	1. Spasm of precapillary sphincters & venules (especially in the splanchnic region) 2. Sever cerebral ischemia 3. Sever cardiac ischemia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