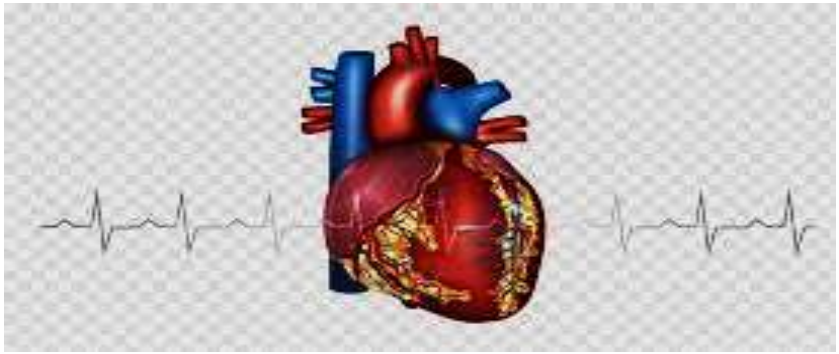
13- Pathophysiology of Shock



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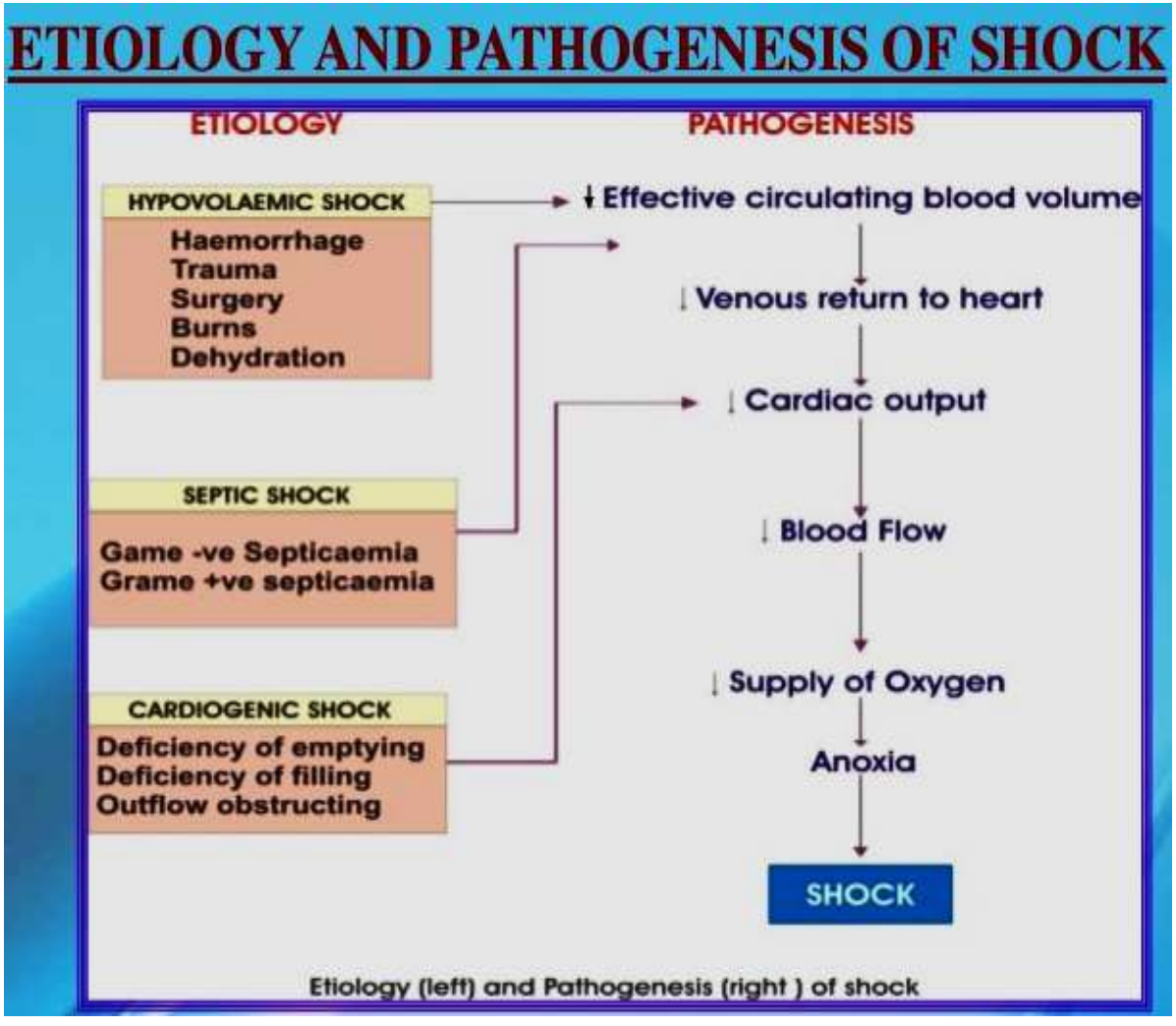
Shock

Definition: Circulatory shock means inadequate tissue perfusion with blood due to decreased CO & ABP.



-Types and causes of shock:

(I)Low-resistance shock: (primary shock) (Normo-volumic shock):

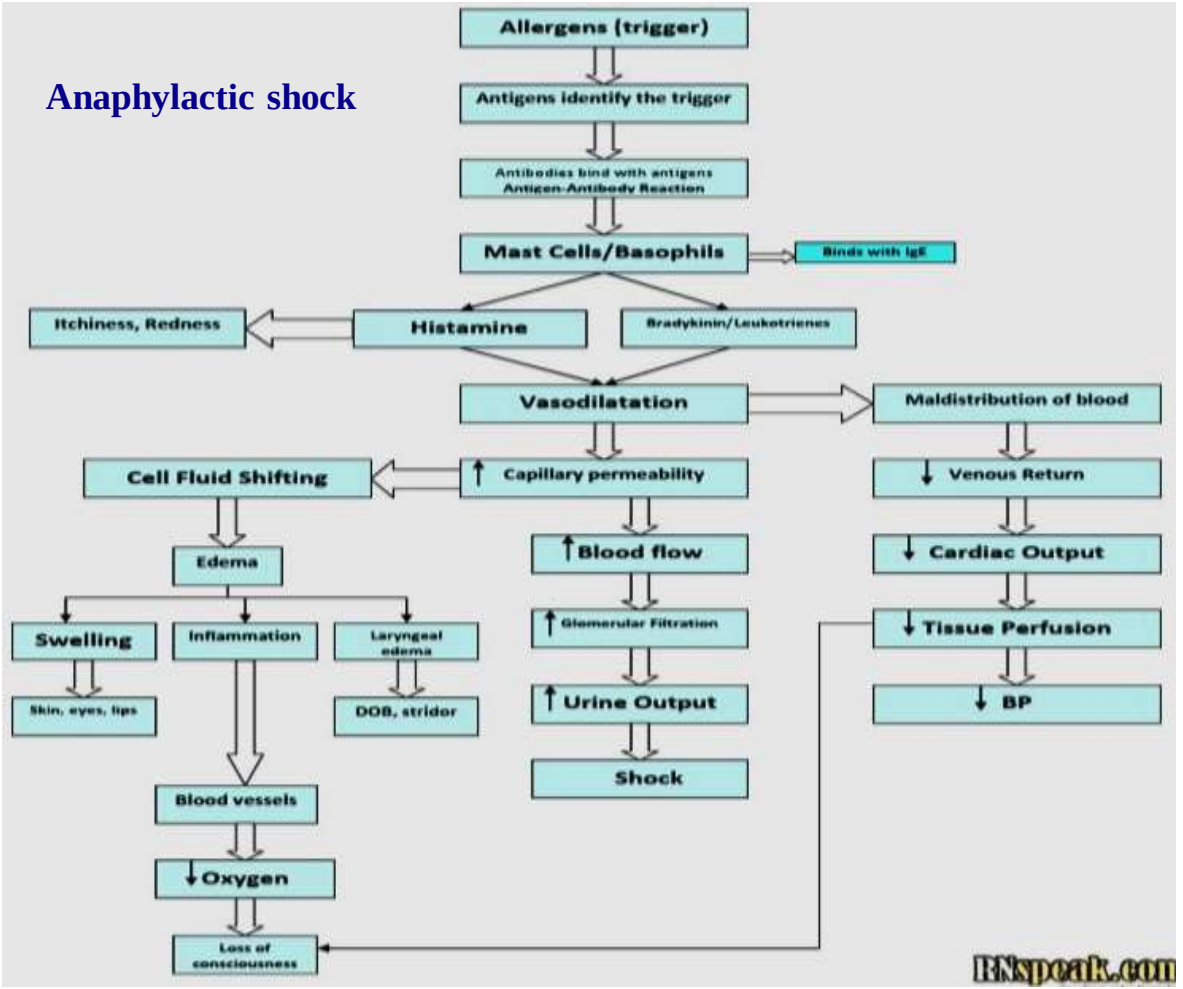
It is caused by severe VD (bl. volume is normal) - e.g.:

(1) Neurogenic shock:

Sever emotions (vago-vagal syncope)→ vaso& venodilatation of skeletal blood vessels & bradycardia → ↓ ABP and shock.

(2) Anaphylactic shock:

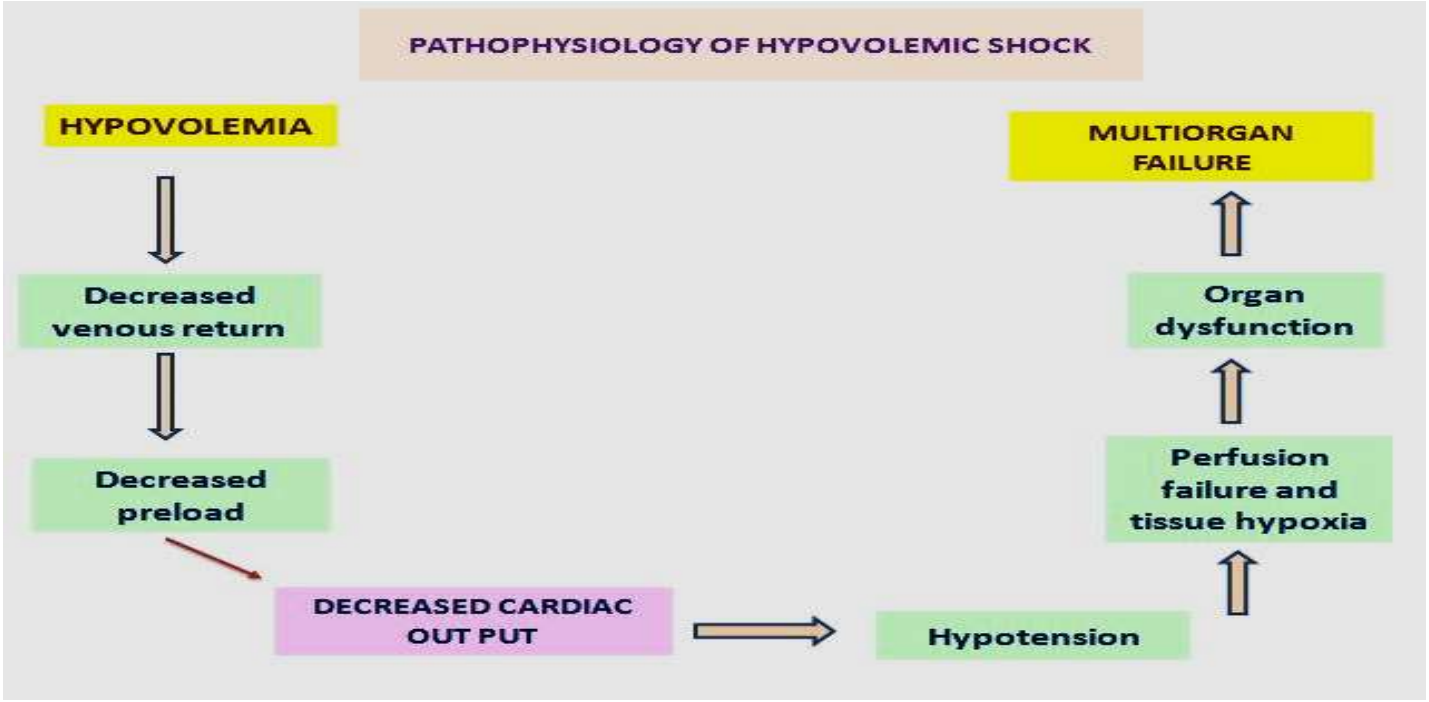
Due to exaggerated antigen-antibody reaction with release of histamine or kinin causing vasodilation with drop in blood pressure.



(3) Septic shock:

Severe infection → bacterial endotoxin →depress the vasomotor center with resulted VD of arterioles and capillaries →↑ capillary permeability → ↓ blood pressure → shock

(II) *Hypovolemic shock:* (Secondary shock) (Cold shock)



Caused by **loss of blood or plasma or extracellular fluid**. e.g.

(1)Post- haemorrhagic shock with failure of compensatory mechanisms.

(2) Burn shock: loss of plasma (**Exeamia**) & VD.

(3)Traumatic shock: Haemorrhage, pain, loss of plasma to tissue.

(4) Dehydration: severe vomiting, diarrhea or sweating.

(III) *Cardiogenic shock:*

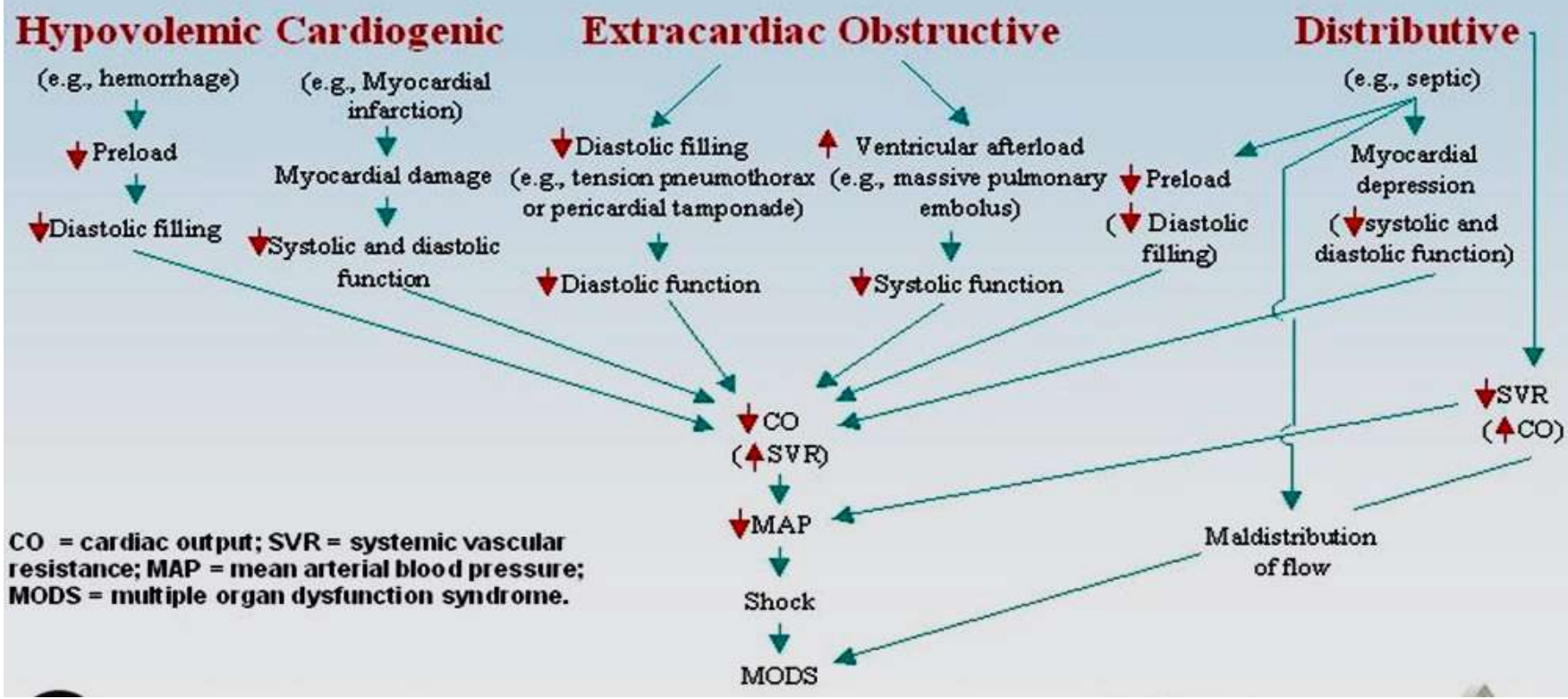
As in infarction, heart failure or arrhythmia → ↓ CO → shock.

(IV) *Obstructive shock*:

due to obstruction of the blood flow at the centers of circulation which hinders blood flow to tissue: -In the **lung**: as in cases of the pulmonary embolism, thrombosis, and tension pneumothorax with marked elevation of the intrathoracic pressure.

-In the **heart** : as in cardiac tamponade (massive pericardial effusion) with fibrosis and constrictive pericarditis, which prevent cardiac filling and contraction.

Classification of Shock



-Prognosis of shock:

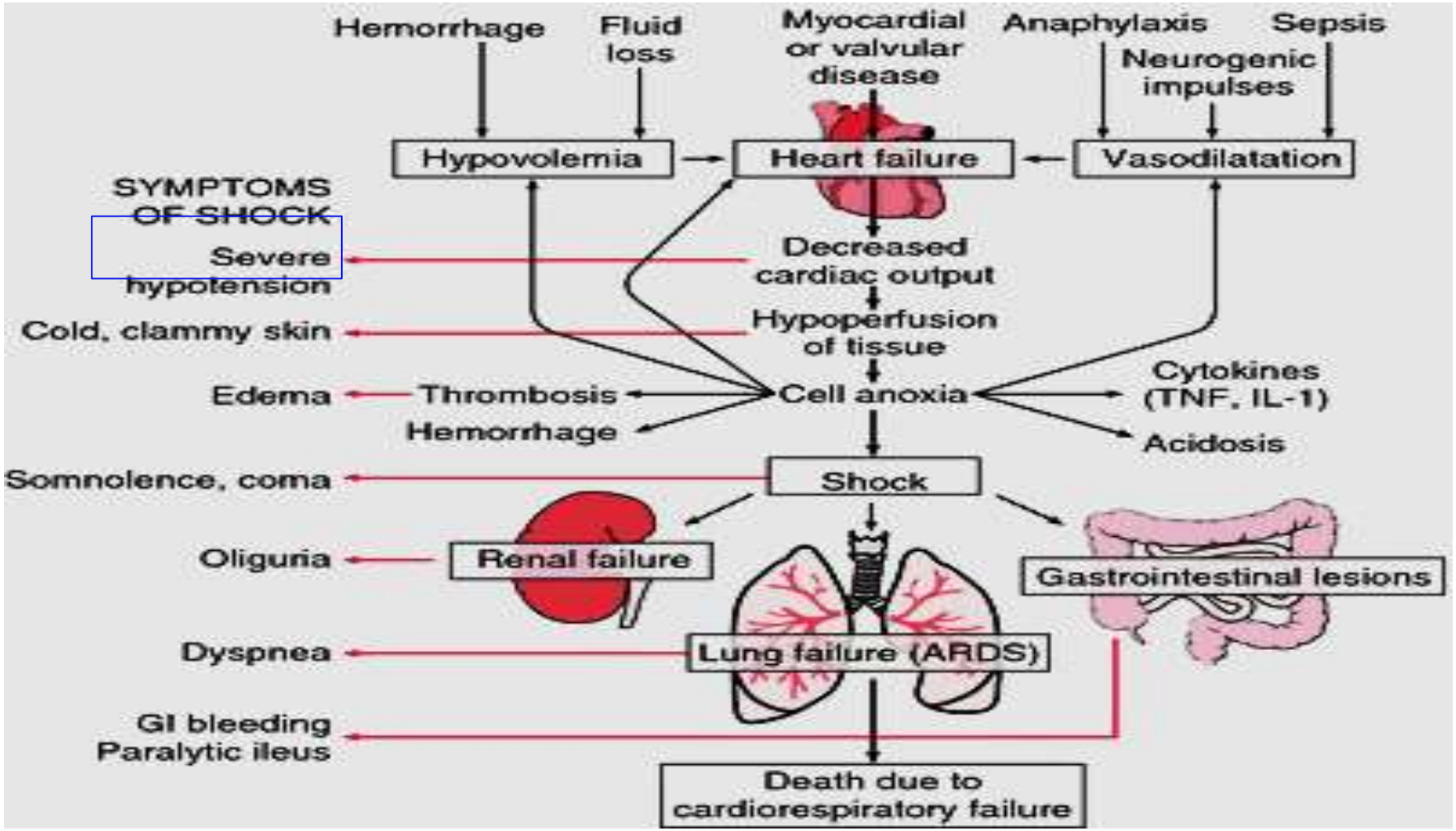
Its severity depends largely on the degree and rate of blood pressure drop and it may be either:

(A) Reversible (compensated) shock:

The compensatory mechanisms (immediate and delayed) gradually restore the ABP up to normal level in negative feedback control.

(B) Irreversible (Refractory or decompensated) shock:

This occurs in severe causes of shock and the patient not be treated for about 3-5 hours → progressive decrease in cardiac output and ABP in a +ve feed back mechanisms .


Hemorrhage
Fluid loss
Myocardial or valvular disease
Anaphylaxis
Sepsis
Neurogenic impulses
Hypovolemia
Heart failure
Vasodilatation
SYMPTOMS OF SHOCK
Severe hypotension
Decreased cardiac output
Cold, clammy skin
Hypoperfusion of tissue
Edema
Thrombosis
Hemorrhage
Cell anoxia
Cytokines (TNF, IL-1)
Acidosis
Somnolence, coma
Shock
Oliguria
Renal failure
Gastrointestinal lesions
Dyspnea
Lung failure (ARDS)
GI bleeding
Paralytic ileus
Death due to cardiorespiratory failure

***Mechanisms that lead to death in refractory shock:**

(1) Cardiac depression:

• Severe decrease in ABP → coronary blood flow → myocardial ischemia → cardiac contraction →COP → ABP and so on → myocardial infarction.

• Cardiac depression by myocardial toxic factor or other bacterial toxins released during shock.

(2) Cerebral depression:

• Severe decrease in ABP → cerebral bl. flow → depression of vasomotor center → no correction of decreased ABP → more decrease in ABP & so on → cerebral damage.

(3) Dilatation of precapillary sphincter:

• After haemorrhage → reflex sympathetic spasm of precapillary sphincters and venules especially in splanchnic area, after that dilatation of precapillary sphincter occurs by metabolites or toxins but venules remaining constricted → ↓ VR→ more decrease in bl. pr →more spasm of venules → more ↓ VR .

• ↑ Capillary filtration → ↑ loss of plasma in tissue space → ↓ bl. volume → ↓ VR → ↓ COP → ischemia of the capillary wall → more filtration.

(4) Release of toxins by ischemic tissues:

• Myocardial toxic factor:

Extreme pancreatic ischemia → trypsin enzyme is released from pancreas → degeneration of pancreatic tissue → release of myocardial toxic factor → direct depression of the heart contractility.

•Endotoxin: released from intestinal bacteria under ischemia → absorbed to bl. → severe VD and cardiac depression → severe shock.

•Free radicals: ↓ COP → tissue hypoxia→ injury of vessels → adherence of granulocytes to vessels→ free radicals which causes more damage of vessels and more adherence of granulocytes and more free radicals and so on.

(5)Thrombosis of small vessels: due to sluggish circulation with activation of clotting factors and platelet aggregation. This leads to more tissue ischaemia.

(6) Acidosis: ↓O2 supply → lactic acid accumulation also ↑ CO2 → H2CO3.
This acidosis leads to tissue damage and activation of intracellular proteolytic enzymes with auto-destruction.
(7) Acute respiratory failure: due to damage of capillary endothelium and alveolar epithelium in the lung with release of cytokines.
(8) Acute renal failure: due to:
-Severe **renal vasoconstriction** causes renal ischaemia and tubular necrosis.
-Muscular tissue damage leading to accumulation of **myoglobin** which enhance the damage in the kidney tissue with **decrease renal plasma flow** and **glomerular filtration rate** and the renal functions are severly impaired with uraemia and anurea.
*** Treatment of shock:** Treatment of the **cause**…….
1) Warming the body (in hypovolemic shock) and raising the lower limb by 30 cm →↑ VR.
2) O2 therapy and glucose, saline IV injection.
3) Keep open air way and guard against pneumonia
4) Low resistance shock is treated by: Corticosteroids, Anti-histamincs, sympathomimetics.

Thank You