# SHOCK


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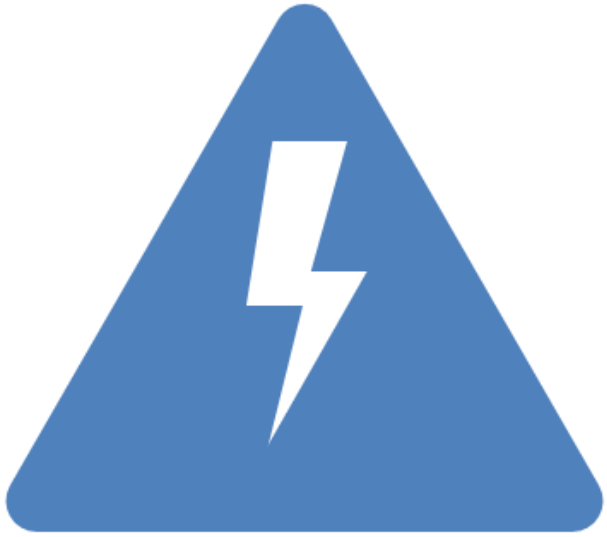
# Agenda

- Definition
- Types of Shock
- Pathogenesis and Causes
- Stages of shock
- Clinical features
- Complications



# SHOCK

Shock is a life-threatening clinical syndrome of cardiovascular collapse characterized by: an acute reduction of effective circulating blood volume (hypotension) an inadequate perfusion of cells and tissues (hypoperfusion)

# Types of shock

**Initial OR Primary Shock :**

- Immediately following trauma emotional over reaction such as due to fear, sorrow or surprise

**True OR Secondary OR Circulatory Shock**

- circulatory imbalance

# Initial OR Primary Shock

vasovagal attack resulting from sudden reduction of venous return to the heart caused by neurogenic vasodilatation and consequent peripheral pooling of blood

Attack lasting for a few seconds or minutes and develop

Unconsciousness, weakness, sinking sensation, pale and clammy limbs, weak and rapid pulse, and low blood pressure

True OR Secondary OR Circulatory shock

Circulatory imbalance between oxygen supply and oxygen requirements at the cellular level

If uncompensated, these mechanisms may lead to

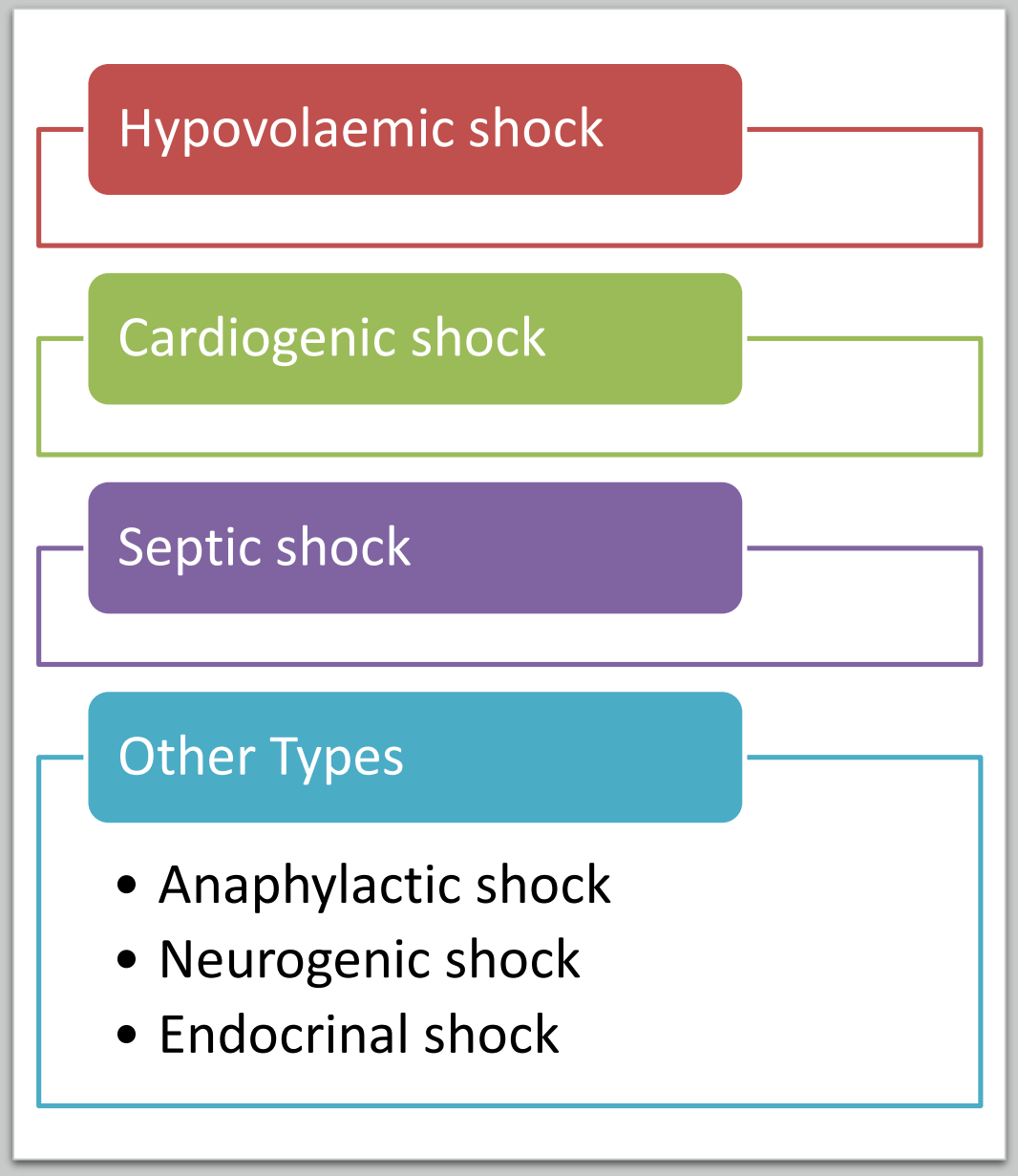
| | |
|---|---|
| circulatory imbalance between oxygen supply and oxygen requirements at the cellular level | impaired cellular metabolism and death |

# Circulatory shock

O2 DEMAND

O2 SUPPLY

# Types of shock



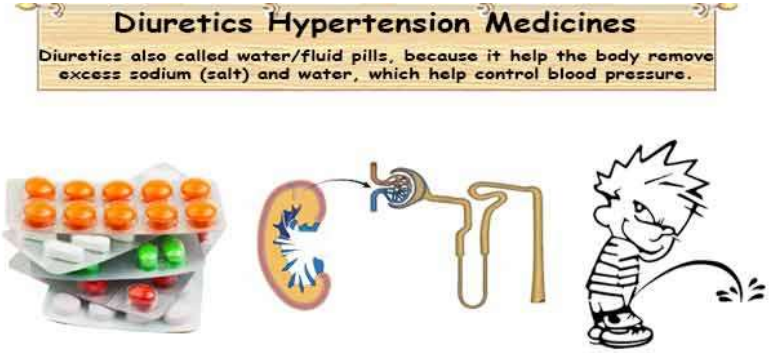
# Classification and Etiology Hypovolaemic shock

Results when anyone lose more than 20 % of there body's blood or fluid supply

This severe fluid loss makes it impossible for the heart to pump a sufficient amount of blood to circulation








# Causes of Hypovolaemic shock

# PATHOGENESIS OF HYPOVOLAEMIC SHOCK

Hypovolaemic shock occurs from inadequate circulating blood volume

The major effects of decreased cardiac output and low intracardiac pressure

The severity of clinical features depends upon degree of blood volume lost, haemorrhagic

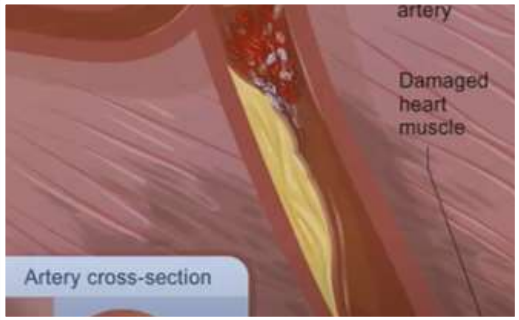
Shock is divided into 4 types:

| Compensated | Mild | Moderate | Severe |
|---|---|---|---|
| < 1000 ml | 1000-1500 ml | 1500-2000 ml | >2000 ml |

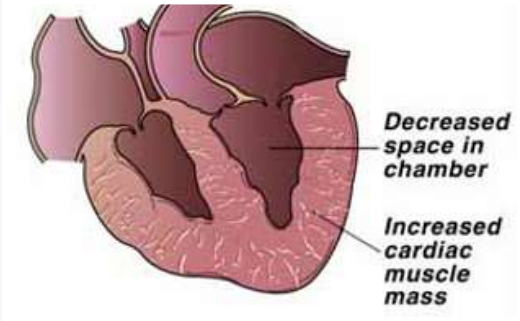
# Cardiogenic shock

Acute circulatory failure with sudden fall in cardiac output from diseases of the heart without actual reduction of blood volume (normovolaemia) results in cardiogenic shock

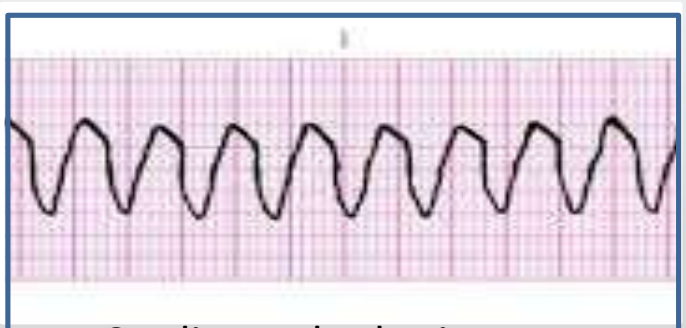
# Cardiogenic Shock
# Deficient Emptying




Myocardial infarction



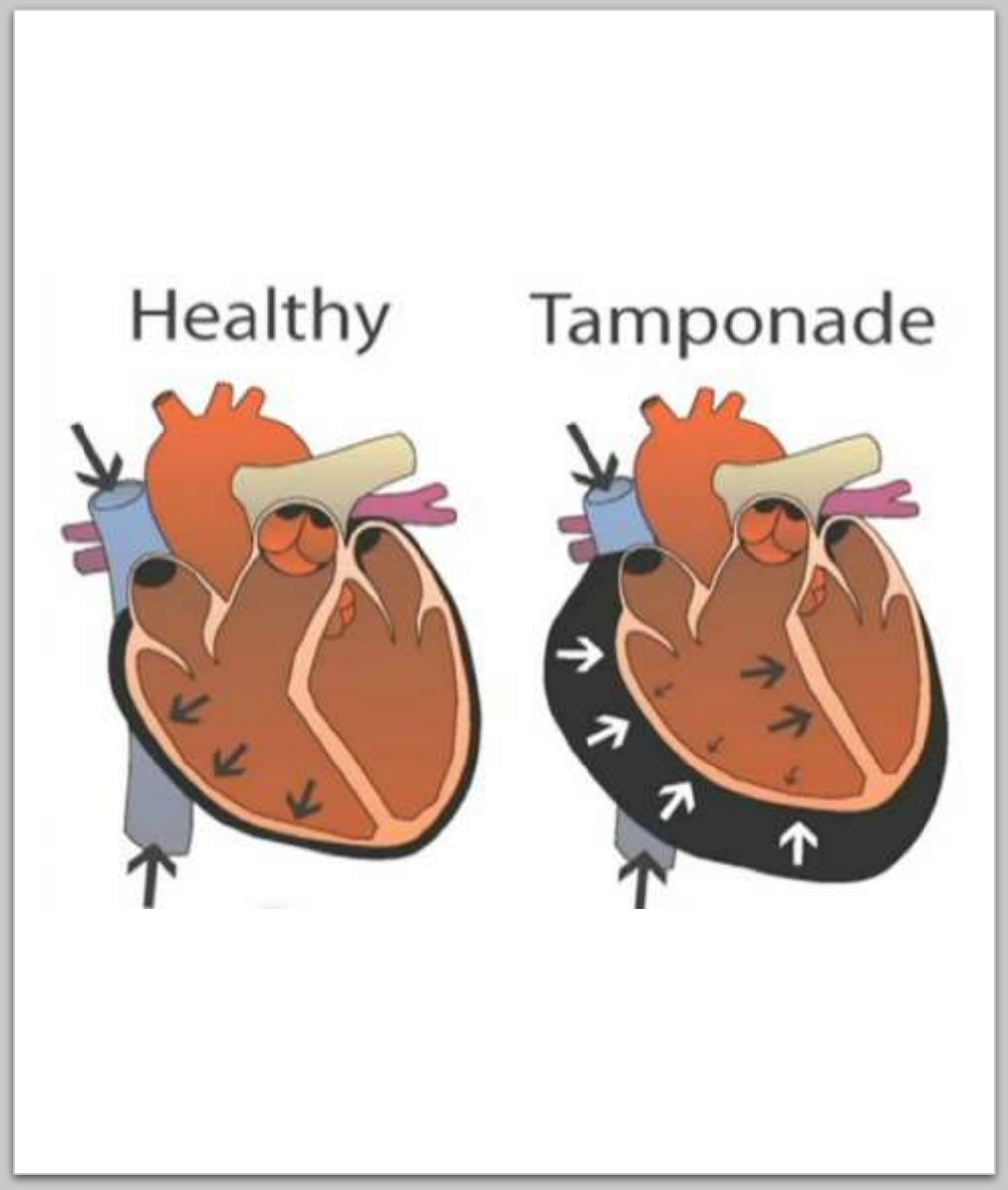

Cardiomyopathies



Cardiac arrhythmias

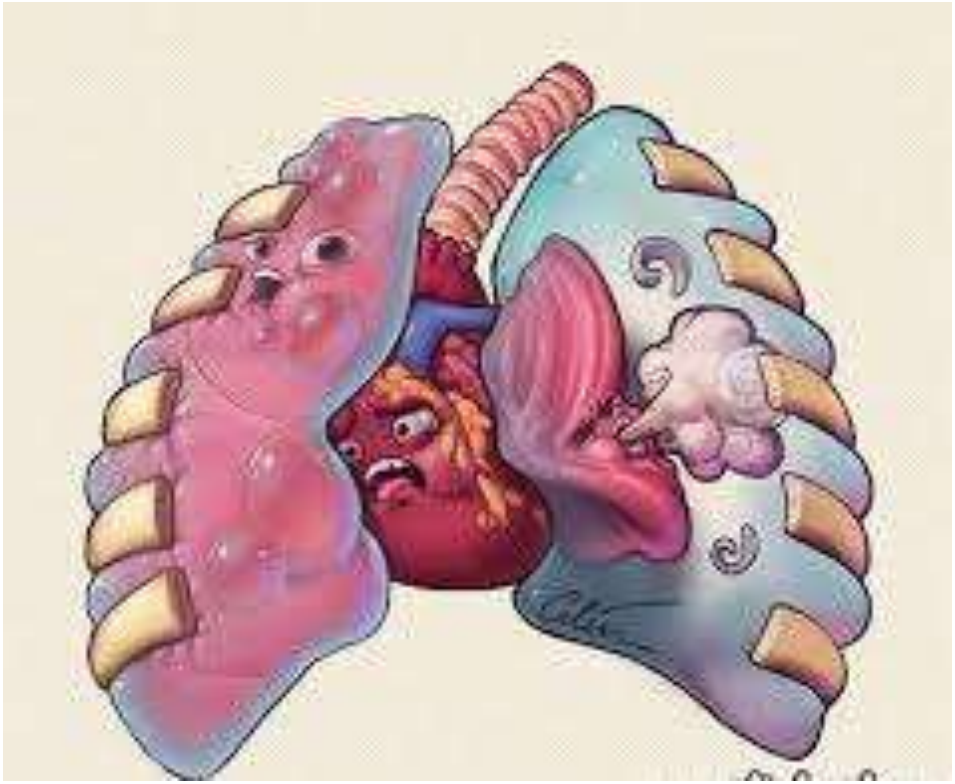
# *DEFICIENT FILLING*

**Cardiac tamponade from haemopericardium**



# Obstruction to the outflow

- Pulmonary embolism after DVT
- Ball valve thrombus
- Tension pneumothorax
- Dissecting aortic aneurysm



# PATHOGENESIS OF CARDIOGENIC SHOCK

- Cardiogenic shock results from a severe left ventricular dysfunction

↓ Cardiac output

↓ tissue perfusion and movement of fluid

- From pulmonary vascular bed into pulmonary interstitial space initially (interstitial pulmonary oedema) and later into alveolar spaces (alveolar pulmonary oedema)

# SEPTIC SHOCK

## Gram-negative septicaemia (endotoxic shock)

- Infection with E. coli, Proteus, Klebsiella, Pseudomonas .

## Gram-positive septicaemia (exotoxic shock)

- Infection with streptococci, pneumococci,Staphylococi.

# PATHOGENESIS OF SEPTIC SHOCK

Septic shock results most often from Gram-negative bacteria entering the body from genitourinary tract, alimentary tract, respiratory tract or skin, and less often from Gram-positive bacteria

In septic shock, there is immune system activation and severe systemic inflammatory response to infection

# PATHOGENESIS OF SEPTIC SHOCK

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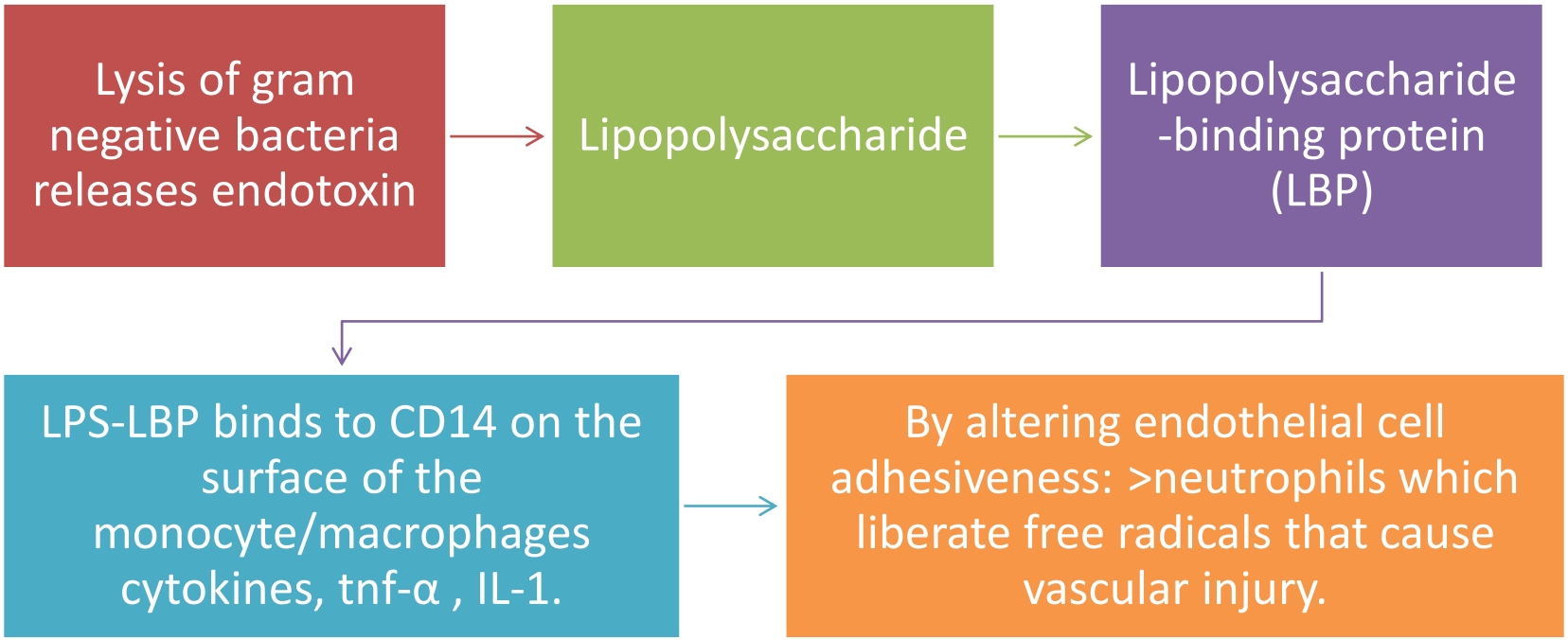
# Activation of macrophage-monocytes



# Activation of other inflammatory responses

## Activation of complement pathway

- C5a and c3a induce microemboli and endothelial damage.

## Activation of mast cells

- Histamine is released which increases capillary permeability.

## Activation of coagulation system

- Thrombi

## Activation of kinin system

- Released bradykinin cause vasodilatation &
- increased capillary permeability in septic shock

## Profound peripheral vasodilatation and pooling of blood causes

- Hyperdynamic circulation in septic shock

# At the septic shock

Increased vascular permeability causes development of inflammatory oedema

Disseminated intravascular coagulation (DIC) is prone to develop in septic shock due to endothelial cell injury by toxins.

Reduced blood flow produces hypotension, inadequate perfusion of cells and tissues, finally leading to organ dysfunction

# Pathophysiology (Stages of Shock)

Compensated (non progressive, initial, reversible) shock

Progressive decompensated shock

Irreversible decompensated shock

# COMPENSATED (NON-PROGRESSIVE, INITIAL, REVERSIBLE) SHOCK

To maintain adequate cerebral and coronary blood supply

by redistribution of blood so that the vital organs (brain and heart) are adequately perfused and oxygenated.

activation of various neurohormonal mechanisms causing

vasoconstriction and by fluid conservation by the kidney.

If the condition that caused the shock is adequately treated, recovery and reestablish the normal circulation REVERSIBLE

These compensatory mechanisms causes

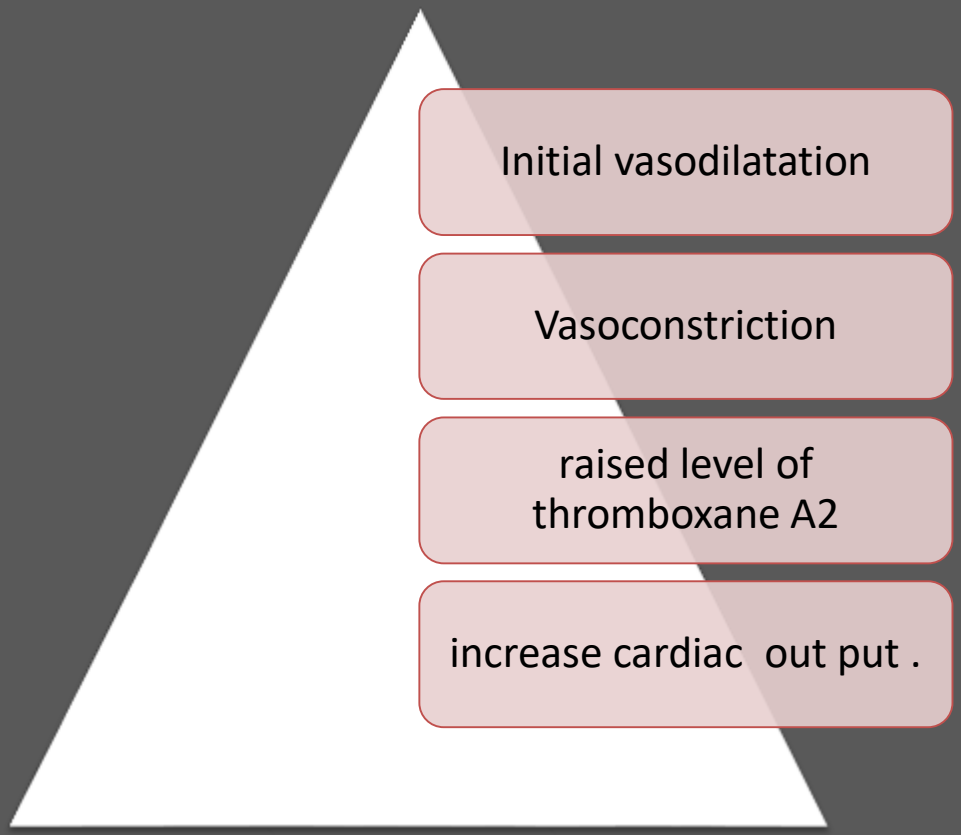
Vasoconstriction

Flud conservation

All these bring about vasoconstriction, particularly in the vessels of the skin and abdominal viscera. Widespread vasoconstriction is a protective mechanism as it causes increased peripheral resistance, increased heart rate (tachycardia) and increased blood pressure

Clinically cutaneous vasoconstriction is responsible for cool and pale skin in initial stage of shock

# Compensatory mechanism in septic shock



# Fluid conservation by the kidney

In order to compensate the actual loss of blood volume in hypovolaemic shock, to restoring the blood volume and improve venous return to the heart

Release of aldosterone from hypoxic kidney by activation of renin-angiotensin-aldosterone mechanism.

Release of ADH due to decreased effective circulating blood volume.

Reduced glomerular filtration rate (GFR) due to arteriolar constriction.

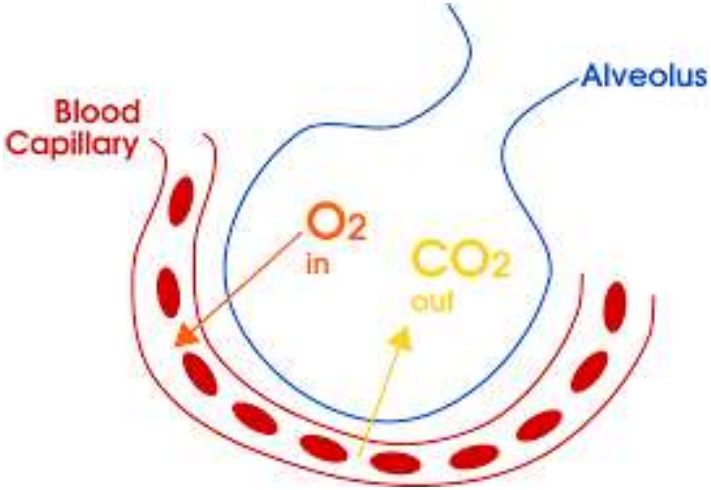


Shifting of tissue fluids into the plasma due to lowered capillary hydrostatic pressure (hypotension). Stimulation of adrenal medulla.

In response to low cardiac output, adrenal medulla is stimulated to release excess of catecholamines (epinephrine and non-epinephrine) which increase heart rate and try to increase cardiac output.

# PROGRESSIVE DECOMPENSATED SHOCK

- Previous H/O-cardiac disease, COPD like diseases so that there is progressive deterioration. The effects of progressive decompensated shock due to tissue hypoperfusion are as under:
- **iPulmonary hypoperfusion**
  - Decompensated shock worsens pulmonary perfusion and increases vascular permeability resulting in tachypnoea and adult respiratory distress syndrome (ARDS)
- **Tissue ischaemia**
  - Impaired tissue perfusion causes Switch from aerobic to anaerobic glycolysis resulting in Metabolic lactic acidosis
  - Lactic acidosis lowers the tissue pH which in turn makes the vasomotor response ineffective
  - This results in vasodilatation and peripheral pooling of blood
  - Clinically at this stage the patient develops confusion and worsening of renal function



# IRREVERSIBLE DECOMPENSATED SHOCK

When the shock is so severe & no recovery takes place, it is called decompensated or irreversible shock.

Its effects due to widespread cell injury include the following:

- Progressive vasodilatation.
  - During later stages of shock, anoxia damages the capillary and venular wall and arterioles become unresponsive to vasoconstrictors listed above and begin to dilate. Vasodilatation results in peripheral pooling of blood which further deteriorate the effective circulating blood volume.
- Increased vascular permeability.
  - Anoxic damage to tissues releases inflammatory mediators which cause increased vascular permeability. This results in escape of fluid from circulation into the interstitial tissues thus deteriorating effective circulating blood volume.

# Irreversible shock

## Myocardial depressant factor (MDF).

- Progressive fall in blood pressure and persistently reduced blood flow to myocardium → coronary insufficiency and myocardial ischaemia due to release of myocardial depressant factor (MDF)→ reduced cardiac output and decreased blood flow.

## Worsening pulmonary hypoperfusion.

- Further pulmonary hypoperfusion causes respiratory distress due to pulmonary oedema, tachypnoea and adult respiratory distress syndrome (ARDS).

# Irreversible shock

## Anoxic damage to heart, kidney, brain.

- Progressive tissue anoxia causes severe metabolic acidosis due to anaerobic glycolysis.
- There is release of inflammatory cytokines and other inflammatory mediators and generation of free radicals.
- Since highly specialised cells of myocardium, proximal tubular cells of the kidney, and neurons of the CNS are dependent solely on aerobic respiration for ATP generation, there is ischaemic cell death in these tissues.

## Hypercoagulability of blood.

- Tissue damage in shock activates coagulation cascade with release of clot promoting factor, thromboplastin and release of platelet aggregator, ADP, which contributes to slowing of blood-stream and vascular thrombosis. In this way, hypercoagulability of blood with consequent microthrombi impair the blood flow and cause further tissue necrosis

# Clinical features of Shock

**Mild shock**

Collapse of subcutaneous veins of extremities especially hand and feet ,

pale face, sunken eyes, weakness,

cold and clammy skin.

Sweat on forehead ,hand & feet .

Urine output ,B p Pulse rate normal .

Patient feels thirsty .

**Moderate Shock**

mild shock features + drowsy & confused oliguria

pulse rate increase< 100/min

Bp normal initaly then fall down in later stage .

**Severe shock**

Unconscious,

Shallow and sighing respiration

, Feeble and irregular pulse,

Anuria ,Rapid pulse, Profound hypotension

**Multi organ failure**

Acute respiratory distress syndrome (ARDS)

,Disseminated intravascular coagulation (DIC) ,Acute renal failure (ARF) Multiple organ dysfunction syndrome (MODS)With progression of the condition, the patient may develop stupor, coma and death

# Mnemonic for shock

**I**nitially

**S**econdary

**H**igh

**S**chool and

**C**olleges try to

**C**ompensate his fee but his

**P**arent chose to

**D**onate it

# References


- Textbook of Pathology by Robins
- Textbook of Pathology by Harsh Mohan
- https://www.slideshare.net

# THANK YOU

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