

Shock:

Shock is a condition of severe impairment of tissue perfusion leading to cellular injury & dysfunction

→ most common cause of death among surgical patients

↳ a systemic state of low tissue perfusion that is inadequate for normal cellular respiration.

With insufficient delivery of oxygen & glucose, cells switch from aerobic to anaerobic metabolism.

If perfusion is not restored in a timely fashion, cell death ensues.

Cellular Pathophysiology:

Reduced tissue perfusion

Oxygen deprivation

Cells switch from aerobic to anaerobic respiration

Production of lactic acid
[Lactic acidosis]

Glucose within cells gets exhausted

anaerobic respiration ceases

Failure of Na^+/K^+ ATPase pump in cell membrane & organelles

Intracellular lysosomes release autodigestive enzymes causing cell lysis

Intracellular contents (including K^+) are released into the bloodstream.

Microvascular Pathophysiology:

Progression of tissue ischaemia

Changes in local milieu

Activation of immune & coagulation systems

Hypoxia, acidosis

Complement & prime leukocyte activation

Generation of oxygen free radicals, cytokines

Injury of capillary endothelial cells

Activation of immune & coagulation systems

Damaged endothelium loses integrity & becomes leaky

Spaces between endothelial cells allow fluid to leak out causing tissue edema

Exacerbation of cellular hypoxia

Systemic Pathophysiology:

Cardiovascular

- reduced preload & afterload
- compensatory baroreceptor reflex action
- increased sympathetic activity



increased release of catecholamines into circulation

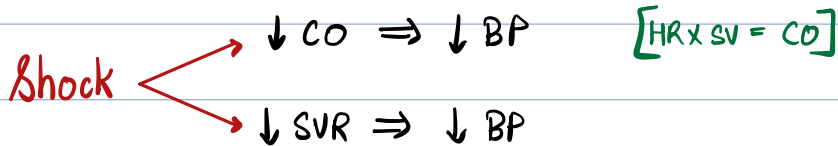
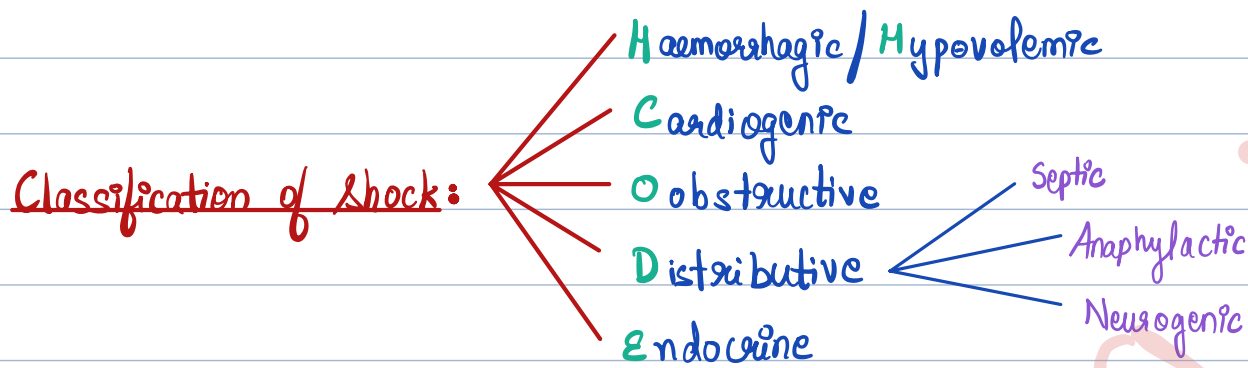
- Tachycardia
- Systemic vasoconstriction (except sepsis)

Respiratory

- metabolic acidosis
- increased sympathetic response \Rightarrow increased respiratory rate, minute ventilation
- compensatory respiratory alkalosis \Rightarrow to increase CO_2 excretion.

Renal

- decreased perfusion pressure of kidney
- reduced filtration at glomerulus, decreased urine output
- stimulation of RAAS.
- reabsorption of Na^+ , H_2O from collecting systems.



Hypovolemic Shock:

due to reduced circulating volume

→ hypovolemia can have haemorrhagic & non-haemorrhagic causes.

Non - Haemorrhagic Causes:

- poor fluid intake
- 3rd degree burns
- bowel obstruction
- excessive fluid loss due to vomiting, diarrhoea, urinary loss (diabetes)
- acute pancreatitis
- diabetic ketoacidosis (glucosuria, polyuria)

Haemorrhagic Causes:

- GI bleed
- Trauma
- Ectopic pregnancy
- Abdominal aortic aneurysm
- Post partum haemorrhage
- Hemoptysis

→ regardless of the cause of shock, hypovolemia must be treated

↓ blood volume



sensed by baroreceptors in aortic sinus & carotid body



stimulate the medullary centres

Release of norepinephrine
at vascular smooth
muscles

↓
vasoconstriction

↓
↑ SVR

↓
↑ BP

↑ contractility of heart
& ↑ HR



attempt at ↑ CO;
but diastolic filling is low
∴ no ↑ in CO

Manifestations:

- ↓ CO
- ↑ SVR
- ↑ HR
- CBC $\begin{cases} \uparrow \text{PCV} \Rightarrow \text{hemoconcentration due to excess fluid (plasma) loss} \\ \downarrow \text{PCV} \Rightarrow \text{hemodilution due to loss of blood} \end{cases}$
- Cyanosis
- Hypoxia

Management: IV

- Crystalloids (normal saline, Ringer's Lactate)
- Colloids (albumin / hetastarch) \Rightarrow to stabilize plasma oncotic pressure

Cardiogenic Shock:

due to primary failure of heart to pump blood to tissues

Causes:

- MI
- Cardiac dysrhythmia
- Valvular heart disease
- Blunt myocardial injury
- Cardiomyopathy
- Endogenous factors - bacterial or humoral agents released in sepsis
- Exogenous factors - pharmaceutical agents / drug abuse.

Obstructive Shock:

due to reduction in preload owing to mechanical obstruction of cardiac filling

Causes:

- Cardiac tamponade
- Tension pneumothorax
- Massive pulmonary embolus

→ Reduced filling \Rightarrow low cardiac output

Distributive Shock:

- pattern of cardiovascular responses characterized by
 - shock
 - anaphylaxis
 - spinal cord injury
- inadequate organ perfusion + vascular dilatation, hypotension, low systemic vascular resistance, inadequate afterload \Rightarrow High cardiac output
- In anaphylaxis, vasodilation is due to histamine release
- In spinal cord injury - failure of sympathetic outflow, adequate vascular tone
[Neurogenic Shock]
- In sepsis, release of bacterial endotoxins & activation of cellular & humoral components.
- • Maldistribution of microvascular blood flow
 - Arteriovenous shunting
 - Cellular dysfunction in utilizing O_2
 - In later phase of septic shock \Rightarrow hypovolemia due to fluid loss into interstitial spaces.

Endocrine Shock:

may present as a combination of Hypovolemic, Cardiogenic or Distributive Shock

Causes:

- Hypothyroidism
- Hyperthyroidism
- Adrenal insufficiency

state of shock due to disorder vascular, cardiac responsiveness to circulating catecholamines

→ decreased cardiac output, bradycardia

(thyrotoxicosis causes high output cardiac failure)

→ Adrenal insufficiency (Addison's disease / Sepsis) ⇒ hypovolemia, poor response to circulating catecholamines

Effects of Organ Failure:

- Heart - CV failure
- Lung - ARDS [acute respiratory distress syndrome]
- Kidney - ARI [acute renal injury]
- Liver - liver failure, coagulopathy
- Brain - cerebral swelling & dysfunction

Clinical Consequences of Shock :

- unresuscitatable shock
- ischaemia (reperfusion & SIRS)
- multiple organ failure

Unresuscitatable Shock : patients in profound shock for a prolonged period of time.

- Cellular ischaemia \Rightarrow cannot compensate \Rightarrow cell death
- Heart - myocardial cell death due to poor coronary perfusion, myocardial depression due to acidemia, hyperkalemia
- poor CO - limited response to fluid & inotropic therapy.
- \downarrow SVR \Rightarrow \downarrow BP
- Systemic ischaemic injury \Rightarrow Inevitable death.

Ischaemia - Reperfusion & SIRS:

- during systemic hypoperfusion, cellular & organ damage progresses (owing to direct effects of tissue hypoxia & local activation of inflammation)
- further injury occurs once normal circulation is restored to these tissues (reperfusion injury)
- built-up acid & potassium can lead to myocardial depression, vasodilation & \therefore further hypotension

Ischaemia - Reperfusion Injury: \rightarrow contributes to tissue damage during myocardial & cerebral infarction following therapies that restore blood flow.

- Restoration of blood flow to ischaemic tissues can promote recovery of cells if they are reversibly injured, but can also paradoxically exacerbate cell injury & cause cell death

Mechanisms:

- OXIDATIVE STRESS \Rightarrow New damage may be initiated during reoxygenation by increased generation of ROS & RNS (reactive nitrogen species)
- INTRACELLULAR CALCIUM OVERLOAD
- ACTIVATION OF COMPLEMENT SYSTEM \Rightarrow some IgM antibodies have a propensity to deposit in ischaemic tissue
 \Rightarrow on reperfusion, circulating complement proteins bind to the deposited antibodies

Multiple Organ Failure :

- Result of prolonged systemic ischaemia & reperfusion injury is end organ damage & multiple organ failure.
- Multiple organ failure = ≥ 2 failed organ systems
- Management - support of organs, ventilation, cardiovascular support, hemodialysis
- Mortality of multiple organ failure = 60%

Compensated Shock:

- as shock progresses, cardiovascular & endocrine compensatory responses reduce flow to non-essential organs to preserve preload & improve blood flow to lungs, brain, kidney.
- Tachycardia, cold peripheries (but this state is only maintained by reducing perfusion to skin, muscle, gut where cells respire anaerobically to sustain ischaemic damage)

Decompensated Shock:

- further loss of circulating volume overload's the body's compensatory mechanisms leading to progressive decompensation.
- Loss of 15% circulating blood volume is within normal compensatory mechanisms
- BP falls after 30-40% of circulating blood volume is lost.

Mild (Compensated) Shock:

- initially • tachycardia • tachypnoea • mild reduction in urine output • ^{mild} anxiety.
- BP = maintained, PP = decreasing
- cool, sweaty peripheries (except septic distributive shock)

Moderate shock:

- as shock progresses, renal compensatory mechanisms fall - renal perfusion falls, urine output dips below 0.5 ml/kg/hr
- tachycardia, falling BP, drowsy, mildly confused.

Severe Shock:

- profound tachycardia & hypotension
- urine output = zero
- unconscious, laboured respiration

Clinical features:

- Capillary refill
- Tachycardia
- Blood Pressure

Capillary Refill: capillary refill time varies (not a specific marker)

- Hypovolemic shock \Rightarrow cool, pale peripheries & prolonged capillary refill time
- Distributive shock \Rightarrow warm peripheries & brisk capillary refill (despite profound shock)

Tachycardia: may not always accompany shock

- ↳ especially in patients on β -blockers / implanted pacemakers \Rightarrow cannot show tachycardia
- Trauma \Rightarrow haemorrhage \Rightarrow relative bradycardia

Blood Pressure: hypotension = last sign of shock.

- Children & fit young adults can maintain BP until final stages of shock by dramatic increase in stroke volume & peripheral vasoconstriction (\therefore normal BP even in profound shock)
- Elderly patients who are normally hypertensive may present with "normal" BP for the general population, but be hypovolemic & hypotensive relative to their usual BP.
- β -blockers prevent tachycardic response