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 lysozomes 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 <u>leaky</u>

↓

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 ⟹ increased respiratory rate, minute ventilation

→ compensatory respiratory alkalosis ⟹ to increase CO_2 excretion.

Renal

→ decreased perfusion pressure of kidney

→ reduced filtration at glomerulus, decreased urine output

→ stimulation of RAAS.

→ resorption of Na^+, H_2O from collecting systems.

Classification of Shock:

- Haemorrhagic / Hypovolemic
- Cardiogenic
- Obstructive
- Distributive
  - Septic
  - Anaphylactic
  - Neurogenic
- Endocrine

Shock
- ↓ CO ⇒ ↓ BP [HR x SV = CO]
- ↓ SVR ⇒ ↓ BP

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
  - ↑ PCV ⇒ hemoconcentration due to excess fluid (plasma) loss
  - or
  - ↓ PCV ⇒ hemodilution due to loss of blood
- Cyanosis
- Hypoxia

Management: IV

- crystalloids (normal saline, Ringer's Lactate)
- colloids (albumin / hetastarch) ⇒ 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 ⟹ 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 ⇒ 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 ⇒ 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 ⟹ cannot compensate ⟹ 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.

→ ↓ SVR ⇒ ↓ BP

→ Systemic ischaemic injury ⟹ 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 & ∴ further hypotension

Ischaemia-Reperfusion Injury: → 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 ⇒ New damage may be initiated during reoxygenation by increased generation of ROS & RNS (reactive nitrogen species)
- INTRACELLULAR CALCIUM OVERLOAD
- ACTIVATION OF COMPLEMENT SYSTEM ⇒ some IgM antibodies have a propensity to deposit in ischaemic tissue
  ⇒ 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, git 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 fail - 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 ⇒ cool, pale peripheries & prolonged capillary refill time

→ Distributive shock ⇒ warm peripheries & brisk capillary refill (despite profound shock)

Tachycardia: may not always accompany shock

↳ especially in patients on β-blockers / implanted pacemakers ⇒ cannot show tachycardia

→ Trauma ⇒ haemorrhage ⇒ 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 (∴ 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