Shock

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Definition

- A state of cellular and tissue hypoxia due to reduced oxygen delivery
- Shock is initially reversible, but may progress to irreversible organ dysfunction

Pathogenesis

- Characterised by systemic hypotension due to
  - Reduced cardiac output
  - Reduced blood volume
- Consequences are
  - Impaired tissue perfusion
  - Cellular hypoxia

Types

- Low blood flow
  - Cardiogenic
  - Hypovolemic
- Maldistribution of blood flow
  - Septic
  - Anaphylactic
  - Neurogenic

Cardiogenic shock

- The heart cannot pump enough blood to meet the body's needs
- Decreased cardiac output and evidence of tissue hypoxia in the presence of adequate intravascular volume

Causes

- Myocardial infarction
- Cardiomyopathy
- Blunt cardiac injury
- Severe systemic or pulmonary hypertension
- Cardiac tamponade
- Myocardial depression from metabolic problems

MI

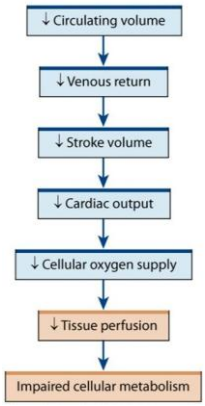
- Cardiogenic shock is the leading cause of death in acute myocardial infarction
- AMI leads to LV dysfunction + systemic hypoperfusion
- Stress on the LV mounts eventually lading to decreased cardiac output
- Decreased cardiac output worsens hypoperfusion and acidemia
- This process spirals creating a vicious cycle

Hypovolaemic shock

- Results when you lose more than 20 percent (one-fifth) of body's blood
- This severe fluid loss makes it impossible for the heart to pump a sufficient amount of blood to body

Causes

- Haemorrhage
- Non haemorrhagic
  - GI loss
  - Hyperglycemia
  - Severe burns

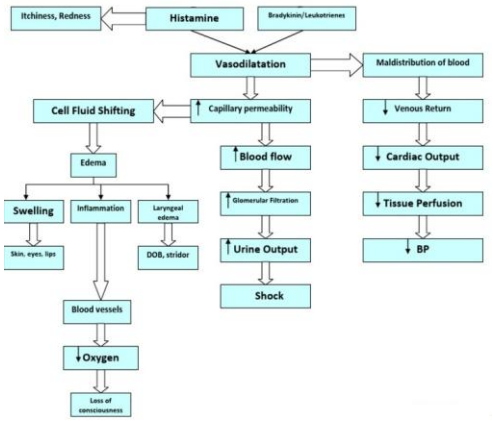


Neurogenic shock

- Neurogenic shock is caused by the sudden loss of the autonomic nervous system signals to the smooth muscle in vessel walls
- Loss of background sympathetic stimulation→ which is responsible for maintenance of tone of blood vessels
- Loss of vascular tone →vessels relax → sudden decrease in peripheral vascular resistance → decreased blood pressure

Anaphylactic shock

- Anaphylaxis is a severe systemic hypersensitivity reaction
- Systemic release of mediators from mast cells and basophils
- Histamine is the primary mediator of anaphylactic shock



Septic shock

- Sepsis, severe sepsis, septic shock
- Sepsis occurs when chemicals released into the bloodstream to fight the infection trigger inflammatory responses throughout the body. This inflammation can trigger a cascade of changes that can damage multiple organ systems, causing them to fail.
- If sepsis progresses to septic shock, blood pressure drops dramatically, which may lead to death.

Sepsis

- To be diagnosed with sepsis, you must exhibit at least two of the following symptoms, plus a probable or confirmed infection:
- Body temperature above 101 F (38.3 C) or below 96.8 F (36 C)
- Heart rate higher than 90 beats a minute
- Respiratory rate higher than 20 breaths a minute

Severe sepsis

- Your diagnosis will be upgraded to severe sepsis if you also exhibit at least one of the following signs and symptoms, which indicate an organ may be failing:
- Significantly decreased urine output
- Abrupt change in mental status
- Decrease in platelet count
- Difficulty breathing
- Abnormal heart pumping function
- Abdominal pain

Septic shock

- Signs and symptoms of severe sepsis
- Extremely low blood pressure that doesn't respond to fluid replacement
- Causative agents
  - Gram positive organisms
  - Gram negative organisms

Septic shock

- Systemic vasodilation & pooling of blood in the periphery → tissue hypoperfusion
- Widespread endothelial cell activation and injury→hypercoagulable state→DIC

Stages of shock

- Shock is a progressive disorder, if uncorrected, leads to death
- Shock evolves through 3 general phases
  - Nonprogressive phase
    - Reflex compensatory mechanisms are activated & perfusion of vital organs is maintained
  - Progressive stage
    - Tissue hupoperfusion & onset of worsening circulatory & metabolic imbalances, acidosis
  - Irreversible stage
    - Sets in after the body has incurred cellular & tissue injury so severe that even if the haemodynamic defects are corrected, survival is not possible

Compensation for shock

- Regardless of the cause, when tissues are hypoxic, the body attempts to recover by compensatory mechanisms
  - Neural
  - Hormonal
- The end goal – to increase COP & blood vessel tone for better O_2 supply

Compensatory mechanisms

- Acute compensatory mechanisms
  - Catecholamines
  - Cortisol
  - Transcapillary shifts
- Moderate compensatory mechanisms
  - Angiotensin II
  - Vasopressin
- Chronic compensatory mechanisms
  - Aldosteron
  - ADH

Catecholamines

- Mediated by the sympathetic nervous system
- COP↓→ impulses from BR at carotid sinus ↓
- Vasomotor center in brain → SNS signals to release→ norepineph from adrenals
- Hypoxia→chemo receptors (carotid artery)→stimulate VMC
- Norepinephrine
  - Vasoconstriction –cold clammy skin
  - Increase HR & contractility

Cortisol

- Stress→Hypothalamus →CRH →Pit→ACTH →Adrenals→ cortisol
- Glucose surge:
  - Energy to endothelial cells
  - Myocardial cells
  - Brain cells to function

Transcapillary shifts

- Shifting of fluid from the interstitium to the vasculature
- This occurs at the capillary level (esp in hypovol. Shock)
- Hypovolaemia→Pressure within capillaries ↓
  - Vasoconstrictive shunting of blood
  - Starling's forces
- Fluid will move from interstitium (high pressure) to vessels (low pressure)

Angiotensin II

- Happens a little later 10 min to 1 hour after the body enters the shock state
- Low COP→Low blood flow→BR in the JGA in renal glomerulus→Renin
- A.gen→A.sin I : A.sin I → A.sin II
  - Blood vessels→vasoconstriction
    - Redistribution of blood from splanchnic circ to brain & heart
  - Retains water and sodium

Vasopressin

- ↑Osmolality→OR in hypothalamus→Post pituitary
- Binds to V1 receptors in arterioles → Vasoconstriction

Alodosterone

- If the patient survives, the final stage of compenstation involves replacing blood volume
- Takes place from 1 to 48 hours
- A.sin II →adrenals→aldosterone
- Sodium reabsorption↑ in the distal convoluted tubule→ water follows sodium

ADH

- Vasopressin has another action on water
- Vasopressin→collecting ducts→reabsortption of water→↑blood volume

Progression

- If the underlying causes are not corrected shock passes to the progressive phase
  - Widespread tissue hypoxia
- Persistent O_2 deficit→intracellular aerobic respiration is replaced by anaerobic glycolysis & production of lactic acid

Irreversible decompensated shock

- Hypoxic endothelial cell injury
- DIC
- Widespread tissue anoxia
- Lyosomal enzyme leakage
- Complete renal shut down

Irreversible decompensated shock

- Clinically:
  - Anuria
  - Severe hypotension
  - Comatose state
  - Labored respiration
  - Increased pulse rate
  - Lactic acidosis
- Organ damage, metabolic disturbance are so severe that survival is not possible