Emergency and Critical Care

Acute and Septic Shock

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Objectives

- Describe the different types of shock.
- Describe the management of the patient experiencing shock.
- Describe the management of the patient in multisystem organ failure
- Describe the management of the patient in sepsis

Review previous lectures

- Describe the different types of shock:
  - Hypovolemic
  - Cardiogenic
  - Septic
  - Neurogenic
  - Anaphylactic
- List one cause for each type of shock
- What is the mechanism of action and dosing for vasoactive drugs:
  - Dopamine
  - Dobutamine
  - Noradrenaline/adrenaline
  - GTN/Nitroglycerine

Shock

‘momentary pause in the act of death’

If not treated & managed quickly and appropriately will be followed by the grim reality

John Collins Warren 1895

Shock - Definition

- Inadequate perfusion of tissues
- Tissue perfusion is inadequate to supply & nutrients to body cells
- imbalance between oxygen supply & demand results in functional impairment of cells, tissues, organs & eventually body systems

Classifications

- Hypovolaemic ‘empty tank’
- Cardiogenic ‘defective tank pump’
- Distributive ‘wrong size tank’
  - Septic
  - Neurogenic
  - Anaphylactic

Signs and Symptoms of Shock

- pallor
- cool/cold, clammy skin - good early sign
- weak, thready pulses
- tachycardia - only 60% of patients
- tachypnoea - good early sign
- hypotension - late sign
- postural hypotension - good early sign
- altered mental status

Stages of Shock

1. Initial
2. Compensatory
3. Progressive
4. Refractory

Initial Stage

- ↓ CO & tissue perfusion
- ↓ O2 delivery & other nutrients
- altered cellular function
- no S & S yet

Compensatory Stage

- Vascular response
  - Begins immediately
  - Peripheral vasoconstriction
  - Arterioles constrict
  - Body is attempting to increase blood pressure and improve venous return to the right atrium

Compensatory stage

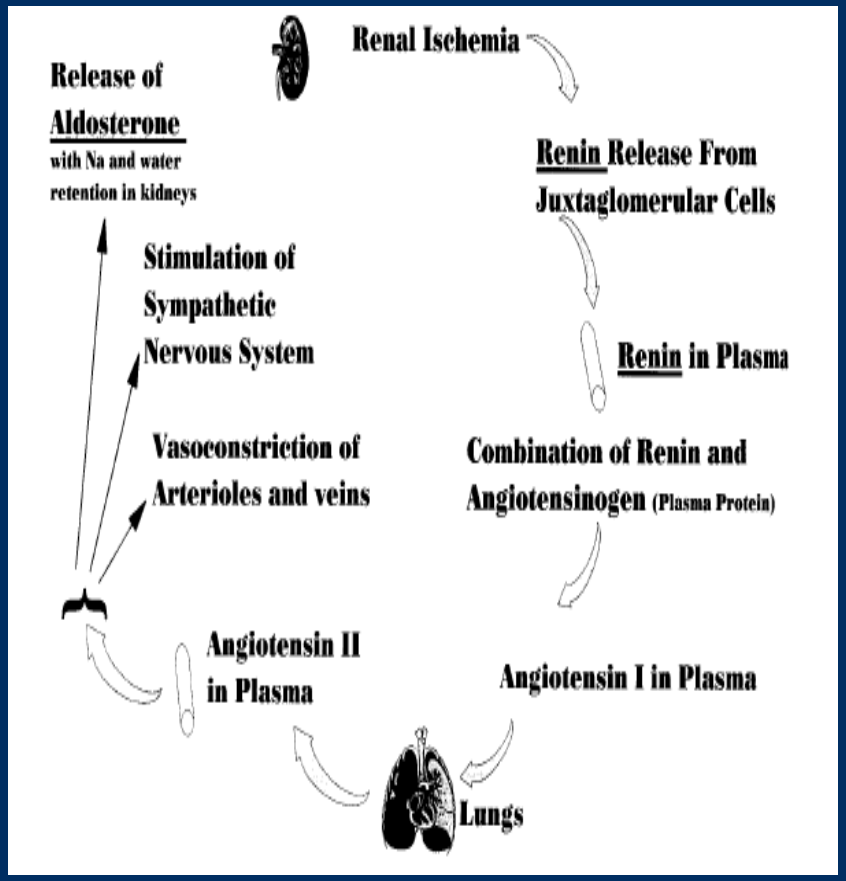
- Chemoreceptor response
  - Occurs as a result of low levels of oxygen and high levels of carbon dioxide
  - Occurs with arterial blood pressure less than 80 mmHg
  - Result is vasoconstriction
  - Begins within seconds of changes in blood pressure

Compensatory stage

- Cerebral response
  - Goal is to maintain perfusion to brain, heart, and lungs
  - Brain and heart blood vessels autoregulate blood flow based on needs of the tissues
  - Blood pressures below 50 mmHg leads to cerebral ischemia

Compensatory stage

- Renal response
  - Blood flow is decreased to the kidneys, which activates the release of renin.
  - Vasoconstriction of arterioles and some veins
  - Retention of water by kidneys
  - Decreased urinary output



Compensatory stage

- Adrenal response
  - Increased release of catecholamines (epinephrine and norepinephrine)
    - Goal is to increase cardiac output and improve blood pressures
    - Causes vasoconstriction
  - Cortisol is released
    - Increased blood sugar and increased insulin resistance
    - Kidneys retain water and sodium

Compensatory stage

- Hepatic response
  - Glycogneolysis is activated by release of epinephrine, break glycogen down into glucose
  - Hepatic vessels constrict

Compensatory stage

- Pulmonary response
  - Tachypnea
    - Attempt to correct metabolic acidosis
    - Maximize oxygen delivery to the aveoli

Progressive Stage

- compensatory mechanisms begin to fail
- loss of autoregulation
- ↓ BP
- ↓ coronary artery perfusion
- ↑ myocardial O_2 consumption exceeds O_2 supply
- myocardial depression - failure
- ↓ cerebral blood flow - LOC

Progressive Stage

- severe hypoperfusion of tissue/organs
- anaerobic metabolism - acidosis
- ↓ renal blood flow - ↓UO – acute tubular necrosis
- increased capillary permeability
- spillage of cellular contents
- acute pulmonary oedema, respiratory failure, arrthymias
- cell death

Refractory Stage

- severe cell destruction
- multiple system failure
  - cardiac, renal, hepatic, pancreatic, intestinal, haematological, neurological
- non-responsive to conventional treatment
- severe hypoxaemia refractory to O2 therapy
- ultimately death