Q2 September 2009

Describe the physiological consequences and responses after an acute haemorrhage of 2.0 litres in a healthy 70kg adult if there is no immediate fluid resuscitation.

2.0L haemorrhage in a 70kg adult represents 40% of blood volume, and therefore is a severe haemorrhage. It causes a significant decrease in cardiac output and arterial blood pressure, causing hypotension

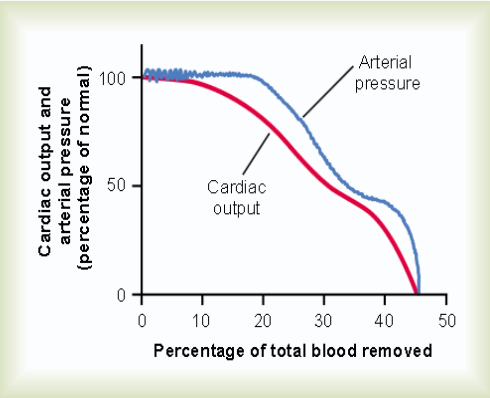

Diagram 24-1 from Guyton & Hall 11th Ed

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Sympathetic response

- Decreased arterial pressure causes powerful sympathetic reflexes initiated by baroreceptors
- Main effects:
  - Arterial vasoconstriction → increased peripheral vascular resistance
  - Venous constriction → increased venous return
  - Increased heart rate up to 160-180
- Maximally effective by 30-60s
- Note does not cause vasoconstriction of coronary or cerebral vasculature → these are still governed by autoregulation, at least until SBP 70mmHg

Compensatory mechanisms

- Baroreceptor reflexes → sympathetic stimulation
- Central nervous system ischaemic response:
  - Occurs at arterial BP ~50mmHg
  - Extreme sympathetic response stimulation when brain suffers from decreased O_2 or increased CO_2
- Reverse stress-relaxation of circulation:
  - Vessels constrict around diminished blood volume
  - Response time 10-60 mins
- Formation of angiotensin by kidneys via RAS:
  - Vasoconstriction and conservation of H_2O and Na^+
  - Response time 10-60 mins
- Formation of antidiuretic hormone by posterior pituitary:
  - Vasoconstriction; water retention by kidneys
  - Response time 10-60 mins
- Compensatory mechanisms to increase blood volume:
  - Resorption of fluid from intestines and interstitial spaces
  - Increased retention of water by kidneys → decreased GFR leading to decreased urine formation
  - Increased thirst (if conscious)
  - Response time 1-48 hours

Physiological consequences

- Shock can become progressive via positive feedback
- Cardiac depression:
  - If arterial pressure falls too low, coronary blood flow decreases below the required level for adequate nutrition of myocardium
  - Causes weakening of myocardium → further decrease in cardiac output
- Vasomotor failure:
  - Diminished blood flow to vasomotor centre of brain causes it to become progressively inactive, eliminating sympathetic stimulation
- Blockage of vessels:
  - Metabolism continues in microcirculation despite low blood flow, causing metabolites to accumulate, and local acidosis → clots
- Increased capillary permeability:
  - Capillary hypoxia and lack of nutrients causes increased capillary permeability causing large transudation → decreased blood volume
- Release of toxins by ischaemic tissue:
  - Shocked tissues release toxic substances e.g. histamines, and tissue enzymes that cause further damage to circulatory system
  - Endotoxin released by dead intestinal bacteria causes cardiac depression
- Generalised cellular deterioration:
  - Mitochondrial depression; release of intracellular hydrolase enzymes
  - In liver → depression of metabolic/detoxification functions
  - Lungs → development of oedema and inability to oxygenate blood
  - Heart → further depression of contractility