2006b(15)/1998b(5): Explain how a metabolic acidosis develops in hypovolaemic shock. Describe consequences to the body

General: A metabolic acidosis is a process which results in ↓pH through the depletion of bicarbonate.

- Hypovolaemic shock occurs when there is diminished intra- (± extra-) vascular volume leading to inadequate tissue perfusion to meet metabolic demand with respect to the provision of substrates and the removal of waste.

Aspects which contribute to the formation of a metabolic acidosis

1. Increased metabolic acid production (Type A lactic acidosis)
   - Insufficient O₂ delivered to tissues Flux = Q x O₂ content
   - ↑anaerobic metabolism through glycolysis with the formation of lactic acid from pyruvate. This is an energy-inefficient process (yields 2ATP for each mol of glucose) resulting in the formation of large amounts of lactic acid.
   - Hepatic hypoperfusion means that lactic acid is unable to enter the Cori cycle to convert the lactic acid back to glucose.

Effect on the body:

- **Respiratory:**
  - ↓pH detected by peripheral & central chemoreceptors. Activate respiratory centre of the medulla and ↑MV through hyperventilation rapidly ↓PaCO₂. ↓pH by 1 → ↑MV by 3L/min
    - May lead to eventual exhaustion
    - Hyperventilation ↑metabolic demand of mm of respiration further compounding acidosis

- **Cardiovascular:**
  - Compounding ‘shocked’ state
  - ↓pH is negatively inotropic
  - Vascular resistance:
    - acidosis → direct ↓SVR (metabolic autoregulation), ↑PVR
    - Hypovolaemia → ↑SVR 2° ADH (V1 receptor), ATII (AT₁R) activation
  - Impaired SNS response, as ↓response to catecholamines <7.2
  - Depressed myocardial function and ↑arrythmogenicity through ↑K⁺ (through cellular H⁺/K⁺ exchange) and ↑Ca^{2+} (through dissociation from albumin)

- **Right-shift** of O₂Hb-dissociation curve, ↑O₂ delivery to tissues
- **CNS effects:**
  - Impaired consciousness

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