Shock

Definition of shock
- Circulatory shock: an abnormality of the circulatory system that results in reduced organ perfusion and tissue oxygenation
- Emotional shock: an acute stress reaction arising in response to a traumatic event, often erroneously used interchangeably with the term ‘shock’ in popular culture; this will not be discussed further

Causes of shock

Reduced cardiac output (CO):
- Hypovolaemic shock
  - Haemorrhage
    - External, including gastrointestinal
    - Internal
      - Chest
      - Abdomen
      - Pelvis
      - Retroperitoneum
      - Long bones
  - Vomiting
  - Diarrhoea
  - Diuresis
  - Burns
- Cardiogenic shock
  - Myocardial infarction
  - Myocardial contusion
  - Myocarditis
  - Cardiac arrhythmia
    - Unstable tachyarrhythmias
    - Unstable bradyarrhythmias
  - Negatively inotropic drug overdose
    - Beta blockers
    - Calcium channel blockers
- Obstructive shock
  - Tension pneumothorax
  - Massive PE
  - Cardiac tamponade

Reduced systemic vascular resistance (SVR):
- Septic shock
- Anaphylactic shock
- Neurogenic shock

Pathophysiology of shock
- Blood pressure (BP) is related to cardiac output (CO) and systemic vascular resistance (SVR) by the following equation:

\[ BP = CO \times SVR \]
CO is the volume of blood pumped by the heart per minute and is in turn related to heart rate (HR) and stroke volume (SV) as follows:

\[ CO = HR \times SV \]

SV is the volume of blood pumped by the heart per contraction and is determined by:
- Preload
- Myocardial contractility
- Afterload

Preload is the ventricular wall tension at the end of diastole and reflects the degree of myocardial muscle fibre stretch; it is determined by volume status, venous capacitance and the difference between mean venous pressure and right atrial pressure.

Preload is related to SV by the Frank-Starling mechanism; increased fibre length initially leads to an increased SV but above a certain point, the fibres become overstretched and further filling results in a decreased SV, as is the case in cardiac failure.

Myocardial contractility is the intrinsic ability of the heart to work independently of preload and afterload; positive inotropes increase the contractility, shifting the Frank-Starling curve upwards.

Afterload is the ventricular wall tension at the end of systole and is the resistance to anterograde blood flow.

Regardless of the cause of shock, inadequate organ perfusion and tissue oxygenation results in cells switching from aerobic to anaerobic metabolism.

This generates a lactic acidosis that disrupts the cellular environment and causes myocardial depression.

**History in a shocked patient**

- Assessment of severity
  - Dyspnoea
  - Confusion
  - Light-headedness
  - Drowsiness
  - Oliguria/anuria

- Symptoms of the cause

**Examination of the shocked patient**

- Airway
  - May be compromised by reduced conscious level

- Breathing
  - Hypoxia secondary to:
    - Cause
    - Airway compromise
    - Apparent hypoxia due to ineffective pulse oximetry from peripheral shutdown
  - Tachypnoea
  - Kussmaul's breathing: hyperventilation to compensate for metabolic acidosis manifesting as ‘air hunger’

- Circulation
  - Cold, pale peripheries
  - Prolonged capillary refill times (CRT > 2 s)
  - Tachycardia
  - Hypotension
<table>
<thead>
<tr>
<th>Classification of haemorrhagic shock</th>
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</thead>
<tbody>
<tr>
<td><strong>Type I</strong></td>
</tr>
<tr>
<td>Volume of blood loss (ml): &lt;750</td>
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<tr>
<td>Percentage blood loss (%): &lt;15</td>
</tr>
<tr>
<td>Heart rate (beats/min): &lt;100</td>
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<tr>
<td>Blood pressure: normal</td>
</tr>
<tr>
<td>Pulse pressure: normal/increased</td>
</tr>
<tr>
<td>Respiratory rate (breaths/min): 14-20</td>
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<tr>
<td>Urine output (ml/hour): &gt;30</td>
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<tr>
<td>Mental state: slightly anxious</td>
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<tr>
<td><strong>Type II</strong></td>
</tr>
<tr>
<td>Volume of blood loss (ml): 750-1500</td>
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<tr>
<td>Percentage blood loss (%): 15-30</td>
</tr>
<tr>
<td>Heart rate (beats/min): 100-120</td>
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<tr>
<td>Blood pressure: normal</td>
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<tr>
<td>Pulse pressure: decreased</td>
</tr>
<tr>
<td>Respiratory rate (breaths/min): 20-30</td>
</tr>
<tr>
<td>Urine output (ml/hour): 20-30</td>
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<tr>
<td>Mental state: mildly anxious</td>
</tr>
<tr>
<td><strong>Type III</strong></td>
</tr>
<tr>
<td>Volume of blood loss (ml): 1500-2000</td>
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<tr>
<td>Percentage blood loss (%): 30-40</td>
</tr>
<tr>
<td>Heart rate (beats/min): 120-140</td>
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<tr>
<td>Blood pressure: decreased</td>
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<tr>
<td>Pulse pressure: decreased</td>
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<tr>
<td>Respiratory rate (breaths/min): 30-40</td>
</tr>
<tr>
<td>Urine output (ml/hour): 5-15</td>
</tr>
<tr>
<td>Mental state: anxious, confused</td>
</tr>
<tr>
<td><strong>Type IV</strong></td>
</tr>
<tr>
<td>Volume of blood loss (ml): &gt;2000</td>
</tr>
<tr>
<td>Percentage blood loss (%): &gt;40</td>
</tr>
<tr>
<td>Heart rate (beats/min): &gt;140</td>
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<tr>
<td>Blood pressure: decreased</td>
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<tr>
<td>Pulse pressure: decreased</td>
</tr>
<tr>
<td>Respiratory rate (breaths/min): &gt;35</td>
</tr>
<tr>
<td>Urine output (ml/hour): negligible</td>
</tr>
<tr>
<td>Mental state: confused, lethargic</td>
</tr>
</tbody>
</table>

**Investigation of shock**
- Bloods including blood gas to check pH and lactate
- Electrocardiogram (ECG)
### Initial management of shock
- Assess the patient from an ABCDE perspective
- Maintain a patent airway
  - Use manoeuvres, adjuncts, supraglottic or definitive airways as indicated and suction any sputum or secretions
- Deliver high flow oxygen 15L/min via reservoir mask to keep sats over 94%
- Attach monitoring
  - Pulse oximetry and non-invasive blood pressure
  - Three-lead cardiac monitoring
- Request 12 lead ECG and portable CXR
- Obtain large-bore intravenous (IV) access and take bloods including blood gas to check pH and lactate
- Fluid resuscitation IV
- Urethral catheterisation and fluid balance monitoring aiming for a urine output >0.5 ml/kg/hour
- If BP fails to respond consider referral to HDU/ICU for
  - Central line insertion with central venous pressure (CVP) and central venous oxygen saturation ($SvO_2$) monitoring
  - Arterial line insertion and invasive arterial BP monitoring
  - Vasopressor and/or inotrope infusion

### Further management of shock
- Identify and treat the cause
  - Haemorrhagic shock
    - Identify the source(s) of bleeding and achieve haemorrhage control e.g. direct compression, pelvic binder, splinting of long bone fractures, surgical ligation of bleeding vessels
    - Restoration of adequate circulating volume
      - Cross-match blood and activate the major haemorrhage protocol
      - Transfuse O negative blood initially, followed by type-specific and fully cross-matched blood as soon as it is available; aim for permissive hypotension
    - Correct coagulopathy by transfusion of platelets, fresh frozen plasma and cryoprecipitate as appropriate
    - RBC: FFP ratio should be between 1:1 and 1:2, the optimum ratio is uncertain. The key is to give FFP early with RBC. Cryo if fibrinogen<1.5.
  - Antibiotics and source control for septic shock
  - Adrenaline 0.5 mg intramuscular (IM) for anaphylactic shock
  - Needle thoracocentesis and intercostal chest drain insertion for tension pneumothorax
  - Pericardiocentesis and thoracotomy for cardiac tamponade
  - Thrombolysis for massive PE
  - Synchronised direct current (DC) cardioversion for unstable tachyarrhythmias
  - Pacing for unstable bradyarrhythmias