



### DEFINITION

Stated simply, shock is inadequate peripheral tissue perfusion (hypoperfusal state) resulting in lack of oxygen and nutrient supply to the cell

### PATHOPHYSIOLOGY

There are four stages of shock

#### 1. *Initial*

- hypoperfusal state causes hypoxia
- results in mitochondria not being able to produce adenosine triphosphate (ATP)
- cell hypoxia leads to cell membrane destruction and become 'leaky' to extracellular fluid
- anaerobic respiration occurs
- build up of lactic and pyruvic acid results in metabolic acidosis

#### 2. *Compensatory*

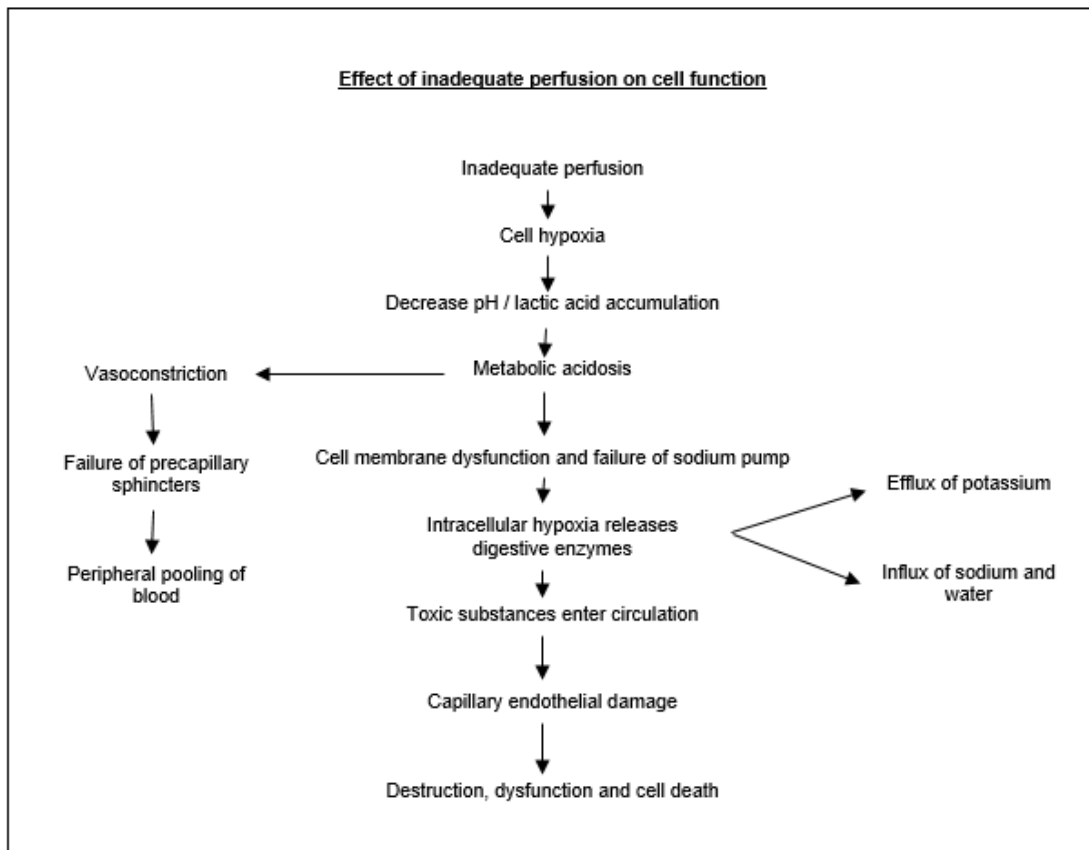
- neural, hormonal and biochemical mechanisms attempt to reverse the above
- acidosis results in hyperventilation to rid of carbon dioxide to correct the pH
- baroreceptors in the arteries detect hypoperfusion and cause release of adrenaline (increases heart rate) and noradrenaline (causes vasoconstriction) resulting in an increase in blood pressure. This is known as the Cushing reflex
- rennin-angiotensin axis is activated and anti-diuretic hormone (ADH) is released to conserve fluid via the kidney
- all these hormones cause vasoconstriction of the kidneys, gastrointestinal tract, and skin to divert blood to the brain, heart and lungs.

#### 3. *Progressive*

- compensatory mechanisms fail
- decreased perfusion of cell results in sodium influx and potassium efflux
- anaerobic metabolism continues, increases metabolic acidosis, arteriolar smooth muscle and pre-capillary sphincters relax and blood pools in the capillaries
- hydrostatic pressure increases, and with histamine release results in leakage of fluid and protein into the surrounding tissues
- blood concentration and viscosity increases, causing sludging of the microcirculation
- prolonged vasoconstriction leads to vital organ compromise

#### 4. *Refractory*

- vital organs fail and shock can no longer be reversed



## TYPES OF SHOCK

### 1. **Hypovolaemic** [*student is referred to Trauma notes*]

- Most common type
- Due to insufficient circulating volume, most commonly blood loss

### 2. **Cardiogenic**

- Failure of the heart to pump effectively
- Massive myocardial infarction, arrhythmias, cardiomyopathy, cardiac valve problems are common causes

### 3. **Distributive**

'Relative hypovolaemia' as a result of dilation of blood vessels which diminishes the systemic vascular resistance

- Septic shock — caused by overwhelming systemic infection
- Anaphylactic shock ~ anaphylactic reaction to an allergen, antigen, drug

- Neurogenic shock — trauma to spinal cord with loss of autonomic and motor reflexes

### 4. **Obstructive** [*student is referred to Trauma notes*]

The flow of blood is obstructed which impedes circulation

- Cardiac tamponade
- Tension pneumothorax
- Massive pulmonary embolism
- Aortic stenosis

## SEPTIC SHOCK

### Definitions

- Severe sepsis: acute organ dysfunction secondary to infection
- Septic shock: severe sepsis plus hypotension not reversed with fluid resuscitation

To diagnose septic shock, the following two criteria must be met:

- Evidence of infection

- Refractory hypotension (hypotension despite adequate fluid resuscitation and cardiac output)
  - In adults defined as systolic blood pressure < 90 mmHg, or a mean arterial pressure < 60 mmHg, without the requirements for inotropic support, or a reduction in 40 mmHg from baseline

In addition to the two criteria above, two or more of the following must be present:

- Tachypnoea (> 20 breaths per minute). or, on blood gas, a PCO<sub>2</sub> less than 32 mmHg
- White blood cell count < 4000 cells/mm<sup>3</sup> or > 12 000 cells/mm<sup>3</sup>
- Heart rate > 90 beats per minute
- Temperature > 38.0 °C or < 36.0 °C

### Pathophysiology

- Most cases of septic shock are caused by endotoxin-producing gram-negative bacilli
- Endotoxins are bacterial wall lipopolysaccharides (LPS) consisting of a toxic fatty acid (lipid A) core common to all gram-negative bacteria, and a complex polysaccharide coat (including O antigen) unique for each species
- Analogous molecules in gram-positive bacteria and fungi can also elicit septic shock
- Free LPS binds to circulating LPS-binding protein -> the complex binds to specific receptor (CD14) on monocytes, macrophages and neutrophils.
- This results in intracellular production of effector cytokines such as IL-1 and TNF
- These cytokines act on endothelial cells and reduce synthesis of anticoagulation factors such as tissue factor pathway inhibitor and thrombomodulin
- They also cause systemic vasodilatation and diminished cardiac contractility

- The coagulation system is activated and systemic leukocyte adhesion occurs resulting in DIC
- The hypoperfusion, resulting from the combined effects of widespread vasodilatation, myocardial pump failure and DIC, causes multiorgan system failure that affects the liver, kidneys, and central nervous system among others
- Unless the underlying infection (and LPS overload) is rapidly brought under control, the patient usually dies

### Treatment

The treatment primarily consists of the following: The mnemonic OVERS may be helpful.

Begin resuscitation immediately in patients with hypotension or metabolic acidosis or elevated serum lactate > 4 mmol/ L

1. **Oxygen administration and airway support**

2. **Volume resuscitation (crystalloids or colloids)**

- Target a central venous pressure of 8-12 mmHg
- Maintain a mean arterial pressure of > 65mmHg (vasopressor of choice are adrenaline)
- Urine output > 0,5 mL/kg/hr
- Central venous or mixed venous oxygenation saturation > 70 % or > 64 %, respectively
- Haematocrit > 30%

3. **Early antibiotic administration**

- Intravenous antibiotic therapy should be started as early as possible and within the first hour of recognition of septic shock or severe sepsis without septic shock
- Broad-spectrum: one or more agents active against most likely bacterial or fungal pathogens

- Appropriate cultures (blood always, urine and sputum if indicated) before initiating antibiotic therapy
4. Rapid source identification and control
- Specific anatomical diagnosis of infection requiring consideration for emergent source control (e. g. necrotising fasciitis, diffuse peritonitis) be sought and diagnosed or excluded as rapidly as possible
  - All patients presenting with severe sepsis be evaluated for the presence of a focus of infection amenable to source control measures
5. Support of major organ dysfunction
- Mechanical ventilation for sepsis induced acute lung injury acute respiratory distress syndrome
  - Adequate sedation and analgesia
  - Glucose control
  - Renal replacement therapy
  - Deep vein prophylaxis
  - Stress ulcer prophylaxis

Early goal-directed resuscitation has been shown to improve survival for emergency department patients presenting with septic shock. Resuscitation directed toward the previously mentioned goals for the **initial 6-hr period** of resuscitation reduces 28-day mortality rate. Surgical intervention (debridement of necrotising fasciitis, drainage of an abscess and laparotomy for peritonitis) forms part of the resuscitation of the septic patient.



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