The shocked patient

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Abstract

Prompt recognition of the shocked patient and administration of therapy is essential. Haemodynamic stabilization and correction of the underlying cause should be based on the pathophysiological processes that are occurring. Monitoring the patient's response to treatment depends on careful observation in a high-dependency area along with serial lactate measurements. By optimizing treatment of circulatory shock, its significant morbidity and mortality can be improved. Here, we give an overview of circulatory shock, recognition of the shocked patient and principles of treatment, and explore some of the underlying causes of shock and their management.

Keywords Circulatory shock; hypovolaemia; lactate; resuscitation; sepsis

Key points

- Circulatory shock is a common, life-threatening condition associated with high morbidity and mortality.
- Rapid recognition of the shocked patient is essential to instigate immediate treatment and provide the best outcome possible.
- The classification of shock states includes four categories: hypovolaemic, obstructive, cardiogenic and distributive.
- It is essential to identify the underlying cause of shock and to rectify it in a timely fashion.
- Measuring venous lactate aids the diagnosis of shock as it is typically raised.
- The 'VIP' mnemonic outlines the initial management of shock.

Introduction

Circulatory shock is a common, life-threatening condition associated with high morbidity and mortality. It is best defined as acute circulatory failure resulting in inadequate cellular oxygen utilization.[1] Shock can lead to multiorgan failure and ultimately death. Early recognition of the shocked patient and the underlying causes is essential to allow rapid intervention and afford the best possible outcome.

Pathophysiology

The initial stages of shock are characterized by hypoperfusion and hypoxia leading to cellular ischaemia as oxygen demand outweighs supply. Previously thought to be the underlying pathophysiological process, it is now appreciated that this is simply the catalyst for a complex chain of events. Cellular hypoxia leads to local vasoconstriction, thrombosis and release of superoxide radicals causing direct cellular damage and endothelial dysfunction.[1] Neutrophil activation and pro-inflammatory cytokines cause cellular injury and organ dysfunction. It is therefore essential to restore tissue perfusion to prevent this inflammatory cascade.

Lactic acidosis in shock is the result of anaerobic respiration causing an accumulation of pyruvate. Hypoxia slows the entry of pyruvate into the Krebs cycle, and this is converted into lactate. To maintain electroneutrality of the blood the cation hydrogen is released into the bloodstream with lactate, which reduces the pH.

Classification

The classification of shock states, proposed in 1972 by Hinshaw and Cox, includes four categories: hypovolaemic, obstructive, cardiogenic and distributive (Figure 1). Hypovolaemic, cardiogenic and obstructive shock result in low cardiac output states caused by different physiological changes. In distributive shock, there is decreased systemic vascular resistance and impaired oxygen extraction at a cellular level, usually with high cardiac output. Endocrine shocks have been given their own classification to aid recognition, but the underlying mechanism is either cardiogenic or distributive.

Shock states are not mutually exclusive and can coexist. For example, sepsis results in distributive shock. However, hypovolaemia caused by extravasation of fluid and cardiogenic shock resulting from myocardial depression can also be present.

Epidemiology and prognosis

Shock is a common condition with increasing incidence. The exact characteristics of non-traumatic shock presenting to the emergency department are not well described but represent 1–2% of emergency medicine service contacts. Hypovolaemia is the most common form of shock. Sepsis occurs more frequently in older people and is present in around 2% of hospital admissions. Cardiogenic shock complicates 5–10% of acute myocardial infarctions. Up to a third of patients admitted to intensive care units are shocked. Sepsis is the most common cause (62%), followed by cardiogenic causes (17%) and hypovolaemia (16%). Mortality is high, with cardiogenic shock carrying as much as 60% mortality, and septic shock 30–50%.

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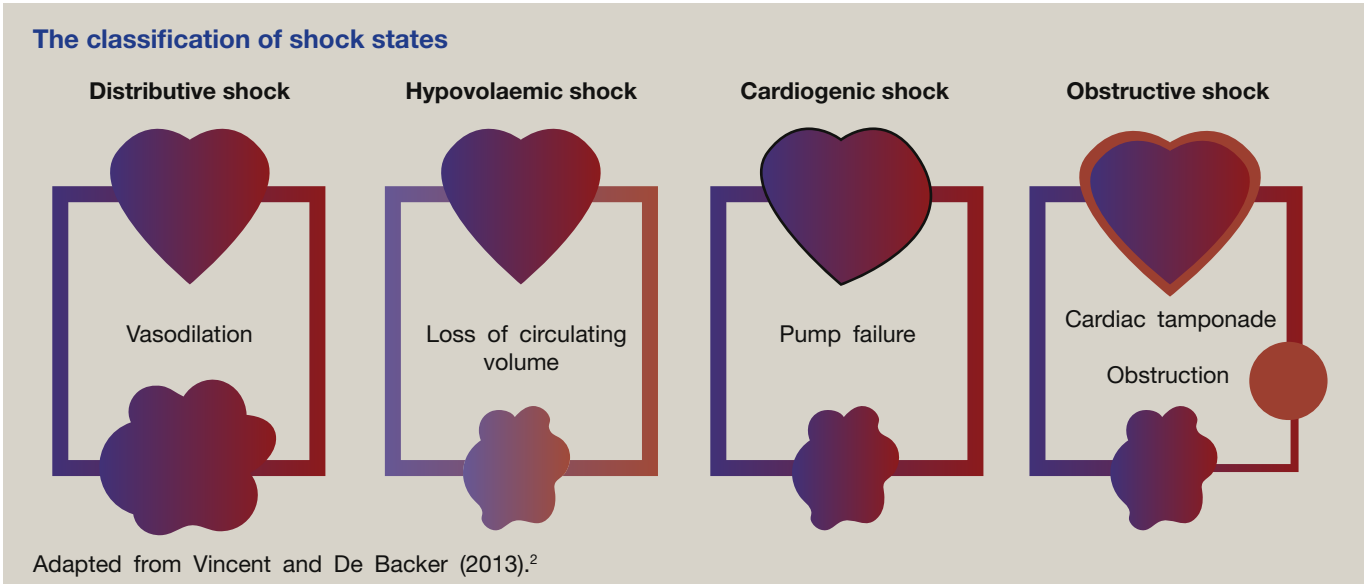

Figure 1

Recognition of the shocked patient

Rapid recognition of the shocked patient is essential to instigate immediate treatment and provide the best outcome possible. It may be obvious from clinical history that a patient is at risk of shock, for example after gastrointestinal haemorrhage. However, the cause of shock and its presence are not always apparent. The diagnosis of shock is based on clinical, haemodynamic and biochemical parameters. A standardized 'ABCDE' assessment, including a thorough fluid status assessment, allows the detection of these clinical signs and can also identify the possible underlying cause.

Although arterial hypotension (systolic blood pressure <90 mmHg) is a cardinal sign of shock, it may not initially be present because of peripheral vasoconstriction. Care should be taken, especially in older patients or patients with hypertension, in whom an apparently 'normal' blood pressure reading can represent relative hypotension and a shock state. Cardiorespiratory parameters, which can be present earlier in the clinical course of shock, include tachycardia, tachypnoea and a postural fall in blood pressure or rise in heart rate. Table 1 outlines the clinical findings in different classes of shock.

Clinical signs of tissue hypoperfusion can be apparent on assessment through the 'three windows of the body'.[2] Cutaneous hypoperfusion can be recognized by skin changes such as cold and clammy peripheries, delayed capillary refill, cyanosis and mottling, which is a late and sinister sign especially when present centrally. Oliguria is an important sign of renal hypoperfusion and is recognized by a urine output of less than 0.5 ml/kg per hour. Finally, cerebral hypoperfusion can be recognized by altered mental state, confusion or obtundation.

Measuring venous lactate aids the diagnosis of shock as it is typically raised. A normal lactate concentration is around 1 mmol/litre, with a value greater than 2 mmol/litre being significant. Severity of hyperlactataemia is related to worse outcomes, and even modest rises in lactate can predict increased mortality. Serial measurement of venous lactate can also be used as a marker of response to treatment.

Echocardiography can be useful to establish the underlying diagnosis by assessing filling status, ventricular size and function, and the presence of pericardial effusion. Table 2 outlines the differential diagnosis for each of the classifications of shock. Table 3 shows the investigations that should be performed as part of the initial work-up for the shocked patient.

Classes of shock and clinical findings

Class	Blood loss	Heart rate (bpm)	Blood pressure	Respiratory rate (per minute)	Capillary refill	Urine output	Mental state
I	<15% (<0.75 litre)	<100	Normal	14–20	Normal (<2 seconds)	>30 ml/hour	Normal/agitated
II	15–30% (0.75–1.5 litres)	>100	Postural fall	20–30	Sometimes delayed	20–30 ml/hour	Agitated
III	30–40% (1.5–2 litres)	>120	Low	30–40	Usually delayed	5–20 ml/hour	Confused
IV	>40% (>2 litres)	>140	Profoundly low	>40	Always delayed	Anuria	Obtunded

Modified from American College of Surgeons. Shock. In: American College of Surgeons, ed. Advanced trauma life support, 1990:59–73.

Table 1

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