The shocked patient

Craig Prescott
Emma Rowland

Abstract

Prompt recognition of shocked patients 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 the treatment of circulatory shock, its significant morbidity and mortality can be improved. Here, we give an overview of circulatory shock, recognition of shocked patients 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 shocked patients 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 rectify it in a timely fashion
- 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 utilization of oxygen.[1] Shock can lead to multiorgan failure and ultimately death. Early recognition of shocked patients and the underlying causes is essential to allow rapid intervention and provide 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 proinflammatory 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, being converted into lactate. To maintain the electroneutrality of the blood, the cation hydrogen is released into the bloodstream with lactate, which reduces the pH.

Craig Prescott *MB BS BMedSci FRCP (Acute) PGDip is a Consultant Acute Physician at University Hospitals Dorset, UK. He has a special interest in venous thromboembolism and stress echocardiography. Competing interests: none declared.*

Emma Rowland *BM BS MRCP is a Medical Registrar at University Hospitals Dorset, UK. Competing interests: none declared.*

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 one-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%.

Recognition of shocked patients

Rapid recognition of shocked patients 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,

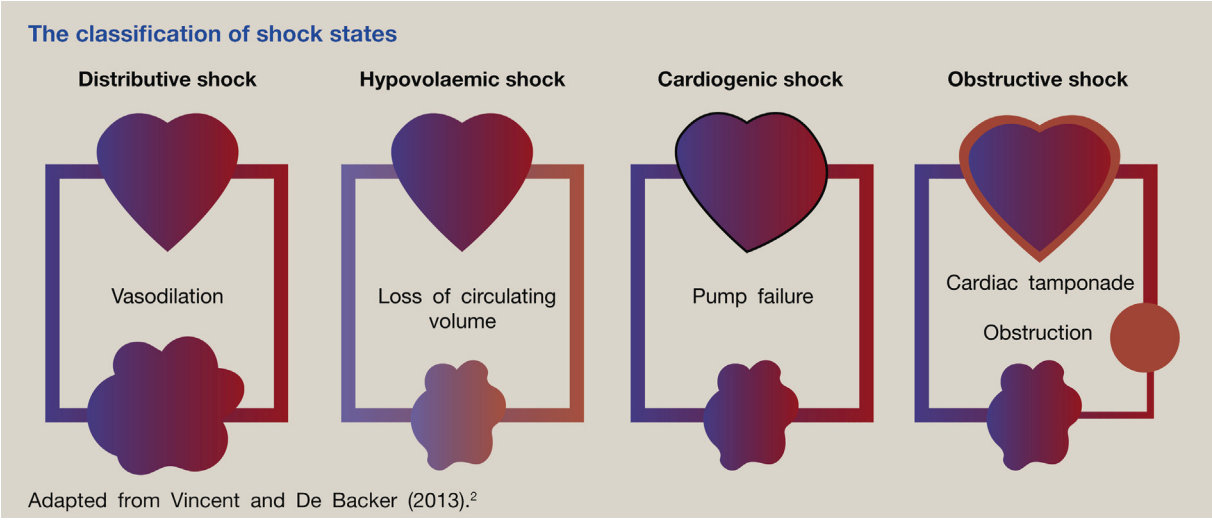

Figure 1

including a thorough fluid status assessment, allows these clinical signs to be detected 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 individuals or those 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 <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 lactate is typically raised. A normal lactate concentration is around 1 mmol/litre, with a value >2 mmol/litre being significant. Severity of hyperlactataemia is related to worse outcomes, and even modest rises in concentration 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 shocked patients.

Therapeutic strategy

The principles of initial resuscitative therapy for shocked patients are not based solely on the underlying cause. The aim is correction of the pathological processes to improve cellular oxygenation and respiration. Once the underlying cause has been identified, it must be corrected immediately.

The 'VIP' mnemonic outlines the initial management of shock: Ventilate (oxygen administration), Infuse (fluid resuscitation) and Pump (use of vasoactive agents).[2]

Ventilate

High-flow oxygen should be administered to reverse known risks associated with hypoxia. There are risks associated with excessive oxygenation, as evidenced after cardiac arrest. In practice it

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

Classifications of shock with differential diagnoses

Type of shock	Differential diagnosis
Hypovolaemic	• Haemorrhage • Gastrointestinal loss • Dehydration • Diabetic ketoacidosis • Burns • Environmental exposure
Cardiogenic	• Myocardial ischaemia • Arrhythmia • Cardiomyopathy • Valvular disease • Drugs
Obstructive	• Pulmonary embolism • Cardiac tamponade • Tension pneumothorax • Aortic dissection
Distributive	• Sepsis • Anaphylaxis • Poisoning • Drugs • Neurogenic
Endocrine	• Hypo/hyperthyroidism • Relative/absolute cortisol deficiency

Table 2

Investigations for the shocked patient

Study type	Investigations
Blood tests	• **Full blood count** • Coagulation studies • **Renal function** • **Glucose** • Ketones • **Liver function** • Amylase • **Blood gases** • **Lactate** • Thyroid function tests • Cortisol/short synacthen test • Group and save • Troponin • B-type natriuretic peptide
Microbiology	• Blood cultures • Urinalysis and culture • Gram stain of secretions or fluid • Coronavirus disease (COVID-19) swab
Imaging	• **Chest radiograph** • Plain radiograph of extremities • Echocardiography • Abdominal ultrasound • Abdominal CT • Computed tomography pulmonary angiography
Bedside tests	• **ECG**

Investigations in bold are essential. All others should be guided by clinical findings.

Table 3

is difficult to monitor oxygen saturations in shock because of reduced peripheral perfusion. Therefore, the use of arterial blood gases is warranted, which allows monitoring of controlled oxygen delivery. If it is not possible to deliver adequate oxygenation, urgent referral for intensivist support with mechanical ventilation is necessary.

Infuse

In order to provide fluid resuscitation, secure vascular access must be obtained. This can be problematic in shocked patients because of peripheral vasoconstriction. It can require support from an experienced physician, ultrasound guidance, central venous catheterization or intraosseous access.

A strategy of fluid challenges using rapid infusions of 250 −500 ml of crystalloid (normal saline, Hartmann's solution) allows assessment of the response while decreasing the risk of iatrogenic harm. After this, the patient should be further reassessed using an ABCDE approach. The aim of treatment is to increase blood pressure (aiming for a mean arterial pressure >65 mmHg), decrease heart rate and increase urine output. Worsening pulmonary oedema and respiratory function after fluid administration can indicate a need for change in strategy and should prompt seeking expert help.

Further fluid boluses should be administered if the patient remains hypovolaemic on reassessment. If 2 litres have been administered and there is still evidence of shock (failure to improve physiological parameters, a lactate concentration that is not improving), expert help should be sought.[3]

When managing shock, the presence of oedema can cause confusion as it can be a result of redistribution of fluid or cardiogenic shock. In both situations, there can be depleted intravascular volume; therefore careful fluid administration should not be withheld for worry of worsening the peripheral oedema. The exception is evidence of pulmonary oedema causing respiratory compromise (see Cardiogenic shock, below).

Pump

Vasoactive agents can be used to improve hypotension in patients who are resistant to fluid administration so that minimum perfusion pressure can be maintained. Noradrenaline (norepinephrine) is the first-choice vasopressor because of its predominant α- and moderate β-adrenergic properties, which cause increased systemic vascular resistance while helping to maintain cardiac output. Vasopressors can be started early while fluid resuscitation is continuing, in order to support blood pressure, with the aim of stopping them once hypovolaemia and physiological parameters have been corrected.

Inotropic therapy can be used in cardiogenic shock, although few controlled trials have been performed. Therefore agents such as dobutamine can be used under specialist guidance. In all cases, vasoactive agents should be initiated only by appropriately trained specialists in high-dependency clinical areas.

Specific situations

It is essential to identify the underlying cause of shock and rectify it in a timely fashion. There are specific treatments for different causes of shock, which should be administered alongside initial resuscitative measures. In this section, we focus on medical causes of shock and their management.

Hypovolaemic shock can be divided into haemorrhagic and non-haemorrhagic. In haemorrhagic shock, as in gastrointestinal bleeding, trauma or aneurysm rupture, control of the haemorrhage is essential. Gastrointestinal bleeding needs the input of an endoscopist.

Initial resuscitation of shocked patients with massive upper gastrointestinal haemorrhage should include transfusion of red cells. It is also important to transfuse platelets and clotting factors in line with the local policy on major haemorrhage, and to keep the platelet count $>50 \times 10^9$/litre, prothrombin time >1.5 seconds and fibrinogen >1.5 g/litre.

Any anticoagulation the patient is taking must be reversed, using prothrombin complex concentrate and vitamin K for warfarin, idarucizumab (Praxbind) for dabigatran, or adexanet alfa (Ondexxya) for gastrointestinal bleeds on apixaban or rivaroxaban in line with the ANNEXA-4 trial inclusion criteria; other anticoagulants should be discussed with a haematologist as there is no specific antidote for factor Xa inhibitors.

There is no evidence for using a proton pump inhibitor before endoscopy, as these have no impact on patient outcomes. In suspected variceal bleeding, terlipressin and broad-spectrum antibiotics should be administered. Tranexamic acid is no longer recommended in the treatment of upper gastrointestinal bleeding as there no benefit for mortality but an excess of venous thromboembolic events, as established by the HALT-IT study.

Non-haemorrhagic hypovolaemic shock occurs through loss of body fluid volume or loss of intravascular fluid volume via fluid shifts into the extravascular space, for example in pancreatitis. Uncontrolled gastrointestinal loss through diarrhoea and vomiting or excessive urinary losses requires careful fluid balance measurement to ensure replacement matches output.

Cardiogenic shock can be the result of any cause of 'pump failure'. When the cause of shock is recognized as acute left ventricular failure, the patient should be reassessed after initial fluid therapy as they might require diuretic therapy, especially in the presence of pulmonary oedema. Shocked patients with a reversible cause of acute heart failure can be considered for inotropic therapy. In left ventricular dysfunction resulting from myocardial infarction, reperfusion therapy with primary angioplasty, or thrombolytic therapy if this is not available within 120 minutes, is the mainstay of treatment, and patients should be urgently referred to cardiology.

Shock resulting from right ventricular infarction must be recognized as management is different from that of left-sided failure; it can be corrected with fluid resuscitation to maintain right ventricular preload before considering reperfusion therapy.

Cardiac dysrhythmias causing shock should be corrected urgently as per Resuscitation Council UK guidelines and can warrant urgent synchronized DC cardioversion for tachyarrhythmias, and atropine or external pacing for bradyarrhythmias. Other causes, such as acute valvular or obstructing lesions, need urgent discussion with a cardiologist and can require surgical intervention.

Obstructive shock caused by pulmonary embolus should be treated with thrombolytic therapy without confirmation using computed tomography pulmonary angiography, if cardiac arrest is imminent. Tension pneumothorax should be diagnosed clinically and treated with urgent needle decompression using a wide-bore cannula inserted in the second intercostal space in the mid-clavicular line. Chest drain insertion should immediately follow needle decompression. Cardiac tamponade requires urgent needle pericardiocentesis under echocardiographic guidance. This should only be performed by a competent practitioner.

Distributive shock is most commonly encountered as sepsis. The definitions of sepsis and septic shock were updated in 2016 to improve the recognition and management of patients with sepsis.[4] The Surviving Sepsis Campaign bundle should be completed within 3 hours, ideally within 1 hour. This includes intravenous antibiotic therapy, two sets of blood cultures before antibiotic administration, lactate measurement and administration of crystalloid 30 ml/kg for sepsis-induced hypoperfusion manifested by organ dysfunction, hypotension or raised serum lactate.[5]

It is important that the source of the sepsis is identified and controlled. In anaphylaxis, the treatment is immediate intramuscular adrenaline (epinephrine), with removal of the trigger if it is still present. Intravenous chlorphenamine and hydrocortisone should also be administered. ◆

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TEST YOURSELF

To test your knowledge based on the article you have just read, please complete the questions below. The answers can be found at the end of the issue or online here.

Question 1

A 54-year-old woman presented with epigastric abdominal pain and vomiting, She collapsed on the ward. She had a history of alcohol excess and varices seen on previous oesophago-gastro-duodenoscopy (OGD).

On clinical examination, her Glasgow Coma Scale (GCS) score was 8, she was very pale, and the airway was patent. Her heart rate was 125 beats/minute and regular, blood pressure 81/54 mmHg, which did not improve significantly with two 500 ml fluid boluses, heart sounds normal, respiratory rate 14/minute, and oxygen saturations 97% on air. She was passing large amounts of dark tar-like stool.

The GCS remains at 8. What is the most important initial management for this patient?

A. Cross-matched blood transfusion
B. Major haemorrhage protocol
C. Aggressive fluid resuscitation
D. A proton pump inhibitor
E. Tranexamic acid

Question 2

An 87-year-old man presented with acute confusion. His carers described finding him slumped in a chair and talking incoherently; they had been unable to move him so called an emergency number.

On clinical examination, his temperature was 38.3°C, blood pressure 94/70 mmHg, and heart rate 115 beats/minute. He had suprapubic fullness.

Investigations

- ECG showed atrial fibrillation
- Bladder scanning showed 2 litres of urine in an enlarged bladder

What is the most important initial management step?

A. Urine dip ± microscopy, culture and sensitivity
B. Catheter insertion
C. 500 ml crystalloid intravenously
D. Blood cultures
E. Broad-spectrum antibiotics

Question 3

A 63-year-old man presented as an emergency after collapsing unconscious in the street. He came round quickly and described a sharp pain that was worse on breathing in, and feeling very dizzy on any ambulation. He had recently taken a flight home from New Zealand where he had been visiting his son.

On clinical examination, his temperature was 37.0°C, heart rate 112 beats/minute and regular, blood pressure 110/89 mmHg, respiratory rate 20/minute, and oxygen saturations 95% on 2 litres of oxygen.

While a CT pulmonary angiogram was being organized he started to feel very unwell, developed a new central crushing chest pain, became sweaty and clammy, and appeared grey. The blood pressure dropped to 86/54 mmHg, and heart rate rose to 130 beats/minute.

After pain relief, what is the most appropriate management for this patient?

A. Give treatment-dose enoxaparin
B. Await the computed tomography pulmonary angiography (CTPA) result, and if it confirms pulmonary embolism arrange for thrombolysis
C. Give treatment-dose enoxaparin and acute coronary syndrome treatment if the ECG changes or troponin increases
D. Thrombolyse immediately, without waiting for CTPA
E. Arrange for CT of the whole aorta to rule out dissection, and give aggressive fluid resuscitation