Shock: causes, initial assessment and investigations

Paul Teirney

Bilal Ahmed

Alistair Nichol

Abstract

Shock may result from a number of distinct disease processes and it is commonly associated with trauma, infection and cardiovascular dysfunction. Shock results in significant morbidity and mortality and is a leading cause of death in hospital patients. In order to improve patient outcomes it is important to recognize shock early, then assess and treat the shocked patient in a systematic way. While the cause of the shocked state is sometimes obvious, in more difficult situations the use of the clinical classification of shock into cardiogenic, obstructive, hypovolaemic or distributive shock can help the clinician to discover the underlying cause of the shock. However, it is important to note that while this is a framework in practice there if often considerable overlap between these different types of shock in clinical practice. After identification of patients in shock, immediate life-saving resuscitation with directed therapy to prevent further deterioration, worsening organ failure and to improve outcome is vital. An ABCDE approach can be a useful systematic way for initial assessment and resuscitation. Basic monitoring should be instituted as soon as possible and in severe or unresponsive shock this should be escalated to invasive monitoring. Immediate generic laboratory, microbiological and radiological tests should be carried out as soon as possible and should include a blood lactate level. Further targeted tests should then be tailored to the history, clinical findings and presumed aetiology of the shocked state. These targeted investigations should help to pin point the specific cause of the shock and guide definitive management.

Keywords Assessment; critically ill; sepsis; shock; treatment

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Paul Teirney *MB FCARCSI is an intensive care specialist registrar at the St Vincent University Hospital, Dublin, Ireland. Conflicts of interest: none declared.*

Bilal Ahmed *MBBS FCARCSI is an intensive care specialist registrar at the St Vincent University Hospital, Ireland. Conflicts of interest: none declared.*

Alistair Nichol *BA MB BCh BAO FCARCSI FCICM FJFICMI PhD is a Consultant Intensivist at St Vincent's University Hospital, Dublin, Ireland and Honorary Intensivist at the Alfred Hospital, Melbourne, Australia; Chair of Critical Care, University College Dublin and an Associate Professor of the Australian and New Zealand Intensive Care-Research Centre in the School of Public Health and Preventive Medicine, Monash University, Australia. Conflicts of interest: none declared.*

Learning objectives

After reading this article, you should understand that:

- Shock is associated with significant morbidity and mortality, which may be reduced with early recognition and appropriate treatment
- A generic approach to assessment and treatment of the shocked patient is essential to facilitate rapid diagnosis of the underlying cause while providing stabilizing interventions
- A classification of shock system can provide a rapid *aid memoire* to the most likely underlying cause and most likely effective interventions
- Targeted investigations directed towards determining the suspected cause of the shock

Introduction

Shock is one of the leading causes of death in hospital patients. Shock is commonly associated with trauma, infection and cardiovascular dysfunction. Traumatic injury is the leading cause of death worldwide among persons between 5 and 44 years of age.[1] The most common cause of death after traumatic injury is haemorrhagic shock. Shock associated with severe infection, which is also known as septic shock, has received much attention recently due to high profile campaigns.[2] The mortality rates associated with severe sepsis are 25–30%, but the development of shock is associated with a mortality rate up to 70%. In the United States the treatment of patients with severe sepsis costs an average of $2200 per case, with an annual total cost of $16.7 billion nationally.[3] Cardiogenic shock complicates 7–9% of patients presenting with myocardial infarction.[4] The mortality of cardiogenic shock is high, but has come down in recent years from 60% in 1995 to 48% in 2004.[4]

It is clear that shock is common and associated with significant morbidity and mortality. In an attempt to improve outcomes it is vital that clinicians recognize shock early, rapidly assess and treat patients in a systematic manner.

Recognition of shock

Definition

Shock is defined as acute circulatory failure with inadequate or inappropriately distributed tissue perfusion resulting in generalized cellular hypoxia.

Clinical features of shock are usually those of tissue hypoperfusion (see Table 1). This is most easily detected in the skin as central pallor, peripheral cyanosis, and increased capillary refill time. It is important to note that the traditional vital signs are less reliable indicators of shock and shock cannot be excluded solely on the basis of normal blood pressure (systolic blood pressure (SBP) <90 mmHg, mean arterial pressure (MAP) <60 mmHg) or a reduction of >40 mmHg from baseline. The complex interplay between the sympathetic and parasympathetic autonomic nervous system can produce pulse rates and blood pressures that are normal, high, or low. Furthermore, in shocked patients with end-organ hypo-perfusion, oxygen delivery to the tissues is not always reduced, and indeed may even be increased in some classes of shock (i.e. severe sepsis).[5]

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Signs of reduced end organ perfusion

Organ system	Clinical features
Neurological	• Reduced level of consciousness • Encephalopathy (confusion, agitation and/or drowsiness)
Cardiovascular	• Reduced capillary refill (>3 seconds) • Cold peripheries • Central pallor
Respiratory	• Tachypnoea >20 breaths/minute • Central and peripheral cyanosis • Desaturation (SpO_2 (peripheral oxygen saturation) <90%)
Renal	• Urinary output <0.5 ml/kg per hour

Table 1

Clinical classification

Clinically, it is common to subdivide shock into cardiogenic, obstructive, hypovolaemic or distributive causes. These classes of shock can be separated based on the main mechanism of the shock (i.e. cardiogenic-pump failure) and the resulting clinical presentation (see Table 2). However, in practice there can be considerable overlap between the different types of shock. Shock can arise through a variety of mechanisms simultaneously in a patient. For instance, in septic shock, there may be a combination of reduced preload from increased vascular permeability and vasodilatation (hypovolaemic), impaired myocardial contractility (cardiogenic) caused by inflammatory mediators reducing pump function, and inappropriate distribution of blood flow to tissue beds (distributive).

Hypovolaemic

Hypovolaemic shock is usually as a result of uncontrolled haemorrhage, but it can be due to excessive fluid loss from the gastrointestinal and urinary tracts, and even from the skin in severe burns.

Obstructive

Obstructive shock occurs when pump failure is due to extrinsic cardiac obstruction, rather than primary myocardial pathology. The most common and potentially reversible causes being pulmonary embolus, tension pneumothorax and cardiac tamponade.

Cardiogenic

Cardiogenic shock is the result of intrinsic myocardial disease, e.g. infarction, myocarditis. However, valvular abnormalities, e.g. mitral regurgitation due to papillary muscle rupture, can also lead to cardiogenic shock.

Distributive

Distributive shock can be due to sepsis, neurogenic (spinal) or anaphylactic shock. This type of shock can also occur with an adequate or increased cardiac output.

General approach to a shocked patient

Philosophy of approach

The goal is to identify patients that are in shock, provide immediate resuscitation with the aim to prevent further deterioration and restore the systemic circulation to a level that meets the body's tissue oxygen requirements (see Table 3).[6] In tandem it is vital to rapidly identify the cause of shock and to perform appropriate tailored investigations and to institute definite management.

Immediate management

ABCDE approach

The A-B-C-D-E (Airway, Breathing, Circulation, Disability and Exposure) principle should be applied for initial assessment and resuscitation. It is important that this process be iterative with frequent reassessments of the patient's condition and response to initial therapies.

Airway management

Consider early intubation and ventilation for severe shock if there is respiratory distress, severe hypoxaemia, pronounced acidosis, or coma. Intubation ensures protection from aspiration in the presence of a reduced conscious level. Where agitation is attributable to cerebral hypoxia, intubation and ventilation facilitates rapid treatment without precipitating further respiratory compromise.

Management of oxygenation and ventilation

Once intubated, inspired oxygen can be maximized to 100% to optimize oxygen delivery to the tissues. Mechanical ventilation will reduce oxygen consumption by the respiratory muscles at a time when their oxygen supply is compromised.

Fluid resuscitation

Conditions that are associated with actual or relative hypovolaemia respond well to restoration of intravascular volume. Titration of fluid administration to heart rate, blood pressure, urine output, lactate concentration is a good starting point prior to invasive monitoring. However, prediction of ongoing fluid responsiveness by objective measures is more difficult. For this purpose, passive leg raise test, pulse pressure and stroke volume

Classes of shock and resulting clinical presentation

Variable measured	Hypovolaemic	Obstructive	Cardiogenic	Distributive
Blood pressure	↓	↓	↓	↓
Central venous pressure [preload]	↓↓	↑	↑	↓
Capillary refill time	↑	↑	↑	↓
Skin temperature	↓	↓	↓	↑

Table 2

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