

Recognition and management of the patient with shock

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Introduction

The term shock probably originates from an inappropriate English translation of the word *choc*, which was used by the French army surgeon Henri Francois Le Dran to describe the collapse of vital functions culminating in death that he observed in soldiers who had been struck by missiles.

In the 1800’s John Collins Warren described shock as a ‘momentary pause in the act of death’, a description which remains accurate to this day if the diagnosis is missed or delayed.

Shock can be considered as the final pathway through which a variety of pathological processes lead to cardiovascular failure and death. Given the enormous spectrum of disease that can lead to shock it is not surprising that it is an extremely common cause of admission to the intensive care unit.¹

Definition

Shock is best defined as the inadequate delivery or utilisation of oxygen for cellular metabolic needs, i.e. any process which results in hypoxia at a cellular level. Most often shock arises as a result of ineffectual perfusion rather than impaired cellular oxygen consumption. It is important to appreciate that ineffectual perfusion can occur in the absence of hypotension, therefore a normal blood pressure does not exclude shock.

Classification

Traditionally shock is classified into four groups according to the main mechanism of decompensation (Table 1).

In **cardiogenic shock** the principle problem is the failure of the heart to act as an effective pump; this is commonly seen following acute myocardial infarction (MI). Over the last 20 years the incidence of shock complicating acute MI has been between 6% and 9%.

Hypovolaemic shock occurs as a result of a decreased circulating volume, which may arise as a consequence of acute bleeding, loss of plasma, (as in acute burns) or third space fluid losses such as those that can occur in pancreatitis.

Obstructive shock refers to those pathologies that restrict blood flow from the heart, for example massive pulmonary embolus (PE) or cardiac tamponade.

Distributive shock is characterised by maldistribution of blood flow, with septic shock being the most common example. Septic shock is defined as shock state in which there is a Systemic Inflammatory Response Syndrome (SIRS) associated with infection. This definition was adopted following a consensus conference in 1991.² However, recently this approach has been challenged³ and new criteria proposed.⁴ Neurogenic shock is also an example of distributive shock and can occur following spinal injury where there has been disruption of the sympathetic outflow from T1 to L2. In this situation unopposed vagal tone leads to a shock state, which is characterised by hypotension, bradycardia and hypothermia.

This classification provides the basis of a useful ‘clinical sieve’ for the differential diagnosis of shock that can be applied at the bedside. However it is important to realise that there may be considerable overlap between shock states classified in this way.

Cardiogenic	Obstructive	Hypovolaemic	Distributive
Myocardial infarction	Pulmonary embolus	Haemorrhagic:	Sepsis
Myocardial contusion	Cardiac tamponade	Trauma	Neurogenic
Myocarditis	Tension Pneumothorax	Gastrointestinal	Anaphylaxis
Acute valvular failure		Retroperitoneal	
Acute ventricular septal defect		Fluid depletion:	
Arrhythmia		Vomiting	
		Diarrhoea	
		Polyuria	
		Burns	

Table 1. Classification of shock

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Key Points

- A normal blood pressure does not exclude shock
- History, examination and simple investigations are usually sufficient to diagnose the presence and cause of shock.
- Echocardiography has an expanding role and can be invaluable
- Distinguishing those patients who are critically ill is a crucial first step. For such patients resuscitation runs in parallel to the diagnostic process.
- Early liaison with intensive care service is essential
- Lactate and base deficit describe severity of and predict outcome from shock
- Therapy titrated to central venous oxygen saturation (ScvO₂) can reduce mortality from septic shock
- Failure to improve on treatment should prompt review of therapy and diagnosis.

Thus, although septic shock is predominantly caused by abnormal vascular tone and maldistribution of blood flow, it frequently co-exists with myocardial depression and relative hypovolaemia.⁵ Recent evidence suggests that a significant proportion of patients with shock following myocardial infarction were septic,⁶ while profound myocardial depression inevitably accompanies the terminal stages of all shock states.

Pathophysiology

Fundamentally different diseases lead to the development of shock because they share the common features of either inadequate oxygen delivery or utilisation by tissues. Depending on severity and duration a cascade of events is triggered which leads to cell damage, systemic inflammation, organ failure and death.

At a cellular level there is a switch from aerobic to anaerobic metabolism with an accumulation of lactate, hydrogen ions and inorganic phosphates. If this situation persists an imbalance occurs between ATP synthesis and demand precipitating an energy crisis within cells with multiple downstream effects. There is a loss of membrane integrity resulting in the accumulation of sodium, calcium and water resulting in generalised cell swelling. Protein synthesis is impaired and there is an imbalance between free radical generation and scavenging resulting in oxidative stress. The latter accelerates cell damage through lipid peroxidation. Mitochondrial dysfunction occurs as a result of changes in membrane permeability, increased cytosolic calcium, free radicals and lipid peroxidation, eventually leading to the initiation of apoptosis.⁷

Clinical approach

Recognising the patient with shock is generally straightforward when there is a suggestive history and corroborative clinical and investigative findings. The diagnosis of hypovolaemic shock is self evident in the setting of haematemesis, hypotension and lactic acidosis. However the diagnosis may be much more difficult with a limited history, confounding co-morbidity such as hypertension or 'occult' shock revealed only by invasive haemodynamic monitoring.⁸ Moreover the predominant mechanism of shock may be difficult to untangle in the presence of 'dual' pathologies such as myocardial infarction complicating gastrointestinal bleeding or sepsis precipitating diabetic ketoacidosis.

The first step in approaching any acute medical admission regardless of the diagnosis, is to decide whether or not that patient is critically ill and in need of immediate intervention. Rapidly assimilating all of the available history and conducting a brief examination, which focuses on key cardio-respiratory and neurological observations will usually accomplish this. These include assessment of airway patency, respiratory rate, heart rate, blood pressure, conscious level and, if available, urine output.

In recent years deviations from the 'normal' range for these observations have been used as an alerting mechanism for potential critical illness, either independently e.g. Medical Emergency Team call out criteria,⁹ or as a composite score such as the Modified Early Warning Score.¹⁰ Whilst this approach has the virtue of simplicity, these systems do not take into account clinical acumen or subtle signs of serious illness. Moreover, co-morbidity and drug therapy may confound the 'normal' ranges for the scored observation. Despite these limitations in the right context the adoption of an early warning score may be a useful initial triage tool.

Clinical findings, which accompany critical illness and suggest shock include: hypotension (SBP <90 mmHg), tachypnoea (Respiratory Rate >25/min), tachycardia (Heart Rate > 120/min) altered mental status, delayed capillary refill (> 2 seconds), decreased urine output (< 0.5 ml/Kg/Hr), cool skin, and cold extremities. Taken independently none of these clinical findings have high sensitivity or specificity for shock. Although a capillary refill time of >2 seconds is widely quoted as being indicative of hypovolaemia, when this hypothesis was tested in healthy volunteers it was found that using the 2 second rule to define delayed capillary refill resulted in a false positive rate of 13% for adult females and 30% in the elderly.¹¹ Tachypnoea is a particularly sensitive indicator of critical illness¹² and is invariably present in shock states.

The sensitivity and specificity of the clinical findings associated with shock are greatly improved if they are considered all together. The presence of two or more of these signs strongly suggests that the patient is critically ill and at increased risk of death.¹³

Following initial triage patients will either be undergoing a modified diagnostic process in parallel with resuscitation or will be subjected to a more traditional clinical approach. Essentially three questions need to be addressed (**Figure 1**):

1. Is the patient critically ill?
2. Does the patient have shock?
3. If so what type of shock is it?

Some physical findings may help discriminate between the different shock states:

- The jugular venous pressure may be elevated in cardiogenic and obstructive causes of shock but will be low in hypovolaemia.
- The patient with septic shock may be peripherally warm with bounding pulses although in the later stages of the disease the same patient may become cool with small volume pulses ('cold shock').
- The characteristic purpuric rash that accompanies meningococcal septicaemia.

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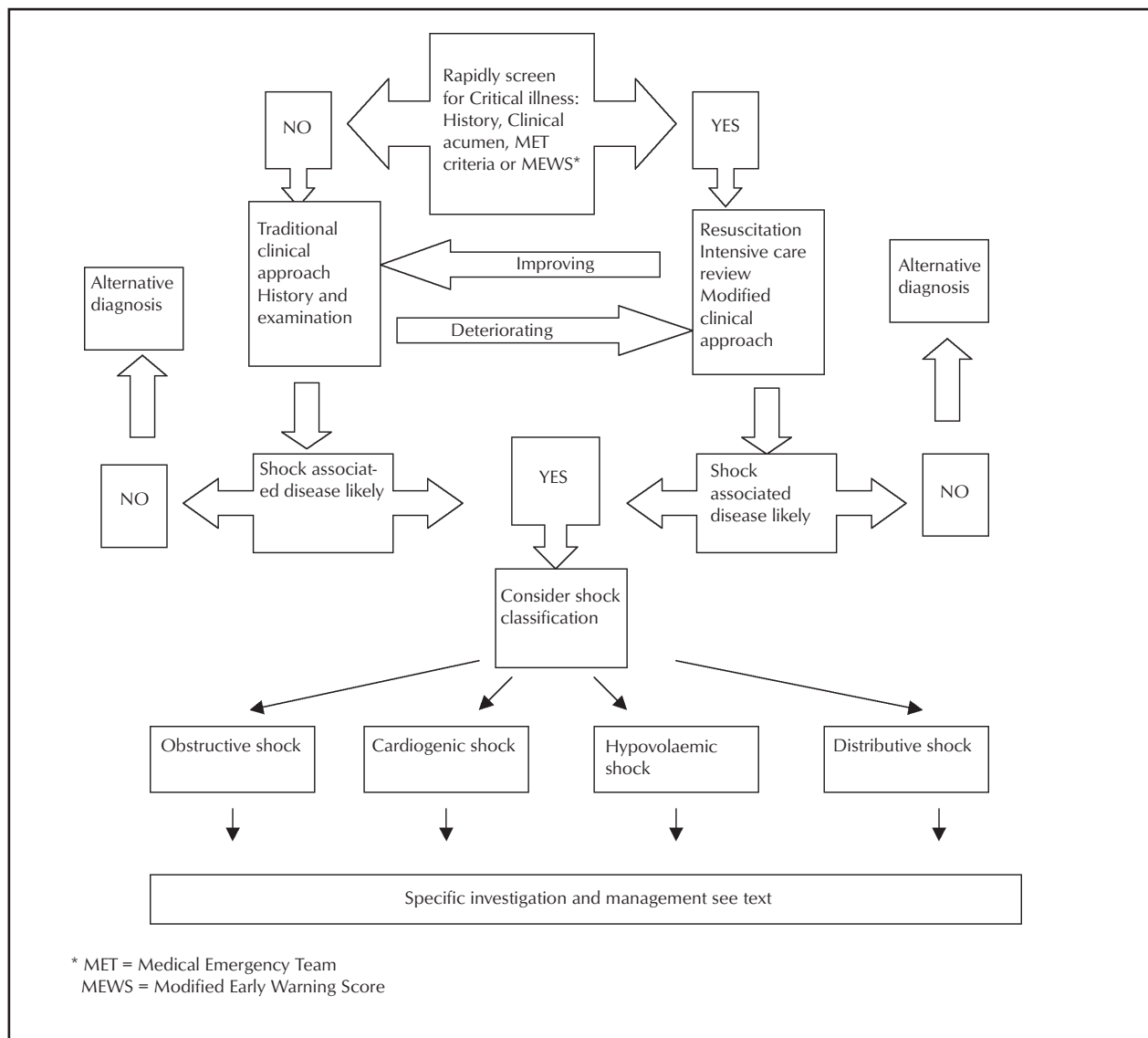


Figure 1. Initial approach to the acute medical admission with potential shock

- Beck's triad of hypotension, muffled heart sounds, and raised jugular venous pressure, associated with cardiac tamponade.

Investigations

Routine laboratory and radiological investigations may be informative in establishing the cause of most shock states. Thus chest X-ray, electrocardiograph, full blood count, arterial blood gas (including lactate), urea and electrolytes, c-reactive protein, troponin, blood clotting screen as well as appropriate microbiological cultures may all be helpful.

The blood lactate level is particularly useful in assessing the severity of shock, as hyperlactaemia develops as a result of a switch from aerobic to anaerobic metabolism. For many years now it has been recognised that increased blood lactate levels and a failure to normalise lactate levels during treatment have been associated with an increase in both mortality and morbidity.¹⁴

Transthoracic echocardiography is an invaluable tool for identifying causes of left ventricular failure, providing information on ventricular preload and circulatory status,¹⁵ as well as diagnosing tamponade. Echocardiography is also very useful in the diagnosis of shock attributable to pulmonary embolus because it can be performed at the patient's bedside and usually in a more timely fashion than CT pulmonary angiography (CTPA). Delayed treatment of massive PE carries an extremely high mortality. Echocardiographic findings suggestive of PE include: a dilated hypokinetic right ventricle, specifically with apical sparing, an increased RV/LV ratio, dilated proximal pulmonary arteries and an increase in tricuspid regurgitant flow velocity. There may also be loss of collapse of the inferior vena cava, which is usually seen on inspiration.¹⁶

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Management

General measures:

For patients who have been triaged as critically ill, resuscitation should be prompt, as should measures specific to the underlying diagnosis. However, general principles of resuscitation should not be ignored. Measures should be taken to ensure the airway is patent, supplemental oxygen is applied with adequate ventilation, and appropriate restoration of circulation.

The patient may require intubation either because they have a reduced conscious level and are unable to protect their airway or they are in respiratory failure unresponsive to other therapy. Mechanical ventilation optimises delivery and reduces consumption of oxygen by muscles of the respiratory pump at a time when oxygen delivery is compromised. Moreover, additional investigation and interventional procedures are safer and easier to carry out on an intubated patient rather than one who is confused or agitated.

It is imperative that the patient is managed in an area where there is appropriate medical and nursing expertise, and suitable monitoring. Minimum monitoring requirements are: Continuous electrocardiography, pulse oximetry, blood pressure, respiratory rate, urine output and Glasgow Coma Score (GCS). However, central venous access and an arterial line with a pressure transducer should be introduced as quickly as possible. If shock does not resolve rapidly with simple measures or if vasoactive drugs are required, then additional haemodynamic monitoring may be required to provide information on cardiac output and vascular resistance.

Invasive haemodynamic monitoring:

An arterial cannula allows continuous monitoring of blood pressure, as well as sequential blood gas analysis. Serial values of pH, lactate and base deficit allows response to treatment to be tracked. Failure to detect an improvement in these variables should lead the physician to reconsider if the diagnosis and treatment are correct.

Central venous access allows measurement of central venous pressure (CVP) as well as central venous oxygen saturations (ScvO₂). The latter gives a measure of the balance between oxygen delivery and extraction by the tissues and will be low (<70%) in the majority of shock states. A normal value for ScvO₂ does not exclude shock, but a low value should prompt either a change or review of therapy, particularly if other physiological variables are abnormal. In septic shock the ScvO₂ may be low, normal or elevated (>75%). The latter represents either maldistribution of flow through the microcirculation and/or an inability of cells to utilise oxygen. In the past true mixed venous oxygenation saturations (SvO₂) were obtained through a pulmonary artery (PA) catheter. Use of PA catheters is generally diminishing in the UK and Europe in favour of other methods of haemodynamic monitoring.

Central venous pressure is often regarded as a measure of preload for the heart, however any given value does not predict preload responsiveness. Thus a CVP of 8mmHg

may represent hypovolaemia, euvolaemia or hypervolaemia in three different patients. Fluid is, with the exception of cardiogenic shock, required in most forms of shock. Monitoring CVP may be useful in guiding initial therapy after a fluid challenge. Following a rapid infusion of 250-500ml of fluid a rise in the CVP of 2-3mmHg should represent an adequate increase in preload. The response to treatment may be measured against variables such as increases in blood pressure, urine output, conscious level and a fall in heart rate and lactate.

Measurement of cardiac output should always be considered if there is not a rapid response to therapy and resolution of the shock state. Traditionally the pulmonary artery catheter has been used to estimate cardiac output in critically ill patients. This method is now less frequently used in favour of other devices and include oesophageal Doppler, Lithium Dilution Cardiac Output (LiDCO) and Pulse Contour Cardiac Output (PiCCO) systems.

Fluid Therapy

Fluid may be administered as crystalloid, colloid or blood products. In general the UK favours colloids for resuscitation, whereas in the USA crystalloids are more likely to be given. Each type of fluid has advantages and disadvantages over the others. In many shock states the coexisting inflammatory response means that much of the administered fluid may leak through capillaries and therefore have less effect on plasma expansion than in "normal" individuals. Another important consideration is that many colloids consist of osmotically active molecules (starch, gelatin, Dextran, albumin) in normal Saline rather than physiologically balanced solutions. Although not within the scope of this review large volume resuscitation with fluids rich in normal saline can lead to a metabolic acidosis.¹⁷ It is important to realise that this acidosis is the result of fluid therapy rather than the shock state. It may be avoided by using more physiological solutions such as Hartmann's solution.

Specific therapy

Distributive shock: Septic shock is the commonest form of distributive shock. Although mortality for individual episodes is falling, changes in patient demographics mean that the overall incidence of septic shock and death are increasing.¹⁸ In 2004 a number of learned societies introduced a set of guidelines, which aimed to improve patient mortality.¹⁹ These guidelines are currently being updated and should be available later this year. A more detailed account of the management of septic shock and the surviving sepsis campaign is found in the article on p68.

Hypovolaemic shock: In addition to fluid resuscitation treatment of fluid losses should focus on the underlying diagnosis (e.g. treating infective diarrhoea, diabetic ketoacidosis, or relieving bowel obstruction) whilst paying careful attention to and correcting electrolyte abnormalities. Patients with burns should be referred to a specialist centre.

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Cardiogenic shock: may require support with inotropic drugs such as adrenaline or dobutamine. Once again familiarity of using a drug and with haemodynamic monitoring, are probably more important than differences between drugs. In the setting of an acute myocardial infarct (MI) thrombolysis, angioplasty or occasionally coronary artery bypass grafting may be required. Surgery is also indicated for cardiogenic shock as a result of acute valve lesions or septal defects complicating MI. An intra-aortic balloon pump may be used as a short term bridge to either surgery or recovery of hibernating myocardium.

Obstructive shock: requires the insertion of an appropriate drain when this results from either a tension pneumothorax or cardiac tamponade. Pulmonary embolism causing shock requires thrombolysis or embolectomy if the former either fails or is contraindicated, followed by anticoagulation. One caveat is that thrombolysis is of proven benefit only when there is hypotension and right ventricular dysfunction on Echocardiography (massive PE). The risk benefit ratio of thrombolysing patients with normal blood pressure, but other features of shock such as a raised lactate and right ventricular dysfunction (submassive PE), probably does not favour the administration of these drugs, though this is somewhat controversial.^{20,21}

Conclusion

Shock is a medical emergency which, regardless of its aetiology is associated with a high mortality. Early recognition, accurate diagnosis of the underlying cause and prompt treatment can improve the outcome for these critically ill patients.

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Further Information

www.survivingsepsis.org – Current guidelines on the management of septic shock.
www.srlf.org – Details of the 8th International Consensus Conference: Haemodynamic monitoring in shock and implications for management. Paris, April 27th–28th 2006.