Breathlessness and cough in the acute setting

Ben G Marshall
Veronica White
Jo Loveridge

Abstract

The symptom of breathlessness is well-recognized as part of the presentation of a wide range of conditions. It can be a manifestation of a life-threatening emergency. In the acute medical setting, the priority is to quickly recognize patients who are critically unwell and require emergency treatment. For these individuals, rapid initial assessment and immediate treatment are essential. However, once the symptoms have stabilized, or in a less acute setting, a more thorough assessment is required. Cough is a common respiratory symptom, often part of a symptom complex, which is troublesome for the patient. It is important to recognize worrying associated features to prompt further investigation.

Keywords Acute cough; acute setting; breathlessness

Key points

Breathlessness

- Patients with breathlessness can present acutely unwell
- An urgent assessment of airway, breathing and circulation should be made in the first instance
- A detailed history and examination should be made when the patient is more stable, along with more detailed investigations such as lung function and echocardiography. This usually leads to the correct diagnosis

Acute cough

- Patients with acute cough associated with haemoptysis, fever, chest pain, breathlessness or weight loss should have an urgent chest X-ray (CXR)
- Patients are unlikely to present acutely with the sole symptom of chronic cough. However, if it is part of a symptom complex as an acute presentation, the patient should be referred for a CXR. If this is normal, spirometry is non-invasive and readily available
- Serious conditions that can present with an isolated cough include malignancy, tuberculosis, interstitial lung disease and foreign body
- Asthma, postnasal drip and gastro-oesophageal reflux disease are the most common pathologies causing cough in a patient with no systemic symptoms and a clear CXR
- If the cause of a patient's cough cannot be identified or they fail to respond to a therapeutic trial, they can be referred to a specialist cough clinic

Breathlessness

Breathlessness is a common symptom in patients presenting in the acute secondary care setting (as well as in primary care). Patients can present acutely or with worsening of more chronic symptoms. Symptoms range from mild worsening of the patient's normal level of function through to a life-threatening medical emergency. Breathlessness is a recognized symptom of cardio-respiratory disease but can also be present in other systemic conditions, such as anaemia or endocrine disorders. Competent assessment and management of the breathless patient is essential to reduce morbidity and mortality.

Definition

'Breathlessness' or dyspnoea is a symptom that is complex to define; it is not a clinical sign. It is used to describe discomfort or distress associated with breathing. This includes the sensation of suffocation, choking or 'air hunger', inability to take an adequate breath, rapid breathing or increased effort related to breathing. It can also be used by patients to describe chest tightness. It is important to understand and help patients define what they mean when they describe breathlessness.

Pathophysiology

The physiology of normal ventilation and respiration is complicated, and the detailed pathophysiology of breathlessness is not fully understood. Unlike pain, there are no specific 'breathlessness' receptors or nerves; neither is there a location within the brain that can be recognized as a breathlessness centre. A number of distinct stimuli trigger a variety of receptors; the afferent input leads to the sensation of 'a need to breathe' (like thirst or hunger sensations). Breathlessness can arise because of either an increased drive to breathe or increased work of breathing. It is a subjective sensation that can be perceived only by the sufferer, further complicated by the fact that social, cultural, emotional and situational factors influence how the sensation is perceived.

Ben G Marshall *PhD FRCP DTM&H is a Consultant Chest Physician at University Hospital of Southampton, and Honorary Senior Lecturer at the University of Southampton, UK, leading subspecialist services in tuberculosis and interstitial lung diseases. His research interests also lie in these disease areas with projects and themes to identify the pathophysiological mechanisms responsible for causing progressive lung damage. He is involved in education on the Southampton Graduate entry programme. Competing interests: none declared.*

Veronica White *MSc MD is the Clinical Director of Respiratory Medicine at St Bartholomew's Hospital, Barts Health NHS Trust, UK. She trained in North East Thames in Respiratory and General Medicine and sub-specialised in tuberculosis (TB); she studied for an MSc in Medical Anthropology at UCL in 1999 and MD thesis at QMUL (2005). Competing interests: none declared.*

Jo Loveridge *MBChB(Hons) MRCP DTMH Diploma in Clinical Education is a Consultant in Acute Medicine at King's College Hospital, London, UK. Competing interests: none declared.*

Aetiology

Because the sensation of breathlessness can be provoked by various mechanisms, many different disease states present with it. Breathlessness is usually caused by cardiac or pulmonary disorders. However, it can also be present in metabolic upset, anaemia and neuromuscular weakness, as well as being a consequence of pain, anxiety and physical deconditioning (Table 1).

Assessment

If patients can present acutely unwell, the conventional approach of a history followed by examination and then investigations may not be appropriate. Instead, the 'ABC' approach (Table 2) can be used, with immediate interventions administered as required. This urgent assessment aims to detect and correct life-threatening disturbances in physiology and commence emergency treatment. Initial investigations include an urgent electrocardiograph (ECG) and chest X-ray (CXR), and blood tests (Figure 1).

ABC assessment

Airway: in most cases, the airway is patent, but rapid assessment for airway obstruction should be undertaken (Table 3). In breathlessness, immediate oxygen should be given with target oxygen saturations of 94–96%, except for decompensated chronic obstructive pulmonary disease (COPD), when the target is 88–92%.[1] It is important to assess whether the patient can talk and to examine for foreign bodies and secretions such as blood or vomit around the mouth. With obstruction, immediate suctioning can be needed. In severe obstruction, paradoxical movement of the chest wall and abdomen may be seen. Breathing can be noisy if the airway is partially obstructed, with audible stridor, gurgling or grunting. Airway manoeuvres (head tilt and chin lift) or airway adjuncts may be required to keep the airway clear, and immediate anaesthetic help can be needed. Laryngeal oedema and airway compromise can occur with anaphylaxis; this should be managed according to recommended guidelines. If an infective cause of airway obstruction is suspected, examination of the oropharynx should be undertaken in an area where the patient can be immediately intubated as manipulation of the airway can cause further obstruction. Antibiotics are the mainstay of treatment, but corticosteroids are sometimes used to treat airway oedema.

Causes of breathlessness

Acute	Subacute	Chronic
Airways obstruction	Pneumonia	COPD
Anaphylaxis	Exacerbation of COPD	Pleural effusion
Asthma	Angina	Malignancy
Pneumothorax (including tension)	Cardiac tamponade	Chronic pulmonary embolus
Pulmonary embolus	Metabolic acidosis	Restrictive lung disorders
Myocardial infarction	Pain	Congestive cardiac failure
Pulmonary oedema	Pontine haemorrhage	Valvular dysfunction
Arrhythmias	Acute hypersensitivity pneumonitis	Cardiomyopathy
Anxiety		Diastolic dysfunction
Anaemia		Neuromuscular disorders
		Deconditioning
		Interstitial lung disease
		Abdominal distension, e.g. ascites

Table 1

The ABC approach

Airway	Is the airway patent? Is the patient talking? Is there noisy breathing or drooling?
Breathing	Check the respiratory rate. Measure oxygen saturations. Look at, feel and listen to the chest
Circulation	Measure heart rate blood pressure and capillary refill time. Check for pallor, sweating and warmth

Table 2

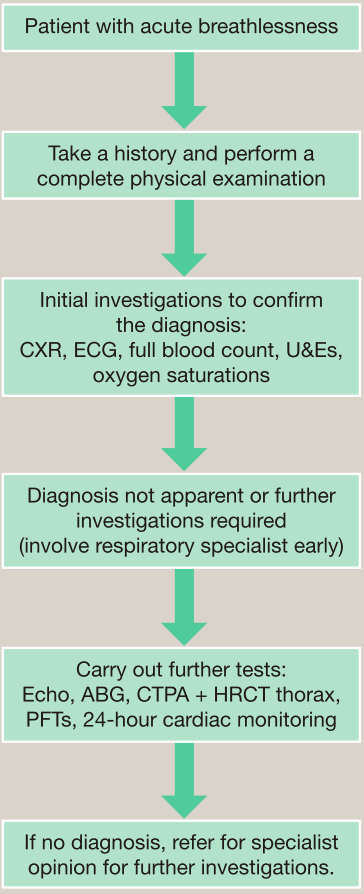
Breathing: most patients complaining of breathlessness have a raised respiratory rate and may be using their accessory muscles. All patients should be assessed for whether they can easily speak in sentences. Poor respiratory effort is always worrying as the patient may be tiring, and intensive therapy unit help may be needed. If expiratory wheeze is heard on auscultation of lung fields, bronchodilator therapy and corticosteroids should be given following guidelines for asthma or COPD.[2]

Continuous monitoring of oxygen saturations is essential for all acutely unwell patients with breathlessness. For patients with hypercapnic respiratory failure, who are not critically ill, target oxygen saturations should be 88–92%, using prescribed controlled 24–28% oxygen via a Venturi mask. Blood gas analysis guides further management, including the need for non-invasive or invasive ventilation if adequate oxygenation is not possible because of hypercapnia and acidosis. Examination of the chest can reveal unilateral hyperresonance and reduced breath sounds suggestive of a pneumothorax. If there are signs of 'tension' (tracheal deviation, tachycardia, hypotension), immediate needle thoracocentesis should be performed using an 18G cannula in the second intercostal space in the mid-clavicular line, followed by definitive chest drain insertion.

Patients with pulmonary oedema can present *in extremis*, unable to complete full sentences and with diffuse widespread inspiratory crepitations prompting immediate treatment with diuretics, nitrates and opiates. Rapid improvement may be seen, but if the patient remains hypoxic, continuous positive airway pressure should be considered. Tachypnoea and hypoxia with signs of increased vocal resonance and bronchial breath sounds can indicate infective consolidation. Signs of associated systemic sepsis should be sought and, if present, treated according to 'surviving sepsis' guidelines. Blood cultures should be taken and appropriate antibiotic therapy given.

Circulation: immediate intravenous access should be obtained and haemodynamic compromise addressed. Continuous blood pressure and ECG monitoring with oximetry is essential to monitor progress in acutely unwell patients. As arrhythmias can cause breathlessness, patients should be assessed for associated

Investigation algorithm for the management of acute dyspnoea



ABG, arterial blood gases; CTPA, computed tomographic pulmonary angiography; Echo, echocardiography; HRCT, high-resolution computed tomography; PFT, pulmonary function test; U&Es, urea and electrolytes.

Figure 1

features of chest pain, altered mental state, signs of cardiac failure and hypotension. Arrhythmias should be managed according to current guidelines.

Cardiac ischaemia can be silent with no chest pain and present only as breathlessness (especially in patients with diabetes mellitus). An urgent ECG should be performed as this can show ischaemic change, axis change or an arrhythmia, and guide diagnosis and management. Pulsus paradoxus is an exaggerated inspiratory fall in systolic blood pressure (>10 mmHg). It is not easily elicited but can be a worrying sign in severe asthma or cardiac tamponade. With cardiac tamponade, there can be associated hypotension, tachycardia with an elevated jugular venous pressure (rising further with inspiration) and diminished heart sounds or a pericardial rub (Kussmaul's sign). If it is suspected, urgent echocardiography with drainage of the effusion is indicated. Pulmonary embolus often presents with breathlessness, and computed tomographic pulmonary angiography (CTPA) is the investigation of choice.[3] All patients should be treated immediately; those with suspected massive pulmonary embolus can require thrombolysis. This treatment should always be conducted with the involvement of a senior specialist in a high-dependency setting.

Causes of airway obstruction

- Blood
- Vomit
- Secretions
- Foreign body
- Airway oedema (with anaphylaxis or infection, e.g. epiglottitis)
- Central nervous system depression and loss of airways reflexes
- Malignancy

Table 3

Further assessment of stable patients

In patients who have been stabilized and those who are less acutely unwell, a detailed history of the presenting complaint should be explored. This should include pre-existing cardiac or pulmonary disease, and standard questions about the onset and duration of the breathlessness, whether it occurs on exertion or at rest, and whether there is orthopnoea or paroxysmal nocturnal dyspnoea. Additional cardiorespiratory questions should cover cough, sputum, fever, chest pain, pleurisy, haemoptysis and oedema. A smoking history should not be missed. Non-cardiopulmonary causes of breathlessness are rare, but underlying neurodegenerative disease (e.g. motor neurone disease) can present with breathlessness (related to increased effort).

On examination, special attention should be paid to the cardiorespiratory system, supported by a full physical examination. Anxiety or hyperventilation can lead to a sensation of breathlessness, but physicians should ensure that other causes of lung disease are ruled out before making such a diagnosis; it should not be made routinely in the acute setting. These patients often require specialist referral.

Cough

Definition

Cough is 'a forced expulsive manoeuvre, usually against a closed glottis, that is associated with a characteristic sound'. It is a protective reflex to help keep the airways clear and expel aspirated foreign bodies. Acute cough is defined as lasting less than 3 weeks, whereas chronic cough persists for more than 8 weeks. The 'grey' area in between is often termed subacute cough.

Pathophysiology

Cough receptors are present in the larynx, carina and branching points of the proximal airways, and are triggered by mechanical or chemical irritants. Afferent nerves carry information to the cough centre in the brainstem. Efferent outflow leads to a rapid inspiration and rise in intrapleural pressure, resulting in a forced expiration that expels foreign matter. Patients with chronic cough often demonstrate a hypersensitive cough reflex that probably relates to increased sensitivity of the receptors in the upper or lower airways, or to changes of processing in the

Causes of cough

Causes of cough	Examples
Respiratory	Viral or bacterial infection, bronchospasm, COPD or chronic bronchiolitis, malignancy, interstitial lung disease, pleural disease
Upper airways disease	Postnasal drip, sinusitis, hyperreactive upper airways syndrome
Cardiovascular disease	Left ventricular failure with pulmonary oedema, mitral stenosis (with elevated pulmonary venous pressures)
Gastro-oesophageal disease	Gastro-oesophageal reflux disease
Neurological disease	Recurrent micro-aspiration
Drugs and irritants	Angiotensin-converting enzyme inhibitors, cigarette smoke

Table 4

brainstem. A poor cough reflex leads to atelectasis of lung subsegments and predisposes to recurrent infection. Conversely, excessive or vigorous coughing can result in complications, including urinary incontinence, hernia or rib fracture. The cough reflex can be in part controlled voluntarily to either produce or suppress a cough.

Epidemiology

Cough is one of the most common reasons for consulting a general practitioner, but is not a common presentation in the acute medical setting, except as part of a symptom complex. The economic burden of cough is huge based on the cost of consultations, the lost days of work and the fact that approximately £100 million is spent per year on over-the-counter preparations.

Aetiology

Acute cough usually results from a viral upper or lower respiratory tract infection and is self-limiting. The common causes of chronic cough with a clear CXR are smoking, asthma, postnasal drip and gastro-oesophageal reflux disease. Other conditions that present with cough are potentially serious and should be excluded during clinical assessment (Table 4).

Assessment of acute cough

Cough in the acute setting is often part of an acute symptom complex, such as an influenza-like syndrome, patients presenting with fever, upper airway symptoms, myalgia and cough. It is important to elicit associated worrying symptoms that merit further assessment. In particular, patients with associated haemoptysis, breathlessness, fever, chest pain or weight loss require a CXR. Serious conditions associated with an isolated acute cough include neoplasms, tuberculosis, foreign body inhalation and interstitial lung disease.[4] Patients at greater risk in the UK include current or ex-smokers, immunosuppressed individuals and contacts of patients with pulmonary tuberculosis. Those with suspected foreign body inhalation should be referred for further specialist review and bronchoscopy by a thoracic or ENT surgeon. If there are no significant associated symptoms or worrying findings, the patient can usually be reassured that the cough is viral in origin and will be self-limiting, usually within 3 weeks (although post-viral cough can sometimes take longer to settle). The opportunity to advise smokers to quit should always be taken. Antibiotics are generally not indicated, but over-the-counter preparations can offer symptomatic relief. The patient should be advised to return if the cough persists or they develop associated worrying symptoms. ◆

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The shocked patient

Craig Prescott
Stuart Ruff

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%.

Craig Prescott *MB BS BMedSci MRCP (Acute) PGDip is a Consultant Acute Physician at Poole Hospital, UK, and Clinical Lead for AKI and DVT. He has a special interest in stress echocardiography. Competing interests: none declared.*

Stuart Ruff *MB ChB MRCP PGDip is a Specialist Registrar in Geriatric Medicine at The Royal Bournemouth Hospital, UK. He has a special interest in medical ethics and law. Competing interests: none declared.*

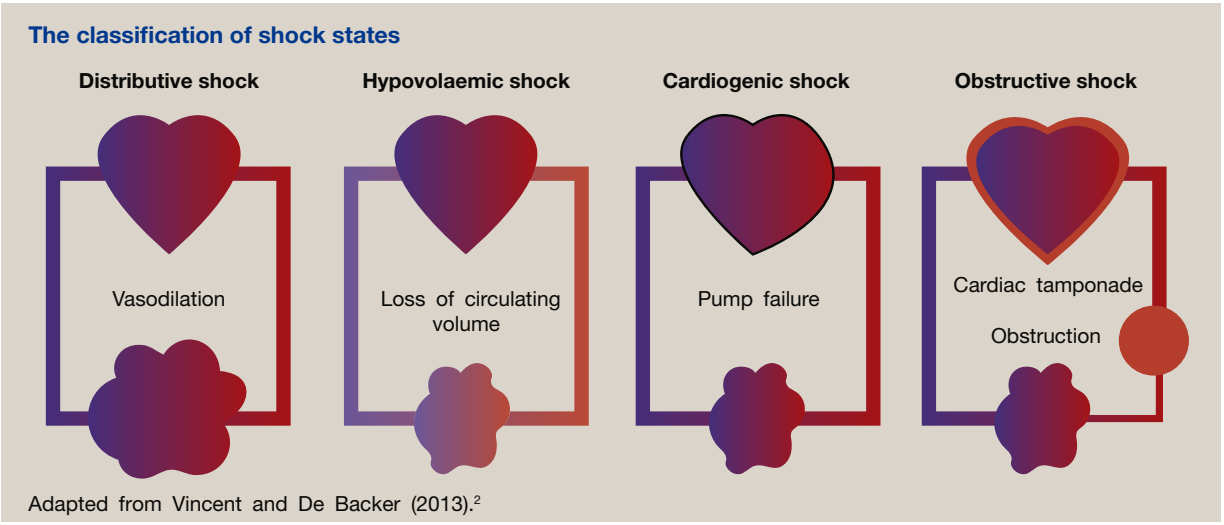

Figure 1

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

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
Bloods	• **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
Imaging	• **Chest radiograph** • Plain radiograph of extremities • Echocardiography • Abdominal ultrasound • Abdominal CT • CTPA
Bedside tests	• **ECG**

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

Table 3

Therapeutic strategy

The principles of initial resuscitative therapy for the shocked patient 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]

Ventilation

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 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. In the shocked patient, this can be problematic 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) allows assessment of response while decreasing the risk of iatrogenic harm. Following 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 following fluid administration can indicate a need for change in strategy and should prompt seeking expert help. Further fluid boluses should be administered if on reassessment the patient remains hypovolaemic. If 2 litres have been administered and there is still evidence of shock (failure to improve physiological parameters or a lactate concentration that is not improving), expert help should be sought.[3]

Patients with oedema can cause confusion when managing shock. Oedema 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 would be evidence of pulmonary oedema causing respiratory compromise (see Cardiogenic shock, below).

Pump

Vasoactive agents can be used to improve hypotension in patients who are fluid-resistant 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 initiated early while fluid resuscitation is continuing, in order to support blood pressure, with the aim of cessation once hypovolaemia has been corrected and physiological parameters have normalized. Inotropic therapy can be used in cardiogenic shock, although few controlled trials have been performed. Therefore agents such as dobutamine and (unlicensed indication) levosimendan 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 to 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, such 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 major haemorrhage policy, to keep the platelet count greater than 50×10^9/litre, prothrombin time greater than 1.5 and fibrinogen greater than 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 and discussion with a haematologist regarding factor Xa inhibitors as there is no specific antidote.

There is no evidence for use of 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. Studies are continuing regarding tranexamic acid as evidence of a beneficial effect is not conclusive.

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 to 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 may 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 consideration of reperfusion therapy.

Cardiac dysrhythmias causing shock should be corrected urgently as per Resuscitation Council 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 (CTPA), 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. Although the definitions of sepsis and septic shock have recently been updated, management has not changed.[4] The Surviving Sepsis Campaign bundle should be completed within 3 hours, ideally within 1 hour. It includes intravenous antibiotic therapy, blood cultures before antibiotic administration, lactate measurement and crystalloid administration 30 ml/kg for hypotension or lactate concentration greater than 4 mmol/litre.[5] It is important that the source of sepsis is identified and controlled. In anaphylaxis, the treatment is immediate intramuscular adrenaline, with removal of the trigger if it is still present. Intravenous chlorphenamine and hydrocortisone should also be administered. ◆

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Medical complications of surgery: an orthogeriatrician's perspective

Mark A Baxter

Abstract

Hip fracture is one of the most common admission diagnoses in the UK, accounting for more than 75,000 hospital admissions per year in England and Wales. Fractured neck of femur can be classified as a 'frailty' presentation because of an average age of over 80 years, a clear association with 'pathological falls', including syncope, and a high number of co-morbidities. The combination of traumatic injury, frailty and surgery presents significant challenges to the managing team and requires an multidisciplinary approach to reduce the risk of perioperative complications, most commonly postoperative delirium, sepsis, cardiac complications, stroke and metabolic disturbance. Close monitoring with geriatric co-management has been shown to reduce complications and resultant morbidity, length of stay and mortality. Many lessons learnt from the experience of managing these very challenging patients are transferable to other frail patients undergoing surgery.

Keywords Delirium; falls and syncope; frailty; hip fracture; orthogeriatrics; rehabilitation

Key points

- The incidence of older people undergoing surgery is increasing
- Older people with frailty present specific challenges from co-morbidity, polypharmacy and physiological changes
- Older people undergoing surgery require a coordinated, multidisciplinary approach to their care
- Close perioperative monitoring reduces the risk of post-operative complications
- Early mobilization and promotion of functional independence gives the best chance of meaningful recovery

Introduction

Hip fracture is a common presentation across the UK, with approximately 65,000 admissions across England, Wales and Northern Ireland in 2015[1] and accounting for more than 4000 inpatient beds at any given time. Over the last 25 years, it has become increasingly recognized that these patients represent a significant challenge in their medical management owing to the prevalence of frailty and multiple co-morbidities in this group.[2] This has led to the development of models of geriatric co-management or orthogeriatric services to provide multidisciplinary team (MDT) care to improve outcome in terms of functional recovery and mortality.

Many hip fracture patients would be classified as acute medical admissions but, because they have sustained a traumatic injury, they will come under the care of non-medical specialists. They should be assessed in the same way as any older person admitted with complex problems, while understanding the need for early surgical intervention. When treating these frail and complex patients, it is best to break the stages of admissions down and address each problem associated with these aspects:

- preoperative:
  - presenting complaint
  - relevant co-morbidities
  - pre-optimization
- perioperative:
  - early postoperative complications
  - management of risk
- postoperative:
  - early postoperative complications
  - late postoperative complications.

Preoperative assessment

Falls and syncope

Fractured neck of femur is classically a low-trauma fracture, that is, an injury sustained from a fall from standing height or less. As this presentation can indicate underlying pathological processes, a thorough medical assessment is mandatory. A formal history of falls can highlight a syncopal event and potential cardiogenic causes, with a particular focus on 'red flags' associated with cardiogenic syncope.[3] Any evidence of cardiogenic syncope or underlying cardiac abnormalities increases the risk of perioperative cardiac complications. In addition, as is common with many admissions for frail older people, falls represent the presenting complaint for many underlying pathologies, such as neurological, musculoskeletal or acute decompensation of underlying chronic disease. A comprehensive medical history and examination highlights any acute illness causing the fall and fracture. Prompt management of these reduces the risk of complications with anaesthesia and surgery.

Highlight and address relevant co-morbidities

As previously stated, hip fracture is a 'frailty' presentation and as such many of the patients have multiple co-morbidities (Table 1), over one-third having one or more significant co-existing conditions. A thorough assessment and wherever possible optimization of these pre-empt possible and hence reduce potential complications.

The management of each co-existing illness should be reviewed, paying close attention to the potential risks associated

Mark A Baxter *MBChB FRCP is a Consultant Orthogeriatrician at University Hospital Southampton and Clinical Lead for Trauma and Orthopaedic Medicine, UK. Conflicts of interest: none declared.*

Co-morbidities in hip fracture patients

Co-morbidity	Percentage (%)
Cardiovascular disease	24
Stroke	13
Respiratory disease	14
Renal disease	3
Diabetes	9
Rheumatoid disease	3
Parkinson's disease	4
Malignancy	8
Paget's disease	1
Current smokers	10
Enteral steroids	1
No. of co-morbidities	
1	35
2	17
3 or more	7

Adapted from Roche et al[2]

Table 1

with surgery and anaesthesia. Particular challenges are associated with perioperative management of the following.

Atrial fibrillation

Many surgical patients with atrial fibrillation experience a fast ventricular rate postoperatively. Patients should be assessed on a case-by-case basis, excluding potential causes such as sepsis, venous thromboembolism and acute cardiac events. Effective management of hydration and analgesia reduces the risk.

Obstructive lung disease

Many patients with underlying lung disease are at increased risk of exacerbations during the perioperative phase. Regular prescription of inhaled medication, close attention to inhaler technique and possible perioperative nebulizers for those unable to use inhalers help to reduce exacerbations. Early mobilization and chest physiotherapy are used where indicated.

Congestive cardiac failure

These patients are among the most challenging to manage. Strict fluid balance is paramount given the blood pressure changes associated with surgery and anaesthesia. Preoperative assessment of fluid status, judicious use of intravenous hydration and up-to-date echocardiography are vital. Many medications, such as antihypertensives and diuretics, should be stopped in the early phase but be introduced at the earliest opportunity when safe to do so.

Delirium prevention

Development of perioperative delirium has been linked to increased morbidity, length of stay and mortality, and poorer functional outcome. Once established, delirium is extremely challenging to treat, so prevention is best. The multidisciplinary management has been outlined in the HELLP study and UK NICE guidelines.

Dementia

Up to 40% of patients with fractured neck of femur have cognitive impairment, with more than 25% having a pre-existing diagnosis of dementia. This is the group with the highest perioperative morbidity and mortality, and specialist support is required to ensure appropriate management. This includes delirium prevention.

Operation within less than 36 hours

For many years, it has been accepted that delays in surgery for hip fracture lead to increased morbidity, length of stay and mortality, with poorer functional outcome. Although specifically related to this injury, the underlying principles are relevant to other surgical conditions. Physiological stress resulting from untreated surgical conditions can lead to decompensation in chronic medical conditions, counteracting attempts at optimization. It is therefore vital that pre-optimization bears close attention to this and does not lead to unnecessary delay.

Perioperative care

Perioperative care of complex medical patients undergoing surgical procedures requires close collaboration with the anaesthetic and surgical teams to reduce the risk of perioperative complications. The occurrence of these is directly related to the underlying co-morbidities, physiological status, frailty and surgical procedure undertaken. Careful preoperative assessment can highlight potential risks, allowing potential tailoring of intraoperative care. Particular issues that can arise are as follows.

Acute kidney injury

Many patients undergoing surgical procedures develop postoperative acute kidney injury. This is multifactorial but significant factors include;

- **intraoperative hypotension** – prolonged renal hypoperfusion can lead to acute tubular necrosis and associated oliguria/anuria. Close perioperative management of hypotension with strict fluid balance and aggressive fluid resuscitation can reduce the duration and severity. Hypotension can be absolute or relative, and controversy remains over what is classified as intraoperative hypotension
- **nephrotoxic medication** – some medications required intraoperatively, including antibiotics, analgesics and some anaesthetic drugs, have nephrotoxic effects. Close attention should also be paid to regular medication, with suspension of antihypertensives and potential nephrotoxic agents in the perioperative phase. Reintroduction should be monitored and gradual.

Postoperative delirium

This is a common complication of surgery and needs a multidisciplinary approach to management. It has distinct presentations:

- **hyperactive** – the classically recognized presentation, with acute confusion, agitation and distractibility. This is associated with visual hallucinations and is generally easily recognized
- **hypoactive** – associated with being withdrawn, 'flat' and confused, with reduced engagement with eating, drinking and rehabilitation. This is underrecognized as patients are

quiet. It presents many clinical challenges, with dehydration, fatigue and weight loss because of poor oral intake. Lack of engagement with rehabilitation can lead to poor outcomes with increased risk of pressure damage, infections and functional decline.

As with the management of all delirium, it is important to exclude underlying causes such as infection, medication (particularly opiate analgesics) and metabolic disturbance and treat them aggressively. Delirium can be prolonged and lead to chronic cognitive decline. A multidisciplinary approach to management should be instituted as per NICE guidance.

Metabolic abnormalities

Surgery places significant physiological stress on the body, and the response to this can be unpredictable, particularly in elderly or medically complex individuals. The most frequently seen metabolic problem in routine clinical practice is postoperative hyponatraemia, although the mechanism is unclear. Depending on the procedure, there can be significant fluid shifts in addition to hypotension and effects of medication. This phenomenon can be transient but should be addressed aggressively by:

- suspension of potential culprit medications, including:
  - diuretics
  - angiotensin-converting enzyme inhibitors
  - proton pump inhibitors
  - psychotropics
- careful assessment of fluid status and appropriate management
- consideration of testing for:
  - paired serum and urine osmolalities
  - paired serum and urine sodium
  - cortisol.

Postoperative complications

The aim of coordinated postoperative care is to enable rapid recovery to achieve optimal function, which is clearly affected by the occurrence of perioperative complications and early aggressive rehabilitation. In frail older people, late postoperative complications are often seen in conjunction with delirium, slow progress and poor engagement with rehabilitation.

A systemic stress response occurs in conjunction with trauma and surgery, resulting in endocrine, metabolic and immunological changes that lead to a catabolic state. With elective procedures, preoperative optimization, good perioperative medical care and following enhanced recovery principles reduces the impact of these; however, there is limited opportunity for this in trauma, although some aspects can be implemented. These changes can lead to fatigue and accelerated muscle wasting, and in the older population, this can be devastating given their limited reserve.

Early rehabilitation

Prolonged inactivity with bed rest and immobility worsens the catabolic effects on muscle. This creates more pronounced atrophy, leading to a vicious 'frailty cycle'. This greatly increases the risk of later complications:

- pressure sores
- poor respiratory function and respiratory infections
- urinary infections
- poor functional recovery.

A coordinated co-management with an MDT approach is required to reduce the risk of this occurring:

- **Early aggressive mobilization** – patients should be mobilized at the earliest opportunity postoperatively, which for many is on the day after surgery. This necessitates appropriate analgesia and pain management.
- **Early promotion of functional independence** – the aim of surgical intervention should be to restore the patient, as far as possible, to their premorbid state. The ward team should therefore aim to re-establish functional independence at the earliest opportunity. Without this clear approach, patients can become rapidly institutionalized and unable to regain independence.
- **Strict monitoring of food and fluid intake** – close attention should be paid to nutritional intake as this is often poor, particularly with coexisting delirium. Supplementation should be considered early in these cases, and enteral feeding if appropriate.
- **Clear goal-setting in rehabilitation** – as already stated, the aim is to restore functional independence. Clear setting of goals with the MDT can facilitate this more effectively. ◆

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