Management of the circulation on the intensive care unit

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Abstract

The management of the circulation in critically ill patients presents significant challenges. Shock is a potentially reversible life threatening physiological state characterized by end organ dysfunction due to an imbalance in oxygen delivery (DO_2) and tissue demand (VO_2). Independent of its aetiology, untreated shock precipitates a cascade of pro-inflammatory mediators resulting in cellular damage and end organ dysfunction. Thus it is the duty of all clinicians to promptly recognize, diagnose and initiate treatments to halt this process. Despite optimum management shock can progress to multi-organ failure necessitating critical care admission and advanced haemodynamic management. This article will classify shock syndromes, discuss the principles of diagnosis, use of haemodynamic monitoring and management strategies for circulatory failure in the critically ill patient.

Keywords Acute circulatory failure; cardiac output monitoring; haemodynamic assessment; haemodynamic monitoring; shock; shock management; shock pathophysiology

Definition and classification of shock

Shock is a syndrome characterized by clinical signs and biochemical markers of end organ hypoperfusion. It is defined as a life-threatening, generalized form of acute circulatory failure associated with inadequate oxygen utilization by the tissues resulting in cellular dysoxia.[1] Reductions in blood pressure (e.g. systolic BP <90 mmHg, mean arterial pressure <60 mmHg or a decrease of >40 mmHg from baseline) are often a late sign in shock and defining shock in terms of absolute blood pressure values is less meaningful and no longer form part of the diagnostic criteria. See Boxes 1 and 2 for the clinical and biochemical markers of tissue hypoxia.

There are three major subtypes of shock as detailed in *Haemodynamics and cardiovascular shock* (pages 467–473 of this issue); hypovolaemic, cardiogenic, and vasodilatory. They can be divided into those with a low cardiac output state characterized by peripheral vasoconstriction (hypovolaemic and cardiogenic) or hyperdynamic cardiac output state with peripheral vasodilatation (vasodilatory). Although this is a useful theoretical approach it is an oversimplification of the pathophysiology as that they frequently co-exist (e.g. septic shock in a patient with chronic heart failure).

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Assessment and investigation of the shocked patient

Initial assessment of the shocked patient must include a systematic ABCDE approach even when the cause of shock appears obvious (Table 1).

Investigations

The request for an investigation must account for clinical features and assist in achieving a diagnosis. Listed below are some of the investigations considered when assessing a shocked patient.

Blood tests

- Full blood count, coagulation screen, electrolytes, urea, creatinine (Note creatinine is a poor biomarker for detecting acute kidney injury and a rise of up to 50% above baseline indicates acute kidney injury and is associated with a increased mortality), liver function tests, amylase, inflammatory markers (C-reactive protein or procalcitonin). Cardiac troponin levels are often raised in critically ill patients thus cardiac ischaemia should be diagnosed integrating clinical features and 12-lead electrocardiogram findings.

Septic screen

- Fluid culture (blood, sputum, urine, ascites, pleural, cerebrospinal or surgical drain fluid), indwelling catheters/devices, prosthetic implants and surgical wounds must be reviewed for the signs of infection. Seemingly innocuous areas of infection can be the source of sepsis in immunosuppressed patients.
- Fungal and viral infections should be considered in immunosuppressed patients prompting specialist microbiological consultation regarding appropriate sampling and treatment.
- HIV status should be verified in patients who are high risk, those who fail to respond to standard antimicrobial therapy and in specific indicator conditions (e.g. Guillain–Barre syndrome, community acquired pneumonia) as outlined by the European Society of Clinical Microbiology and Infectious diseases.[2]

Clinical and biochemical markers of cellular hypoxia[1]

- Tachycardia
- Reduced systolic blood pressure or mean arterial pressure, narrow pulse pressure
- Delineating levels of cutaneous vasoconstriction in limbs
- Altered mentation — coma, disorientation or confusion
- Capillary refill time >2 s
- Urine output (<0.5 ml/kg/h)
- Lactate >2 mmol/L
- Metabolic acidosis
- Central venous oxygen saturation ($ScVO_2$) <70%
- Veno-arterial carbon dioxide gap (pCO_2) >6 mmHg

Box 1

Biochemical markers of cellular hypoxia

Metabolic acidosis and lactate. During the early stage of shock as DO_2 reduces due to falling haemoglobin, hypoxia or cardiac output the tissues compensate by increasing oxygen extraction resulting in a reduction in venous oxygen saturations. As DO_2 becomes critically low and insufficient to meet tissue demands, anaerobic respiration increases resulting in an elevation in plasma lactate levels (>2 mmol/L) until oxygen delivery is restored. Elevated plasma lactate levels pre- and post-resuscitation are associated with increased mortality. Numerous studies have utilized lactate to guide and monitor response to resuscitation with positive impact on organ failure. In light of this a recent consensus statement recommends monitoring plasma lactate levels every 2 hours during the first 8 hours and 8—12 hours thereafter.[1]
Although plasma lactate is a better prognostic marker than blood pressure there are limitations to its use as elevated levels are found in situations where there is neither elevated production or reduced elimination (e.g. type D lactic acidosis and metformin toxicity).

Venous oxygen saturation. Mixed venous oxygen saturation (SvO_2) is the oxygen saturation of haemoglobin in the pulmonary artery. SvO_2 is a marker of oxygen flux and when low indicates a disruption in tissue oxygen supply demand ratio. Low SvO_2 levels (less than 70%) are associated with increased mortality in ICU patients with septic shock and are a marker of increased severity of injury in trauma. With a reduction in the use of the pulmonary artery flotation catheter, central venous oxygen saturation ($ScvO_2$) from a catheter in the superior vena cava is an alternative. Studies have confirmed the correlation of SvO_2 and $ScvO_2$ and suggest $ScvO_2$ is a reliable surrogate for SvO_2. $ScvO_2$ differs due to incomplete mixing of venous blood from the upper and lower body and is typically 5—10% lower than SvO_2 in steady state. A $ScvO_2$ of less than 70% suggests oxygen delivery is insufficient to meet metabolic demands of the tissues and its therapeutic utility gained widespread popularity following its adoption by the Surviving Sepsis Campaign guidelines.[3—5] A normal or elevated $ScvO_2$ in the context of hyperlactatemia does not assure adequate tissue oxygenation as local hypoperfusion or inability to extract oxygen can occur limiting its utility.

Veno-arterial carbon dioxide gap (pCO_2) is a relatively new monitoring modality that measures the difference between mixed or central venous and arterial CO_2 to identify patients who are under resuscitated. In the context of a $ScvO_2$ >70% a veno-arterial CO_2 gap of >6 mmHg has been suggested to represent tissue hypoperfusion.[1]

Box 2

Imaging

- Point-of-care ultrasound for assessing critically ill patients has increased in recent years. Ultrasonography may be used to diagnose cardiac tamponade, pneumothoraces, free intra-abdominal fluid and basic cardiac parameters. Focused assessment sonography in trauma (FAST) is a useful adjunct in the primary survey of polytrauma patients and has superseded diagnostic peritoneal lavage as a method of identifying patients with intraperitoneal fluid/blood.

  In the 2014 European Society of Intensive Care Medicine consensus guidelines on shock, echocardiography is the recommended primary method of evaluating cardiac output owing to its minimally invasive nature and ability to differentiate shock subtypes.[1]
- Computed tomography (CT) is a vital diagnostic modality commonly used in the assessment of the shocked patient. Modern multi-slice detectors provide detailed images and greatly reduce the requirement for diagnostic laparotomy, which is now reserved for the unstable shocked polytrauma patient.
  CT pulmonary angiogram can reliably exclude central pulmonary emboli.
- Radiology may also be employed as a therapeutic aid (e.g. drainage of intra-abdominal collections) providing source control in sepsis and control of haemorrhage that would otherwise require surgical intervention (e.g. mesenteric angiography and embolization). These techniques are particularly useful in high-risk patients.

Principles of shock management

With a good understanding of cardiovascular physiology the management of shock can be reduced to three general principles.

1. Increasing the oxygen content of the existing circulation

Oxygen is a widely available, well-tolerated drug that should be administered to all shocked patients. With a flow of 15 L/min via a non re-breath face mask up to 85% oxygen can be delivered to the patient. As the oxygen content of the circulation is of equal importance as cardiovascular optimization this simple step should not be overlooked. Often concerns exist regarding patients with chronic obstructive airways disease where removal of the hypoxic respiratory drive may precipitate type 2 respiratory failure. These concerns should be noted but in the context of shock, global tissue hypoxia will have a more devastating consequence than hypercarbia. A pragmatic approach is titrating oxygen to achieve saturations of 92—95%.

2. Restoration of the circulating blood volume

Absolute or relative hypovolaemia is common amongst critically ill patients independent of the aetiology of shock and when unrecognized is associated with poor outcome. The simplest method of improving cardiac output is by increasing cardiac preload utilizing the Frank—Starling principle to improve stroke volume. A fluid challenge should be considered in all shocked patients to assess fluid responsiveness, the notable exceptions being uncontrolled traumatic haemorrhage or ruptured aortic aneurysm where aggressive resuscitation may disrupt the primary clot.[1]

Fluid responsiveness is the ability to increase the cardiac output and its surrogate markers such as blood pressure and peripheral perfusion following fluid administration. For most patients a suitable fluid challenge is the rapid infusion of 250 ml of fluid followed by re-assessment of clinical indices after 15—30 minutes. In circumstances where there are concerns that repeated fluid loading may precipitate cardiac decompensation 100 ml aliquots can be given or a passive leg raise test (Box 3) can be performed.