The emergency management of the patient presenting with shock

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Introduction

Shock may not be the commonest scenario encountered within the average clinical take, but unless rapidly recognised and managed appropriately, it carries an extremely high mortality.

Shock is defined as inadequate tissue perfusion and is a consequence of a number of different clinical conditions. Hypotension is often, but not always, present. Blood pressure (BP) is a product of cardiac output (CO) and peripheral resistance (PR) as defined in the following equation:

$$BP = CO \times PR$$

An inadequate cardiac output with intense vasoconstriction can therefore result in tissue hypoxia in the absence of hypotension. The recognition of shock, its causes and initial management will be discussed in this article.

Recognition of shock

The most important step in the recognition of shock is to consider it; the obtunded patient who is hypotensive and tachycardic is recognisable to all, but this is often indicative of a very late and potentially irreversible stage of shock. The early identification of patients with more subtle signs may allow interventions to prevent further deterioration (see Box 1).

What are the biochemical markers of shock?

Clinical features indicative of shock can be reinforced by biochemical evidence of inadequate tissue perfusion. We are not particularly concerned here with specific indicators of organ-specific hypoperfusion but with global markers of anaerobic metabolism. This is important as significantly abnormal markers of tissue perfusion may develop in the presence of subtle clinical signs of shock prompting timely rescue management.

System	Features
Cardiovascular	Cool peripheries, delayed capillary refill Tachycardia or bradycardia Narrow pulse pressure Hypotension, mottled skin
Respiratory	Tachypnoea, laboured breathing
Abdo/GU	Urine output <0.5ml/kg/hr
Neurological	Confusion, agitation, drowsiness

Box 1. Clinical indicators of shock.

Markers of anaerobic metabolism

There are two easily available markers of global anaerobic metabolism:

1. Raised lactate
2. Metabolic Acidosis

In the shocked state, hydrogen ions and lactate are generated in large quantities due to the inability of pyruvate (the product of glycolysis) to enter the Krebs cycle, the normal process of aerobic respiration. Pyruvate is converted, by lactate dehydrogenase, to lactic acid, which in aqueous solution dissociates to lactate and hydrogen ions.

Lactate is metabolised (back to pyruvate) in the liver (70%), by mitochondrial rich tissue such as skeletal and cardiac myocytes (25%) and by the kidneys (5%). The generation of lactate in excess of the tissues ability to metabolize it leads to a rise in serum lactate concentration.[1] The accumulation of lactate in the setting of shock is referred to as 'type A' lactic acidosis.

Acidotic states are often well compensated for in the early stages of illness, by both increased respiratory effort and plasma buffers, such that the pH initially remains within the normal range. The 'standard' base deficit (or excess) and bicarbonate, as measured on a blood gas sample, are derived parameters excluding respiratory compensation, and thus reveal the underlying metabolic state.

What are the different types of shock?

In order for the correct management to occur it is vital that the clinician has a basic knowledge of the different types of shock.

Cardiogenic Shock

You are asked to review a 57 year old woman who has suffered an acute anterior myocardial infarction with a markedly elevated troponin level. She is hypotensive,

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tachycardic and desaturating despite receiving high flow oxygen via a re-breath system.

The patient described above almost certainly has *cardiogenic shock*, defined as 'persistent hypotension and tissue hypoperfusion due to cardiac dysfunction in the presence of adequate intravascular volume and left ventricular filling pressure'.[2]

Causes of cardiogenic shock are summarised in Box 2.

- Acute myocardial infarction – pump failure or mechanical complications – papillary muscle rupture, ventricular septal defect, free wall rupture
- Acute on chronic valve disease
- Outflow tract obstruction - aortic stenosis, hypertrophic cardiomyopathy
- Myocarditis
- Endocarditis
- Pericardial tamponade
- Life threatening arrhythmias

Box 2. Examples of causes of cardiogenic shock.

Cardiogenic shock and established septic shock may be difficult to distinguish (or even co-exist) so context is critical. The presence of pulmonary oedema may help in differentiation.

How should cardiogenic shock be managed?

Initial management includes oxygen, reduction of anxiety, and immediate referral to Cardiology and Critical Care. Definitive care may include coronary intervention, cautious vasodilatation and/or inotropic support.

Inotropic support

Dobutamine is one of the few inotropes commonly used outside of the critical care complex and is useful in the management of cardiogenic shock. It should only be used with senior medical input in a closely monitored area with trained nursing staff experienced in its use. Its mechanism of action is via stimulation of both β_1 and β_2 receptors of the sympathetic nervous system. Clinically it has relatively weak inotropic effects and is a vasodilator – an ideal combination in a patient with low cardiac output and high vascular resistance. Dobutamine can be administered peripherally but should only be given in a controlled environment such as a Coronary Care Unit or appropriately trained and skilled Acute Care area of an AMU. Frequent review of pulse rate, blood pressure, urine output and clinical response is required. Like all inotropic drugs, there is a real potential to provoke myocardial ischaemia which is particularly relevant in the setting of cardiogenic shock. While reduced afterload may balance the inotropic effect, a major problem with dobutamine is a significant chronotropic effect, with a marked increase in heart rate potentially limiting use. The higher the dose, the greater the incidence of side effects of the drug, including hypotension or hypertension, tachycardia and malignant arrhythmias.

Hypovolaemic Shock

A patient in the resus department is drowsy, hypotensive and tachycardic; shortly after your arrival he passes a large volume of offensive black tarry stool, accompanied by 250 ml of dark red vomit.

The history suggests hypovolaemic shock secondary to large volume blood loss from an upper gastrointestinal bleed. The initial management consists of resuscitation with crystalloid and/or colloid quickly followed by blood products to correct any anaemia or coagulopathy.

Hypovolaemic shock may be caused by obvious or covert haemorrhage, diarrhoea and vomiting or increased insensible losses. The management consists of targeted fluid resuscitation that is guided by frequent clinical assessment of the patient and restoration of normal haemodynamic indices.

Distributive Shock

You are asked to review an 18 year old college student who is drowsy, hypotensive, tachycardic and tacyhypnoeic with a rapidly spreading maculapapular rash. He is pyrexial at 38.6°C and feels warm peripherally but his capillary refill is markedly delayed.

Distributive shock, at least initially, is a consequence of insufficient intravascular volume due to both decreased vascular tone and a variable amount of capillary leak. There are several causes of distributive shock (see Box 3) however the initial management is often the same, consisting of fluid resuscitation and early involvement of the critical care team to provide targeted organ support.

- Systemic inflammatory response syndrome
- Septic Shock
- Neurogenic (spinal trauma above T4)
- Anaphylaxis

Box 3. Examples of causes of distributive shock.

Systemic inflammatory response syndrome (SIRS)

SIRS is an inflammatory state affecting the body that has many causes e.g. massive blood transfusion, pancreatitis, burns and severe infection. It can be defined by ≥2 of the following criteria;

- Temperature <36°C or >38°C
- Heart rate >90 beats per minute
- Respiratory rate >24 pm or $PaCO_2$ <4.3kPa (in a non ventilated patient)
- White cell count <4 x 10^9 or >12 x 10^9 or >10% immature forms

Management consists of treatment of the underlying cause where possible and fluid resuscitation as guided by clinical and laboratory indices. SIRS due to septic shock will be discussed in more detail below. The management of anaphylaxis will be covered in a separate article.

Septic shock

Septic shock is defined as SIRS due to severe infection complicated by hypotension (systolic BP <90mmHg, MAP <60mmHg or fall in systolic BP ≥40mmHg from baseline) which is refractory to adequate fluid resuscitation.

The patient described in the vignette above is demonstrating many clinical signs of shock as evidenced by drowsiness, hypotension, tachycardia and tachypnoea. The rapidly spreading rash and pyrexia indicates that septic shock is a strong possibility. Many, but not all, patients with septic shock will be vasodilated and feel warm peripherally at presentation. They may well however have delayed capillary refill. Initial management includes fluid resuscitation and early implementation of the surviving sepsis guidelines.[3]

Septic shock is a pathological, overwhelming inflammatory response to severe infection. Bacteria and fungi are most usually implicated but viruses or parasites can also evoke the same reaction. An individual's response to such insults is probably genetically mediated. Some show little haemodynamic or systemic instability whereas others rapidly deteriorate and develop multi-organ failure following infection with the same organism.

Management of septic shock as an Acute Medicine trainee requires adequate fluid resuscitation (at least 40ml/kg of crystalloid/colloid), early broad spectrum antibiotics following culture of relevant sites, source control where possible and early referral to intensive care. The profound vasodilation associated with this condition often requires the use of drugs with vasoconstrictive properties (vasopressors) such as noradrenaline. This sort of care can usually only be delivered safely in a fully monitored Critical Care environment.

Obstructive Shock

Obstructive shock is defined as inadequate cardiac output due to pathology outside of the heart. Tension pneumothorax and acute massive pulmonary embolism are two examples.

General Management of shock

The descriptions of shock above illustrate how fundamentally different clinical pathologies may all result in tissue hypoxia and that there is no generic approach to the management of shock. Early clinical suspicion and recognition of shock must be followed by thoughtful appropriate targeted management.

When reviewing a shocked patient, consideration of the following formulae may help if the aetiology is unclear:

BP = Cardiac Output x Peripheral Resistance

Cardiac Output = Stroke Volume x Heart Rate

Box 4 summarises how these formulae may be applied to the clinical syndromes of shock, allowing both identification of the type of shock and subsequent targeted management.

Type of shock	Problem	Initial management
Hypovolaemic	Decreased stroke volume	Fluid bolus
Cardiogenic	Decreased cardiac output	Early echocardiogram Inotropes Urgent cardiology review
Obstructive	Inadequate cardiac output due to increased peripheral resistance	Rapid diagnosis paramount Tension pneumothorax – needle decompression Pulmonary embolism - thrombolysis
Distributive	Decreased stroke volume Decreased peripheral resistance	Fluid bolus Consider critical care input for vasopressors

Box 4. Mechanisms and initial management of different types of shock

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