Management of shock in trauma

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Abstract

Shock is defined as the failure of the circulatory system to provide the organ perfusion and tissue oxygenation required to meet cellular metabolic demands. Traumatic shock is most commonly associated with haemorrhage, although non-haemorrhagic shock can be found in trauma in the form of cardiogenic or neurogenic shock. Over the last decade evidence has demonstrated that trauma patients have an acute traumatic coagulopathy (ATC) caused by the injury process itself. This has been fundamental to the development of the current approach to management of traumatic shock, known as damage control resuscitation (DCR). DCR encompasses three key resuscitative strategies, permissive hypotension, haemostatic resuscitation (the use of blood products as primary resuscitative fluids) and damage control surgery. The implementation of DCR alongside the creation of trauma networks has been revolutionary in the management of the shocked trauma patient. Current focus is on evolving and refining these strategies including identifying the subsets of patients at greatest risk as early as practicable following injury.

Keywords Coagulopathy; haemorrhage; hypovolaemia; lethal triad; massive transfusion; shock; trauma; trauma team

Royal College of Anaesthetists CPD Matrix: 1A01; 1B04; 1H02; 2A02; 2A05; 3A10; 3A14

Learning objectives

After reading this article, you should be able to:

- Describe the physiological response to haemorrhage
- Describe the lethal triad of trauma
- Describe the clinical management of traumatic shock
- Explain the role and benefits of the trauma team

Shock

Shock is defined as a failure of the circulatory system to provide adequate organ perfusion to meet the oxygen demand of cellular metabolism. Shock in trauma is most commonly due to haemorrhage accounting for up to 40% of deaths following trauma. It remains the leading preventable cause of trauma-related death. It is vital to remember that not all shock results from blood loss. Cardiogenic shock can result from blunt force trauma, obstructive shock from cardiac tamponade and tension pneumothorax, neurogenic shock may be associated with acute spinal cord injury. This article will focus on haemorrhagic shock in trauma.

Physiological response to haemorrhage

Significant haemorrhage will lead to a transient fall in blood pressure that triggers sympathetic stimulation. This results in an increased cardiac output (through a rise in heart rate and contractility) as well as arterial and venous constriction with the overall aim being to maintain blood flow to the vital organs. This cannot occur indefinitely, severe blood loss reduces oxygen delivery to the tissues resulting in anaerobic cell respiration generating a lactic acidosis.

The lethal triad

Prolonged acidosis (pH $< 7.2/[H^+] > 63$ nM/L) inhibits platelet and clotting factor function as well as impairing ventricular function. The reduced platelet and clotting factor activity secondary to the metabolic acidosis results in a coagulopathy. In turn this leads to further bloodloss worsening the metabolic acidosis. Poor tissue perfusion coupled with removal of clothing, environmental exposure and administration of cold fluids results in hypothermia again worsening enzyme function. This results in a vicious cycle where physiological derangement reduces the ability of the body to maintain homeostasis. The interplay of these three factors is known as the 'lethal triad' consisting of acidosis, coagulopathy and hypothermia that if allowed to continue results in death.

Acute traumatic coagulopathy

In 2003 Brohi et al. demonstrated that coagulopathy was present in up to one-third of trauma patients (Injury Severity Score >15) presenting to the emergency department even before the administration of fluids. This has led to the concept of acute traumatic coagulopathy (ATC). Patients with ATC have a significantly higher mortality and are at greater risk of multiorgan failure. ATC occurs as part of the body's response to injury and is directly related to the degree of tissue damage and duration of shock.

The exact pathophysiology remains unclear as there are a number of conflicting processes at work, some resulting in consumption of factors due to procoagulation and others favouring an anticoagulant process.

Tissue damage exposes tissue factor (TF). This drives local thrombin and fibrin production. Platelets are activated by thrombin and adhere to the damaged tissue in turn amplifying thrombin generation. Recent studies have shown that despite this there are still adequate amounts of clotting factors and preserved thrombin generation in patients with ATC.

Conversely hypoperfusion (through hypoxia, and epinephrine and vasopressin release) activates tissue plasminogen activator (t-PA) and inhibits plasminogen activator inhibitor (PAI), thus promoting hyperfibrinolysis. This may explain the survival benefit from tranexamic acid administration. Additionally, there is

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evidence that activated Protein C (aPC) levels are increased adding to the anticoagulant element of ATC by cleaving factors Va and VIIIa as well as binding PAI and further encouraging fibrinolysis.

Platelet function is reduced early in the period following trauma, often despite normal platelet counts, possibly due to a blunted thrombin activation effect.

Haemodilution of coagulation factors occurs through the administration of crystalloids, colloids or packed red cells, whilst hypothermia has a negative effect on platelet function and the enzymes of the clotting cascade. This all exacerbates the coagulopathy of ATC.

Clinical management of traumatic shock

Damage control resuscitation (DCR)

Over the past decade the multiple interventions and stages of trauma resuscitation have been unified under one concept, damage control resuscitation (DCR). The UK military definition of DCR encompasses all care from point of injury through to post surgical care on the Intensive Care Unit. The overall aim being 'to minimize blood loss, maximize tissue oxygenation and optimize outcome'. This entails haemorrhage control with the early use of tourniquets and haemostatic dressings at the point of wounding, alongside the use of permissive hypotension. The use of advanced pre-hospital teams allows the early commencement of haemostatic resuscitation and rapid sequence induction. These techniques are continued and expanded upon in the Emergency Department with prompt access to diagnostic and interventional imaging. In combination, these strategies aim to reverse the lethal triad and return the patient to homeostasis. In shocked patients whose organ perfusion cannot be maintained with ongoing resuscitation, rapid surgical intervention is required.

Permissive hypotension (PH)

The aim of PH is to minimize blood loss prior to control of bleeding. This can be temporary control with tourniquets, pelvic binder or permanent surgical control. 250 ml fluid boluses are given to achieve the target systolic blood pressure of 80–90 mmHg. A radial pulse can be used as a surrogate until a blood pressure is available. However, whilst absence of a radial pulse signifies significant shock, its presence does not guarantee a blood pressure of 80 mmHg. If there is no head injury fluid can be titrated to maintain consciousness.

The concept is based on the principle that the first clot is the best clot. By allowing a state of 'controlled shock' perfusion to vital organs can be maintained, whilst the risk of clot disruption by increasing blood pressure is reduced. Evidence for improved mortality from PH is still lacking particularly in blunt trauma. Evidence in animal studies using pigs suggests hypotension can be maintained for up to 60 minutes, but past this point the oxygen debt of the tissues may be impossible to overcome even with aggressive DCR.

PH is contraindicated in patients with head injuries or pregnancy both of which require higher blood pressures. There is little guidance in the paediatric population where its use is still debated.

Haemostatic resuscitation (HR)

The aim of haemostatic resuscitation is to restore tissue perfusion by replacement of blood volume, ensure optimal oxygen delivery by the replacement of red blood cells and reverse coagulopathy by the replacement of clotting factors and platelets. It may only be possible to stop or slow the coagulopathy prior to control of bleeding. The use of crystalloid fluid replacement in trauma can have a detrimental effect on coagulation. The early use of blood products is advocated.

Haemostatic resuscitation consists of two phases: fluid administration before and after control of bleeding. Whilst bleeding is ongoing blood products are administered at fixed ratios targeted to maintain a systolic blood pressure of 80–90 mmHg. Once bleeding is controlled a more targeted approach can be undertaken with the use of viscoelastic tests such as thromboelastography (TEG) or rotational thromboelastometry (ROTEM).

Attention must be given to biochemical correction of the patient. The use of large volumes of stored blood products will lead to hypocalcaemia and hyperkalaemia, both of which need to be managed, preferably in a pre-emptive manner.

Massive haemorrhage protocol

The logistical challenges in undertaking massive transfusions are significant. To allow teams to concentrate on other pressing clinical issues and release 'cognitive bandwidth', the use of massive transfusion protocols is advised. Each institution will have their own protocol with subtle variations due to the set up of the blood bank service but a common theme runs through them all. Once the recognition or expectation of a massive transfusion is present, one call to a dedicated phone line can be made to trigger the protocol. Blood boxes containing pre defined blood products are released until the protocol is terminated by the treating clinicians. This allows rapid administration of fixed ratios of Fresh Frozen Plasma (FFP), Packed Red Blood Cells (PRBC) and platelets. While there is significant ongoing haemorrhage, reliance on blood test results to guide resuscitation may not be helpful, even tests with a fast turnaround time are likely to provide 'historical' results. During this phase a fixed ratio transfusion is recommended.

The exact ratio is debated with the PROPPR Trial comparing 1:1:1 or 1:2:1 (FFP:PRC:PLTs).[1] There was no difference in the 24 hour or 30 day mortality with each ratio but haemostasis was achieved in more patients in the 1:1:1 group. Findings in the observational ACIT trial however found increased survival in the arm with a higher ratio of platelets and plasma to blood.[2]

Hypocalcaemia complicates massive transfusion due to citrate in stored blood products binding calcium. In hypocalcaemia fibrin will not polymerize and platelet function is reduced as well as myocardial contractility and systemic vascular resistance. During the phase of rapid administration Calcium (10 ml 10% calcium chloride) should be given with every four units of FFP and four units of PRBC as a minimum.

Part of the coagulopathy in trauma is caused by fibrinolysis. To prevent this 1 gram of intravenous tranexamic acid should be given as early as possible, preferably in the pre hospital phase. The strongest evidence for use in the CRASH 2 trial came in those given it within the hour. There was some benefit if given between 1 and 3 hours of injury, after this period it is associated with an increase in mortality.

Targeted resuscitation

Once bleeding is controlled a more tailored approach is employed with both the volume and the type of blood products

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