Fluid management for trauma; where are we now?

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Resuscitation from haemorrhagic shock remains one of the primary tasks of the traumatologist, whether practising in the emergency department (ED), the operating theatre or the intensive care unit (ICU). Diagnosis of haemorrhage, surgical strategy, choice of fluids to administer, monitoring and optimal endpoints for resuscitation are all controversial, and recommendations in each of these areas have evolved substantially in the past decade. Actions taken in the first minutes of care may profoundly affect the patient’s subsequent clinical course, putting a greater focus than ever on the dynamic process of early resuscitation. This article will review the pathophysiology of haemorrhagic shock and will briefly address emerging and controversial therapies.

**Defining haemorrhagic shock**

In simple terms, shock is failure of adequate oxygen delivery to the tissues of the body. Hypoperfusion leads to cellular ischaemia, leading in turn to ‘hibernation’ (the cell reduces its level of metabolic activity), anaerobic metabolism (absent oxygen, the cell must produce lactic acid as it generates energy), apoptosis (the cell begins a programmed shutdown process) and outright necrosis (cell death). The ischaemic cell takes up interstitial fluid (perhaps to dilute accumulating metabolic poisons) and swells, reducing perfusion to its neighbours. Lactate, and other toxic metabolites, poison cells not affected by the initial ischaemia, and mediators released in response to metabolic stress trigger a response from immune system cells. The net effect is a biological cascade that, if unchecked, can be fatal.

Uncontrolled haemorrhage leads to acutely fatal shock, characterized by complete failure of the cardiovascular system: loss of contractile power in the heart and great vessels, inappropriate vasodilation, loss of response to catecholamines and, eventually, brain death. Even when haemorrhage is corrected and the macrocirculation restored, shock can still prove fatal through the pathophysiology of organ system failure, beginning with the lungs and progressing to the renal, gut, immune and cardiovascular systems. Figure 1 is a crude representation of the shock cascade, demonstrating the amplification that begins with a single ischaemic cell but can extend to affect the entire body.

Clinically, shock is characterized by the symptoms of the body’s response to hypoperfusion. Low blood pressure, tachycardia, decreased urine output, pale skin and diaphoresis are all characteristic. However, it is important to remember that hypotension is not synonymous with shock. Low blood pressure may occur in the absence of hypoperfusion, as in the vasodilated but euvolemic patient undergoing general anaesthesia. Normal blood pressure may also mask hypoperfusion, as in the intensely vasoconstricted young patient with normal vital signs despite loss of blood volume approaching 40%.

**Anaesthetic and surgical strategy**

As with septic shock, the most important component of resuscitation from haemorrhagic shock is source control. The Advanced Trauma Life Support curriculum of the American College of Surgeons emphasizes the ABC of trauma care: Airway, Breathing, and Circulation. Control of the airway facilitates patient management and ensures adequate oxygen uptake by the blood stream. Rapid assessment of the chest will also identify tension pneumothorax or pericardial tamponade as a mechanical source of hypoperfusion.
Once these preliminary steps are taken, the focus of care shifts to a search for haemorrhage as the cause of circulatory shock. Bleeding must be diagnosed and treated as swiftly as possible, and this priority should inform all subsequent decision-making. Clinically significant haemorrhage occurs into one of only five compartments: the thoracic cavity, the peritoneum, the retroperitoneal space, the tissue compartments of the thigh and outside the body. These are investigated by direct inspection of the patient, chest X-ray, abdominal sonography, pelvic X-ray and computer tomography. External bleeding is managed by direct pressure and ligation of exposed cutaneous vessels. Haemorrhage in the chest is managed initially by tube thoracostomy, as bleeding from the low-pressure pulmonary circuit will usually resolve spontaneously unless a major vessel has been injured. Bleeding in the abdomen or persistent bleeding in the chest is addressed by surgical exploration. Pelvic or retroperitoneal haemorrhage is difficult to access surgically; where logistically possible, angiographic embolization is the preferred approach. Bleeding into the thigh may be substantial at the time of injury (up to 1500 ml) but will almost always resolve spontaneously through tamponade in the muscle compartment and vasoconstriction of the feeding vessels. Traction, splinting or external fixation of fractures will facilitate spontaneous haemostasis in the associated soft tissue beds.

For the patient who requires exploratory surgery, the emphasis is on ‘damage control.’ While the patient is hypoperfused and resuscitation is under way, the goal is to perform the fastest surgery possible to achieve control of haemorrhage. Large vessels are ligated or shunted whenever possible, and not reconstructed or bypassed. Bleeding from the spleen, kidney, lung-lobe or bowel segments is managed by excision, without anatomical reconstruction. Hepatic or retroperitoneal haemorrhage is controlled with cautery, topical haemostatic agents and packing. When haemostasis is achieved, the chest or abdomen is drained, packed open and covered with a temporary sterile dressing. This allows monitoring of recurrent or ongoing haemorrhage, permits tissue oedema without fear of compartment syndrome and offers easy access for subsequent therapeutic or reconstructive surgery. The patient is moved through theatre and angiography suite or both as swiftly as possible, subsequently returning to the trauma bay or ICU for completion of resuscitation.

Surgical strategy for managing haemorrhagic shock must be focused on control of haemorrhage. The same is also true of the

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**Fig. 1** The shock cascade. Ischaemia in one organ system triggers a systemic response that persists even after adequate resuscitation. This is the pathophysiology of the multiple organ system failure that commonly follows severe haemorrhagic shock.
anaesthesia plan. While not as obvious as the ligation of a bleeding vessel, the actions of the anaesthetist may play just as great a role in the patient’s survival. Fluid administration is one aspect of this, titrated to maintain or improve perfusion while at the same time encouraging native haemostasis. Maintenance of body temperature, blood composition, electrolyte balance, respiratory function and depth of muscle relaxation and anaesthesia are also critical tasks. Finally, the appearance in clinical practice of systemic haemostatic agents (i.e. recombinant human coagulation factor VIIa) offers a new option. Most important of all, perhaps, is the anaesthetist’s ability to facilitate the patient’s movement between the ED, operating theatre, angiography and ICU such that no moments are wasted in the organized care of the actively haemorrhaging patient.

Fluids in resuscitation

Quantity of fluid

One of the most remarkable changes in resuscitation practice has been the growing realization in the past decade that too much of a good thing can itself become a bad thing. Although isotonic fluid administration is clearly necessary for a complete recovery from shock, due both to haemorrhagic losses from the circulation and the tendency of ischaemic cells to take up interstitial fluid, it is clear that the indiscriminate administration of crystalloid fluid has the potential to make the patient’s condition worse, especially early in resuscitation. I.V. fluid administration increases ventricular preload, resulting in an immediate increase in blood pressure. This may reverse vasoconstriction that was contributing to haemostasis and may directly displace early fibrin clots. Furthermore, isotonic crystalloid solutions dilute the oxygen-carrying capacity of the blood and the concentration of clotting factors and platelets. Unless rigorous attention is paid to warming infused fluids (unlikely in early resuscitation), significant hypothermia may also develop, contributing to both metabolic acidosis and coagulopathy. The clinical result is a downward spiral of hypotension, fluid bolus, re-bleeding and recurrent hypotension.

Bench research in the 1980s and 1990s demonstrated decreased survival with aggressive fluid administration in a variety of uncontrolled haemorrhage models in swine, rats, sheep and dogs.\(^5\)\(^6\) Optimal survival was attained with fluid therapy titrated to lower than normal blood pressure and cardiac output, for the duration of active haemorrhage. This concept of ‘deliberate hypotensive resuscitation’ has been tested in two noteworthy clinical trials (Table 1). Bickell and colleagues\(^4\) randomized victims of penetrating trauma to ‘fluid’ or ‘no fluid’ in the pre-hospital and ED phases of care, eventually documenting a significant improvement in outcome with the experimental therapy. Dutton and colleagues\(^5\) studied both blunt- and penetrating-injured patients in haemorrhagic shock using a fluid resuscitation protocol titrated to maintain a systolic blood pressure of 70–80 mm Hg until definitive control of bleeding. This smaller study showed no difference in mortality, despite a higher average injury severity in the low pressure group, suggesting that this approach was at least worthy of consideration. While neither study was scientifically definitive, largely owing to the tremendous logistical difficulty in studying an intrinsically heterogeneous problem, clinical practice has now evolved to a much more careful administration of fluids in early resuscitation (especially isotonic crystalloid solutions). Fluids are now administered in prescribed small boluses and titrated to a specific physiological endpoint such as blood pressure or, even better, base deficit and lactate.

Type of fluid

The composition of fluid administered to the actively haemorrhaging patient is as important as the rate and quantity. While isotonic crystalloid solutions are important for making up ‘third space’ losses, and are inexpensive and readily available, they do not adequately replace the whole blood that the patient is losing. One concern is that the intravascular persistence of these solutions is low, with estimates of as little as 12% of an administered bolus of 0.9% saline remaining in the circulation 30 min later. Colloidal solutions, such as hypertonic saline-dextran, have been recommended for early resuscitation; however, so far, there has been no definitive evidence of benefit. The recently completed safe KS albumin fluid evaluation (SAFE) trial in Australia showed no difference in outcomes among ICU patients receiving crystalloid vs colloid (albumin) as their primary resuscitative fluid.\(^6\)

Early transfusion therapy with red cells, plasma and platelets is thus essential to successful resuscitation. While in the long term blood transfusion has been associated with an increased incidence of organ system failure and death in closely matched groups of patients, as well as profound immune suppression, in the short term there is no available substitute. Early replacement of oxygen-carrying capacity, in the form of red blood cells, may be life-saving. Many trauma centres maintain a supply of un-cross-matched, type-O red blood cells available in the ED for immediate use in patients presenting with severe haemorrhagic shock. The safety of this therapy has been well established, and the advantage of immediate transfusion vs waiting 30 min for group-specific or 60 min for cross-matched blood may be substantial.

Table 1 Clinical trials of deliberate hypotensive resuscitation

<table>
<thead>
<tr>
<th>Trial</th>
<th>Bickell et al.(^4)</th>
<th>Dutton et al.(^5)</th>
</tr>
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<tbody>
<tr>
<td>Mechanism of injury</td>
<td>Penetrating</td>
<td>Blunt and penetrating</td>
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<tr>
<td>Site of resuscitation</td>
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<td>Trauma resuscitation unit, operating room</td>
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<td>Study mechanism</td>
<td>No fluid given</td>
<td>Blood pressure titration</td>
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<td>N</td>
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<tr>
<td>Study Group Mortality</td>
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</tr>
<tr>
<td>Control Group Mortality</td>
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<td>7%</td>
</tr>
<tr>
<td>P-value</td>
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<td>Not significant</td>
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Early transfusion of plasma and platelets is similarly advantageous, as coagulopathy complicating haemorrhagic shock is easier to prevent than to reverse. For the patient who requires a transfusion of >1 blood volume (approximately 10 units of red blood cells) in a short period, dilutional coagulopathy is almost certain. Our own preference is to begin therapy with plasma and platelet concentrates as soon as this need is recognized, with maintenance over the course of early resuscitation of an approximately 1 : 1 : 1 ratio of blood components. Once haemorrhage has been anatomically controlled, further transfusion therapy can be managed in a much more conservative fashion and titrated to specific levels of haemoglobin, prothrombin time and platelet concentration. Haemoglobin as low as 60 g litre\(^{-1}\) is now routinely tolerated in an asymptomatic patient, while plasma and platelets are rarely given to the stable patient without evidence of haemorrhage.

Finally, the anaesthetist must monitor the other components of blood. Electrolyte concentration should be followed closely during early resuscitation, with particular attention to the blood calcium concentration. Rapid transfusion can lead to intravascular chelation of free calcium (with a negative effect on myocardial performance) because banked blood components are packaged with citrate to prevent clotting. The anaesthetist must monitor ionized calcium closely and replace it as needed during rapid transfusion. Respiratory acidosis should be managed by adjustment of mechanical ventilation, while metabolic acidosis can only really be treated by control of haemorrhage and restoration of adequate intravascular volume. The use of bicarbonate to elevate serum pH has been advocated in the past, but has not been found beneficial in the treatment of haemorrhagic shock. On the other hand, there is mounting evidence that close management of serum glucose concentrations with i.v. insulin improves patient outcomes. Although a definitive study in early resuscitation has not yet been completed, tight glucose control is an emerging standard of care in most centres.

**Completion of resuscitation**

After definitive control of haemorrhage, the emphasis of resuscitation shifts to the restoration of normal tissue perfusion. The phenomenon of ‘occult hypoperfusion’ has been used to describe patients (specially young patients) who reach the ICU with normal vital signs, but persistently elevated serum lactate.\(^7\) These patients are hypovolaemic owing to under-resuscitation, but supporting their blood pressure on the basis of profound vasoconstriction. If not promptly recognized, this situation creates the potential for sustained shock, organ system failure and death. When receiving such a patient, it is imperative that the ICU practitioner examines the patient’s arterial blood gas and serum lactate concentration for any evidence of persisting anaerobic metabolism. If present, the patient should be aggressively fluid resuscitated until the lactate concentration has cleared to normal.\(^8\) Warming to normal body temperature and adequate systemic analgesia will also help reverse vasoconstriction.

It is not unusual to see ‘overshoot’ in the vital signs after resuscitation from severe haemorrhage, characterized by hypertension, tachycardia, fever and other signs of a hyperdynamic circulation. While this phenomenon has been associated with improved survival from shock, attempts to artificially create it (through the use of inotropic agents) have not generally been successful. Optimal results depend on adequate fluid loading, with inotropes reserved for those patients who do not resuscitate (clear their lactate) despite adequate intravascular volume as measured by pulmonary artery catheter or trans-oesophageal echocardiography.

**The future of resuscitation**

Future efforts to improve outcomes from haemorrhagic shock will focus on more rapid diagnosis and control of bleeding (as with recombinant Factor VIIa or various topical haemostatic agents), better monitoring of the shock state allowing more precise limitation of fluid administration during active haemorrhage and active manipulation of the inflammatory cascade illustrated in Fig. 1. The success of activated protein-C infusion in mitigating the severity of the sepsis syndrome\(^9\) is the tip of an iceberg of practice that will one day include assessment of the patient’s genomic and proteonomic inflammatory predispositions, direct assay of serum mediators and manipulation of the level of inflammatory response during each hour and day after the initial shock insult. In the meanwhile, informed resuscitation from shock in both the early and late phases will help improve outcomes and save lives.

**References**


Please see multiple choice questions 4–8.