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Identification and Resuscitation of the Trauma Patient in Shock

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Trauma is a major source of morbidity and mortality in the United States and world-wide. When analyzed from a global perspective, the World Health Organization estimates that over 5 million people died of traumatic injury in the year 2000, accounting for 9% of global mortality and 12% of the global disease burden [1]. In the United States, traumatic injury is the fifth most frequent cause of death, with roughly 10% of the population suffering from some type of traumatic injury in any given year [2]. Although trauma impacts all age groups, it has an especially significant impact on the younger patient demographic, with roughly 50% of those who die being between 15 and 44 years of age [3].

Regardless of the mechanism of injury, hemorrhage is a leading cause of death following trauma [4–7]. Injury-induced hemorrhage accounts for the largest proportion of mortality within the first hour of trauma center care, causes 50% of injury-associated death within the first 24 hours of trauma care, and claims more lives than any other injury-induced pathology within the first 48 hours of care [7,8]. Moreover, hemorrhage-induced hypotension in trauma patients is predictive of greater than 50% mortality. Unlike more

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insidious processes, traumatic hemorrhagic shock kills quickly, with the bulk of its victims succumbing within the first few hours of emergency department arrival [9]. Though hemorrhage remains the most common etiology of trauma-related shock, other forms of shock such as obstructive, cardiogenic, or neurogenic may occur and need to be considered as well.

This article focuses on rapid diagnosis and treatment of the patient suffering from trauma-related shock, including early identification of patients at risk for occult hypoperfusion. Resuscitation strategies (delayed resuscitation, damage control resuscitation), end points of resuscitation, and the role of blood products and pro-coagulants for resuscitation are discussed.

Early identification of shock

One of the most crucial elements in determining the etiology of shock is to first recognize its presence. Overt hypotension clearly identifies the shocked patient; however, not all patients in shock initially exhibit hypotension. Shock is defined most simply as inadequate tissue perfusion. Particularly in the early stages of disease, determination of tissue hypoperfusion is oftentimes challenging, as the body will immediately engage in compensatory mechanisms designed to prevent cardiovascular collapse. For the clinician, recognizing this compensatory phase of shock is essential to early diagnosis and management (Table 1).

Most commonly, the etiology of shock in trauma is related to hypovolemia caused by uncontrolled hemorrhage. The Advanced Trauma Life Support guidelines illustrate the progressive phases in the development of shock by dividing degrees of hemorrhage into four classes. These classes have been essentially derived from expert opinion and do not necessarily apply to each individual, as physiology varies; however, the insidious progression from class I to class IV hemorrhagic shock is illustrated in Table 2.

Hypotension upon presentation represents decompensated shock and carries little ambiguity as to the perfusion status of the trauma patient. The mortality in patients with hemorrhagic shock presenting with hypotension exceeds 50% overall [9], and this figure is even higher in the elderly patient [10,11]. The definition of initial hypotension has arbitrarily been defined as below a systolic pressure of 90 mm Hg; however, recent investigators have challenged this parameter, reporting that a more appropriate cut point may be a systolic pressure of less than 109 mm Hg on initial presentation. Investigators based this potentially new cut point on data indicating that patients whose presentation systolic blood pressure (SBP) ranges from 91 mm Hg to 109 mm Hg have an increased mortality, compared with their counterparts with systolic pressures greater than 109 mm Hg [12]. The retrospective nature of this study renders this data preliminary at best, but serves as a careful reminder of the arbitrary nature of the 90 mm Hg cut point. Heart rate is a more sensitive indicator of inadequate

Table 1
Indicators of hypoperfusion in the trauma patient

Monitoring method	Indicators of hypoperfusion
Physical examination	• Cool, clammy skin • Change in mental status (anxiety, confusion, lethargy, obtundation, coma) • Decreased urine output • Prolonged capillary refill
Vital signs	• May be normal initially • Tachycardia, bradycardia • Hypotension • Tachypnea • Hypothermia • Shock index (heart rate/systolic blood pressure) > 0.9
Metabolic markers	• Metabolic acidosis • Increased lactate • Increased base deficit

perfusion than hypotension; however, this variable can also be deceptive at times. In a study by Victorino and colleagues [13], upwards of 35% of trauma patients with hypotension did not display tachycardia. Trauma patients without hypovolemia may display tachycardia because of fear and

Table 2
Classes of hemorrhage

Signs monitored on initial presentation[a]	Class I	Class II	Class III	Class IV
Blood loss (mL)	Up to 750	750–1500	1500–2000	>2000
Blood loss (% blood volume)	Up to 15%	15%–30%	30%–40%	>40%
Pulse rate	<100	>100	>120	>140
Blood pressure	Normal	Normal	Decreased	Decreased
Pulse pressure (mm Hg)	Normal or increased	Decreased	Decreased	Decreased
Respiratory rate	14–20	20–30	30–40	>35
Urine output (mL/hr)	>30	20–30	5–15	Negligible
CNS/Mental status	Slightly anxious	Mildly anxious	Anxious, confused	Confused, lethargic
Fluid replacement (3:1 rule)	Crystalloid	Crystalloid	Crystalloid and blood	Crystalloid and blood

The guidelines in Table 2 are based on the 3-for-1 rule. This rule derives from the empiric observation that most patients in hemorrhagic shock require as much as 300 mL of electrolyte solution for each 100 mL of blood loss. Applied blindly, these guidelines can result in excessive or inadequate fluid administration. For example, a patient with a crush injury to the extremity may have hypotension out of proportion to his or her blood loss and require fluids in excess of the 3:1 guidelines. In contrast, a patient whose ongoing blood loss is being replaced by blood transfusion requires less than 3:1. The use of bolus therapy with careful monitoring of the patient's response can moderate these extremes.

[a] For a 70-kg man.

From American College of Surgeons Committee on Trauma. Advanced trauma life support for doctors. 7th edition. Chicago (IL): American College of Surgeons; 2004; with permission.

pain, whereas those with hypovolemia may display ''relative bradycardia'' from paradoxically increased parasympathetic tone, age extreme, or medications (ie, beta blockers) [13,14].

The term ''shock index'' refers to the ratio of heart rate to SBP, and this variable may help identify hypoperfused patients with more subtle vital sign abnormalities. A shock index of greater than 0.9 has been found to be more sensitive than traditional vital sign analysis in identifying disease severity in a heterogeneous group of patients presenting to the emergency department [15]; however, a large retrospective study was unable to demonstrate an advantage of shock index over traditional vital sign analysis in trauma patients [16]. Prospective study of this variable in trauma patients is needed to determine if there is a role for this index beyond simple vital sign analysis. While the presence of vital sign abnormalities may indicate shock, the absence of these abnormalities does not completely exclude occult hypoperfusion in the traumatic patient [17].

Occult hypoperfusion

Occult hypoperfusion (inadequate organ and tissue perfusion in the presence of normal or relatively normal vital signs) can be identified through a careful physical examination plus an evaluation of metabolic markers of tissue hypoperfusion (see Table 1) [17]. Patients should be examined for physical manifestations of poor perfusion, such as cool and clammy skin, mental status changes, and decreased urine output. Metabolic markers of hypoperfusion include bicarbonate, base deficit, and lactic acidosis. With inadequate perfusion, cells will begin anaerobic metabolism and generate lactic acid as well as other acidic by-products. Several investigators have recently described the existence of occult hypoperfusion in the trauma patient. Brown and colleagues [14] studied 139 patients with penetrating abdominal trauma who had normal hemodynamics upon presentation and were taken to the operating room for laparatomy. Of the 139 patients studied, 82% (114) had less than 750 mL^3 of free intraperitoneal blood, 11% (15) had 750 to 1500 mL^3, and 7% (10) had more than 1500 mL^3 at laparotomy. Thus, despite seemingly reassuring vital signs, a number of these patients had a significant amount of intra-abdominal hemorrhage.

Parks and colleagues [18] recently further validated the clinically accepted concept that hypotension is a late sign of shock. In this study, researchers analyzed data from the National Trauma Data Bank and found that tissue hypoperfusion preceded overt hypotension in the majority of patients. Among the subjects reviewed, ''mean and median systolic blood pressure did not decrease to less than 90 mm Hg until the base deficit was worse than −20, with mortality reaching 65%.'' These findings illustrate that significant pathology and hypoperfusion can be present in the normotensive patient, and that tissue hypoperfusion precedes hypotension in many patients. Occult hypoperfusion may be particularly concerning in the elderly

as well, as Callaway and colleagues [19] report a mortality of 38% in normotensive elderly trauma patients with initial lactic acid levels of >4 mmol/dL. Thus, the use of lactic acid and base deficit can serve as metabolic markers of tissue hypoperfusion and should serve as adjuncts to a careful history and physical examination. Once shock is identified, either by overt vital sign abnormalities or through more subtle signs of tissue hypoperfusion, a search for the etiology and immediate measures to reverse hypoperfusion should begin.

Differential diagnosis of shock

Given that shock in the traumatically injured patient is generally a result of hypovolemia due to hemorrhage, the major focus of this article is on identifying and managing that entity; however, other possible etiologies can exist and need to be immediately considered (Box 1). The nonhemorrhagic causes of shock include obstructive, cardiogenic, distributive, and neurogenic. Differentiation of shock begins with the basic history and physical examination and can be supplemented with additional modalities, such as the bedside ultrasound, diagnostic peritoneal lavage, computed tomography, and explorative laparotomy.

Obstructive shock

Causes of trauma-induced obstructive shock include cardiac tamponade and tension pneumothorax. Chest trauma may cause cardiac tamponade or

Box 1. Differential diagnosis for trauma-related shock

Obstructive shock
- Cardiac tamponade,
- Tension pneumothorax
- Pulmonary thromboembolism[a]

Cardiogenic shock
- Cardiac contusion
- Acute coronary syndrome[a]

Distributive shock
- Sepsis[b]
- Adrenal insufficiency[b]

Hypovolemic shock
- Hemorrhage

Neurogenic shock
- Spinal cord injury

[a] Unlikely in acute trauma unless a precipitant to the trauma.
[b] Not observed in early trauma.

tension pneumothorax. Cardiac tamponade caused by penetrating or blunt injury may present with Beck's triad of hypotension, neck vein distention, and diminished heart tones [20]. However, the triad is often difficult to interpret in the trauma patient because hypovolemia may mask neck vein distention [21]. Furthermore, muffled heart tones are difficult to determine in the often noisy emergency department setting. Cardiac echocardiography has rapidly become an important modality for early and accurate diagnosis of tamponade. When available in the resuscitation room, echocardiography can reveal fluid in the pericardial sac and allow for rapid diagnosis and treatment [22,23]. In the setting of cardiac tamponade, collapse of the right atrium typically can be seen and aid in diagnosis. Note that the sensitivity of echocardiography is reduced in the presence of bilateral hemothoraces, a potential result of significant thoracic trauma [24]. Tension pneumothorax classically presents with diminished breath sounds on the side of the pnemothorax, hyperressonance to percussion, and tracheal deviation in the nonintubated patient. In the clinical setting, these classic findings can sometimes be deceiving, and clinical judgment and high clinical suspicion should ultimately guide the decision to immediately perform needle decompression and chest thoracostomy. When the patient is unstable, immediate needle decompression should be performed followed by chest tube thoracostomy. These interventions should be performed before radiologic confirmation, as a tension pneumothorax is a time-dependent, life-threatening lesion.

Cardiogenic shock

Cardiogenic shock would be a rare finding in the trauma patient but could occur from a significant cardiac contusion, a myocardial infarction resulting from the stress of the traumatic injury, or from myocardial ischemia as the inciting event leading to the trauma. Cardiac contusion, often referred to as blunt cardiac injury (BCI), is unlikely to occur as an isolated entity and is most often associated with other coaxial injuries [25]. While there are no established diagnostic criteria for BCI, clinicians concerned about this entity in patients with direct trauma to the chest should obtain a 12-lead electrocardiogram (ECG) upon arrival in the emergency department; echocardiography may be considered depending on the clinical scenario and hemodynamic status of the patient [21]. Several studies have examined the role of ECG and cardiac serum biomarkers (creatine phosphokinase-MB isoenzyme, troponin I) in the evaluation and disposition of patients with BCI. Wisner and colleagues [26] retrospectively reviewed data on 2252 blunt trauma patients admitted to their hospital and found that no patients who were hemodynamically stable on admission with a normal ECG had clinically significant complications. A meta-analysis of retrospective and prospective studies on BCI found that an abnormal ECG, and abnormal CK-MB isoenzymes, correlated with intervention-requiring complications,

while patients with a normal ECG and CPK-MB levels did not develop clinically significant complications. In the prospective trials, 58 out of 2210 patients (2.6%) developed complications, while in the retrospective studies 112 out of 2471 patients (4.5%) developed complications related to BCI; the majority of complications were related to arrhythmias (76% and 77%, respectively) [27]. More recent studies in the trauma literature have addressed the value of cardiac troponin and have generally concluded that a normal ECG and normal troponin are reassuring for ruling out significant BCI [28–30]. However, the issues of length of telemetry monitoring and whether serial cardiac biomarkers need to be obtained and trended over time remain controversial.

In theory, the stress of a traumatic injury could result in myocardial infarction with resultant cardiogenic shock, though this would be very rare. Echocardiography, ECG, and cardiac enzymes would help in defining this particular entity. Finally, myocardial infarction may have lead to the traumatic injury and is and thus mislead the clinician. Shock out of proportion to the mechanism of injury or physical exam findings should raise suspicion for this etiology and is addressed specifically in a subsequent section, ''Clinical mimicry of trauma-induced shock.''

Treatment of cardiogenic shock from cardiac contusion is with intravenous fluids and inotropes, as needed. Some patients may require interventions, such as a cardiac balloon pump [31–33]. Early consultation with a cardiologist should be considered. Cardiogenic shock from an etiology such as a primary myocardial lesion is difficult to treat in the presence of traumatic injury, because modalities such as anticoagulation cannot be employed.

Neurogenic shock

Neurogenic shock is caused by an insult to the spinal cord, often at the cervical or high thoracic level, resulting in a loss of sympathetic tone and creating a state of peripheral dilatation with subsequent hypotension. Typically, bradycardia rather than tachycardia accompanies the hypotension. Even in cases with suspected spinal trauma, it is critical to exclude hemorrhagic or obstructive causes of shock, as these are more common and require immediate intervention. Treatment of neurogenic shock includes hemodynamic support with intravenous fluids as well as atropine, vasopressors, or inotropes as needed. The use of high-dose corticosteroids in this condition remains controversial and is beyond the scope of this article.

Clinical mimicry of trauma-induced shock

While the vast majority of trauma patients presenting with shock have an etiology related to the immediate trauma, a minority of patients may develop shock from another cause with resultant trauma. Clinicians should

consider a nontraumatic etiology of shock when the physical examination and the mechanism of injury do not correlate with the presence of shock. For example, a patient in shock who was involved a single car crash without significant damage to the vehicle or significant bodily injury, should raise suspicion for a competing etiology. A 12-lead ECG or computed angiography of the chest may surprisingly reveal that the patient in a motor vehicle crash who loses consciousness has an acute myocardial infarction or a pulmonary embolism. Endocrinologic emergencies, such as diabetic ketoacidosis and myxedema coma, can result in trauma or actually be precipitated by trauma [34]. Rarely, anaphylactic shock caused by drug and latex allergy could develop in the resuscitation bay by unsuspected iatrogenic mechanisms [35,36]. While these scenarios remain rare, consideration should be given when the shock state is not consistent with the severity of trauma or the identification of traumatic injury.

Management of hemorrhagic shock

Sequential assessment of the airway, breathing, and circulation should rapidly identify nonhemorrhagic causes of shock requiring immediate intervention, such as tension pneumothorax or cardiac tamponade. Persistent hypotension or hypoperfusion from hemorrhagic shock requires simultaneous resuscitation and a definitive strategy to obtain hemostasis. The following sections will focus upon identifying the source of hemorrhage, resuscitation strategies, end points of resuscitation, and adjunctive measures for resuscitation and achieving hemostasis (blood products and procoagulants) for patients with hemorrhagic shock.

Identifying the source of hemorrhage

While in many cases the etiology of hemorrhage is easily identifiable, such as a penetrating gunshot to the abdomen, the origin of hemorrhage may be unknown. The focused assessment with sonography for trauma (FAST) examination can rapidly identify free fluid in the abdominal cavity indicative of hemorrhage. Fluid, or blood, can be visualized using four main windows: perihepatic, perisplenic, suprapubic, and pericardial. Sensitivity and specificity may depend on experience of the operator and amount of free fluid present, but generally fall in the range of 87% to 100% for nonradiologists (generally emergency medicine physicians and surgeons) evaluating blunt thoracoabdominal trauma patients [37–39]. Moreover, nonradiologists have been found to have equivalent accuracy to radiologists in evaluation of the FAST examination [40]. However, despite well-trained operators, small amounts of blood may not be detected by FAST; Branney and colleagues [41] prospectively determined that only 10% of observers were able to detect intraperitoneal fluid (infused by diagnostic peritoneal lavage) with fluid volumes of less than 400 mL.

For some patients, the source of hemorrhage will remain unclear after history, physical examination, and FAST examination. In those who are hemodynamically stable, computed tomography of the chest and abdomen can identify the source of bleeding. For the unstable patient, immediate transfer to the operating room for control of hemorrhage should be considered. Hemorrhage from the thoracic cavity or from within the peritoneum can be controlled with laparotomy; however, consideration should be given to other sources of occult hemorrhage before transport to the operating room. Specifically, a pelvic fracture with retroperitoneal hemorrhage can be the source of life-threatening hemorrhage and shock. The definitive therapy, in that case, may be angiography and embolization rather than operative intervention. A diagnostic peritoneal lavage (DPL) can be performed in the resuscitation room to determine whether or not intraperitoneal hemorrhage is present. With the advent of the FAST examination, the use of the DPL has decreased because intraperitoneal fluid is most often determined with ultrasound. However, DPL can be useful in the event that the FAST is negative or indeterminate, and the origin of hemorrhage remains unclear. In circumstances when pelvic or retroperitoneal hemorrhage is not suspected and the source of blood loss is from either the chest or abdomen, a DPL can be performed in the operating room before determining the choice of first incision (chest versus abdomen).

Resuscitation strategies

The guiding principle in management of shock is to restore tissue perfusion and control the source of the insult. Hemorrhagic shock poses the particular challenge of balancing the timing and kind of resuscitation in relation to the achievement of hemostasis. The traditional strategy of early resuscitation beginning in the field and continuing into the operating room has recently been challenged, specifically in penetrating trauma. A prospective trial by Bickell and colleagues [4] compared immediate versus delayed fluid resuscitation in hypotensive patients with penetrating torso injuries. The investigators reported that patients in whom fluids were restricted until arrival in the operating room had lower mortality, fewer postoperative complications, and shorter hospital length of stay. The proposed mechanism for the reported improved outcomes in the delayed resuscitation group stems from the concept that under-resuscitation allows for blood coagulation followed by "auto-resuscitation," such that blood pressure is restored. In a follow-up study, Dutton and colleagues [42] performed a prospective trial to compare resuscitation strategies and divided patients into either restrictive resuscitation (goal SBP greater than 80 mm Hg) versus liberal resuscitation (goal SBP greater than 100 mm Hg). They did not find any difference in mortality between groups but did find that hemorrhage took longer to control in the group with the liberal fluid strategy.

The overall strategy of delayed resuscitation remains controversial and the original Bickell study had several limitations. First, in the delayed group Bickell and colleagues [4] note that the initial systolic pressures were 59 mm Hg in the field, yet improved to 113 mm Hg in the preoperative setting. The increase in overall pressure in the group reflects several issues, including the fact that patients may have been hypotensive from tension pneumothorax or cardiac tamponade, rather than hypovolemia from on-going hemorrhagic shock. Once reversed, high-volume resuscitation was not necessary. Study investigators conceded that many of the patients examined were not in hemorrhagic shock, and they used the term ''post-trauma hypotension'' rather than hemorrhagic shock to describe them. In addition, in explaining the phenomena of increased blood pressure between the field and preoperative setting, study investigators noted that this group included ''some with severe hypotension, who had died by this time.'' Perhaps this subgroup is the most concerning: could early resuscitation in these profoundly hypotensive patients have supported perfusion and allowed for the possibility of survival? Bickell and colleagues also report that a number of patients received fluid in the delayed resuscitation group (8%), though the remainder reportedly only received antibiotics and intravenous contrast. The 8% protocol violations, though small, raise the potential for bias and provision of fluid to those who may have truly needed it. Finally, the study was performed at a single center, focused on penetrating torso trauma only, and has yet to be reproduced. Despite the shortcomings, there may be some important concepts that can be derived from the Bickell study. Prior to achievement of hemostasis, high-volume resuscitation in the presence of relatively normal blood pressure could potentially be detrimental, and treatment of occult hypoperfusion may be more appropriate after hemostasis is obtained. However, recognition and identification of occult hypoperfusion is still essential and early operative intervention for on-going hemorrhage remains paramount.

In seeming contrast to Bickell's work, recent publications have reported on the concept of damage control resuscitation [43]. Damage control resuscitation refers to early field and emergency department high-volume resuscitation, predominately with blood and procoagulant blood products (ie, plasma, platelets, cryoprecipitate) rather than crystalloid. The concept of damage control resuscitation emerged from surgeons in the battlefields in Iraq and Afghanistan and reflects a strategy employed for the most profoundly wounded patients, such as those with anticipated massive transfusions. On the battlefield, approximately 7% of traumatically injured patients will require massive transfusion, in contrast to 1% in the civilian population. The focus of damage control resuscitation is to provide blood products and procoagulants rather than crystalloid to resuscitate the patient, as well as prevent the dilutional effects of crystalloid on coagulation and core temperature. The end points of resuscitation, proposed by the proponents of this approach, consist of metabolic goals (relief of lactic

acidosis, normalization of base excess) in addition to such traditional goals as urine output and blood pressure normalization. A standard order set for damage control resuscitation consists of 6 units of packed red cells, 6 units of fresh frozen plasma, 12 units of platelets, and 10 units of cryoprecipitate to be infused as a pre-emptive modality during early resuscitation. Though surgeons in the field report better efficacy with this approach, little data has yet to be published, but promises to be forthcoming.

Combining the concepts of delayed resuscitation, damage control resuscitation, and traditional advanced trauma life support resuscitation strategies may be seemingly difficult, yet invoking the concept of therapy tailored to the individual may help guide clinical management. In hemorrhagic shock, the profoundly injured and profoundly hypotensive patient would seemingly not do well with a delayed resuscitation strategy. For this population, invoking a damage control resuscitation strategy, consisting of a high volume of blood products, in addition to emergent operative control may be life saving. For the less severely ill, such as those with occult hypoperfusion or transient hypotension, high-volume resuscitation before achievement of hemostasis may not be warranted. Definitive control of hemorrhage should be obtained immediately. After hemostasis is obtained, adequate resuscitation guided by the end points of resuscitation can be achieved. For those in the in-between category, the exact resuscitation strategy remains unclear; however, the authors would favor providing the necessary volume resuscitation to maintain adequate pressures to allow for safe delivery of the patient to the operating room. Again, after achievement of hemostasis, resuscitation strategies can continue based on the end points of resuscitation discussed in the next section.

The choice of fluid for initial resuscitation has also been the focus of much debate. The concept of damage control resuscitation mitigates the use of crystalloid in favor of blood products, stating that plasma may be the ultimate resuscitation fluid providing both volume and procoagulant properties. Crystalloid may rapidly and temporarily restore pressure and perfusion, but is limited by the dilutional effect and the inability of crystalloid to remain in the intravascular space. Differences between lactated ringers and normal saline are minimal and probably not clinically significant, particularly in the early resuscitation period. Compared to normal saline, lactated ringers are slightly hypotonic and could theoretically result in less capacity to remain intravascular. Conversely, normal saline contains 154 milliequivalents of chloride, and infusion of high quantities may result in a hyperchloremic, metabolic acidosis. The clinical significance of this induced acidosis remains unknown. Hypertonic solutions, such as hetastarch and albumin, have also been used and studied. The debate between crystalloid and colloids is extensive and a comprehensive review is beyond the scope of this article. However, there has yet to be a study to show an advantage of hypertonic or colloid solutions over crystalloid resuscitation.

A recent randomized, controlled trial with albumin in a heterogenous population failed to show any advantage over crystalloid [44]. Moreover, hetastarch may result in coagulopathy and renal damage, thus raising the possibility of harm for the patient in hemorrhagic shock. Thus, no clear advantage exists in the usage of hypertonic or colloid solutions as compared with crystalloid. Blood products and procoagulant colloids may serve as ideal fluid for those with severe hemorrhage, with the advantage of reversing coagulopathy, restoring hemoglobin, and avoiding dilutional coagulopathy. However, in patients with less severe hemorrhage, exposure to blood products may result in potentially unwanted effects on the immune system and risk for associated lung injury. Again, a proper assessment and balance based on the degree of shock and illness of the individual patient is required.

End points of resuscitation

The goal of resuscitation is to restore and maintain adequate tissue perfusion; however, determining when perfusion has been restored may be difficult. As discussed in the previous section, the exact timing for aggressive resuscitation and achievement of end points of resuscitation remains controversial and should be considered for each individual. The following section will discuss the various indicators or end points of resuscitation without regard to timing. Perhaps one of the most important distinctions that needs to be made by the clinician is the difference between perfusion and pressure. Oftentimes, pressure variables are used as surrogates for perfusion; however, this strategy can sometimes lead to erroneous assessment of adequate perfusion. Examples of pressure variables include blood pressure, central venous pressure, and pulmonary capillary wedge pressure. There is no perfect measurement of perfusion; however, indicators of perfusion include metabolic parameters (bicarbonate, base deficit, lactic acidosis), physical exam findings (cool, clammy skin), and urine output (reflecting low flow to the kidneys). Each of these end points of resuscitation has both advantages and limitations, and perhaps the best approach is to take all factors into account at the bedside of the individual patient.

Heart rate and blood pressure

As discussed in the section on identification of shock, overt vital sign abnormalities are the frontline in determination of the perfusion status of the trauma patient. Likewise, the restoration of blood pressure and heart rate to normal values serves as an important end point of resuscitation. However, as noted earlier, the normality or relative normality of vital signs does not necessarily equate to normal perfusion. Therefore, volume replacement should aim to restore blood pressure and pulse rate; after these ends have been achieved the clinician needs to evaluate the patient for any other indicators of ongoing tissue hypoperfusion.

Central venous pressure

Central venous pressure monitoring as a guide to resuscitation has been suggested in certain circumstances and is discussed in the current Advanced Trauma Life Support guidelines. However, use of this variable, particularly in the early resuscitation phase of the patient with hemorrhagic shock, may be fraught with pitfalls. Again, the following discussion assumes hemorrhagic shock and not other etiologies. If an accurately calibrated central venous pressure is initially very high, one would have to immediately consider such entities as pericardial tamponade or tension pneumothorax, conditions which should hopefully be diagnosed without the use or need of central venous pressure measurements.

Central venous pressure represents a pressure and not a perfusion variable. As such, an adequate central venous pressure can be present with poor perfusion; conversely a low central venous pressure can be present in patients with normal perfusion. Understanding this basic concept is essential to use of this variable. The normal central venous pressure range is 0 mm Hg to 6 mm Hg. This means that a central venous pressure in the patient with traumatic shock should be low, and this would not be unexpected in either the well- or hypoperfused patient. In contrast, many conditions can increase central venous pressure regardless of underlying volume status or perfusion. For example, patients with chronic obstructive pulmonary disease may have very high baseline central venous pressures. Patients receiving positive pressure ventilation, particularly those requiring positive-end expiratory pressure, will have increased central venous pressures, as will patients requiring any form of vasopressor therapy. Moreover, acidosis may result in central vascular vasoconstriction despite hypovolemia [45]. Not surprisingly, oftentimes central venous pressure calibrations are inaccurate or erroneously calculated. In essence, central venous pressure is an indirect measure of volume affected by other causes of pressure elevation, including erroneous calculation. In the initial stages of hemorrhagic shock, this variable should be used with caution. With very rare exception, patients in the early stages of hemorrhagic shock with persistent hypotension or hypoperfusion should be provided with volume resuscitation, regardless of central venous pressures. In the later stages of shock or in shock of a mixed etiology, central venous pressure could be used to help guide management, however interpretation should be taken into the context of patient characteristics, other hemodynamic variables, and external physiologic factors.

Urine output

Urine output remains an important, noninvasive end point of resuscitation in the patient with shock, and typically a urine output of less than 0.5 mL per kilogram per hour is considered inadequate in the shock state. Decreased perfusion to the kidneys results in decreased urine output and provides an excellent measure of resuscitation. However, this end point is

limited in the early stage of trauma, as the evaluation of urine output requires the tincture of time. Also, other causes of decreased urine output exist, such as underlying renal disease (ie, end-stage renal disease) or acute tubular necrosis. In unclear cases, diagnostic and clinical measures should be employed to distinguish whether oliguria is flow related (ie, prerenal) versus nonflow related; however, these are beyond the scope of the current article. Finally, in some cases, normal or high urine outputs (particularly in the initial time period) can occur in the face of inadequate perfusion. Therefore, urine output, like all end points of resuscitation, needs to be taken in context of the overall clinical picture and in conjunction with other measures of resuscitation.

Lactate clearance and base deficit

Lactic acidosis may indicate tissue hypoperfusion even when vital signs are initially normal or relatively normal. In addition to allowing for identification of shock or hypoperfusion, lactic acid levels can also provide an end point of resuscitation and indicator of restored perfusion. Effective lactate clearance has been found to be associated with lower mortality levels in trauma, sepsis, and postcardiac arrest [46–48]. Therefore, effective lactate clearance in the patient with traumatic shock signifies a more favorable overall outcome in the long-term, and provides an end point of resuscitation in the short-term. Like all of the end points discussed, lactate clearance has shortcomings. For unclear reasons, some patients with significant hypoperfusion do not mount a high lactate level. Conversely, some conditions are associated with elevated lactic acid levels without associated tissue hypoperfusion; examples of these conditions include seizure activity, severe respiratory distress (work of breathing), certain medications (ie, anti-retrovirals, metformin, linazolid, albuterol), thiamine deficiency, carbon monoxide or cyanide toxicity, and diabetic ketoacidosis.

In addition to lactate clearance, base deficit may provide yet another metabolic end point of resuscitation. Arterial base deficit represents the degree of metabolic acidosis, calculated as how many milliequivalents of base would need to be added to a liter of blood to normalize the pH. A retrospective analysis of prospectively collected data on 100 trauma patients by Kincaid and colleagues [49], revealed that patients with persistently elevated base deficit had higher rates of multiple organ failure and death when compared with those with a low base deficit. Davis and colleagues [50] found the base deficit calculated on admission for trauma patients was predictive of need for early transfusion of blood products, as well as increased complications, including adult respiratory distress syndrome and multisystem organ failure. The most significant shortcoming of base deficit is that other diseases and physiologic states may alter base deficit without reflecting an alteration of perfusion; several examples of this include renal failure, chronic obstructive pulmonary disease, or acidosis from any of a number of additional nonperfusion related causes.

Lactate and base deficit can be used both in the initial resuscitation period, as well as during the subsequent inpatient hospitalization. A recent study by Martin and colleagues [51] compared simultaneously measured serum lactate levels and arterial base deficit levels in 1298 patients in the surgical intensive care unit, 79% of which were trauma patients, and found that in approximately one third of the cases there was disagreement between the two measures. Additionally, lactate was more predictive of morbidity and mortality than was base deficit among their study population.

Lactic acidosis and base deficit arguably remain the best metabolic markers of tissue hypoperfusion that we can measure and follow. As with any other laboratory test, these variables must be interpreted and incorporated with the entire clinical context when managing critically ill trauma patients.

Achieving hemostasis

Perhaps the most important aspect of the management of hemorrhagic shock is to obtain hemostasis. Obtaining hemostasis can be definitively achieved by proper identification of the source of hemorrhage and an immediate measure to control it. Operative therapy is required for uncontrolled thoracic or intraperitoneal hemorrhage, whereas angiography and cauterization is typically employed for retroperitoneal bleeding from a pelvic source. While active resuscitation and a move toward definitive hemostasis is being made, consideration can be given to adjuncts of hemostasis and resuscitation, such as blood products and procoagulants.

Blood and blood products in traumatic shock

Since World War I, human blood products have been the gold standard in large volume resuscitation efforts. However, the reliance on human blood products is limited by several factors, including availability, relatively short shelf life, and potential to transmit disease. Moreover, packed red blood cell units contain scant amounts of platelets and coagulation factors, thereby contributing to the dilutional element of hemorrhage-related coagulopathy, particularly when dosed in the absence of procoagulation adjuncts. At present, human red cell alternatives have been unsuccessful in matching the efficacy of human red cells in the setting of massive volume replacement [52].

Despite these shortcomings, transfusion of packed red cells remains the gold standard in resuscitation of the actively hemorrhaging patient. Though controversy exists as to when transfusion should be undertaken, most would agree that transfusion should be provided in ongoing hemorrhage if hemodynamic instability persists after approximately 2 L of crystalloid infusion. Hypoperfusion in the absence of hypotension should be considered a possible indication for transfusion as well. Ultimately, bedside clinical judgment will determine the need for transfusion. In critically ill hemorrhaging patients, a very low threshold for transfusion should be maintained.

To mitigate coagulopathy and assist in hemostasis, additional blood components should be considered when providing large volume resuscitation, including fresh frozen plasma (FFP), platelets, and fibrinogen concentrate or cryoprecipitate [2,53,54]. Definitive evidence regarding the optimal timing, dose, and combination of these agents remains elusive. Gonzalez and colleagues [54] performed a retrospective study evaluating the timing of FFP administration, and found that the severity of coagulopathy in trauma patients upon admission to the intensive care unit is associated with decreased survival. Based on these findings, the investigators suggest earlier use of FFP and argue that more aggressive use of FFP could improve mortality and reduce total in-hospital transfusion requirements. However, outcome data is limited and systematic review of known studies in this area failed to show significant improvement in blood saving or promotion of hemostasis [55]. However, only one of these studies was focused specifically on trauma resuscitation.

In addition to FFP, replacement of individual coagulation factors, such as factor VIIa, have been studied and used in the setting of traumatic hemorrhage over the past decade. Interest in factor VIIa for hemorrhagic shock surged following a case report from Israel, where an exsanguinating soldier who sustained a penetrating inferior vena cava tear failed traditional hemostatic efforts, only to be stabilized by two doses of factor VIIa [56]. Evidence supporting the use of factor VIIa is relatively sparse but perhaps encouraging, with retrospective and small prospective studies suggesting mitigation of coagulopathy and perhaps improved outcome [57–60]. Despite evidence of potential efficacy, large randomized controlled trials have yet to be performed and the universal application of factor VIIa remains unknown [61–64]. Future prospective trials will hopefully help to identify the value of factor VIIa in the treatment of traumatic hemorrhage.

Another proposed method of reversing the coagulopathy of trauma is the use of antifibrinolytic agents, such as aprotinin and tranexamic acid (TXA). These drugs are lysine analogs, substituting for lysine in plasminogen and thereby promoting fibrinolysis [65]. Both of these compounds have proven to significantly reduce perioperative bleeding and intraoperative transfusion requirements in a variety of surgical settings [66,67]. However, data in traumatic hemorrhage is limited and insufficient to provide any recommendations either for or against the use of these adjuncts [68]. A randomized, controlled trial of TXA in hemorrhagic shock is currently underway [69].

At this time, transfusion of FFP, cryoprecipitate, and platelets remain the traditional adjuncts in minimizing the coagulopathy associated with hemorrhagic shock. The optimal dosages and timing of administration remains unknown; however, we would support early administration of products to minimize coagulopathy. Other adjuncts, such as factor VIIa and antifibrinolytics, continue to be studied as outcome data remains lacking for these therapies.

Summary

Management of the critically ill trauma patient requires rapid identification and differentiation of the patient in shock. Overt shock with accompanying hypotension is not difficult to identify, though occult hypoperfusion (normal pressure in the presence of tissue hypoperfusion) may be subtle. Most often, patients with shock following trauma are suffering from ongoing hemorrhage, though other etiologies may be present. The initial examination should rapidly reveal nonhemorrhagic causes of shock. Once the etiology of shock is identified, definitive therapy should be initiated. Achievement of hemostasis and restoration of tissue perfusion remain paramount for the treatment of hemorrhagic shock. The timing and kind of resuscitation of hemorrhagic shock may vary depending on the severity of injury and patient physiology, though delivery of a viable patient to the operating room is the primary goal of the emergency physician. Delayed resuscitation in penetrating torso trauma remains controversial, and the potential application of this approach is discussed in detail in the main portion of this article. Damage control resuscitation, consisting of high volume blood products and procoagulants, may be lifesaving for the profoundly injured and profoundly hypotensive patient. Once hemostasis is achieved, resuscitation aimed at specific end points should be used. Each end point of tissue perfusion has advantages and disadvantages, and the optimal resuscitation strategy should take all parameters into account in context of the individual patient. The rapid identification, differentiation, resuscitation, and achievement of hemostasis will ultimately lead to optimal outcome in the management of the patient with traumatic shock.

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