The Science of Shock and Fluid Resuscitation

1

Eileen M. Bulger

Introduction

Shock is defined simply as inadequate tissue perfusion. This is an important definition as many think of shock as hypotension, but inadequate tissue perfusion can occur long before hypotension is evident. Inadequate tissue perfusion leads to anaerobic metabolism due to inadequate oxygen delivery and activation of the host inflammatory response, which can further exacerbate tissue injury and lead to the development of organ failure. Thus early recognition and management of shock is critical to preventing these sequelae. There are many compensatory mechanisms to preserve vital organ perfusion in the setting of hypovolemia. These include increased heart rate, increased sympathetic tone, and peripheral vasoconstriction. As a result, most patients will not manifest significant hypotension until they have lost more than 30 % of their blood volume. This is described as Class 3 shock. Class 1 shock involves a loss of 10–15 % of circulating blood volume (500–700 mL), Class 2 20–30 % (750–1,500 mL), Class 3 30–40 % (1,500–2,000 mL), and Class 4 >40 % (>2,000 mL). Cardiac output is dependent on both the heart rate and stroke volume. As a result, most healthy patients will compensate for decreased stroke volume by increasing tachycardia and this is usually the first sign of shock.

Etiology of Shock

The most common etiology of shock in the emergency setting is hypovolemic shock due to blood loss from trauma, spontaneous hemorrhage, or insensible fluid losses such as burns. Bleeding may be localized to the abdomen or thorax based on mechanism of injury but it is important to remember after trauma there are also significant losses due to hemorrhage from skin and extremity lacerations. There may also be issues of prehospital intravascular volume depletion due to prolonged extrication, long transport times, or delay in resuscitation in the field. Many medications used for general anesthesia cause peripheral vasodilatation and loss of sympathetic tone and thus patients with hypovolemia may become hypotensive only upon induction as they lose these compensatory mechanisms.

The second most common etiology of shock in the emergency setting is septic shock. The urological emergencies of Fournier's gangrene, upper urinary tract infection, and severe genital infection all may be associated with septic shock and are reviewed later in the text. Sepsis is defined as clinical signs consistent with the systemic inflammatory response syndrome (SIRS) together with definitive evidence of infection [1]. A diagnosis of SIRS includes two or more of the following: temperature

E.M. Bulger (✉)
Department of Surgery, Harborview Medical Center, University of Washington, 325, 9th Avenue, Box 359796, Seattle, WA 98104, USA
e-mail: ebulger@u.washington.edu

H. Wessells (ed.), *Urological Emergencies: A Practical Approach*, Current Clinical Urology,
DOI 10.1007/978-1-62703-423-4_1,

>38 or <36 °C, heart rate >90 beats/min, respiratory rate >20 breaths/min or $PaCO_2$ < 32 mmHg, and WBC > 12,000 cells/mm^3 or <4,000 cells/mm^3 or >10 % immature forms. Severe sepsis is sepsis associated with organ dysfunction, hypoperfusion, or hypotension. Septic shock is sepsis with hypotension despite adequate fluid resuscitation. Septic shock can be divided unto early versus late septic shock. In the early phase, patients will be hyperdynamic with increased cardiac output, but inadequate tissue perfusion due to peripheral vasodilation and poor utilization of oxygen in the tissues. These patients are often hypovolemic as well and require initial fluid resuscitation prior to considering administration of vasopressors. In the late phase of septic shock, patients have impaired myocardial contractility and vasoconstriction, which can lead to progressive organ dysfunction. Exacerbation of the inflammatory response can also result in increased microvascular permeability which creates a "capillary leak" with loss of intravascular fluids into the interstitial spaces. As discussed in detail below, early goal-directed therapy for these patients has been associated with improved outcome.

Other less common causes of shock in trauma patients include cardiogenic shock, neurogenic shock, and hypoadrenal shock. Cardiogenic shock results from impaired cardiac function leading to inadequate cardiac output, usually as a result of chronic congestive heart failure or acute myocardial ischemia. Cardiogenic shock can also occur in the setting of compression of the venous outflow from increased intrathoracic pressure such as a tension pneumothorax. In this circumstance the patient is usually not hypovolemic. Neurogenic shock results from disruption of the spinal cord usually in the high cervical region resulting in loss of sympathetic tone and diffuse vasodilation. Finally, hypoadrenal shock may be due to acute adrenal insufficiency, which can result from adrenal infarction, withdrawal from long-term corticosteroid therapy, or critical illness-related corticosteroid insufficiency. For the purposes of this chapter we will focus on the management of the three leading causes of shock in the trauma patient, hypovolemic, septic, and cardiogenic, but it is important to remember neurogenic and hypoadrenal shock in the differential diagnosis especially for acutely injured patients.

Initial Management of Shock

The initial management of shock involves a high index of suspicion to allow early recognition of shock and initial fluid administration to begin to replenish intravascular volume and evaluate the patient's response to fluid challenge. The key is to quickly determine the etiology of shock in this patient so as to allow initiation of the treatment for the underlying problem. For example in a patient with hypovolemic shock the focus should be on finding the source of the bleeding and controlling that source. For a patient with septic shock, early administration of antibiotics and control of the source of the infection are paramount.

Patients can be grouped based on their initial response to a fluid challenge as responders, transient responders, and nonresponders. This distinction is important as it allows identification of patient who may require rapid surgical intervention. A responder is a patient who upon receipt of a fluid challenge normalizes their vital signs and does not show evidence of ongoing instability. A transient responder will show improved vital signs for a brief period of time but will again deteriorate suggesting an ongoing source of volume loss. Finally, a nonresponder will show no improvement in vital signs and likely represents a patient with significant hemorrhage or another etiology of shock such as cardiogenic or neurogenic. As a surgeon the focus must be on ruling out life-threatening hemorrhage in this group of patients. Patients who do not have evidence of ongoing hemorrhage may require invasive monitoring to further evaluate preload, such as monitoring of central venous pressure (CVP) or pulmonary wedge pressure, and cardiac function, such as measurement of cardiac output.

Management of Hypovolemic Shock

When hypovolemic shock is evident the treatment involves controlling any sources of hemorrhage and repleting fluid losses with crystalloid and blood products. There is no data to support colloids as the primary resuscitation fluid for trauma or surgical

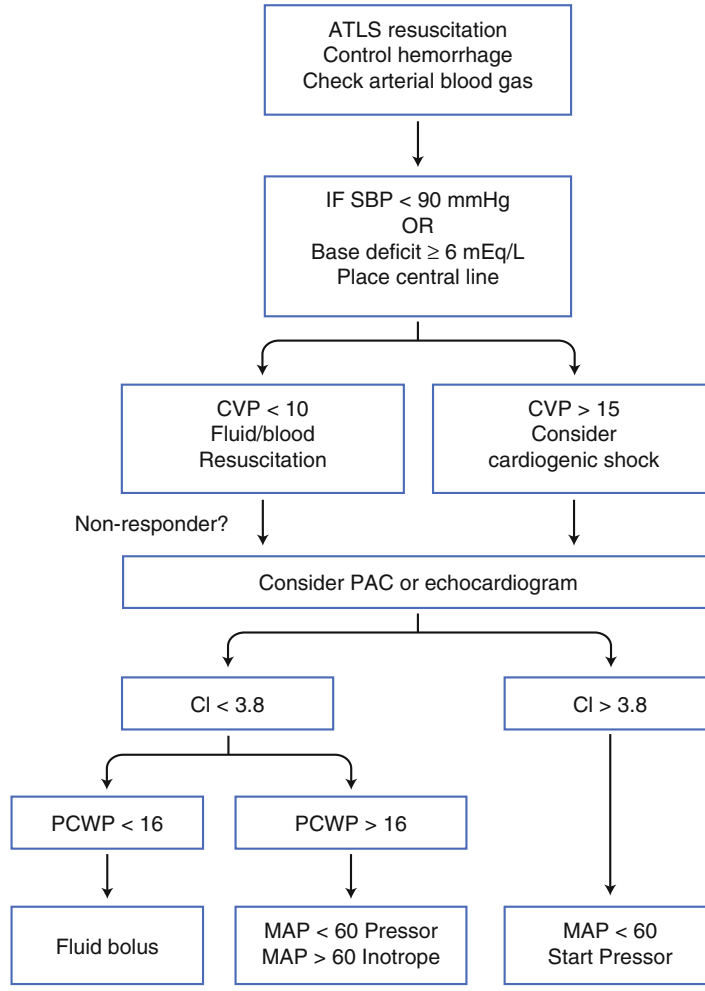

Fig. 1.1 Resuscitation algorithm for patients with shock after traumatic injury. *SBP* systolic blood pressure; *CVP* central venous pressure; *PAC* pulmonary artery catheter; *CI* cardiac index; *PCWP* pulmonary capillary wedge pressure; *MAP* mean arterial pressure (adapted from [28])

patients [2]. There is data to suggest that it is reasonable to limit volume resuscitation in the actively bleeding patient until surgical or angiographic hemostasis can be achieved. This is based on data from animal models that suggest that with a major vascular injury, rapid fluid administration can lead to increased blood pressure that may lead to increased hemorrhage in this situation [3]. Data from a clinical trial of patients with penetrating torso trauma suggests that very limited fluid resuscitation prior to surgical control of hemorrhage was beneficial [4]. This has also been shown to be the case in the setting of a ruptured abdominal aortic aneurysm [5, 6]. This approach is termed hypotensive resuscitation and implies that limited fluid is given to target a lower threshold for systolic blood pressure until hemorrhage control is achieved. There are also several animal studies supporting the use of small volume hypertonic saline solutions for resuscitation of hemorrhagic shock [7, 8]. However, recent clinical trials of prehospital resuscitation with hypertonic saline in severely injured patients have failed to demonstrate any benefit [9–11]. Figure 1.1 illustrates the resuscitation protocol advocated by the Inflammation and Host Response to Injury: Glue Grant Consortium [12].

There is also recent data to suggest that the development of coagulopathy occurs very early after injury in the setting of significant blood loss [13, 14]. This has led to the recommendation to consider early administration of blood products, especially fresh frozen plasma (FFP) in these

patients. Several retrospective studies from both civilian and military cohorts have suggested that patients requiring a massive transfusion who receive a higher ratio of plasma to packed red blood cells (PRBC) have a better outcome than those receiving a lower ratio [15–19]. Another recent study has also suggested that the ratio of platelets to PRBC may also play an important role [20]. These retrospective studies all suffer from survival bias, in that patients had to live long enough to get the higher ratio of products, but in general there is a sense that in selected patients minimizing crystalloid in favor of early administration of blood products with higher ratios of FFP to PRBC than traditionally used may be beneficial. A randomized controlled trial to determine the optimal FFP and platelet to PRBC ratio will likely begin enrollment in 2012. In the meantime, implementation of a standardized algorithm for the approach to as massive transfusion patient has been associated with improved outcome [21, 22]. Thus each institution should have a protocol, which can be activated as needed, to provide rapid availability of blood products to these patients. Finally, attention should be paid in these patients to avoid hypothermia, which has been clearly associated with increased coagulopathy and higher mortality.

In the setting of hypovolemic shock exacerbated by coagulopathy, acidosis, and hypothermia, damage control principles should be employed in the operating room [23]. The goals of a damage control procedure are to get control of the surgical hemorrhage and minimize GI contamination while minimizing the time in the operating room by delaying definitive repair and abdominal closure until after the patient has stabilized with correction of coagulopathy and rewarming to normothermia.

Management of Septic Shock

Recent data has suggested that early recognition of septic shock and aggressive management is associated with improved outcome. This is largely based on the study by Rivers et al. [24] that promoted an algorithm for early goal-directed therapy, which resulted in lower mortality. This includes early assessment of serum lactate to help identify patients with inadequate tissue perfusion, early administration of appropriate antibiotics, and early fluid administration and CVP monitoring to a goal CVP of 8–12 mmHg prior to initiation of vasopressors. The algorithm also emphasizes a targeted resuscitation to an $ScvO_2$>70 % (Fig. 1.2). Once fluid resuscitation goals have been met one can consider vasopressor support. The first line agent is commonly norepinephrine with additional vasopressin as needed.

Management of Cardiogenic Shock

Cardiogenic shock is less common but can be a source of sudden intraoperative shock in a patient suffering an acute myocardial infarction or pulmonary embolism. In this setting, one should rule out hypovolemia by administration of volume and consider early invasive monitoring to evaluate preload and cardiac function. When a patient has a sudden decline in the operating room it is also important to consider other problems that can cause acute hypotension such as a tension pneumothorax. If there is high suspicion for a pulmonary embolus the case should be aborted and if feasible anticoagulation should be considered. If this is not feasible, one may consider angiographic attempts to retrieve the thrombus and placement of an IVC filter to prevent additional thrombi. In these setting of cardiovascular collapse, surgical retrieval of the thrombosis may also be considered. If there is high suspicion for acute coronary ischemia, the case should be aborted and consideration made for cardiac catheterization. Most patients with cardiogenic shock will have adequate preload. Once this has been assured one should consider inotropic support if tissue perfusion remains inadequate.

Hemodynamic Monitoring

Most patients with shock especially those not responding to initial resuscitation efforts will require hemodynamic monitoring to guide the resuscitation. Data that can be useful to collect include markers of preload such as CVP or pulmonary artery wedge pressure, assessment of

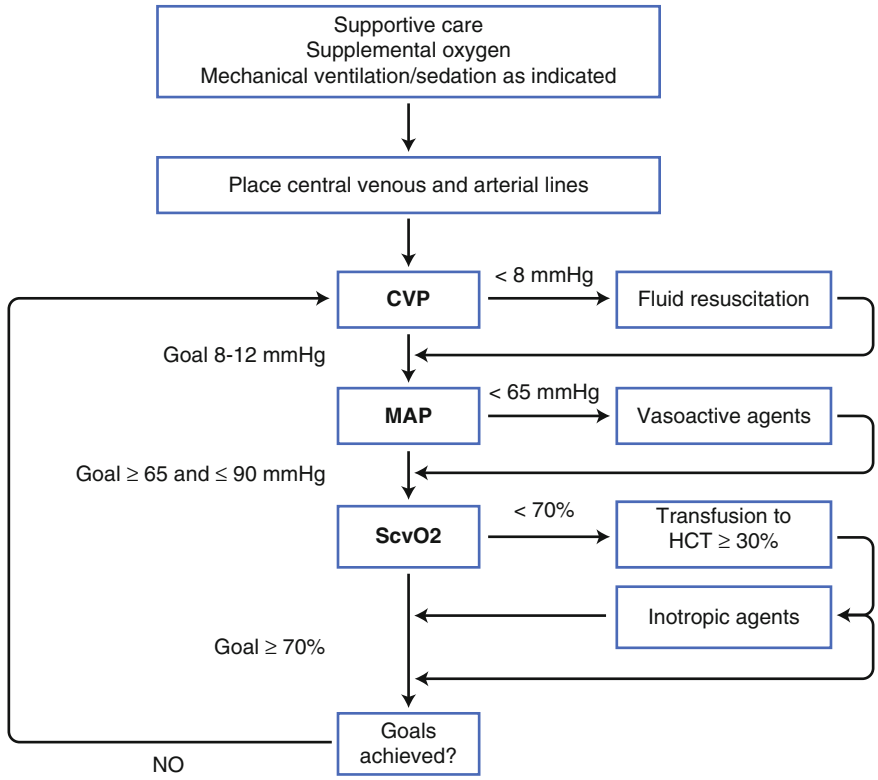

Fig. 1.2 Resuscitation algorithm for patients with septic shock. *CVP* central venous pressure; *MAP* mean arterial pressure; *$ScvO_2$* central venous oxygen saturation; *HCT* hematocrit (early goal-directed therapy, adapted from [24])

tissue oxygen delivery and utilization such as the central venous oxygen saturation and the cardiac output and systemic vascular resistance (SVR). Table 1.1 illustrates the changes one would expect in these parameters for each type of shock. There are a variety of monitoring devices now available to assess these parameters ad some authors have also advocated the use of bedside ultrasound or echocardiography as another means to assess preload.

Endpoints of Resuscitation

There has been much debate in the literature regarding the optimal endpoints to determine that an effective resuscitation has been achieved. Urine output is a good marker for restoration of renal perfusion and improved intravascular volume as long as there has not been a prolonged period of hypovolemia leading to acute renal failure, or underlying chronic renal disease. Urine output targets of 30 mL/h for adults and 1–2 mL/kg/h for children are accepted. More invasive monitoring allows for evaluation of preload based on venous filling pressures and cardiac output over time. As noted in the sepsis studies, many authors have advocated central mixed venous oxygen saturation as a target for resuscitation with a goal $ScvO_2 > 70$ %. Finally many authors target resolution of metabolic acidosis by trending either the arterial base deficit or the serum lactate level [25–27].

Summary

In summary, shock is simply inadequate tissue perfusion. The major etiologies for shock are hypovolemic/hemorrhagic, septic, cardiogenic, neurogenic, and hypoadrenal. The leading cause of shock in emergency patients is hypovolemic. Management of shock hinges on identifying the underlying cause and treating it (i.e., stop the bleeding) while pursuing

Table 1.1 Hemodynamic changes based on type of shock

Type of shock	CVP/ PCWP	Cardiac output	SVR	SvO_2
Hypovolemic	↓	↓	↑	↓
Early septic	↑↓	↑	↓	↑
Late septic	↑↓	↓	↑	↓
Cardiogenic	↑	↓	↑	↓
Neurogenic	↓	↓	↓	↓
Hypoadrenal	↑↓	↓	↓	↓

fluid resuscitation to restore intravascular volume. Resuscitation can be guided by hemodynamic monitoring and the clinical response to fluid resuscitation including improved urinary output and resolution of metabolic acidosis. Early recognition of shock is critical to allow intervention before there is significant organ injury.

References

1. Sihler KC, Nathens AB. Management of severe sepsis in the surgical patient. Surg Clin North Am. 2006;86(6):1457–81.
2. Barron ME, Wilkes MM, Navickis RJ. A systematic review of the comparative safety of colloids. Arch Surg. 2004;139(5):552–63.
3. Mapstone J, Roberts I, Evans P. Fluid resuscitation strategies: a systematic review of animal trials. J Trauma. 2003;55(3):571–89.
4. Bickell WH et al. Immediate versus delayed fluid resuscitation for hypotensive patients with penetrating torso injuries. N Engl J Med. 1994;331(17):1105–9.
5. Roberts K et al. Hypotensive resuscitation in patients with ruptured abdominal aortic aneurysm. Eur J Vasc Endovasc Surg. 2006;31(4):339–44.
6. van der Vliet JA et al. Hypotensive hemostasis (permissive hypotension) for ruptured abdominal aortic aneurysm: are we really in control? Vascular. 2007;15(4):197–200.
7. Tobias TA et al. Comparative effects of 7.5% NaCl in 6% Dextran 70 and 0.9% NaCl on cardiorespiratory parameters after cardiac output-controlled resuscitation from canine hemorrhagic shock. Circ Shock. 1993;39(2):139–46.
8. Kien ND et al. Cardiac contractility and blood flow distribution following resuscitation with 7.5% hypertonic saline in anesthetized dogs. Circ Shock. 1991;35(2):109–16.
9. Bulger EM et al. Out-of-hospital hypertonic resuscitation following severe traumatic brain injury: a randomized controlled trial. JAMA. 2010;304(13):1455–64.
10. Bulger E et al. Hypertonic resuscitation of hypovolemic shock after blunt trauma: a randomized controlled trial. Arch Surg. 2008;143:39–148.
11. Bulger EM et al. Out-of-hospital hypertonic resuscitation after traumatic hypovolemic shock: a randomized, placebo controlled trial. Ann Surg. 2011;253(3):431–41.
12. Inflammation and host response to injury clinical guidelines [cited 30 Sept 2009]. Available from: http://www.gluegrant.org/clinical-protocols.htm.
13. Hess JR et al. The coagulopathy of trauma: a review of mechanisms. J Trauma. 2008;65(4):748–54.
14. Hess JR, Holcomb JB, Hoyt DB. Damage control resuscitation: the need for specific blood products to treat the coagulopathy of trauma. Transfusion. 2006;46(5):685–6.
15. Borgman MA et al. The ratio of blood products transfused affects mortality in patients receiving massive transfusions at a combat support hospital. J Trauma. 2007;63(4):805–13.
16. Gonzalez EA et al. Fresh frozen plasma should be given earlier to patients requiring massive transfusion. J Trauma. 2007;62(1):112–9.
17. Borgman MA et al. The effect of FFP: RBC ratio on morbidity and mortality in trauma patients based on transfusion prediction score. Vox Sang. 2011;101(1):44–54.
18. Holcomb JB et al. Increased plasma and platelet to red blood cell ratios improves outcome in 466 massively transfused civilian trauma patients. Ann Surg. 2008;248(3):447–58.
19. Gunter Jr OL et al. Optimizing outcomes in damage control resuscitation: identifying blood product ratios associated with improved survival. J Trauma. 2008;65(3):527–34.
20. Inaba K et al. The impact of platelet transfusion in massively transfused trauma patients. J Am Coll Surg. 2010;211(5):573–9.
21. Nunez TC et al. Creation, implementation, and maturation of a massive transfusion protocol for the exsanguinating trauma patient. J Trauma. 2010;68(6):1498–505.
22. Holcomb JB. Optimal use of blood products in severely injured trauma patients. Hematology Am Soc Hematol Educ Program. 2010;2010:465–9.
23. Brasel KJ, Weigelt JA. Damage control in trauma surgery. Curr Opin Crit Care. 2000;6(4):276–80.
24. Rivers E et al. Early goal-directed therapy in the treatment of severe sepsis and septic shock. N Engl J Med. 2001;345(19):1368–77.
25. Bannon MP et al. Central venous oxygen saturation, arterial base deficit, and lactate concentration in trauma patients. Am Surg. 1995;61(8):738–45.
26. Callaway DW et al. Serum lactate and base deficit as predictors of mortality in normotensive elderly blunt trauma patients. J Trauma. 2009;66(4):1040–4.
27. Husain FA et al. Serum lactate and base deficit as predictors of mortality and morbidity. Am J Surg. 2003;185(5):485–91.
28. Moore FA et al. Inflammation and the host response to injury, a large-scale collaborative project: patient-oriented research core-standard operating procedures for clinical care III. Guidelines for shock resuscitation. J Trauma. 2006;61(1):82–9.