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

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The challenges of this guideline include: a) early identification of high risk patients, b) implementation in environments that are suboptimal for monitoring resuscitation, c) early identification of resuscitation “non-responders” that require more aggressive interventions, and d) avoiding potentially harmful over zealous interventions.

This guideline is based on the best available evidence and expert consensus discussions supported by the Inflammation and Host Response to Injury Large Scale Collaborative Project award from the National Institute of General Medical Sciences, and is being used in the funded clinical studies.1,2 The following section provides a brief overview of the rationale for specific guideline recommendations. This is followed by two algorithms that depict escalation in interventions and monitoring requirements in the subset of patients who do not respond to ongoing volume loading and/or blood transfusions. With multi institutional experience and critical analysis this resuscitation process may be further refined; at this time it is intended to serve as a template for interventional trials and to test the utility of new monitoring technology. This protocol was designed for blunt trauma patients who are presumed not to have a serious concomitant brain injury. Its purpose is to guide resuscitation as soon as feasible after arrival in the Emergency Department (ED) after control of active torso bleeding.

Protocol Rationale

Early Recognition of Shock in the Emergency Department

Recognizing the presence of shock and assessing its severity are key factors in early identification of high risk patients. Shock often can be detected by simple physical examination findings in the ED resuscitation area. Diminished or absent peripheral (radial, pedal) or central (carotid, femoral) pulses, decreased capillary refill associated with pallor or cool clammy extremities may all denote the presence of shock and hypovolemia. The initial blood pressure (BP) measurement should be performed using a manual cuff because automatic cuff BP measurement devices may over-
Volume 61 • Number 1

Guidelines for Shock Resuscitation

estimate systolic BP (SBP) in hypovolemic trauma patients. A SBP < 90 mm Hg and/or a heart rate (HR) > 130 bpm is generally considered to be indicative of shock. Some patients (especially the young) compensate for hypovolemia and maintain a normal SBP even in the face of significant ongoing hemorrhage although this is often associated with tachycardia. Additionally, because acute massive blood loss may paradoxically trigger a vagal-mediated bradycardia, the traditional inverse correlation between increased HR and decreased effective blood volume may not hold in the early resuscitation period. The initial hemoglobin concentration ([Hb]) is notoriously misleading because there has not been sufficient time for influx of interstitial fluid into the intravascular space and the patient has not yet been volume resuscitated. Therefore, it is important to measure the [Hb] again after the initial 2 L of crystalloid loading, a decrease greater than 2 g/dL is grounds for concern. The magnitude of arterial base deficit (BD) has been shown to be a useful index of the severity of hemorrhagic shock. A BD ≥ 6 mEq/L is indicative of severe shock. Serial BD determinations are important in determining the effectiveness of interventions and lack of response is indicative of a poor prognosis. Other less well studied markers of the severity of shock include venous blood lactate, bicarbonate concentrations and end-tidal CO₂ to PaCO₂ differences.

Volume Loading With Isotonic Crystalloid Fluid

The key step in resuscitation of the injured patient is the control of active hemorrhage. The actively bleeding patient cannot be adequately resuscitated without hemorrhage control. Resuscitation with isotonic crystalloid fluids has been the standard of care in the United States since the late 1960s. The laboratory work of Shires and Moyer demonstrated the best survival was achieved with large volume isotonic crystalloid solution. The basic concept is that interstitial fluid moves into both the intravascular and intracellular spaces in response to shock and that adequate resuscitation requires replenishment of both the intravascular and interstitial spaces. Their studies demonstrated that the optimal ratio of isotonic crystalloid infusion to shed blood infusion was 3 to 1. Subsequent studies demonstrated that the optimal ratio for survival after severe shock increases and can be as high as 8 to 1. Clinical trials were performed in the 1970s and 1980s that compared isotonic resuscitation and colloid resuscitation. Individually, these trials were underpowered and reported conflicting results. When subjected to meta-analysis, they have yielded no consistent differences in overall outcome. When the same data were subjected to subgroup analysis, however, the use of isotonic crystalloids in trauma patients was associated with improved survival. A large clinical trial published in 2004 found no differences in outcome between crystalloid and colloid resuscitation in ICU patients, but again, subgroup analysis demonstrated improved outcomes in trauma patients receiving crystalloid. Although these subgroup analyses are not definitive, they are consistent with the early laboratory studies, which indicated that survival in hemorrhagic shock is improved with large volume crystalloid resuscitation. However, in recent years, “damage control” surgery combined with prompt ICU resuscitation appears to be salvaging more patients who are arriving with exsanguinating hemorrhage. Unfortunately, over zealous crystalloid infusion appears to have adverse consequences, e.g. cerebral edema (increased ICP), acute lung injury (worsened pulmonary edema), and the abdominal compartment syndrome (primary and secondary).

Lactated Ringer’s is the Preferred Isotonic Crystalloid

Although newer formulations (e.g. Ringer’s ethyl pyruvate) are being tested clinically, normal saline (NS) and lactated Ringer’s (LR) remain the most commonly used isotonic fluids. In theory, LR is preferable to NS because it provides a better buffer for metabolic acidosis, but to date, investigators have not documented any important differences in outcome. Moreover, the D isomer of lactate may have adverse immunoinflammatory properties. One laboratory study found that NS and LR were equivalent in the setting of moderate hemorrhagic shock but that in the setting of massive hemorrhage, NS was associated with greater physiologic derangement (e.g. hyperchloremic acidosis) and a higher mortality. Clinical experience confirms the adverse effects of iatrogenic hyperchloremic acidosis. In addition, the potential benefits of using hypertonic saline (HTS) – rapid blood pressure response, decrease in ICP and improved immunologic status – for resuscitation are unproven but currently in clinical trials.

Blood Transfusion to Maintain Hemoglobin Concentration at 10 g/dL

The optimal [Hb] continues to be a subject of intense debate. Early laboratory studies of shock resuscitation suggested that survival was improved when [Hb] was maintained in the range of 12 to 13 g/dL. Subsequent studies using isovolemic hemodilution models indicated that the optimal [Hb] for maintaining oxygen delivery was 10 g/dL, and, until relatively recently, this value was the recommended level for critically ill patients. Currently, there is a growing recognition that administration of stored packed red blood cells (PRBC) can adversely affect outcome by modulating the inflammatory response (by both amplifying early proinflammation and aggravating late immunosuppression) and by impairing tissue perfusion (limiting access to or obstructing the microcirculation as a consequence of decreased RBC deformability). A 1999 randomized trial found that patients who received transfusions according to a restrictive policy (i.e. transfusion when the [Hb] fell below 7 g/dL) did as well as, and possibly better than, patients who received transfusions on a more liberal basis (i.e. transfusion when [Hb] fell below 10 g/dL). However, this study was done in a select group of euvolemic patients in which those with active hemorrhage were ex-
included; thus, it is not applicable to severely injured trauma patients requiring active shock resuscitation, but is cogent as their clinical course progresses. (See SOP for blood transfusions.) In addition, if blood transfusions are to be restricted during active resuscitation, it is not clear which alternative fluids should be used. Colloid solutions have been associated with complications and indiscriminant use of crystalloid fluid is detrimental. Hypertonic saline and hemoglobin based oxygen carriers are attractive alternatives, but additional clinical trials are needed before these could become standard of care. Additionally, maintaining higher [Hb] during active bleeding may facilitate coagulation. Many patients who are resuscitated by this process will also require a massive transfusion (i.e. >10 units PRBC in 24 hour), and are at risk for developing a coagulopathy. This subset of patients may benefit from early fresh frozen plasma (FFP) administration and this should be factored into their volume loading regimen.

Early Central Venous Pressure Monitoring

The most likely etiology of shock following major trauma is hypovolemia secondary to acute blood loss. Therefore, initial volume loading with isotonic crystalloids (1 L boluses in adults and 20 cc/kg in children) is recommended. The response to this empiric volume loading assists in early triage decisions. Prompt correction of abnormal vital signs indicates that a lesser volume deficit (10–20% blood volume) was present and that an expedited trauma evaluation can be safely performed to rule out occult bleeding. Patients who do not respond to empiric volume loading may have severe hypovolemia (30–40% blood volume), cardiogenic shock or neurogenic shock. Given that neurogenic shock is usually well tolerated and typically responds to initial volume loading, the key issue is to quickly distinguish hypovolemic shock from cardiogenic shock. Placement of a catheter that permits reliable measurement of central venous pressure (CVP) following the initial boluses can help differentiate these states. A high CVP (>15 mm Hg) suggests cardiogenic shock (likely etiologies include tension pneumothorax, pericardial tamponade or myocardial contusion/infarction). A low CVP (<5 mm Hg) may indicate acute ongoing blood loss, and mandates focus on identifying occult sources of blood loss. In many instances control of hemorrhage requires operating room (OR) or interventional radiologic (IR) interventions. Patients who initially respond to volume loading, but require ongoing crystalloid volume and/or blood transfusion during expedited trauma evaluation, or who have evidence of severe shock by ABG (i.e. BD ≥ 6 mEq/L), should have a central venous line placed and have continuous CVP measurements displayed to assist with ongoing resuscitation until the patients arrives in the intensive care unit (ICU). In the ICU, with more intensive monitoring available, the decision needs to be made if escalation to pulmonary artery (PA) catheterization is warranted.

Pulmonary Artery Catheterization in the ICU

The primary goal of shock resuscitation is the early establishment of “adequate” oxygen delivery (DO2) to vital organs. The yet to be resolved controversy is what is “adequate.” The calculated variable DO2 is the product of cardiac output (CO) and arterial oxygen content (CaO2). By convention, CO is indexed to body surface area and expressed as cardiac index (CI), and when multiplied by CaO2 yields an oxygen delivery index (DO2I). Normal DO2I is roughly 450 mL/min/m². CaO2 and DO2I are calculated as follows:

\[ \text{CaO}_2 = [\text{Hb}] (g/dL) \times 1.38 \text{ mL O}_2/\text{g Hb} \times \text{SaO}_2(\%) + [\text{PaO}_2(\text{mmHg})] \times 0.003 \text{ mL O}_2/\text{mmHg} \]

\[ \text{DO2I} = (\text{mL/min/m}^2) \times \text{CaO}_2 (\text{mL/dL}) \times 10 \text{ dL/L} \]

where \([\text{Hb}]\) is hemoglobin concentration, \(\text{SaO}_2\) is hemoglobin O2 saturation, \(\text{PaO}_2\) is arterial oxygen tension, and 0.003 is solubility of O2 in blood. Thus, there are four variables (i.e. \(\text{PaO}_2, \text{SaO}_2, [\text{Hb}], \text{ and CI}\)) that determine \(\text{DO2I}\). Of the four variables CI is the most difficult to monitor and manipulate. This is the rationale for liberal use of the PA catheter in severely injured patients. Myocardial dysfunction during traumatic shock resuscitation is common, but usually responds to volume loading. However, once a patient has been volume loaded (CVP >15 mm Hg) and has evidence of ongoing shock (e.g. decreased MAP, increased BD or lactate levels), a PA catheter is warranted to better monitor cardiovascular function, especially filling pressures and CI. Once the PA catheter is placed, the key question is what CI is acceptable. Early work from Shoemaker et al. demonstrated that the “survivor” response to traumatic stress is to become hyperdynamic (CI > 4.5 L/min/m²) and consequently have supranormal DO2I (>600 mL/min/m²). Supranormal DO2I was, therefore, proposed to be the resuscitation goal. Subsequent prospective randomized controlled trials (RCTs), however, failed to demonstrate improved outcome with goal oriented resuscitation to supranormal \(\text{DO2I}\). In fact, several recent studies indicate that this strategy is harmful. Recent studies in which a normal \(\text{DO2I}\) goal of 500 mL/min/m² was used demonstrated that patients achieve similar hyperdynamic responses to standardized interventions, require less volume loading, and have better outcomes than patients resuscitated to a supranormal \(\text{DO2I}\) goal. We recommend using a CI ≥ 3.8 L/min/m² as the resuscitation goal for this resuscitation process guideline. During active resuscitation, most severely injured patients will have \(\text{SaO}_2 > 92\%\) and \([\text{Hb}] > 10 \text{ g/dL}\) and, therefore, \(\text{DO2I}\) will approach 500 mL/min/m². PA catheters capable of continuous monitoring of CO and mixed venous hemoglobin oxygen saturation (SmvO2) are now commonly available and should be utilized. These continuously monitored variables provide rapid feedback that is often necessary to guide effective, timely resuscitation interventions. This approach is based on expert opinion as there are no data to suggest that a PA catheter is either
harmful or beneficial in severely injured patients undergoing shock resuscitation.

Assessment of Pre-Load by PCWP and the use of the “Starling Curve” Intervention

The traditional variable used to assess volume status is the pulmonary capillary wedge pressure (PCWP). It is important to recognize that significant hypovolemia can exist despite reasonable PCWP. Peripheral vasoconstriction of less acutely essential organs (e.g., kidney, gut, muscle, and skin) results in blood volume shifts to maintain the central circulation and perfusion of more essential organs (e.g., heart and brain). With a low CI and a low PCWP (i.e., <10 mm Hg), volume loading should be undertaken. After the PCWP increases to ≥15 mm Hg, the benefits of increasing CI by the Frank Starling mechanism should be considered. If additional CI is needed then a stepwise incremental volume loading intervention is used to identify the optimal CI-PCWP “operating point.” Because the shape and position of the Frank Starling curve is dependent on left ventricular contractility, compliance and afterload, it is difficult to identify optimal or plateau PCWP during volume loading without frequent sequential measurements. In a recent study, one-third of high risk patients had persistently low CO, high systemic vascular resistance (SVR) and high BD despite volume loading to a PCWP ≥ 15 mm Hg. However, these patients did respond well to the “Starling curve” intervention, by increasing CO, decreasing SVR and decreasing BD. This observation is consistent with a recent RCT in which patients who did not respond to initial volume loading (to presumed euvolemia) were randomized to additional volume loading or to a vasodilating inotropic agent. Patients who received further volume loading were found to have a better resuscitation response than those treated with inotropic agents. This favorable response to additional pre-load must, however, be weighed against the risks of increasing hydrostatic pressure leading to increase pulmonary edema, especially in patients with leaky endothelium (e.g., pulmonary contusion). Increasing PCWP >25 mm Hg in attempts to create a “Starling curve” should not be done because of the potential of causing or worsening pulmonary edema.

Use of Vasoactive Agents in Non-Responders

The primary early problem in shock resuscitation is decreased preload with resulting decreased CO. In this setting, the normal “survivor” response is to become hyperdynamic with volume loading. However, as time and severity of shock increases, a complex pathophysiologic interaction evolves that limits CO. Important factors include relative hypovolemia, primary or secondary myocardial dysfunction and excessive peripheral vasoconstriction. With ineffective intervention, shock will ultimately progress into pathologic vasodilation which heralds irreversible shock. Unfortunately, in the later phases of shock, patients who do not respond to volume loading also do not consistently respond to vasoactive agents. Therefore, a PA catheter should be placed to more accurately characterize their hemodynamic profile and monitor their response to vasoactive agent administration. A specific agent with known physiologic actions should then be administered and titrated to a desired effect. (See Table 1.)

For the typical non responding patient (i.e., low CI, adequate PCWP and high SVR), a vasodilating inotrope such as dobutamine (beware of hypotension) or dopamine (beware of tachycardia) is recommended. Patients with a particularly high SVR may respond better to simple afterload reduction with nitropresside. However, additional volume loading may be needed to maintain an adequate PCWP and oxygenation may worsen due to loss of hypoxic pulmonary vasoconstriction (i.e., worsened V/Q mismatch). For patients with low CO and normal SVR, dopamine or lower dose norepinephrine are reasonable choices. For patients who have low SVR and are thus unable to maintain an adequate MAP (≥ 60 mm Hg), higher doses of norepinephrine should be used. It is also important to rule

Guidelines for Shock Resuscitation

| Table 1 Vasoactive Agents Commonly Used in Shock Resuscitation |
|------------------|------------------|------------------|------------------|------------------|
| **Agent** | **Dose Rate (μg/kg/min)** | **Physiologic Action** | **Intended Effects** | **Adverse Effects** |
| Dopamine | Low | < 5 | DA₁ receptor | selective vasodilation | ↑ HR |
| Medium | 5–15 | β₁ receptor | ↑ CI, ↑ MAP, ↑ PCWP | Arrhythmia |
| High | > 15 | α₁ receptor | ↑ MAP | |
| Dobutamine | 5–20 | α₁, α₂ receptors | ↑ CI, ↓ PCWP | ↑ HR, ↓ MAP, arrhythmia |
| Norepinephrine | 0.01–0.05 | α₁, β₁ receptors | ↑ CI | ↑ afterload |
| Nitroprusside | > 0.05 | α₁ receptor | ↑ MAP | Vasodilatation |
| Phenylephrine | 0.25–10 | NO donor | ↓ afterload, ↑ CI | ↓ MAP, ↓ PaO₂ thiocyanate toxicity |
| Vasopressin | 0.04 units/min | Vasopressin receptor | ↑ MAP | Vasodilatation |

α₁, post synaptic receptor on vascular smooth muscle; α₂, pre synaptic receptor; β₁, largely cardiac; β₂, largely smooth muscle receptor of vasculature and bronchial tree; DA₁, dopaminergic post synaptic receptors in renal, splanchnic, cerebral and coronary vessels; CO, cardiac output; MAP, mean arterial pressure; PCWP, pulmonary capillary wedge pressure; HR, heart rate
out relative adrenal insufficiency in patients requiring higher doses of norepinephrine. Additionally, low dose vasopressin (as replacement therapy) may reduce the need for norepinephrine in patients exhibiting impending irreversible shock. Of note, there are no data that demonstrate that use of a specific vasoactive agent in non-responding patients improves outcome. Several studies have demonstrated improved hemodynamic responses to specific agents, and extrapolate this response to an improved outcome.

**Protocol Summary**

**Initial Resuscitation**

Figure 1 depicts initial ED resuscitation and the following text includes explanatory annotations lettered A through F. Variables that drive decision making include SBP, HR, BD, [Hb], CVP and clinical judgment (ever present). Additionally, stopping ongoing active hemorrhage is paramount to the survival of these patients.

A. Major trauma patients arriving in shock (SBP < 90 mm Hg and/or HR > 130 bpm) are initially managed by using Advanced Trauma Life Support (ATLS). Routine monitoring includes frequent vital signs (minimum q 15 minutes), continuous ECG and pulse oximetry (SpO2) and core body temperature. A data flow sheet is necessary to trend physiologic indices, laboratory test results and fluid volume/blood transfusion administration. During the initial ED evaluation an ABG analysis should be obtained in all patients presenting in traumatic shock.

B. Major torso trauma patients who have evidence of shock (documented by early SBP < 90 mm Hg and/or a BD ≥ 6 mEq/L), and who require ongoing resuscitation, should have a central venous line (via subclavian or internal jugular vein) placed in ED. CVP measurements should be used to differentiate the type of shock and to assist with subsequent monitoring of shock resuscitation.

C. Early CVP > 15 cm mmHg (before extensive volume loading) suggests cardiogenic shock.

D. Differential diagnosis of cardiogenic shock following blunt trauma includes: 1) tension pneumothorax, 2) myocardial contusion/infarction, 3) pericardial tamponade (uncommon) and 4) air embolus (rare). Specific diagnosis and treatment is beyond the scope of this protocol. ATLS guidelines should be followed.

E. CVP < 10 mm Hg despite volume loading indicates persistent hypovolemia and this most likely reflects ongoing bleeding. The endpoint of initial resuscitation is controversial and the algorithm statement “resuscitate until stable” is intentionally vague (i.e. requires clinical judgment). The crux of the issue is whether it is preferable to administer fluids to restore DO2 to the vital organs (risking hemodilution and disruption of early hemostatic clots) or to withhold fluid resuscitation until control of hemorrhage (risking prolonged cellular shock to the extent that it becomes irreversible by the time hemorrhage control is accomplished). At present, the rationale compromise is hypotensive resuscitation (SBP > 90 mm Hg and HR < 130 bpm) with moderate volume loading until hemorrhage control is accomplished. This approach is becoming the standard of care for penetrating trauma victims. It is most likely safe for blunt torso trauma patients who do not have significant concomitant brain injuries that could be worsened by permissive hypotension.

F. LR boluses should continue and, when LR infusion exceeds 30 mL/kg, blood should be administered. Earlier empiric blood transfusion is indicated in patients (especially the elderly) who arrive in severe shock or who have injuries associated with significant bleeding (e.g. vertical
shear pelvic fracture or bilateral femur fracture.) Protocols for massive transfusion should be established with the blood bank to ensure prompt availability of blood products for patients arriving with ongoing life-threatening hemorrhage. Among the most devastating complications of massive blood and fluid administration is a coagulopathy. Stored blood is deficient in factors V and VIII and platelets. Timely administration of FFP and platelets will minimize risk of coagulopathy after massive transfusion. Presumptive factor replacement is usually not indicated in the early phase of resuscitation, but may be appropriate in patients with massive hemorrhage caused by significant intracavitary bleeding or an unstable pelvic fracture.24,25

ICU Resuscitation

Hemorrhage control is of paramount importance in the initial management of major torso trauma patients arriving in shock. It is assumed that this issue will have been addressed in the vast majority of patients by the time the patient is admitted to the ICU. The priorities in early ICU care are to: a) optimize resuscitation, b) correct hypothermia, coagulopathy and acidosis and c) monitor for ongoing bleeding requiring OR or IR intervention. Figure 2 depicts ICU resuscitation and the following text includes explanatory annotations lettered A through H. An important early decision is whether to escalate monitoring interventions (i.e. placement of arterial and PA catheters). Variables that drive decisions in the PA catheter algorithm include CI, [Hb], PCWP, BD and clinical judgment.

A. When the patient arrives in the ICU, the physician needs to decide whether to continue resuscitation using serial vital signs, CVP, [Hb] and BD determinations (i.e. CVP Algorithm.) Most patients can be managed using this process, but close observation by a physician at bedside is required because CVP is a very indirect monitor of hemodynamic function.

B. For patients who are not responding to ongoing volume loading/blood transfusion (i.e. low MAP or persistently high BD) and/or are demonstrating secondary organ dysfunctions (i.e. worsening oxygenation or decreased urine output), pulmonary artery catheterization is warranted. The possibility of an impending abdominal compartment syndrome needs to be considered if crystalloid fluid volume loading exceeds 10 L or PRBC transfusion exceeds 10 units. Periodic urinary bladder pressure measurements should be obtained to monitor for onset of abdominal compartment syndrome.9,14

Fig. 2. ICU resuscitation.
C. It is assumed that most patients being treated by this algorithm will be intubated. If not intubated and requiring ongoing volume loading, intubation needs to be considered, because worsening pulmonary function is likely. If intubated and PEEP ≥ 12 cm H₂O, the effects of high mean airway pressures on cardiac function needs to be considered, as does use of PA catheter.

D. If the patient meets the CI goal of 3.8 L/min/m², then the patient should be monitored as depicted in this arm of algorithm. Hemodynamic variables should be assessed hourly, and possibly more frequently. Laboratory variables including [Hb] and ABG (BD) should be determined every 4 hours, and possibly more frequently until the patient is fully resuscitated and stable. Coagulation variables and urinary bladder pressure measurements should be monitored as deemed necessary.

E. Most young patients easily exceed the CI goal of 3.8 L/min/m² with modest crystalloid volume loading and blood transfusion. There is no need to increase PCWP to high levels in responding patients, but [Hb] should be maintained ≥ 10 g/dL during acute resuscitation to assure a safety margin in the event of occult or recurring bleeding. However, once the BD has been normalized and the need for ongoing volume loading has resolved, a lower [Hb] is acceptable. (See SOP for transfusion.)

F. PCWP may not accurately reflect left ventricular end diastolic volume and increasing PCWP to ≥ 15 mm Hg may enhance cardiac performance. The optimal relationship between PCWP and CI can be determined by incrementally increasing left ventricular preload (PCWP) and then measuring cardiac output (CI) in response, i.e. by generating a “Starling curve” to determine the optimal CI-PCWP operating point. This should only be done in patients who are not meeting the CI goal and who have evidence of ongoing shock (i.e. persistently elevated BD.)

G. After obtaining optimal PCWP, if CI < 3.8 L/min/m², then infusion of a vasodilating inotropic agent should be started. Dobutamine is recommended as the preferred inotropic agent. Dobutamine infusion should be started at a dose rate of 5.0 µg/kg/min and increased in increments of 2.5 µg/kg/min to a maximum of 20 µg/kg/min to increase CI. If the patient does not tolerate the vasodilation, dopamine should be considered with progression from low to mid- to high dose, while monitoring for excessive tachycardia.

H. Occasionally, an inotropic agent with vasoconstrictive effects may be needed to maintain MAP ≥ 60 mm Hg, enhance myocardial contractility and maintain coronary perfusion pressure. These agents decrease peripheral perfusion at the microcirculatory level by α₁ vasoconstriction of metarterioles thereby prolonging or exacerbating the effects of shock, and are therefore an intervention of last resort. Norepinephrine is recommended as the preferred agent. Norepinephrine infusion should be started at 0.05 µg/kg/min and increased in increments of 0.05 µg/kg/min as needed to obtain CI ≥ 3.8 L/min/m², and maintain MAP ≥ 60 mm Hg. The maximum recommended norepinephrine dose rate is 0.2 µg/kg/min. Adrenal insufficiency in patients requiring norepinephrine to maintain MAP needs to be ruled out. Low dose vasopressin may decrease the required dose of norepinephrine.

REFERENCES


