# Chapter 32

# DIAGNOSIS AND MANAGEMENT OF HYPOTENSION AND SHOCK IN THE INTENSIVE CARE UNIT

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## INTRODUCTION

Shock is a state of impaired tissue oxygenation and perfusion that can be caused by decreased oxygen delivery, poor tissue perfusion, or impaired oxygen utilization. Hypotension is a sign of shock and an indicator of advanced derangement, requiring immediate evaluation and management. For example, in hemorrhagic shock, hypotension is not present until greater than 30% of blood volume has been lost. Although hypotension and shock are not synonymous, the goals of treatment are the same: to restore the body's oxygen balance and correct hypoperfusion. This chapter will address the categories of shock, initial evaluation of a hypotensive patient, general principles of shock management, and management for specific causes of shock.

## PATHOPHYSIOLOGY AND CLINICAL PRESENTATION

Shock represents a state of hypoperfusion that can be the final pathway for a number of conditions. Hypoperfusion from any cause results in an inflammatory response. A normal physiologic compensation to improve perfusion of vital organs is sympathetic vasoconstriction resulting in an elevated diastolic pressure, narrow pulse pressure, and peripheral hypothermia. There is also a sympathetically mediated tachycardia that helps maintain cardiac output. Hypoperfusion also causes an acidosis induced by lactate production and resulting in compensatory tachypnea as the body attempts to offset the resulting acidosis. The other major effect of the acidosis is a rightward shift of the oxyhemoglobin curve,[1] allowing more of the oxygen that is bound to hemoglobin to be released. Additionally, there is increased shunting of blood to the most vital of organs—the heart and the brain—because of the opening of arteriovenous connections to bypass capillary flow.[2]

As these compensatory mechanisms begin to fail, the clinical signs and symptoms of shock become evident. The most commonly discussed signs of shock are hypotension, altered mental status, and oligura, but dysfunction of any end organ can result. Laboratory abnormalities include lactic acidosis, elevated base deficit, hypoxia, elevated blood urea nitrogen and creatinine, elevated liver-associated enzymes and bilirubin, and coagulation abnormalities. Lactic acidosis and base deficit are more sensitive indicators of severity and prognosis than are blood pressure and urine output (these will be covered in greater detail later in this chapter).[3,4]

## CATEGORIES OF SHOCK

It is helpful to place shock in one of the following four distinct categories: (1) hypovolemic, (2) cardiogenic, (3) distributive, and (4) obstructive. Hypovolemic shock can result from hemorrhage or other forms of intravascular fluid loss such as capillary leak, gastrointestinal losses, or renal losses. Its hemodynamic profile is significant for increased heart rate (HR), decreased cardiac output (CO), increased systemic vascular resistance (SVR), decreased cardiac filling pressures, decreased pulse pressures (PPs), and decreased central venous oxygen saturation (ScvO2). Simply stated, the circulatory system cannot maintain adequate blood flow and the body is compensating by increasing HR in an effort to increase CO and SVR to maintain perfusion. On physical exam, one would expect to see pallor and flat neck veins.

Cardiogenic shock is most often caused by a myocardial infarction, but it can have other causes such as myocardial contusion. Like hypovolemic shock, its hemodynamic profile shows increased HR, decreased CO, increased SVR, and decreased ScvO2. It differs, however, in that cardiac-filling pressures, central venous pressure (CVP), and pulmonary artery occlusion pressure are elevated in cardiogenic shock. In this state, the volume is available, but pump failure causes inadequate blood circulation. Physical exam is significant for distended neck veins, pulmonary edema, and a possible S3 gallop.

Distributive shock is often referred to as high output or hyperdynamic shock because, unlike the other forms of shock, the cardiac output is normal to elevated. The loss of vascular tone that defines distributive shock results in decreased SVR and an increased pulse pressure caused by decreased diastolic pressure. Many causes of distributive shock exist, including early septic shock, neurogenic shock, and anaphylactic shock.

Obstructive shock shares the hemodynamic profile of cardiogenic shock, and the two are often lumped together. The most significant difference between the two is the cause. Obstructive shock is caused by impaired cardiac filling as in cardiac tamponade, or excessive afterload as in a massive pulmonary embolus. Management lies in relieving the obstruction, which is often readily treatable if identified, but can be fatal if not detected.

One must be cognizant that the categorization of

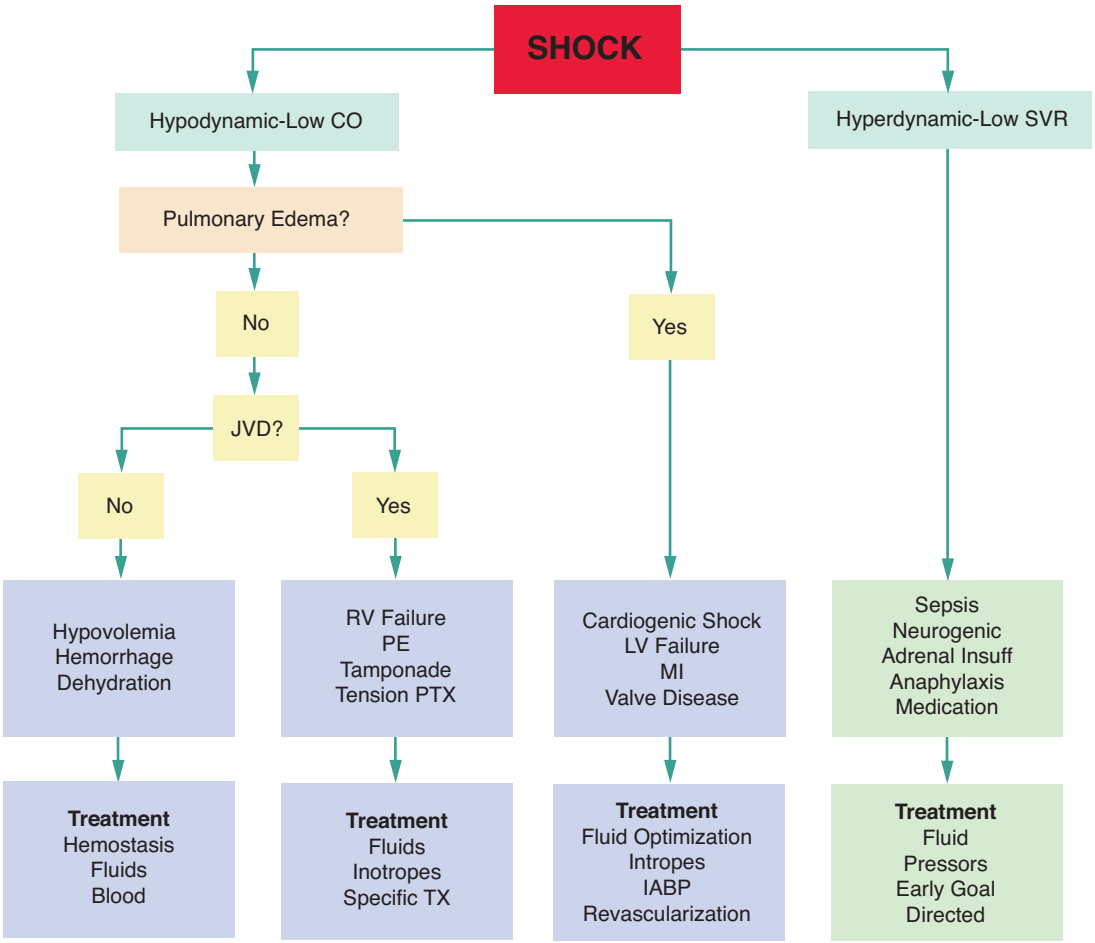


**Figure 32-1.** Diagnosing and treating various forms of hypotension.
CO: cardiac output; IABP: intra-aortic balloon pump; JVD: jugular vein distension; LV: left ventricular; MI: myocardial infarction; PE: pulmonary embolism; PTX: pneumothorax; RV: right ventricular; SVR: systemic vascular resistance; TX: therapeutics

shock is not always clear cut and overlap often occurs. For example, while septic shock is considered distributive shock, there is often a large component of hypovolemia present from third spacing of fluid. Alternatively, a thoracic trauma patient may suffer hemorrhage, causing hypovolemic shock, but may also have cardiac tamponade or a pneumothorax, resulting in obstructive shock. Additionally, shock is a dynamic state so the dominant component may change over time or with treatment. An overview of the causes and treatments of the various forms of shock can be seen in Figure 32-1.

## GENERAL DIAGNOSTIC APPROACH FOR HYPOTENSION

As with any medical illness, diagnosing the source of hypotension should begin with a history and physical examination. The importance of a thorough but focused physical exam must not be underestimated. Vital signs should be obtained. Airway, breathing, and circulation should be immediately assessed and the patient must be fully disrobed and inspected, front and back. Specific findings that may guide the investigation are vital signs, level of consciousness, appearance of neck veins, auscultation of cardiac and breath sounds, sources of external bleeding, assessment of possible internal sources of bleeding, and neurologic status.

Noninvasive vital signs are not adequate to determine the severity of illness or injury. Tachycardia, tachypnea, and hypotension are highly concerning findings, but they likely represent an advanced stage of disease. Consequently, invasive monitoring and laboratory evaluation should be obtained.[4] CVP and ScvO2

can assist with determining the type of shock. Arterial catheterization may be helpful in maintaining a more accurate blood pressure as well as in determining respiratory variation of pressures. There is no apparent benefit to using a pulmonary artery catheter.[5] Lactate and base deficit are important values to obtain.[3,4] They will not assist in determining the cause of hypotension, but they will aid in assessing severity and adequacy of resuscitation. Base deficit has been shown to correlate with greater fluid requirements, ongoing blood loss,[6] and mortality.[7] Lactate has been shown to correlate with the development of multiorgan failure.[8]

Although obtaining a thorough history is not a requirement when assessing a hypotensive critically ill patient, at a minimum one must be aware of allergies and medications. Hypotension can be caused by anaphylaxis or may result from narcotic or sedating medications. Additionally, withdrawal of a chronic medication, such as glucocorticoids, can cause hypotension. If possible, obtaining a thorough trauma history may allow for elucidation of occult injuries.

Radiography is very important in the critically ill trauma patient. Radiographic imaging of the C-spine, chest, and pelvis is generally obtained as part of the initial trauma evaluation, but should be considered in a hypotensive intensive care unit (ICU) patient. A chest radiograph could reveal a pneumothorax, suggest a hemothorax or pericardial effusion, or identify pneumonia in a septic patient. C-spine fractures raise concern about neurogenic shock, and a pelvic fracture may lead to investigation for intraperitoneal hemorrhage. Although these films may guide therapy, it is imperative that obtaining them does not delay any necessary treatment. For example, if a tension pneumothorax is suspected, immediate decompression should be performed without X-ray film confirmation.

The use of ultrasound (US) as a diagnostic tool has dramatically changed the evaluation of hypotension in trauma patients over the past two decades. It can be performed rapidly and repeated frequently without a risk of radiation to the patient. It has many benefits in the acute setting. For example, one can determine whether fluid exists around the heart, if there is impaired cardiac contractility after thoracic trauma, or whether free fluid exists in the abdomen after blunt abdominal trauma. In some cases, a diagnosis can be obtained almost instantaneously. For example, visualization of Morison's pouch can demonstrate free fluid in the abdomen and determine the need for surgery. It is currently taught in Advanced Trauma Life Support[9] and recommended by the Eastern Association for the Surgery of Trauma as the initial test to exclude hemoperitoneum.[10] Physical exam is often of limited value in critically ill trauma patients for many reasons, including medication effect, altered mental status, and distracting injuries. Therefore, to improve diagnostic accuracy, many trauma centers routinely include Focused Assessment Sonography for Trauma (FAST) as part of the physical exam.[11]

The FAST exam consists of four sonographic views to evaluate for pericardial and peritoneal free fluid: (1) pericardial, (2) perisplenic, (3) perihepatic, and (4) pelvic.[11] This exam is most helpful when free fluid is identified. A negative exam is less helpful because of a lower sensitivity. Therefore, the Eastern Association for the Surgery of Trauma guidelines recommend repeat exams and at least 6 hours of monitoring before accepting a negative exam.[10] Similarly, Advanced Trauma Life Support recommends a repeat exam in 30 minutes.[9] The pericardial view allows for identification of a pericardial effusion, but if there is concern for myocardial contusion, a formal echocardiogram should still be obtained.

An extended FAST (eFAST) exam, which includes evaluation of the pericolic gutters and the pleural space, can also be performed. Evaluation of the pleural space with US allows for identification of hemothoraces and pneumothoraces more rapidly than chest radiographs and also has a greater sensitivity.[11] Although a pneumothorax is more easily seen with US, it is more difficult to determine its size this way.[11] As with many traumatic injuries, pneumothoraces are dynamic conditions and repeat exams should be considered. It is also possible to use US to ensure drainage of a pneumothorax. Although not part of the eFAST exam, US can also be used to guide fluid management during resuscitation by measuring the size and collapsibility of the inferior vena cava.[12]

Limitations to the use of US include altered windows caused by obesity, subcutaneous air, or other injuries or dressings. Specific risk factors exist that increase the likelihood of a nondiagnostic US, existence of an injury missed by US, or requirement for a computerized tomography (CT) scan despite US findings.[11] These factors include persistent abdominal pain, seat belt sign, abdominal wall contusion, pulmonary contusion, hematuria, rib fractures, spine fractures, and pelvic fractures. Although false negative rates for screening US in patients with blunt abdominal trauma are low (1%), the risk increases to more than 6% for high-risk patients.[13,14] For a trauma patient with the risk factors listed above, a CT scan should be the initial diagnostic test unless the patient is too unstable for transport to a CT scanner.[15]

CT scans are the most definitive, highest fidelity, noninvasive test for the hypotensive trauma patient.[15] It is important to remember, however, that no unstable patient should go to the CT scanner. Other risks as-

sociated with CT scanning include contrast allergy, contrast nephropathy, and excessive radiation. Because of these risks as well as logistical concerns, CT scans cannot be repeated with the ease of US. Consequently, when scanning, one should consider whether the results would alter management and ensure that all areas of interest are scanned at once.

Diagnostic peritoneal lavage (DPL) was a common step in the evaluation of the hypotensive trauma patient before the evolution of US. DPL remains a reasonable option if US is unavailable, equivocal, or inconsistent with the clinical picture. However, DPL will alter future physical and radiographic exams and it cannot be repeated. Although there are no absolute contraindications to DPL, prior abdominal surgery, abdominal infections, coagulopathy, obesity, and second- or third-trimester pregnancy are all relative contraindications.[16]

## GENERAL PRINCIPLES OF MANAGEMENT OF THE HYPOTENSIVE INTENSIVE CARE UNIT PATIENT

The specific treatments for the various causes of shock may differ, but the overall goal in treating hypotension and shock is to restore oxygen balance and improve tissue perfusion. To do this, one must increase blood pressure, increase cardiac output, optimize oxygen delivery, and decrease oxygen demand. In general terms, fluids and vasopressors are used to increase blood pressure. Fluids should be the initial treatment, with vasopressors added only if the patient is unresponsive to fluids. The point at which vasopressors should be added differs based on the type of shock and will be addressed as such. Fluids and inotropes can be used to increase cardiac output. Oxygen delivery is further optimized by increasing hemoglobin and oxygen supply, and oxygen demand is decreased through the use of sedation, analgesia, and antipyretics. To assess progress, monitoring of arterial blood pressure, pulse oximetry, CVP, urinary output, acid base status, lactate, and base deficit are recommended.[4,17] The trends of the values obtained are often of more benefit than the baseline values.

Hemorrhage control and fluid resuscitation are the mainstays of the management of shock. If bleeding is the cause of shock, hemorrhage control is more important than resuscitation and surgical intervention should be pursued emergently. While awaiting surgery, fluid resuscitation is essential, but it should not delay surgery. Clarke et al showed a 1% increase in mortality for every 3 minutes of resuscitation prior to surgery.[18] A reasonable method to determine adequacy of hemorrhage control is to give 2 L of normal saline. If blood pressure improves, bleeding is likely controlled. If blood pressure improves only temporarily, there is ongoing blood loss. If there is no response, there is high volume blood loss. Transient responders and nonresponders require surgical intervention.[9] Following control of hemorrhage, the priority shifts to fluid resuscitation. Crystalloids and colloids are equally effective, although crystalloids are less expensive.[19]

Blood products must also be considered in the critically ill trauma patient. The goals should be to improve perfusion and oxygenation and decrease coagulopathy as opposed to targeting arbitrary laboratory values. A restrictive resuscitation standard, as discussed in the Transfusion Requirements in Critical Care trial,[20] does not apply to an actively bleeding patient. It is difficult to assess the exact amount of blood loss in trauma patients, so it is not often possible to directly replace lost blood with blood products. Furthermore, it is important to remember that a hematocrit is not an accurate measure of blood loss in an acutely bleeding patient because hemodilution has not yet occurred. Consequently, red blood cell transfusion is indicated in any patient with evidence of hemorrhagic shock.[21] After initial resuscitation and hemostasis, red blood cell transfusion should be considered for hemoglobin less than 7 g, and one unit should be given at a time.[21]

The end points of resuscitation are highly controversial. Over-resuscitation can lead to reversal of vasoconstriction of injured vessels, dislodging of clots, dilution of clotting factors, cooling of the patient, and swelling of visceral organs, possibly leading to abdominal compartment syndrome. It was previously thought that over-resuscitation would also increase intracranial pressure, but the amount of fluid given during resuscitation does not correlate with intracranial pressure.[20] Conversely, under-resuscitation risks poor cerebral perfusion and hypoxic brain injury.

No optimal algorithm for resuscitation exists. A mean arterial pressure of greater than 65 is often considered a goal, but this is highly debatable.[15,17] An individual's baseline blood pressure must be considered as well as the injury or illness. Another frequently used indicator is urine output, but if kidney injury exists, it may not be a viable option. More appropriate, sensitive, and specific indicators of perfusion are lactate and base deficit. The initial lactate level and the response of lactate to resuscitation correlate with multiorgan dysfunction and death.[3] Additionally, lactate has been shown to be noninferior to ScvO2 as a marker for resuscitation in septic shock.[22] Base deficit is also helpful in

the initial assessment of severity of illness or injury as well as progress over time. Base deficit changes over time are more predictive of survival than pH.[23] Base deficit has also been shown to correlate with risk of multiple organ dysfunction syndrome, development of acute respiratory distress syndrome, need for blood transfusion, development of renal failure, coagulopathy, and hospital length of stay.[24,25] Persistent elevation of either lactate or base deficit should prompt a search for an occult injury or the development of a complication such as abdominal compartment syndrome. A reasonable endpoint of resuscitation is normalization of lactate or base deficit. The role of vasopressors and inotropes varies with the type of shock, so these agents will be addressed more directly in the management of specific shock etiologies.

## MANAGEMENT OF SPECIFIC TYPES OF SHOCK

### Hypovolemic Shock

Shock in the trauma patient is considered hypovolemic until proven otherwise. The clinical presentation of the patient in hemorrhagic shock changes as the condition progresses. For the patient with less than 15% blood loss (approximately 750 mL), there will be little evidence of shock. As blood loss increases from 15% to 30%, the patient develops tachycardia, tachypnea, and anxiety. It is not until 30% of blood is lost that hypotension develops. At this point, anxiety has progressed to confusion. In the final stage of shock, more than 40% of blood volume has been lost and this condition is life threatening.[26] This development of hypotension is even more concerning in a young, previously healthy patient because he or she can often compensate until the point of hemodynamic collapse.

When the cause of blood loss is not externally apparent, one must consider four primary sites of massive internal bleeding: (1) long bone fractures (a femur fracture can bleed 2 to 3 units of blood into the thigh), (2) pleural cavities (each cavity can hold 2 to 3 L of fluid), (3) abdominopelvic cavity, and (4) the retroperitoneal space.[15] If bleeding is not the cause of the hypovolemia, gastrointestinal losses, urinary losses, third spacing of fluid, and dehydration must be considered.

The treatment for hypovolemic shock is to stop the volume loss and replace the fluid that has been lost. If it is hemorrhagic shock, hemostasis must be achieved, which may require short-term options such as a tourniquet or pelvic fixation, but surgical intervention may be necessary. Additional hemostatic agents are available, most commonly Quikclot powder and dressings (Z-Medica Corporation, Wallingford, CT) that use the inert mineral kaolin to clot blood.[27] Other developing treatments include recombinant factor VII, tranexamic acid, and red blood cell substitutes, but the roles of these agents are not clear at this time. The 2010 European guidelines, however, make weak recommendations to consider recombinant activated coagulation factor VII if major bleeding in blunt trauma persists despite standard attempts to control bleeding and best-practice use of blood components and that antifibrinolytic agents be considered in the bleeding trauma patient.[28] If hemorrhage is not the cause, other sources of volume loss or underlying disease processes must be controlled. Fluid replacement should resemble fluid lost. For massively bleeding patients, blood products must be delivered. High fresh frozen plasma to packed red blood cell and high platelet to packed red blood cell ratios have demonstrated improved survival.[29] Precise optimal ratios have not been well defined, but it appears that ratios greater than 1:2 are beneficial.[29] As discussed above in the general principles section, optimal resuscitation algorithms do not exist and gastrointestinal and third space losses are difficult to quantify. Consequently, resuscitating to a goal of normalizing lactate or base deficit remains a reasonable option.

### Cardiogenic Shock

Cardiogenic shock is caused by pump failure resulting in decreased forward flow and tissue hypoxia. In a nontrauma population, this can be caused by myocardial infarctions, cardiomyopathies, and arrhythmias. Cardiogenic shock from trauma can result from myocardial contusion, penetrating injury, or traumatic valve injury. The development of shock from blunt cardiac trauma is rare because blunt cardiac trauma is usually self-limited.[30] It should, however, be considered in patients with mechanisms of injury involving high speed frontal impact, particularly if any injury to the sternum or chest wall is noted. Furthermore, the stress response to trauma causes a catecholamine response, which increases HR, contractility, and myocardial oxygen demand. In the patient with underlying atherosclerosis, this may overwhelm the heart's limited blood flow and lead to cardiogenic shock even if there is no direct cardiac trauma.

If a myocardial contusion or valvular trauma is suspected, a formal transthoracic echocardiogram, or transesophageal echocardiogram if possible, should be obtained. Initial treatment of cardiogenic shock includes reperfusion, treatment of arrhythmias, and optimization of fluid and electrolyte status. Reperfu-

sion is available in a great many US hospitals, but is often not possible in more austere combat environments and may be contraindicated with anticoagulant and fibrinolytic drugs. Percutaneous intervention may not be available, and thrombolysis is contraindicated in a trauma patient with head or facial trauma within the past 3 months or with internal bleeding in the past 2 to 4 weeks.[31] If the patient's trauma was mild and no significant bleeding resulted, thrombolytics can still be considered, but a full risk-benefit analysis must be completed. Trials of fluid should be cautious and responses should be monitored closely. Inotropic support may be necessary. A patient's blood pressure may not tolerate the addition of a dobutamine alone because the drug causes vasodilation, so the addition of norepinephrine or dopamine is frequently required. More advanced treatments, such as balloon pumps or ventricular assist devices, may be necessary but are beyond the scope of this chapter.

## Distributive Shock

Many etiologies of distributive shock exist and the treatment for each cause differs. For example, toxins and medication overdoses can result in distributive shock. Although fluid resuscitation is important in this situation, specific antidotes for the toxin will be necessary. Specific toxicology will not be addressed in this chapter. This section will address the treatment of sepsis, anaphylaxis, neurogenic shock, and adrenal crisis.

### *Sepsis*

The term "sepsis" is often used to refer to a disease spectrum that ranges from systemic inflammatory response syndrome to septic shock. Systemic inflammatory response syndrome criteria include hyperthermia (> 38.3°C) or hypothermia (< 36°C), tachycardia (> 90 beats per minute), hyperventilation (respiratory rate >20 breaths per minute or partial pressure of carbon dioxide < 32) and leukocytosis (white blood cells > 12,000) or leucopenia (white blood cells < 4,000).[29] Sepsis is defined as the presence of two or more of these criteria with a source of infection. The diagnosis shifts to severe sepsis when organ dysfunction is evident. The final stage, septic shock, is diagnosed when refractory hypotension is present.[32]

Sepsis is rare in the immediate posttraumatic period. If the cause of hypotension does appear to result from sepsis in the acute setting, a diagnosis of bowel injury should be considered. As a patient's ICU course continues, sepsis becomes a more likely cause of hypotension. Trauma patients at high risk for sepsis include patients with a prolonged ICU stay, dirty wound (eg, dirt, bowel injury), devitalized tissue (eg, crush injuries), and wounds with a high risk of complication (eg, anastomotic leak, pancreatic leak).[30]

The Surviving Sepsis Campaign has created an algorithm for the treatment of sepsis that has changed care in many ICUs. Based on early goal-directed therapy (Figure 32-2), first published by Rivers, the most recent Surviving Sepsis guidelines were published in 2012.[17] The algorithm begins with fluid resuscitation with crystalloid or colloid to a goal CVP of 8 to 12 cm $H_2O$ (12–15 cm $H_2O$ if intubated). If a goal mean arterial pressure of greater than 65 cm $H_2O$ is not reached with fluid resuscitation, vasopressors should be initiated, with norepinephrine and dopamine being the first line agents of choice. Additional resuscitation goals are an ScvO2 greater than 70% and urine output greater than 0.5 mL/kg/h. If the ScvO2 goal is not reached, treatment options include further fluid resuscitation, red blood cell transfusion, or addition of inotropic support with dobutamine. If mean arterial pressure goals are not reached with fluid resuscitation to an adequate urine output and central venous pressure and vasopressor administration is required, 50 mg of hydrocortisone should be given every 6 hours. Adrenocorticotropic hormone (ACTH) stimulation test is not recommended.[14]

While resuscitation is underway, diagnosis and treatment must also be undertaken. Blood cultures should be obtained as well as cultures of other possible sources of infection (urine, cerebrospinal fluid, sputum).[14] If possible, cultures should be drawn prior to antibiotic administration but should not delay antibiotics. Imaging necessary to determine a diagnosis should also be obtained, but again, this should not delay antibiotic administration. Broad spectrum antibiotics (one or more agents directed against suspected organism with good penetration of likely sources) should be initiated within 1 hour once septic shock is suspected. Source control is the next step. All possible sources of infection should be evaluated and managed as necessary. Least invasive yet effective strategies should guide source control, and all potentially infected foreign objects and devices should be removed. Guidelines for management of blood products, mechanical ventilation, sedation, analgesia, glucose, renal replacement, bicarbonate, deep venous thrombosis prophylaxis, stress ulcer prophylaxis, and limiting support are also included but are beyond the scope of this chapter. They can be found at: www.survivingsepsis.org/guidelines.[14]

### *Anaphylaxis*

Anaphylaxis is a severe allergic reaction caused by degranulation of mast cells or basophils. This process

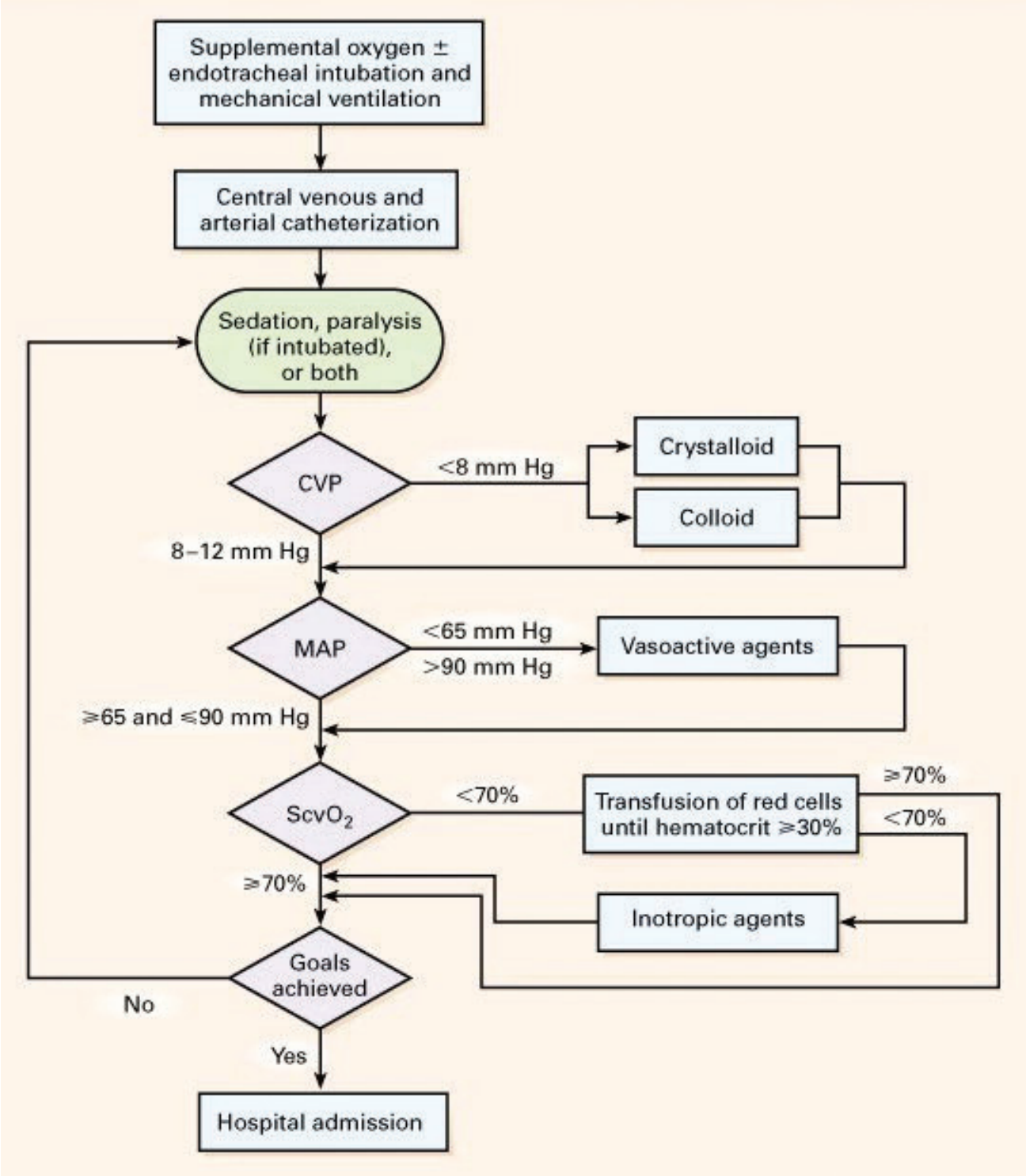


**Figure 32-2**. Early goal-directed therapy in septic shock.
CVP: central venous pressure
MAP: mean arterial pressure
ScvO2: central venous oxygen saturation
Reproduced with permission from: Rivers E, Nguyen B, Havstad S, et al. Early goal-directed therapy in the treatment of severe sepsis and septic shock. *N Engl J Med*. 2001;345:1368–1377.

is mediated by immunoglobulin E. Anaphylactoid reactions present similarly but are not mediated by immunoglobulin E. Common triggers include foods, insect stings, latex, and medications.

The mainstay of treatment for anaphylactic shock is epinephrine. Intramuscular injection (0.3 to 0.5 mg of 0.1% solution) can be used in mild or moderate cases. Slow, continuous intravenous (2 to 10 $\mu$g/min of 0.01% solution)[33] administration is recommended for patients with significant hypotension. Massive fluid shifts can occur with anaphylaxis, and aggressive administration of normal saline should accompany epinephrine. Antihistamines, glucocorticoids, and bronchodilators should also be administered.

### *Neurogenic Shock*

Neurogenic shock can be distinguished from other forms of distributive shock by the relative bradycardia that occurs from loss of sympathetic tone. Neurogenic shock can result from any spinal cord lesion above T6. Penetrating injuries are the most common, but development of a large hematoma with resultant cord compression can also be a cause. Symptoms include hypotension, bradycardia, flaccid paralysis, loss of deep tendon reflexes, and priapism. The goals of treatment are to protect the airway, improve vascular tone, and decrease the potential area of injury by maintaining spinal perfusion. As in other forms of shock, initial treatment is fluid resuscitation, but as hypovolemia is corrected, vasopressors will likely be necessary. Norepinephrine, dopamine, and phenylephrine are all reasonable options. Maintenance of mean arterial blood pressure at 85 to 90 mm Hg for the first 7 days after acute spinal cord injury to improve spinal cord perfusion is recommended.[34] Additionally, atropine may be necessary to combat bradycardia.

### *Adrenal Crisis*

Adrenal insufficiency in the critically ill patient can take many forms. It can be caused by a chronic disease process of the adrenals or of the hypothalamic-pituitary axis, or more acute causes such as medication withdrawal, critical illness, adrenal hemorrhage, hypoperfusion, or direct trauma. Either way, the resulting condition presents with nonspecific findings that can make diagnosis difficult. These findings include weakness, nausea, vomiting, abdominal pain, hypotension, fever, and hypoglycemia. The combined findings of hypotension, hyponatremia, and hyperkalemia should raise suspicion for adrenal crisis, which can be made by completing an ACTH stimulation test. The ACTH stimulation test will not be valid if the patient has received hydrocortisone, so if a patient requires emergent treatment and an ACTH stimulation test is desired later, dexamethasone should be used for steroid replacement. The first line steroid for the treatment of adrenal insufficiency, however, is hydrocortisone, 200 to 300 mg daily, in divided doses.[35] In addition to steroid administration, aggressive fluid resuscitation and determination and treatment of the cause are essential. In the setting of refractory septic shock, an ACTH stimulation test is not recommended, and treatment should be initiated with hydrocortisone at the same dose of 200 to 300 mg daily.[35]

## Obstructive Shock

Obstructive shock is the result of an anatomical impediment such as a pneumothorax, pulmonary embolism (PE), or pericardial effusion that causes decreased venous return, excessive afterload, and/or decreased cardiac filling. The treatments for each of these disorders will be addressed independently. Aggressive fluid resuscitation may be necessary to maintain the patient until the obstruction is relieved, but it is strictly a temporizing measure. It is important to note that obstructive shock is likely to significantly worsen with mechanical ventilation. The sedation associated with the intubation process contributes to the condition, but more importantly, the increased intrathoracic pressure that results from positive pressure ventilation can further decrease preload and ventricular filling and exacerbate the condition.

### *Pulmonary Embolism*

The classical findings of PE include dyspnea, pleuritic chest pain, and hemoptysis. In reality, the findings of PE are much less specific and range from dyspnea to cough to wheezing.[36] Patients may even be asymptomatic. Electrocardiogram may show an S wave in lead I, a Q wave in lead III, and T wave changes in lead III, which indicate right heart strain, but more commonly nonspecific ST changes, tachycardia, or a normal electrocardiogram are noted. Chest X-ray (CXR) may show a pleural-based, wedge-shaped defect, referred to as Hampton's hump, or paucity of vascular markings distal to the site of embolus, referred to as Westermark's sign, but the CXR is more likely to be normal. Given the nonspecific findings, it is important to maintain a high suspicion for PE, particularly in a trauma population. Numerous risk factors exist for PE and many of them are relevant to trauma patients. The trauma itself is a risk factor, but venous injury or repair, central venous catheterization, recent surgery, and immobility are also factors common to critically ill trauma patients.

### *Pneumothorax*

Pneumothoraces are the most common injury resulting after blunt thoracic trauma.[11] Patients at risk must be evaluated for equal bilateral breath sounds, equal chest excursion, jugular vein distension, and mediastinal shift. A CXR is a reasonable test when looking for a pneumothorax, but many pneumothoraces are not seen on a CXR and obtaining a CXR could lead to a delay in treatment. US may be a better diagnostic option given its improved sensitivity in trained providers. Blaivas et al showed 98% sensitivity for US compared to 76% for CXR.[37] Additionally, US can be rapidly performed at bedside. Chest CT is another diagnostic option. Regardless of the diagnostic tool used, if suspicion is high and the patient is unstable, the chest should be decompressed without delay for completion of diagnostic tests. In an emergent setting, needle decompression at the second intercostal space along the midclavicular line can be lifesaving. Definitive management with chest tube placement should follow this decompression. It is important to remember that pneumothoraces are dynamic. Repeat evaluation over time may be necessary. An initial negative test or small pneumothorax does not rule out the development of a tension pneumothorax one hour later.

### *Cardiac Tamponade*

Cardiac tamponade results from accumulation of fluid in the pericardial sac and is most commonly caused by penetrating trauma, but it can also result from blunt thoracic trauma.[15] Physical exam is significant for tachycardia, hypotension, muffled heart sounds, jugular vein distension, and elevated CVP. CXR may show a foreign body such as a bullet or other penetrating fragment or may demonstrate a waterbag heart. Electrocardiogram can range from normal to nonspecific ST changes to electrical alternans. The pericardial views obtained in the FAST exam allow for rapid bedside diagnosis of a pericardial effusion, but cardiac tamponade is a clinical diagnosis determined by hemodynamic compromise. Initial therapy consists of volume expansion to improve cardiac filling and cardiac output. This is only a temporizing measure. Definitive treatment is drainage of the pericardial fluid. This can be done by pericardiocentesis or surgery. Pericardiocentesis risks further injury and it may be difficult to drain any clotted blood. It may, however, be lifesaving in the acute setting. Surgical drainage is preferable in patients with potential intrapericardial bleeding or with clotted blood.[38] It allows for complete visualization, more complete drainage, and surgical correction of the source of bleeding. Surgical drainage may not be available, however, so pericardiocentesis—with or without US guidance—may be necessary to prevent hemodynamic collapse. In patients with cardiac tamponade, hemodynamic collapse can be precipitated by positive pressure ventilation (PPV). PPV should be avoided if at all possible, but at the very least, decompensation should be anticipated and optimization of fluid status should be achieved to ensure continued cardiac filling.

## SUMMARY

This chapter delineates the various possible causes of hypotension in the ICU and discusses the treatments by category. See Figure 32-2, which provides an overview of this discussion. Although treatments vary based on the cause of hypotension, fluid resuscitation can be lifesaving in all forms of shock. After the patient's airway and breathing have been assured and fluid resuscitation has been initiated, diagnostic tests can be completed to further guide treatment.

It is essential to be vigilant to the patient's physiologic changes over time because shock is a dynamic state. Additionally, one must remember that often more than one cause may be contributing to a patient's shock state. For example, trauma patients can suffer from hemorrhagic shock, neurogenic shock, and obstructive shock simultaneously. Therefore, physical assessment must be rigorous and frequent, and physiologic parameters must be monitored concurrently.

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