Shock Overview

Patrícia M. Veiga C. Mello, M.D.,[1,2] Vinay K. Sharma, M.D.,[3] and R. Phillip Dellinger, M.D.[4]

ABSTRACT

Despite improved understanding of the pathophysiology of shock and significant advances in technology, it remains a serious problem associated with high morbidity and mortality. Early treatment is essential but is hampered by the fact that signs and symptoms of shock appear only after the shock state is well established and the body's compensatory mechanisms have started to fail. Although the causes of shock are varied, the basic abnormality in all varieties is tissue and cellular dysoxia. In this overview we discuss the definition, classification and pathogenesis of shock in light of the recent advances in our understanding of its mechanisms. The epidemiology, diagnosis, and management of the various types of shock are also briefly discussed.

KEYWORDS: Shock, hypoperfusion, septic shock, cardiogenic shock, hypovolemic shock

Objectives: Upon completion of this article, the reader will: (1) appreciate divergent pathophysiology of different causes of shock; (2) understand the importance of intervention on systemic manifestations of shock; and (3) be familiar with an algorithmic approach to resuscitation of shock patients.

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Shock is likely the most serious diagnosis made in intensive care units worldwide. Its etiology is varied and complex and optimal resuscitation and intervention varies with etiology. Aggressive diagnostic and therapeutic interventions must occur simultaneously to avoid irreversible cellular injury and microcirculatory failure. Shock remains a major cause of mortality in any setting in which it appears and without the appropriate diagnostic and therapeutic approach it is almost invariably lethal. Despite significant technological advances in critical care medicine, the combination of delay in diagnosis and incomplete understanding of its intricate pathophysiology results in high mortality rates. Optimal management requires a multidisciplinary team, ideally led by an intensivist,[1,2] in a hospital setting with appropriate diagnostic and management capabilities.

HISTORICAL ASPECTS

Hippocrates described a "posttraumatic syndrome" long before *shock syndrome* was used as a medical term. The word *shock* is derived from the French word *choquer,*

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meaning "to collide with." The term *choc* was first used by a French surgeon, Le Dran, to indicate a severe impact or jolt,[3] but it was not until 1867 that the term became popularized when Edwin Morris published his *Practical Treatise on Shock after Operations and Injuries.*[4] He defined it as "a peculiar effect on the animal system, produced by violent injuries from any cause, or from violent mental emotions" calling attention to a body's response to injury for the first time as opposed to focusing on the immediate manifestations of trauma itself. By the late 1800s, Fisher suggested that a generalized "vasomotor paralysis" resulting in splanchnic blood pooling was the cause of shock,[5] and a few years later Maphoter, suggested extravascular leakage of fluids was the cause of the clinical findings seen in traumatic shock.[6]

In the 1900s Cannon, based on his battlefield experience during World War I, attributed the initiation of shock to more than mere blood loss with a disturbance of the nervous system causing relaxation of blood vessels and hypotension.[7] He proposed that a toxic factor was released during shock leading to altered capillary permeability and loss of blood volume from the intravascular space. In 1930, Alfred Blalock challenged Cannon's theory, arguing that blood loss would sufficiently explain the fall in cardiac output.[8] In the 1940s a cardiovascular physiologist, Carl Wiggers, published a series of studies demonstrating that a prolonged shock state could lead to irreversible circulatory failure.[9] Fluid resuscitation became the standard of care in the management of these patients. Hypotension had become not only the hallmark of shock but also the endpoint followed by most physicians while managing these patients.

For a long time shock was considered to occur only as a result of trauma. It was not until ~1898, during the Spanish American War, that sepsis was described to cause shock.[10] In 1906 Rosenau published his observations of a severe reaction occurring after a second injection of some foreign proteins (i.e., anaphylactic shock). In 1935 Tennant and Wiggers demonstrated an immediate drop in myocardial contraction when the heart was acutely deprived of coronary perfusion.[11]

DEFINITION

The definition of shock has continued to change considerably over the years. It can no longer be based on blood pressure alone. Assessment of perfusion independent of arterial pressure has clearly demonstrated that adequate blood pressure does not equal adequate cardiac output or tissue perfusion.[12,13] Seemingly adequate oxygen delivery (DO_2) also does not guarantee oxygen or substrate utilization at a cellular level. In sepsis, there is evidence suggesting that a cellular disturbance may impair oxygen and substrate utilization.[14,15] Cyanide or carbon monoxide intoxication leads to cellular cytotoxic hypoxia, despite the presence of adequate DO_2. Situations as may occur with sepsis and cyanide or carbon monoxide poisoning have led to the concept of "cytotoxic or cytopathic shock." In light of these new concepts, regardless of the mechanism by which it occurs, when cellular dysoxia occurs, a "shock state" is present, which ultimately leads to organ dysfunction and failure.

CLASSIFICATION

Shock has traditionally been classified into four categories: hypovolemic, distributive, cardiogenic, and obstructive shock. More appropriate is to classify shock into five categories to include cytotoxic shock (Table 1).

EPIDEMIOLOGY AND ETIOLOGY

The incidence and prevalence of shock are currently unknown. Several factors make it difficult to perform epidemiological analysis of this entity. Regardless of its etiology, patients may die before getting to the hospital. Furthermore, it is not a reportable diagnosis and there is still a lack of consensus regarding the definition of shock in general, and specific forms of shock. Not surprisingly there is great variability in reported shock incidence and mortality rates. Despite all these epidemiological difficulties, it is well known that all types of shock carry a very high mortality.

Cardiogenic shock is the number one cause of mortality from coronary artery disease in the United

Table 1 Classification of Shock and Its Most Common Etiologies

Hypovolemic	External and occult hemorrhages, skin losses (severe burns), third-spacing (pancreatitis, bowel obstruction, and prolonged abdominal surgery), gastrointestinal tract losses (vomiting, diarrhea), urinary tract losses
Cardiogenic	Acute myocardial infarction and its complications (e.g., acute mitral regurgitation, rupture of the interventricular septum, rupture of the free wall), myocarditis, end-stage cardiomyopathy, myocardial contusion, myocardial dysfunction after prolonged cardiopulmonary bypass, valvular heart disease, and hypertrophic obstructive cardiomyopathy
Obstructive	Cardiac tamponade, massive pulmonary embolism, tension pneumothorax, cor pulmonale, atrial myxoma, coarctation of aorta
Distributive	Septic shock, anaphylactic shock, neurogenic shock, adrenal crisis
Cytotoxic	Cyanide intoxication, carbon monoxide intoxication, iron intoxication

States.[16] Estimated incidence ranges between 6 and 8%,[17–20] and this rate has remained fairly stable from 1975 to 1997.[21] In the largest registry of patients with cardiogenic shock, 75% of patients had predominant left ventricular failure, 8% had acute mitral regurgitation, 5% had ventricular septal rupture, 3% had isolated right ventricular shock, 2% had tamponade or cardiac rupture, and 8% had shock resulting from other causes (such as myocarditis, end-stage cardiomyopathy, myocardial contusion, myocardial dysfunction after prolonged cardiopulmonary bypass, valvular heart disease, and hypertrophic obstructive cardiomyopathy).[22] Early reperfusion strategies have improved survival rates in recent studies but mortality remains high.[21] Several studies have reported lower rates of shock (4–7%) with the use of thrombolytics in myocardial infarction,[20,23–25] although no evidence has been found that this therapy is beneficial once shock has occurred.[17,25] Even lower mortality rates are reported with revascularization strategies.[26,27] Despite advanced supportive care in the management of heart failure and acute myocardial infarction, cardiogenic shock is still the most common cause of in-hospital mortality in transmural myocardial infarction, with overall mortality rates remaining between 70 and 90%.[22,18]

Accurate assessment of the incidence of septic shock is also difficult to ascertain. In a study by the Centers for Disease Control and Prevention, the incidence of sepsis in 1989 was 176 per 100,000.[28] Septic shock is reported as the thirteenth most frequent cause of mortality in the United States.[29] A recent meta-analysis found the mortality rate from septic shock to be $> 40\%$ in most studies analyzed.[30] The mortality rate from septic shock observed in the placebo arms of randomized controlled trials have decreased over time most probably due to advances in supportive care (Table 2). Once the predominant cause of sepsis, gram-negative bacteria now account for $\sim$38% of cases, whereas 52% are due to gram-positive bacteria.[31] There has also been a dramatic (207%) increase in fungi as a cause of sepsis.[31]

Hypovolemic shock remains a major cause of death in trauma patients but may also be seen as a complication of surgery and in patients with burns and gastrointestinal bleeding. Trauma patients may also have obstructive or neurogenic shock. Regardless of comorbidities or injuries, the shock state by itself will greatly affect these patients' prognosis and will be responsible for significant increases in morbidity and mortality.[32,33]

Table 2 Change in Septic Shock Mortality Rates over Time

Period	No. of Studies	No. of Patients	Hospital Mortality Rate (%)
1958–1969*	13	668	61
1970–1979*	17	1378	53
1980–1989*	39	2594	55
1990–1997*	62	6256	45
1997–1992	30	7874	39

*Adapted from Friedman et al.[30]

PATHOPHYSIOLOGY

Cardiogenic shock occurs when myocardial damage (acute or acute on chronic) reaches a point where pump function is markedly impaired. As one enters cardiogenic shock, stroke volume and cardiac output decrease, reducing myocardial perfusion, which, in turn, exacerbates ischemia and creates a downward spiral. Compensatory mechanisms that are activated by decreasing myocardial function eventually become maladaptive. Increased heart rate and increased afterload resulting from catecholamine release increase myocardial oxygen demand and worsen ischemia. Impaired diastolic filling due to tachycardia and ischemia, combined with the kidneys' attempt to increase preload by retaining fluid, result in pulmonary congestion and hypoxia.

Obstructive shock is characterized by inadequate ventricular filling due to cardiac compression or severe obstruction to ventricular inflow or outflow. In cardiac tamponade inadequate heart filling leads to decreased cardiac output, decreased blood pressure, reflex vasoconstriction, and elevated intracardiac pressures despite inadequate filling. Massive pulmonary embolism leads to obstruction of the pulmonary vessels by clot and release of vasoconstrictive mediators. Elevation of right-sided pressures with a normal pulmonary artery occlusion pressure and low cardiac output reflects right ventricular failure due to increased pulmonary resistance.

Hypovolemic shock is characterized by loss of circulating volume. Hypovolemia, tissue injury, and pain result in an increase in sympathetic drive in an attempt to raise blood pressure by increasing heart rate, cardiac contractility, and peripheral vasomotor tone. Although initially beneficial, these adaptive measures can eventually be harmful because the hypermetabolic state induced by the sympathetic drive can make tissues more susceptible to local ischemia. Uneven peripheral vasoconstriction can result in maldistributive microcirculatory flow and tissue hypoxia. Compensatory mechanisms fail when volume loss is $> 25\%$. An important inflammatory component also occurs in severe hypovolemic shock.[34,35] Delays of just 2 hours in appropriate resuscitation from volume losses exceeding 40% may result in inability to effectively correct tissue hypoperfusion.[36] Despite adequate control of volume loss, the patient may die as a consequence of the systemic activation of the inflammatory cascade triggered by the initial insult that can be further aggravated by reperfusion injury phenomenon.[37,38]

The characteristic feature of distributive shock is a decline in peripheral vascular resistance. Septic shock is

the classic example, but several other conditions can lead to a similar hemodynamic profile. Trauma to the spinal cord may lead to neurogenic shock that is characterized by an autonomic dysfunction with loss of peripheral vascular tone with a relative hypovolemic state and severe hypotension. Bradycardia may also be present and further impair cardiac output. In anaphylactic shock, severe immunoglobulin E (IgE)-mediated immediate hypersensitivity leads to massive release of mediators from mast cells and basophiles (especially histamine) resulting in decreased vascular resistance, capillary leak, and impaired contractility. Adrenal crisis is another form of distributive shock, which, when volume resuscitated, evokes a hemodynamic profile similar to septic shock.

Tumor necrosis factor alpha (TNF-α) and interleukin-1 (IL-1) are the dominant cytokines in septic shock.[39] Increased TNF-α levels are also seen in heart failure[40] and hemorrhagic shock.[34] TNF-α is produced by macrophages in response to microbial antigens and other cytokines. It results in the release of additional inflammatory mediators (IL-1β, IL-6, IL-8, thromboxanes, platelet-activating factor, and eicosanoids), which activate the coagulation and complement systems, depress myocardial contractility, and lead to vasodilation through inducible nitric oxide synthase activation.[41,42]

Nitric oxide is a key player in distributive shock where it serves multiple physiological roles, including neurotransmission, regulation of tissue perfusion via vascular tone and responsiveness, platelet responsiveness, renal volume control, and antimicrobial defense.[43,44] Nitric oxide is the major mediator of vasodilation and hypotension in septic shock[45,46] and may also be involved in the development of myocardial depression.[47] It has also been implicated in vascular dysfunction seen in hemorrhagic shock.[35]

Oxygen Metabolism

In general, constant oxygen consumption (VO_2) is maintained over a wide range of DO_2. At some critical point, oxygen extraction cannot increase any further, and reductions in DO_2 will result in a reduction in VO_2 (Fig. 1). This physiological oxygen supply dependency is primarily seen during low output circulatory shock. It was initially thought that a pathological oxygen supply dependency was present in patients with septic shock (i.e., the critical DO_2 point is increased and VO_2 is dependent on DO_2 over a wider range); however, this relationship is unlikely.[48–50]

In shock, decreased perfusion leads to limited oxidative metabolism resulting in lactic acidosis from anaerobic metabolism. The degree of lactate elevation correlates with both the degree of hypoperfusion and the mortality rate.[51] Regional hypoperfusion is indicated by decreased gastric intramucosal pH[52,53] and hepatic venous oxygen desaturation.[54] However, in most patients with septic shock, there also appears to be an inability of the tissues to extract oxygen from the blood.[15] Thus lactic acidosis may occur despite normal cardiac output and mixed venous oxygen saturation (SvO_2). In cardiogenic and hypovolemic shock, lactic acidosis occurs only after severe reduction in SvO_2.

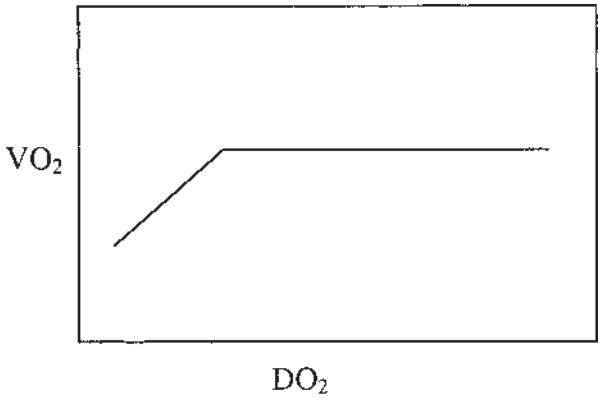

Figure 1 Relationship between oxygen consumption (VO_2) and oxygen delivery (DO_2).

DIAGNOSIS

The first step for successful outcome with a shock state is early recognition. It is important to keep in mind that diabetic, cirrhotic, neutropenic, and elderly patients may develop septic shock without a typical clinical picture or obvious source of infection. Basic evaluation should include metabolic panel, hemogram, arterial blood gas, electrocardiogram, and chest x-ray. It is important to remember that a drop in hemoglobin level occurs late in hemorrhagic shock and volume loss is best assessed by signs of hypoperfusion. The echocardiogram is increasingly being used in the assessment of patients in shock; it is noninvasive, can be performed at bedside, and can immediately reveal or exclude several potential etiologies of the shock state. Recent studies have suggested that procalcitonin level is a good marker of infection and may help differentiate septic from other shock states.[55,56]

Initial physical examination should focus on identifying signs of tissue hypoperfusion and on differentiating cardiogenic shock from other types of shock because initial volume resuscitation may be different in the former. If signs of fluid overload are absent, a possible source of volume loss or infection should be aggressively sought. No sign, symptom, or laboratory test by itself is diagnostic of shock, perhaps with the exception of profound hypotension. Shock is easy to diagnose when a patient arrives in the emergency department with multiple stab wounds, profuse bleeding, and immeasurable blood pressure. The problem is recognizing it in more subtle presentations. It is necessary to maintain a high index of suspicion and be alert to a group of nonspecific signs and symptoms that in the appropriate clinical context permits an early diagnosis of shock.

Table 3 Hemodynamic Profiles and Main Therapeutic Intervention in the Various Shock States

Hemodynamic Profiles of Shock	Cardiac Output	Preload	Afterload	Contractility	Intervention
Hypovolemic	↓	↓	↑	N	Crystalloid or colloid, blood
Cardiogenic	↓	↑	↑	↓	Inotropes, vasopressors
Septic					Fluids, vasopressors
Prior to fluids	↓ to N	↓	↓	↓	
After fluids	↑	N	↓	↓	
Pulmonary Embolism	↓	↓	↑	N	Thrombolytic therapy
Pericardial tamponade	↓	↓	↑	N	Pericardiocentesis
Anaphylactic					Fluids, inotropes, vasopressors
Prior to fluids	to N	↓	↓	↓	
After fluids	↑	N	↓	↓	
Adrenal					Fluids, steroids, inotropes, vasopressors
Prior to fluids	to N	↓	↓	N	
After fluids	↑	N	↓	N	

Hypotension is present in most shock states and will usually catch the attention of the physician, but unfortunately only occurs once the compensatory mechanisms are overwhelmed. In hypovolemic shock, tachycardia occurs after ~15% of the circulating volume is lost. However, despite being a sensitive sign of shock it is nonspecific, and it is important to be aware that this response may be blunted in patients who are on β-blockers or calcium channel blockers. Mottled skin and cold extremities, altered consciousness, thirst, concentrated urine, oliguria, and elevated creatinine may be present. In hemorrhage almost 30% of volume will be lost before the patient becomes hypotensive.[57] An earlier sign is narrowing of the pulse pressure due to catecholamine-stimulated elevation of diastolic blood pressure in response to the low circulating volume.[57] Furthermore, not only can shock occur in the absence of hypotension, it may persist even once hypotension has been reversed. Blood pressure may be maintained with vasopressors at the cost of worsening oxygen debt.

Measurement of lactic acid is a useful tool to assess severity and follow adequacy of therapeutic maneuvers,[58,59] but lactic acid changes may not occur early enough to be a sentinel marker for shock. At least in sepsis, elevated lactic acid levels have been shown to occur with normal intracellular oxygenation.[60] Furthermore, elevated lactic acid levels may occur due to comorbid conditions, especially liver failure because it is cleared by the liver. However, in a recent post hoc analysis of early, goal-directed therapy in septic patients with lactic acidosis (> 4 mmol/L) and mean arterial pressure above 100 mm Hg, patients in the protocol group had significantly lower mortality than those in the standard therapy group.[61]

Measurement of cardiac output and SvO_2, as well as calculation of DO_2, VO_2, and oxygen extraction ratios can be achieved with a pulmonary artery (PA) catheter. Despite the controversies regarding its use,[62,63] the catheter is widely used, and much of its reported negative effect on outcome may be the result of poor understanding and improper utilization of data.[64] In addition to ascertaining filling pressures, flow, and oxygen indices, specific pathological diagnoses linked to shock may be made (Table 3). Elevated right-sided and low PA occlusion pressure in the setting of acute inferior myocardial infarction should raise the suspicion of right ventricular infarct.[65] Central venous oxygen saturation ($ScvO_2$) is easier to obtain than pulmonary artery mixed venous saturation and may potentially be a good surrogate for SvO_2 in septic shock.[66]

Hypoperfusion is the hallmark of shock. Assessment of oxygen transport parameters is the best way of determining the presence of global tissue hypoperfusion. However, regional tissue hypoperfusion may be present despite normal values of oxygen transport variables, base deficit, and lactic acid levels. Gastric tonometry has been shown to predict mortality and may help determine splanchnic perfusion and guide resuscitation.[67,68] However, the difficulty of technique and interpretation of the data, and the increased cost associated with gastric tonometry, have limited its clinical applicability.

Cardiogenic shock may present with signs of increased central venous pressure, pulmonary edema, third heart sound, and peripheral vasoconstriction; although pulmonary edema may be absent in right ventricular infarct. Arrhythmia and mitral regurgitation murmur may also be present. Echocardiography helps evaluate systolic function and can reveal papillary muscle rupture, mitral regurgitation, ventricular septal defects, and free-wall rupture.[69]

Kussmaul's sign, pulsus paradoxus, distant heart sounds on auscultation, and decreased voltage on electrocardiogram may be present in cardiac tamponade. Equalization of pressures is diagnostic of this condition with mean right atrial, right ventricular end diastolic, and PA occlusion pressures within 5 mm Hg of one

another. The central venous pressure tracing may show a rapid "*x*" descent and a blunted "*y*" descent, reflecting ventricular inflow obstruction. Echocardiogram reveals pericardial effusion, and may show ventricular septal deviation to the left and right ventricular collapse during systole.

Distributive shock, frequently referred as "warm shock," is characterized by peripheral vasodilation and a hyperdynamic cardiac status that prevails until later stages when myocardial depression ultimately leads to decreased cardiac output. Evidence of infection, presence of spinal trauma or a trigger for anaphylaxis will help differentiate the underlying etiology. Adrenal crisis may present with abdominal pain, nausea, vomiting, hypothermia, refractory hypotension, hyponatremia, and hyperkalemia.

MANAGEMENT

The approach to the patient with shock must be dynamic with diagnostic and therapeutic maneuvers occurring simultaneously, striving to avoid further injury, by improving tissue perfusion (Fig. 2). With the exception of cardiogenic shock, aggressive fluid resuscitation is

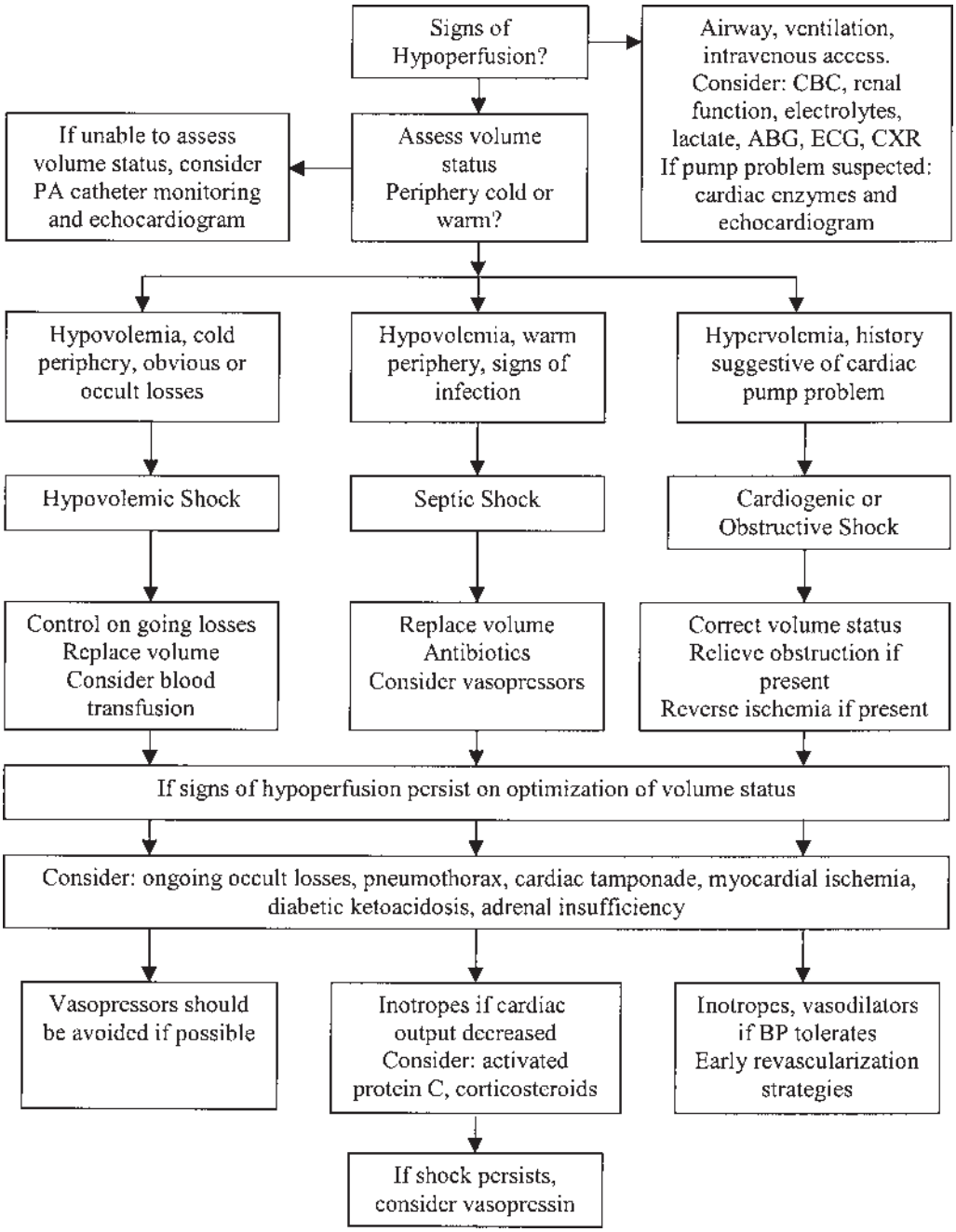

Figure 2 Diagnostic and management approach to shock.

usually required. However, cardiogenic shock due to right ventricular infarction also requires volume resuscitation. Fluids should be given until signs of hypoperfusion resolve or signs of volume overload appear. Constant reassessment is key and the effects of each therapeutic intervention on signs of tissue hypoperfusion should guide therapy. There has been no conclusive evidence favoring the use of either crystalloid or colloid solutions in volume resuscitation.[70] Blood transfusion should be instituted to maintain an adequate DO_2 and/or SvO_2 or when significant blood loss is apparent.[57] Endotracheal intubation and mechanical ventilation are often required.

Adequate assessment of intravascular volume status is a challenge. Occult hypovolemia due to extravascular loss of fluid is frequently underestimated and peripheral edema is often mistakenly used as a sign of intravascular volume overload. Studies have shown that neither physical examination[71,72] nor central venous pressure monitoring[73,74] is accurate in determining left ventricular volume status. For that purpose a PA catheter placement offers a more accurate assessment of intravascular volume status and tissue perfusion, better delineation of the hemodynamic profile, and dynamic observation of the effect of each therapeutic intervention (Table 3). It can help guide both fluid resuscitation and vasopressor or positive inotrope titration. However, use of a PA catheter is controversial given recent studies indicating no benefit or perhaps even harm from placement. The PA catheter has some limitations; cardiac output measured by thermodilution may be falsely increased (e.g., in severe tricuspid regurgitation or in intracardiac shunt). In such instances, cardiac output calculation using Fick's method may be used in addition to the PA catheter to more accurately estimate cardiac output and tissue perfusion. Noninvasive methods of estimating cardiac output include thoracic electrical bioimpedance[75] and pulse contour analysis.[76] However, the accuracy of these systems has been questioned.[77]

In cardiogenic shock, therapeutic measures include early revascularization strategies, emergency surgery, intraaortic balloon pump, and artificial ventricular support devices (LVAD [left ventricular assist device]/BiVAD [biventricular assist device]). Revascularization strategies have proven to be most beneficial if undertaken within the first few hours after the insult.[27,78,79]

Obstructive shock due to cardiac tamponade requires needle, catheter, or surgical drainage of the pericardial fluid. Fluid and vasoactive drugs may be required to support circulation while awaiting decompression. In shock due to pulmonary embolism, cardiac arrhythmias should be corrected, a fluid challenge given, and thrombolytic therapy considered. The role of vasoactive drugs is less clear.

In hypovolemic shock, volume resuscitation is indicated and vasopressors should be avoided if possible, though often administered during initial resuscitation. Several studies performed in the trauma population have shown worse outcome with aggressive volume resuscitation prior to bleeding control, probably due to disruption of the hemostatic plug.[80–82] However, delayed resuscitation may exacerbate the inflammatory component triggered by hypovolemic shock leading to multiple organ failure and death.[37,83] Therefore, it seems that judicious fluid resuscitation to maintain hemodynamic stability with avoidance of overload prior to bleeding control is prudent.

In septic shock, the initial treatment remains antibiotic therapy and source control. Appropriate antibiotic therapy improves outcome.[84,85] In addition, volume resuscitation is paramount. The fluid deficit in septic shock is often 6 L or more in the first 24 hours and vasopressor support should not be utilized in place of adequate intravascular volume. Traditionally, dopamine has been the vasopressor of choice.[86] However, one study has suggested that norepinephrine is more easily titrated to achieve hemodynamic goals and may be associated with better outcome.[87] A recent study has emphasized the importance of instituting aggressive therapy early and targeting normal ScvO2 saturation.[88] Recombinant human activated protein C has been clearly demonstrated to improve outcome in septic shock.[89] In septic shock patients with impaired adrenal responsiveness, hydrocortisone plus fluorocortisone may also improve survival.[90]

It has been suggested that maintaining a supranormal cardiac output in patients with shock should increase DO_2 and VO_2, decrease any oxygen debt present, and potentially improve survival. Randomized controlled trials have been performed to test this strategy but found that supranormal cardiac output and DO_2 did not lower mortality when used to reverse tissue hypoxia (as in septic patients).[91–96] However, when used in a prophylactic approach (i.e., before organ failure develops), there appears to be a survival advantage.[97–101]

When shock occurs due to more than one etiology, a mixture of signs may be present and the hemodynamic profile will not show the classic pattern of one particular type of shock, but a mixture of features. It is important to be able to recognize such a patient and adapt the management accordingly. The physical signs and hemodynamic profiles may also be altered by preexisting comorbidities. Cirrhotic and pregnant patients have a lower systemic vascular resistance and a hyperdynamic hemodynamic profile in the absence of a distributive shock. Pregnant patients have a higher circulating volume that may mask early signs of shock. Athletes have a higher circulating volume and cardiac output, and a lower resting heart rate. Thus signs of shock may also be delayed in these patients. Patients with heart block and a pacemaker may not be able to mount a tachycardic response.

CONCLUSIONS

Shock continues to result in substantial morbidity and mortality despite significant advances in technology and pathophysiological understanding. Initial priority is aimed at the general principles of resuscitation: assuring adequate airway and oxygenation, vascular access, and volume resuscitation. The goal of therapy is to restore adequate tissue perfusion. Manipulation of the blood oxygen content and cardiac ouput may improve tissue perfusion. Early diagnosis, aggressive resuscitation, and interruption or reversal of the insult (i.e., control of bleeding, myocardial revascularization strategies, infection control) is the optimal approach in managing the patient in shock.

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