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TABLE 313-4 Main Types and Key Features of Extracorporeal Gas Exchange TERM DESCRIPTION KEY FEATURES IMPORTANT TECHNICAL NOTES VA-ECMO (venoarterial extracorporeal membrane oxygenation) Deoxygenated blood drains via venous catheter to a pump and membrane oxygenator; blood is then returned to the arterial system VV-ECMO (venovenous-ECMO) Deoxygenated blood drains via venous catheter to a pump and membrane oxygenator; blood is then returned to the venous system ECCO2R (extracorporeal CO2 removal) Venous catheter drains blood to a CO2 removal device; blood then returns via a venous catheter is indicated for patients with hypercapnia after extubation, high-flow oxygen support for all other patients may be preferable given similar efficacy to NIV in preventing reintubation and generally better patient comfort. Although many factors can cause a patient to fail an SBT or require reintubation and continued mechanical ventilation, common processes perpetuating mechanical ventilation include critical ill ness myopathy and polyneuropathy, myocardial ischemia, congestive heart failure, vascular and extravascular volume overload, delirium, malnutrition, and electrolyte abnormalities (hypophosphatemia, hypo kalemia, and hypomagnesemia). These processes should be evaluated and treated, as necessary, in patients failing attempts to discontinue mechanical ventilation. EXTRACORPOREAL GAS EXCHANGE Despite interventions to optimize oxygenation and alveolar ventilation on mechanical ventilation, some patients suffer life-threatening hypox emia, refractory respiratory acidosis, and barotrauma, and may be candidates for salvage therapy with extracorporeal gas exchange, a pro cedure whereby blood continuously circulates outside the body through a device that oxygenates it, removes CO2, and then returns blood to the patient’s circulation. Although often referred to as ECMO, modern gas exchange membranes both deliver oxygen and remove CO2, replacing the gas exchange function of the lung. The main components of an ECMO “circuit” include vascular cannulas to remove and return blood to the patient, a pump to circulate blood, and a gas exchange mem brane. ECMO can provide varying levels of both respiratory and circu latory support depending on the clinical situation (Table 313-4). In a patient both in shock and requiring full respiratory support, the ECMO circuit would include a central venous cannula (V) to remove blood and a central arterial cannula (A) to return oxygenated blood at rela tively high flow rates (up to 6 L/min) providing mechanical circulatory support, so-called VA-ECMO. In the absence of shock, both the drain ing and return vascular cannulas can be central venous, or VV-ECMO, but blood flow is still relatively high (2–5 L/min) to provide adequate oxygen delivery to tissues. In situations where a patient’s lungs can pro vide adequate oxygenation but insufficient CO2 removal, such as severe obstructive lung disease exacerbations, a venovenous

circuit with low blood flows (0.25–2 L/min) is often adequate to remove CO2 and treat refractory respiratory acidosis, a process called extracorporeal CO2 removal (ECCO2R). ECMO continues to evolve, including the use of hybrid circuits. For example, if patients on traditional VA-ECMO have asymmetric hypoxia in the upper body, an additional venous return catheter can be placed in an internal jugular vein to deliver additional oxygenated blood; this hybrid circuit would be V (removal)-VA (dual arterial and venous return)-ECMO. Moreover, several ECMO centers now have mobile ECMO equipment and teams, allowing patients who are too unstable for transfer to an ECMO center to start on ECMO and facilitate transfer to an ECMO center for further care. Although technologic advances in the ECMO pumps, gas exchange membranes, and even vascular catheters have reduced ECMO-related complications, the procedure is resource-intensive and still associated with several adverse events, including cannula site hemorrhage and vascular injury, catheter-related infection, pneumothorax, pulmonary and gastrointestinal hemorrhage, limb ischemia, intracranial hemor rhage, and disseminated intravascular coagulation (DIC). Clinical outcomes for ECMO patients remain promising, including for patients

Circulatory and respiratory support Requires large vascular catheters (16–30 Fr) Higher blood flow rates (2–6 L/min) Respiratory support Requires large vascular catheters (20–30 Fr) Higher blood flow rates (2–5 L/min) Partial respiratory support, CO2 removal only Requires smaller vascular catheters (14–18 Fr) Lower blood flow rates (0.25–2 L/min) with severe respiratory failure from SARS-CoV-2 infection treated with ECMO. However, the overall mortality benefit from ECMO, especially in ARDS, remains unclear. Selecting patients most likely to benefit from ECMO, therefore, is very important, and in addition to exhausting traditional mechanical ventilatory support, patients being considered for ECMO should have a reversible underlying illness or be eligible for organ transplant (heart and/or lung), no chronic severe end-organ disease (e.g., severe kidney disease), no contraindication to systemic anticoagulation, a good functional status before the acute ill ness requiring ECMO, and a good neurologic prognosis. CHAPTER 314 Approach to the Patient with Shock ■ ■FURTHER READING Acute Respiratory Distress Syndrome Network et al: Ventilation with lower tidal volumes as compared with traditional tidal volumes for acute lung injury and the acute respiratory distress syndrome. N Engl J Med 342:1301, 2000. Barrot L et al: Liberal or conservative oxygen therapy for acute respi ratory distress syndrome. N Engl J Med 328:999, 2020. Bertini P et al: ECMO in COVID-19 patients: A systematic review and meta-analysis. J Cardiothorac Vasc Anesth 36:2700, 2022. Girard T et al: An official American Thoracic Society clinical practice guideline: Liberation from mechanical ventilation in critically ill adults. Rehabilitation protocols, ventilator liberation protocols, and cuff leak tests. Am J Respir Crit Care Med 195:120, 2017. Hernandez G et al: Effect of post extubation high-flow nasal can nula vs non-invasive ventilation on reintubation and post extubation respiratory failure in high-risk patients: A randomized clinical trial. JAMA 316:1565, 2016. Moss M et al: Early neuromuscular blockade in the acute respiratory distress syndrome. N Engl J Med 380:1997, 2019. Murphy PB et al: Effect of home noninvasive ventilation with oxygen therapy vs oxygen therapy alone on hospital readmission or death after an acute COPD exacerbation. A randomized clinical trial. JAMA 317:2177, 2017. Tramm R et al: Extracorporeal membrane oxygenation for critically ill adults. Cochrane Database Syst Rev 1:CD010381, 2015. Section 2 Shock and Cardiac Arrest Rebecca M. Baron, Anthony F. Massaro

Approach to the Patient

with Shock Shock is the clinical condition of organ dysfunction resulting from an imbalance between cellular oxygen supply and demand result ing in cellular and tissue hypoxia. This life-threatening condition is common reason for requiring care in the intensive care unit (ICU).

A multitude of heterogeneous disease processes can lead to shock. The organ dysfunction seen in early shock is often reversible with res toration of adequate oxygen supply. Left untreated, shock transitions from this potentially reversible phase to an irreversible phase and death from irreversible multisystem organ dysfunction. The clinician is required to identify the patient with shock promptly, make a pre liminary assessment of the type of shock present, and initiate therapy to prevent irreversible organ dysfunction and death. In this chapter, we review a commonly used classification system that organizes shock into four major types based on the underlying physiologic derangement. We discuss the initial assessment utilizing the history, physical examination, and initial diagnostic testing to confirm the presence of shock and determine the type of shock causing the organ dysfunction. Finally, we will discuss key principles of initial therapy with the aim of reducing the high morbidity and mortality associated with shock.

■ ■PATHOPHYSIOLOGY OF SHOCK The cellular oxygen imbalance of shock is most commonly related to impaired oxygen delivery in the setting of circulatory failure. Shock can also develop during states of increased oxygen consumption or impaired oxygen utilization. An example of impaired oxygen utiliza tion is cyanide poisoning, which causes uncoupling of oxidative phos phorylation. This chapter will focus on the approach to the patient with shock related to inadequate oxygen delivery. PART 8 Critical Care Medicine In the setting of insufficient oxygen supply, the cell is no longer able to support aerobic metabolism. With adequate oxygen, the cell metabolizes glucose to pyruvate, which then enters the mitochondria where ATP is generated via oxidative phosphorylation. Without suf ficient oxygen supply, the cell is forced into anaerobic metabolism, in which pyruvate is metabolized to lactate with much less ATP generation (per mole of glucose). Maintenance of the homeostatic environment of the cell is dependent on an adequate supply of ATP. ATP-dependent ion pumping systems, such as the Na+/K+ ATPase, consume 20–80% of the cell’s energy. Inadequate oxygen delivery and subsequent decreased ATP disrupt the cell’s ability to maintain osmotic, ionic, and intracellular pH homeostasis. Influx of calcium can lead to activation of calcium-dependent phospholipases and pro teases, causing cellular swelling and death. In addition to direct cell death, cellular hypoxia can cause damage at the organ system level via leakage of the intracellular contents into the extracellular space activating inflammatory cascades and altering the microvascular circulation. ■ ■DETERMINANTS OF OXYGEN DELIVERY Because shock is the clinical manifestation of inadequate oxygen deliv ery relative to cellular needs, we will review determinants of oxygen delivery (DO_2). Disease processes affecting any of the components of oxygen delivery have the potential to lead to the development of shock. Disturbances to key determinants of oxygen delivery form the basis of the four major shock types described below. The two major components of DO_2 are cardiac output (CO) and arterial oxygen content (CaO_2): $DO_2 = CO \times CaO_2$ The two components of CO are heart rate (HR) and stroke volume (SV), which can be substituted in the above equation as $DO_2 = (HR \times SV) \times CaO_2$ The major determinants of SV are preload, afterload (systemic vas cular resistance [SVR]), and cardiac contractility. The relationship can be represented as $SV \alpha (Preload \times Contractility)/SVR$ In this equation, preload refers to the myocardial fiber length before contraction (the ventricular end-diastolic volume). Contractility refers to the ability of the ventricle to contract independent of preload and afterload. The SVR represents the afterload, or the force against which the ventricle must contract.

The CaO2 is composed of oxygen carried by convection with hemo globin (arterial oxygen saturation, or SaO2) and oxygen dissolved in arterial blood (partial pressure of oxygen, or PaO2), given as CaO2 (in mL/dL) = (Hb × 1.34 × SaO2) + (PaO2 × 0.003) A disease process that affects these variables (HR, preload, con tractility, SVR, SaO2, or hemoglobin (Hb)) has the potential to reduce oxygen delivery and cause cellular hypoxia. Each of the shock types described below has a distinctive physiologic hemodynamic profile corresponding with alterations in one of the variables affecting oxygen delivery described above. ■ ■CLASSIFICATION OF SHOCK While there is a heterogeneous list of specific conditions that can cause shock, it is helpful to categorize these processes into four major shock types based on the primary physiologic derangement leading to reduced oxygen delivery and cellular hypoxia. The four major shock types are distributive, cardiogenic, hypovolemic, and obstructive. Table 314-1 outlines these major shock types, as well as specific disease processes that can result in that physiologic derangement. Each shock type has a distinct hemodynamic profile (Table 314-2). Familiarity with the major shock types and their unique hemodynamic profile is essential so that when evaluating a patient presenting with shock, the clinician can use the history, physical examination, and diagnostic testing to determine the type of shock present and promptly begin appropriate initial therapy to restore oxygen delivery. Distributive Shock Distributive shock is the condition of reduced oxygen delivery where the primary physiologic disturbance is a reduc tion in SVR. It is unique among the types of shock in that there is a compensatory increase in CO (Table 314-2). The central venous TABLE 314-1 Physiologic Classification of Shock Distributive Septic shock Pancreatitis Severe burns Anaphylactic shock Neurogenic shock Endocrine shock Adrenal crisis Cardiogenic Myocardial infarction Myocarditis Arrhythmia Valvular i. Severe aortic valve insufficiency ii. Severe mitral valve insufficiency Obstructive Tension pneumothorax Cardiac tamponade Constrictive pericarditis Pulmonary embolism Aortic dissection Hypovolemic Hemorrhagic i. Trauma ii. GI bleeding iii. Ruptured ectopic pregnancy GI losses Burns Polyuria i. Diabetic ketoacidosis ii. Diabetes insipidus Abbreviation: GI, gastrointestinal.

TABLE 314-2 Hemodynamic Characteristics of the Major Types

of Shock CARDIAC OUTPUT SYSTEMIC VASCULAR RESISTANCE TYPE OF SHOCK CVP PCWP Distributive ↓ ↓ ↑ ↓ Cardiogenic ↑ ↑ ↓ ↑ Obstructive ↑ ↓↑ ↓ ↑ Hypovolemic ↓ ↓ ↓ ↑ Abbreviations: CVP, central venous pressure; PCWP, pulmonary capillary wedge pressure. pressure (CVP) and pulmonary capillary wedge pressure (PCWP) are usually reduced. The most common cause of distributive shock is sepsis. Sepsis has been redefined as the dysregulated host response to infection resulting in life-threatening organ dysfunction. When this process is accompanied by persistent hypotension requiring vasopres sor support (despite adequate volume resuscitation) with end-organ hypoperfusion as measured by an elevated lactate level, it is classified as septic shock. Other processes that are manifest as cellular hypoxia related to a primary reduction of SVR include pancreatitis, severe burns, and liver failure. Anaphylaxis is predominantly an IgE-mediated allergic reaction that can rapidly develop after exposure to an allergen (e.g., food, medication, or insect bite), in which there is a profound dis tributive type of shock possibly mediated through histamine release. In this setting, there is evidence of both venous and arterial vasodilation. Studies have demonstrated extravasation of up to 35% of the circulat ing blood volume within 10 min. Patients with severe brain or spinal cord injury may have a reduction of SVR related to disruption of the autonomic pathways that regulate vascular tone. In these patients, there is pooling of blood in the venous system with a resulting decrease in venous return and decreased

CO. A final category of patients who pres ent with distributive shock consists of those with adrenal insufficiency. Adrenal insufficiency may be related to chronic steroid use, medi cations (immune checkpoint inhibitor–associated primary adrenal insufficiency), or other processes that might affect the adrenal glands, including metastatic malignancy, adrenal hemorrhage, infection (e.g., tuberculosis, HIV), autoimmune adrenalitis, or amyloidosis. In con ditions of stress (such as infection or surgery), the deficit in adrenal function may become apparent with an inability to increase cortisol leading to vasodilation as well as aldosterone deficiency–mediated hypovolemia. Cardiogenic Shock Cardiogenic shock is characterized by reduced oxygen delivery related to a reduction in CO owing to a primary car diac problem. There is usually a compensatory increase in SVR in cardiogenic shock. When the cardiac process (e.g., myocardial infarc tion) affects the left ventricle (LV), there will be elevation of the PCWP and when it affects the right ventricle (RV), the CVP will be elevated. As detailed above, the CO (and accordingly the DO2) can be reduced by alterations in the SV or HR. In cardiogenic shock, the SV may be reduced by processes that affect myocardial contractility (e.g., myocar dial infarction, ischemic cardiomyopathies, and primary myocarditis) or mechanical valvular disease (acute mitral insufficiency or aortic insufficiency). Both bradyarrhythmias and tachyarrhythmias (from either an atrial or ventricular source) may have associated hemody namic consequences with a reduction in CO. Hypovolemic Shock Hypovolemic shock encompasses disease processes that reduce CO (and oxygen delivery) via a reduction in preload. In addition to the reduced CO, this shock type is character ized by an elevated SVR and low CVP and PCWP related to decreased intravascular volume. Any process causing a reduction in intravascular volume can cause shock of this type. Hypovolemic shock is most com monly related to hemorrhage, which may be external (secondary to trauma) or internal (most commonly upper or lower gastrointestinal [GI]) bleeding). Hypovolemic shock can also be seen with nonhem orrhagic processes. Examples include GI illnesses causing profound emesis or diarrhea, renal losses (e.g., osmotic diuresis associated with

diabetic ketoacidosis or diabetes insipidus), or skin loss (e.g., severe burns, inflammatory conditions such as Stevens-Johnson syndrome).

Obstructive Shock Obstructive shock is also characterized by a reduction in oxygen delivery related to reduced CO, but in this case, the etiology of the reduced CO is an extracardiac pulmonary vascular or mechanical process impairing blood flow. Specific processes that can impede venous return to the heart and reduce CO include tension pneumothorax (PTX), cardiac tamponade, and restrictive pericarditis. Similarly processes that obstruct cardiac outflow, such as pulmonary embolism, venous air embolism, fat embolism (right heart), or aortic dissection (left heart), are included in this shock type category. Mixed Shock The types of shock outlined in this classification scheme are not mutually exclusive; not uncommonly, a patient will present with more than one type of shock. For example, the initial physiologic disturbance leading to reduced perfusion and cellular hypoxia in sepsis might be distributive shock. In this setting, a sepsisinduced cardiomyopathy can develop, which reduces myocardial con tractility, thus producing a cardiogenic component to what now would be described as a mixed type of shock. CHAPTER 314 Undifferentiated Shock Upon initial presentation, many patients have undifferentiated shock in which the shock type and specific dis ease process are not apparent. Using the history, physical examination, and initial diagnostic testing (including hemodynamic monitoring), the clinician attempts to classify a patient with one of the types of shock outlined above so that proper therapy can be initiated to restore tissue perfusion and oxygen delivery. Approach to the Patient with Shock

The epidemiology of shock is dependent on the clinical setting. A 2019 study of the etiology of shock in the emergency depart ment (ED) of a university hospital in Denmark revealed that among 1553 patients with shock, 30.8% had hypovolemic shock, 27.2% had septic shock, 23.4% had distributive nonseptic shock, 14% had car diogenic shock, and only 0.9% had obstructive shock. In the ICU set ting, septic shock predominates. A 2010 study (from eight hospitals) demonstrated that 62% of ICU shock patients had septic shock, 16% hypovolemic shock, 15% cardiogenic shock, and only 2% obstructive shock. Among specialty ICUs, the distribution of shock type differen tiates further. In the medical ICU, the largest number of patients have distributive shock related to sepsis. In contrast, a 2019 study of shock in 16 cardiac ICUs found that 66% of shock patients were assessed as having cardiogenic shock. Mortality associated with shock is high, but differences are seen between the types of shock. Septic shock and cardiogenic shock have the highest mortality rates. In the ED study from Denmark, the 90-day mortality of the septic and cardiogenic patients was 56.2% and 52.3%, respectively. These numbers coincide with other studies. Hypovolemic shock is associated with a lower mortality rate. ■ ■STAGES OF SHOCK Regardless of type, shock progresses through a continuum of three stages. These stages are compensated shock (preshock), shock (decom pensated shock), and irreversible shock. During compensated shock, the body utilizes a variety of physiologic responses to counteract the initial insult and attempts to reestablish the adequate perfusion and oxygen delivery. At this point, there are no overt signs of significant organ dysfunction. Laboratory evaluation may demonstrate mild organ dysfunction (i.e., elevated creatinine or troponin) or a mild elevation of lactate. The specific compensatory response is determined by the initial pathophysiologic defect. In early sepsis with reduction in SVR, there is a compensatory rise in HR (and CO). With early hemorrhagic volume loss, there will be a compensatory increase in SVR and HR. As the host compensatory responses are overwhelmed, the patient transitions into true shock with evidence of organ dysfunction. Appropriate interven tions to restore perfusion and oxygen delivery during these initial two phases of shock can reverse the organ dysfunction. If untreated the patient will progress to the third phase of irreversible shock. At this point, the organ dysfunction is permanent and often the patient pro gresses to multisystem organ dysfunction.

TABLE 314-3 Key Principles in the Treatment of Shock

1. Recognize shock early
2. Assess for type of shock present
3. Initiate therapy simultaneous with the evaluation into the etiology of shock
4. Involve all members of the multidisciplinary team
5. Aim of therapy is to restore oxygen delivery
6. Identify etiologies of shock that require additional lifesaving interventions ■ ■EVALUATION OF THE PATIENT WITH SHOCK The initial evaluation of the patient with shock utilizes the history, physical examination, and diagnostic testing toward two specific aims. The first aim is confirmation of the presence of shock. Given the reversible nature of the organ dysfunction in early shock, it is impor tant that the clinician has a high clinical suspicion for this condition. The possibility of shock should be considered in all patients presenting with new organ dysfunction or hypotension. This early recognition of the presence of shock is an essential tenet of shock care (Table 314-3). The second aim of the initial assessment (history, physical examina tion, and diagnostic testing) is to identify either a specific shock etiol ogy or to determine the type of shock present. In some

patients, the type of shock and etiology will be readily apparent (e.g., the patient with hypovolemic shock from a gunshot wound), but in many cases, the cause is determined only after further evaluation. We will discuss the role of the history, physical examination, and diagnostic testing toward these specific aims. While the assessment of shock etiology is ongoing, the initiation of therapy should not be delayed while the final diagnosis is being determined. Evaluation of shock etiology and initia tion of therapy should be simultaneous and as expedient as possible. PART 8 Critical Care Medicine History Obtaining a concise, focused history is essential. If the patient is unable to provide a history, ancillary information from fam ily or friends accompanying the patient, emergency medical services (EMS) personnel, or nursing facility (if applicable) should be obtained, and a brief chart review should be performed. Often, a patient with shock will present with nonspecific symptoms such as weakness, mal aise, or lethargy. When focal symptoms are reported, it is important to determine whether the symptom is related to the primary process causing shock or a result of inadequate oxygen delivery for cellular metabolic needs. For example, a patient with distributive shock from urosepsis could report chest discomfort in the setting of tissue hypoxia. As the history is being obtained, the clinician must be attentive to any details indicating new organ dysfunction. The most easily identified new organ dysfunction from the history is the presence of a newly altered mental status or decrease in urine output (oliguria). In some cases, the type of shock (and the specific disease process) is apparent from the initial history. Patients with distributive shock from sepsis may present with fever and a history suggesting a focal site of infec tion (e.g., cough, sputum production, abdominal discomfort, diarrhea, flank discomfort, or dysuria). Anaphylactic distributive shock may be suggested by the onset of pruritis, hives, dyspnea, and new facial edema after exposure to common allergens. Cardiogenic shock may be identified by the onset of exertional chest discomfort. The patient with significant arrhythmia may have an initial complaint of palpitations with syncope or presyncope. Hypovolemic shock may be identified in patients who present with a history of trauma (blunt or penetrating) or GI bleed (hematemesis, melena, or bright red blood per rectum). A patient with hypertension and tearing chest or back pain may be presenting with acute aortic dissection and obstructive-type shock. Asymmetric leg swelling, acute-onset chest pain with dyspnea in the setting of immobility, and/or underlying malignancy raises concern for obstructive shock due to pulmonary embolism. For most patients, the specific etiology will be less clear, but the history can be helpful in raising the likelihood of a particular type of shock. As an example, a patient with a preexisting immune dysfunction or medication-induced neutropenia may present with hypoperfusion and new organ dysfunction, in which the clinician must have a high suspicion for septic shock. Similarly, a patient with extensive cardiac disease requires a higher suspicion for cardiogenic shock.

Physical Examination The physical examination can assist in the identification of shock (in both the compensated stage prior to overt evidence of organ dysfunction and decompensated stage). The exami nation also can add insight into what type of shock is present (distribu tive, cardiogenic, hypovolemic, or obstructive). Shock is most commonly seen in the setting of circulatory failure. Vital signs are frequently abnormal. In most cases, this is manifest as hypotension (a systolic blood pressure [SBP] <90 mmHg or mean arterial pressure [MAP] <65 mmHg), but isolated blood pressure measurements below these values do not define shock on their own. Many patients may have underlying conditions such as peripheral vascular disease or autonomic dysfunction or are on

medications that cause longstanding low blood pressure without any evidence of organ dysfunction. Alternatively, patients with underlying hypertension may develop shock and organ dysfunction at higher blood pressures. Evalu ating the patient's current blood pressure in relation to the patient's baseline blood pressure and observing hemodynamic trends and cor relation with end-organ perfusion over short time intervals are more useful than an absolute SBP or MAP value. Tachycardia is a common compensatory mechanism in shock. The absence of an elevated heart rate does not exclude shock as patients with underlying cardiac con duction system disease or on home nodal blocking medications may have a diminished or absent tachycardic response. Alternatively, one cannot be reassured by an elevated heart rate without hypotension, as many younger patients can compensate for an extended period of time before developing hypotension. Tachypnea is another vital sign abnormality seen early in shock as the body compensates for a develop ing metabolic acidosis. While these early compensatory responses are nonspecific, the clinician should recognize these findings early as they may herald the development of end-organ dysfunction if perfusion and oxygen delivery are not restored. The physical examination can confirm the presence of shock prior to the return of laboratory testing. The central nervous system (CNS), kidney, and skin are the organ systems most easily assessed for evidence of organ dysfunction. These organ systems are considered the "windows" through which we can identify organ dysfunction. Decreased oxygen delivery to the brain is manifest as confusion and encephalopathy. In the early stage of shock, the body will redirect blood flow to the CNS to maintain adequate perfusion. In the patient with shock and altered mental status, all the usual compensatory mecha nisms have been outstripped by the magnitude of shock pathophysiol ogy. New encephalopathy represents a manifestation of decompensated shock. To assess renal function during the physical examination, one should evaluate the patient's urine output since the time of presenta tion. If not already present, a urinary catheter should be placed for accurate hourly assessment of urine output. In patients with normal baseline renal function, oliguria (<0.5 mL/kg per h) may indicate shock. Finally, cold and clammy skin is a sign of hypoperfusion with compensatory vasoconstriction to redirect blood flow centrally (brain, heart). Progressive vasoconstriction can lead to mottling of the skin. Capillary refill time (CRT) is the time it takes for color to return to an external capillary bed after pressure is applied. In the setting of shock, the CRT is delayed. Many components of the examination provide insight into hemody namics and assist in elucidating the type of shock present. The physical examination may be used to differentiate shock with high CO (distrib utive) from that with low CO (cardiogenic shock, hypovolemic shock, and obstructive shock). Examination findings suggestive of highoutput shock (distributive) include warm peripheral extremities and large pulse pressure (with low diastolic pressure). Alternatively, cool extremities with narrow pulse pressure would indicate low CO forms of shock. Among types of shock with low CO, the examination can be used to distinguish between conditions with increased intravascular filling pressure (cardiogenic shock, obstructive shock) and intravascu lar volume depletion (hypovolemic shock). Elevation of jugular venous pressure (JVP) and presence of peripheral edema are seen with high right-sided cardiac pressures. The JVP may be elevated in cardiogenic shock (with right-sided failure) and obstructive shock (pulmonary embolism) but reduced (JVP <8 cm) in hypovolemic shock. Similarly,

patients with cardiogenic shock and right-sided cardiac dysfunction may have peripheral edema, but this is not an examination finding typ ically present in acute hypovolemic shock. Distinguishing cardiogenic from obstructive shock can also be aided by physical examination. Rales on pulmonary auscultation may be related to left-sided cardiac dysfunction. The presence of cardiogenic shock

would be further sup ported by an S3 gallop. One must remember, however, that it is well established that patients with chronic heart failure do not present with the classical findings of acute heart failure. At times, the physical examination may identify the specific etiol ogy of shock. This is particularly helpful in the patient who cannot provide a detailed history. The examination may demonstrate the site of an untreated infection (e.g., cellulitis, abscess). The examina tion may reveal a brady- or tachyarrhythmia leading to development of shock. Similarly, large ecchymosis may indicate a significant bleed related to trauma or spontaneous retroperitoneal bleeding. The rectal examination may reveal GI hemorrhage. Pulsus paradox and elevated JVP may suggest the presence of cardiac tamponade. Patients with a tension PTX may have a paucity of breath sounds over the affected side, deviation of the trachea away from the affected side, or subcutaneous emphysema. Combinations of easily assessed examination components have been organized into a scoring system to identify high-risk patient popula tions. The shock index (SI) is defined as the HR/SBP, with a normal SI being 0.5–0.7. An elevated SI (>0.9) has been proposed to be a more sensitive indicator of transfusion requirement and of patients with critical bleeding among those with hypovolemic (hemorrhagic) shock than either HR or BP alone. The SI may also identify patients at risk for postintubation hypotension. This concept of use of a clinical score to identify at-risk patients has been extended to patients with distribu tive shock from sepsis. The quick Sequential Organ Failure Assessment (qSOFA) score is a rapid assessment scale that assigns a point for SBP <100, respiratory rate >22, or altered mental status (Glasgow Coma Scale <15). A qSOFA ≥2 (with a concern for infection) is associated with a significantly greater risk of death or prolonged ICU stay. The Third International Consensus Definition of Sepsis has recommended the use of the qSOFA to identify the most acutely ill subset of patients with sepsis (longer length of stay, increased need for ICU admission, and higher in-hospital mortality). Diagnostic Testing Laboratory evaluation should be initiated promptly in all patients with suspected shock. The laboratory evalu ation is directed toward the dual aim of assessing the extent of endorgan dysfunction and of gaining insight into the possible etiology of shock. Table 314-4 outlines the recommended initial laboratory evalu ation of the patient with undifferentiated shock. BLOOD TESTS Evaluation of lactate, blood urea nitrogen (BUN), creatinine, and transaminases provides an assessment of the extent of end-organ dysfunction related to shock. (See discussion of lactate below.) Urine electrolytes with subsequent calculation of the fractional excretion of sodium (FENa) or fractional excretion of urea (FEUrea) may indicate states of hypovolemia or decreased effective circulating TABLE 314-4 Initial Laboratory Evaluation of Undifferentiated Shock

1. Lactate
2. Renal function tests
3. Liver function tests
4. Cardiac enzymes
5. Complete blood count (with differential)
6. PT, PTT, and INR
7. Pregnancy test
8. Urinalysis and urine sediment
9. Arterial blood gas
10. ECG
11. CXR Abbreviations: CXR, chest x-ray; ECG, electrocardiogram; INR, international normalized ratio; PT, prothrombin time; PTT, partial thromboplastin time.

volume. Elevation of alkaline phosphatase may suggest biliary obstruc tion and may thereby identify a source of infection in patients with distributive shock. Elevation of cardiac enzymes can indicate a primary cardiac problem with myocyte damage related to ischemia, myocar ditis, or a pulmonary embolism. An elevation of the white blood cell count may raise suspicion for an infective process, but this is certainly not diagnostic; an accompanying left shift may improve the sensitivity of this measure. Reductions in hemoglobin and hematocrit are seen in patients with hemorrhagic hypovolemic shock (although an actively bleeding patient may have normal values on initial presentation). While the extent of acidosis may be determined with a venous blood gas (VBG), if there is accompanying hypoxemia, an arterial blood gas should be obtained. For patients with undifferentiated shock, there should always be a high index of suspicion for possible infection. Uri nalysis and urine sediment should be sent to evaluate for pyuria. Blood cultures, urine cultures, and sputum cultures should be obtained. Radiographic evaluation should be directed to seek sources of infection suggested by the history and physical examination.

CHAPTER 314 Lactate measurement has a role in the diagnosis, risk stratification, and, potentially, the treatment of shock. Increased lactate (hyperlacte mia) and lactic acidosis (hyperlactemia and pH <7.35) are common in shock. Lactate is a product of anaerobic glucose metabolism. In glycolysis, the enzyme phosphofructokinase metabolizes glucose to pyruvate. Under aerobic conditions, the pyruvate is then converted (in the mitochondria) to acetyl CoA and enters the Krebs cycle with resulting ATP generation through oxidative phosphorylation. In the setting of cellular hypoxia, the Krebs (tricarboxylic acid) cycle cannot oxidize the pyruvate, and thus the pyruvate is converted to lactate by the enzyme lactate dehydrogenase. Under normal conditions, lactate is produced from skeletal muscle, brain, skin, and intestine. In the setting of reduced oxygen delivery and cellular hypoxia, the amount of lactate produced from these tissues increases (and other tissue can begin to produce lactate). While most of the studies have been performed in patients with septic shock, there is evidence that elevated lactate cor relates with a worse outcome. A recent systematic literature review evaluating the role of lactate measurement in a variety of critically ill populations supported the value of serial lactate measurements in the evaluation of critically ill patients and their response to therapy. Approach to the Patient with Shock Electrocardiogram The electrocardiogram (ECG) is an essen tial part of the evaluation of the patient with shock. There may be a bradycardic or tachycardic arrhythmia causing a reduction in CO. ST-segment elevation myocardial infarction may be identified. The presence of the S1 Q3 T3 pattern would raise concerns for pulmonary embolism. Reduced voltage in the presence of electrical alternans raises the possibility of pericardial tamponade. Chest X-Ray The chest x-ray (CXR) can demonstrate a new focal alveolar or interstitial infiltrate suggesting an infectious process (and possible distributive septic shock). Bilateral cephalization of the pulmonary vasculature, peribronchial cuffing, septal thickening, and intralobular thickening are typical of pulmonary edema and a cardio genic process. The CXR can be used to confirm or exclude the presence of a pneumothorax. CXR findings are neither sensitive nor specific for pulmonary embolism. In select cases, there may be the finding of a peripheral wedge-shaped opacity indicating pulmonary infarction, an enlarged pulmonary artery, or regional vascular oliguria. A chest computed tomography (CT) angiogram may be needed to exclude the diagnosis of pulmonary embolism and further evaluate the thorax. Point-of-Care Ultrasound Point-of-care ultrasound (POCUS) has an increasing role in the evaluation and treatment of shock. Benefits of POCUS include its low cost, rapidity with which it can be obtained, and noninvasive nature. It has diagnostic value in patients who present with undifferentiated shock. In patients with mixed shock, it can give insight into the relative contribution of the individual shock

types. Several structured protocols exist for evaluation of undifferenti ated shock including the Rapid Ultrasound for Shock and Hypotension (RUSH), the Abdominal and Cardiothoracic Evaluation with Sonogra phy in Shock (ACES), and Sequential Echographic Scanning Assessing

Mechanism or Origin of Shock of Indistinct Cause (SESAME). These protocols share common components to assess cardiac function, evaluate intravascular volume status, and identify fluid collections. In a rapid and protocolized manner, views are obtained of the heart, lungs, pleural space, inferior vena cava, abdominal aorta, abdomen, and pelvis. Some of the protocols extend to view the deep veins of the lower extremity.

POCUS transthoracic echocardiography (TTE) is central to the POCUS evaluation of shock. TTE utilizes both the two-dimensional (2D) and M mode. It focuses the examination on LV function, RV function, and the pericardium. The 2D mode can evaluate LV size, wall thickness, and ventricular function. Ventricular size and thickness can suggest longer standing cardiac processes. Evaluation of LV func tion through estimation of left ventricular ejection fraction (LVEF) can identify shock with globally reduced LV function or regional wall motion abnormalities. Similarly, the assessment of RV function also examines RV size and wall thickness (to identify conditions such as elevated pulmonary pressures or suggest pulmonary embolism). An additional important assessment includes evaluation for pericardial tamponade. Two-dimensional echocardiography can also be used to assess valve function, including acute processes, such as mitral valve rupture. Assessment of valvular function is often an evaluation that requires a higher skilled practitioner. The performance of the bedside echocardiogram by the critical care practitioner does not replace for mal examination by the echocardiography service or assessment by a cardiologist. PART 8 Critical Care Medicine Another component of POCUS includes inferior vena cava (IVC) evaluation to assess intravascular filling. A collapsible IVC in sponta neously breathing patients at the end of expiration suggests reduced intravascular volume. Evaluation of the pleural space for effusion has been a longstanding role of ultrasound, and POCUS pleural space evaluation can be more sensitive than CXR for identifying a PTX. Defined views of the abdomen can identify significant intrabdominal fluid collections indicating hemorrhage or possible infection. Exami nations that extend to the proximal deep veins of the lower extremity can identify deep-venous thrombosis, raising the possibility of pul monary embolism as an etiology of shock. While POCUS can aid in determining the etiology of shock, a 2018 international randomized controlled study utilizing POCUS to evaluate undifferentiated shock in 273 ED patients did not demonstrate a benefit in survival at 30 days or hospital discharge. In addition, there was no difference in amount of IV fluids administered, inotrope use, CTs ordered, or need for ICU care or length of stay. One significant limitation of POCUS is that performance and interpretation of testing are operator dependent. Familiarity with basic ultrasound techniques and interpretation is now expected in the ED and critical care setting. Accordingly, competency standards have been proposed for emergency medicine and critical care providers in both basic and advanced POCUS techniques. ■ ■INITIAL TREATMENT OF SHOCK Because shock can progress rapidly to an irreversible stage, a key prin ciple in shock management is to initiate treatment for circulatory shock simultaneously with efforts to elucidate shock etiology (Table 314-3). If the initial history, physical examination, and laboratory evaluation have identified the shock type or the specific etiology, then therapy is directed to reverse the underlying physiologic abnormality causing the hypoperfusion and reduced oxygen delivery. To expedite care, all mem bers of the multidisciplinary team (physicians, nurses, pharmacists, and respiratory therapists) must be involved in the development and delivery of care. Details of the optimal care

for the specific disease pro cesses leading to shock may be found in other chapters of this text. As many patients will present with undifferentiated shock, in this section, we will discuss treatment directed at the patient with undifferentiated shock. At the conclusion of this section, we will highlight etiologies of shock that require initiation of lifesaving specific therapy. A key early consideration is to ensure adequate intravenous access. Placement of two peripheral venous catheters (16 or 18 gauge) will provide initial access for the aggressive volume resuscitation that is

often required for patients with distributive or hypovolemic shock. If there is concern for distributive shock with sepsis, this IV access will also permit prompt antibiotic administration. For patients with ongo ing hypotension despite adequate volume resuscitation, placement of a central venous catheter (CVC) is indicated to provide therapy with vasopressors and inotropes. The CVC will provide a mechanism for hemodynamic monitoring (CVP), as well as a means to obtain cen tral venous oxygen saturations (ScvO2). The ScvO2 is a surrogate of mixed venous oxygen saturation and thus can provide insight into the adequacy of oxygen delivery. Central venous access using a sheath will provide an access point for placement of a Swan-Ganz catheter if more detailed assessment of hemodynamic measurements is required (PCWP, CO, and SVR) and/or if larger bore central access is required for more aggressive volume or blood administration as in hemorrhagic shock. If the patient presents critically ill or in the midst of cardiopul monary arrest, the quickest method of obtaining central access will be through the use of an intraosseous device. Placement of an arte rial line allows for intravascular measurement of blood pressure and continuous determination of MAP. In addition, it can provide insight into the adequacy of volume resuscitation through the measurement of systolic or pulse pressure variation. The arterial line will provide access for determination of arterial oxygen tension, which is helpful since peripheral oximetry measurements (SpO2) can be unreliable in states of tissue hypoperfusion. The arterial line facilitates repeated measures of acid-base status and lactate to assess the impact of treatment. All patients with shock should have a urinary catheter placed to permit hourly assessment of renal function as another potential indication of the adequacy of resuscitation. Volume Resuscitation Initial volume resuscitation has the aim of restoring tissue perfusion and is crucial to optimal shock therapy. Assessment of current intravascular volume status and determination of the optimal amount of necessary volume resuscitation are challeng ing. The physiologic goal of volume resuscitation is to move the patient to the non–preload-dependent portion of the Starling curve. Most patients with any of the four shock types will benefit from an increase in intravascular volume. For patients with distributive shock, the need for early aggressive volume replacement is well established. In the past, the use of early goal-directed therapy (EGDT) in septic shock targeted specific measures of CVP, MAP, and SvO2 to guide volume resuscita tion (and initiation of vasopressors and inotropes). More recent studies have demonstrated that targeted resuscitation using invasive moni toring is not universally required, but in all of these studies, patients in the “usual care” arms of the study received early initial volume resuscitation. For patients with suspected septic shock, a minimum of 30 mL/kg as an initial volume of resuscitation is recommended by the Surviving Sepsis Campaign. While the need for volume resuscitation is most apparent for patients with distributive or hypovolemic shock, even some patients with cardiogenic shock may benefit from cautious volume replacement. In these patients, there should be a careful assess ment of volume status prior to volume administration. In general, volume replacement therapy should be given as a bolus with a predefined endpoint to assess the effect of the volume resuscita tion. Most commonly, the volume resuscitation will begin with crystal loid. In patients with hypovolemic shock due to ongoing hemorrhage, volume replacement with packed red blood cells is

warranted. In cases of massive transfusion, platelets and fresh frozen plasma should be provided to offset the dilution of these components during volume replacement. Because hemoglobin is a key determinant of CaO2, red cell administration may be a part of volume replacement even without hemorrhage in order to optimize oxygen delivery if hemoglobin con tent is <7 g/dL. Assessment of intravascular volume status (and the adequacy of volume resuscitation) begins with the physical examination (described above). The passive leg raise (PLR) test can predict responsiveness to additional IV fluid by providing the patient with an endogenous vol ume bolus. While the patient is resting in a semirecumbent position at a 45° angle, the bed is placed in Trendelenburg position such that the patient's head becomes horizontal and the legs are extended at a

45° angle. There is then an immediate (within 1 min) assessment of changes in CO (or pulse pressure variation as a surrogate). It is impor tant to emphasize that one does not merely look for changes in blood pressure; if the shock patient is mechanically ventilated, there is the option of looking at changes in SV variation (or pulse pressure varia tion) during the respiratory cycle to assess volume responsiveness. A

> 12% SV variation suggests a volume-responsive state. This measure ment requires that the patient be in a volume cycle mode of ventilation, without breath-to-breath variations in intrathoracic pressure and with out arrhythmias. A final caveat to the use of these parameters to assess volume status is that these studies were performed on patients being ventilated with tidal volumes larger than currently used to minimize ventilator-induced lung injury. There is also increased use of echocardiography to assist in determi nation of intravascular fluid status, with a variety of static and dynamic variables that the trained operator can assess. The most commonly used parameters to assess adequacy of volume resuscitation are IVC diameter and IVC collapse. Alternatively, serial assessments of LV function can be performed while volume is being administered. Place ment of a pulmonary artery catheter (PAC) is another tool for assess ment of volume status. This more invasive measure involves placement of the PAC into the central venous circulation and through the right heart. Ports in the PAC (Swan-Ganz catheter) allow for direct measure ment of CVP, pulmonary artery (PA), and PCWPs. The PCWP is used as a surrogate for LA pressure. While studies have not identified a mor tality or length-of-stay benefit with routine use of PA catheterization, there are cases where it may be beneficial. Patients with mixed shock (distributive and cardiogenic) or those with ongoing shock of unclear etiology are examples of situations in which it should be considered. The need for continued volume replacement must be frequently reassessed. As the patient continues to receive treatment for shock, the initial proper strategy regarding volume management may change in light of development of processes that independently require a different volume-management strategy. For patients who initially present with shock but then develop respiratory failure related to acute respiratory distress syndrome (ARDS) or renal failure, it may be reasonable to begin volume removal. Vasopressor and Inotropic Support If intravascular volume status has been

> optimized with volume resuscitation but hypotension and inadequate tissue perfusion persist, then vasopressor and/or ino tropic support should be initiated. The use of vasopressors and ino tropes must be tailored to the primary physiologic disturbance. The clinician must understand the receptor selectivity of various agents and that for some agents the selectivity may be dose dependent. In patients with distributive shock, the primary aim is to increase the SVR. Norepinephrine is the first-choice vasopressor in septic shock, with potent α1 and β1 adrenergic effects. The α1 causes vasoconstric tion, while β1 has positive inotropic and chronotropic effects. At higher doses, epinephrine has a similar profile (at lower doses, the

β effects predominate) but is associated with tachyarrhythmia, myocar dial ischemia, decreased splanchnic blood flow, pulmonary hyperten sion, and acidosis. In distributive shock, vasopressin deficiency may be present. Vasopressin acts on the vasopressin receptors to reverse vasodilation and redistribute flow to the splanchnic circulation. In a randomized trial in patients with septic shock, the addition of lowdose vasopressin to norepinephrine did not reduce all-cause 28-day mortality compared to norepinephrine alone but suggested a poten tial benefit in the less “sick” population. Vasopressin is safe and has a role as a second agent for hypotension in septic shock. Dopamine does not have a role as a first-line agent in distributive shock. A ran domized controlled study in patients with all-cause circulatory shock did not show a survival benefit from dopamine but did reveal an increase in adverse events (arrhythmia). In this study, the subgroup of patients with cardiogenic shock had increased mortality. For patients with cardiogenic shock, dobutamine is a first-line agent; it is a syn thetic catecholamine with primarily β-mediated effects and minimal α adrenergic effects. The β1 effect is manifest in increased inotropy, and the β2 effect leads to vasodilation with decreased afterload; it can

be used with norepinephrine in patients with mixed distributive and cardiogenic shock.

■ ■OXYGENATION AND VENTILATION SUPPORT In addition to the cellular hypoxia caused by circulatory failure, patients with shock may present with hypoxemia. For patients with distributive shock, this may be related to a primary pulmonary pro cess (e.g., pneumonia in a patient with septic shock). For patients with cardiogenic or obstructive shock, hypoxemia may be related to pulmonary edema as a result of LV dysfunction and elevations of PCWP. For patients with all types of shock, there can be develop ment of ARDS and subsequent V./Q. (ventilation/perfusion) mismatch and shunt. Supplemental oxygen should be initiated and titrated to maintain SpO2 of 92–95%. This may require intubation and initiation of mechanical ventilation. If the patient requires intubation and initia tion of mechanical ventilation, this should be provided promptly so as to minimize the duration of tissue hypoxia. Patients with shock may have high minute ventilatory needs to compensate for metabolic aci dosis. As shock progresses, they may not be able to maintain adequate respiratory compensation, which may be a second indication to initi ate mechanical ventilator support. If mechanical ventilation support is initiated, it is important to provide ventilation with lung-protective strategies focused on low tidal volume ventilation and optimization of positive end-expiratory pressure to minimize ventilator-induced lung injury. In addition, there should be daily sedation cessation to assess underlying neurologic function and minimize time on mechanical ventilation. There are currently few data to support the use of nonin

vasive ventilation in the setting of shock. CHAPTER 314 Approach to the Patient with Shock Antibiotic Administration Sepsis is the most common cause of shock. For patients presenting with undifferentiated shock, if the diagnosis of septic shock is being entertained, then broad-spectrum antibiotics should be administered after obtaining appropriate cultures. For patients with sepsis, every hour of delay in appropriate antibiotic administration is associated with an increase in mortality. While it is ideal to initiate antibiotics after appropriate cultures, the inability to obtain cultures should not delay the start of treatment. When sepsis is excluded as a cause of shock, an important aspect of antibiotic steward ship is to stop all antibiotics. Specific Causes of Shock Requiring Tailored Intervention The initial evaluation (history, physical examination, and diagnostic testing) may have identified an etiology of shock that requires urgent lifesaving intervention in addition to the initial treatment steps outlined above. Patients with distributive shock secondary to anaphylaxis require removal of the inciting allergen, administration of epinephrine, and vascular support with IV fluid resuscitation and vasopressors. Adre nal insufficiency requires replacement with IV stress-dose steroids. Cardiogenic shock patients with arrhythmia may require treatment as outlined in advanced cardiac life support algorithms or placement of an artificial pacemaker. In cases of acute ischemic events, consideration must be given to revascularization and temporary mechanical support ive measures. In the case of valve dysfunction, emergency surgery may be considered. Patients with hypovolemic shock due to hemorrhage may require surgical intervention in the case of trauma or endoscopic or interventional radiology procedures in the case of a GI source of blood loss. Sources of occult bleeding can include soft tissue injury sites including bleeding after long-bone fractures, retroperitoneal bleeding, and the GI tract. Among patients with obstructive shock, a tension PTX would necessitate immediate decompression. Proximal pulmonary embolism requires evaluation for thrombolytic therapy or surgical removal of the clot. Dissection of the ascending aorta may require surgical intervention. ■ ■FURTHER READING Benham et al: A standardized and comprehensive approach to the management of cardiogenic shock. JACC Heart Fail 8:879, 2020. Evans L et al: Surviving Sepsis Campaign: International guidelines for the management of sepsis and septic shock. Crit Care Med 49:e1063, 2021.

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