Shock

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Epidemiology of Shock

Shock is a pathophysiological state characterized by inadequate tissue oxygenation. It is a life-threatening condition that may lead to cellular death and vital organ dysfunction. Common clinical manifestations of shock include tachycardia, hypotension, oliguria, and confusion. Laboratory tests often demonstrate signs of end-organ damage such as acute liver or kidney injury, metabolic acidosis, and lactic acidosis.

There are four major categories of shock: cardiogenic, hypovolemic, distributive, and obstructive shock. However, it is important to note that these major categories of shock are not mutually exclusive and combinations with other form(s) of shock may occur.

Cardiogenic shock (CS) is often depicted as shock due to pump failure. The primary disorder in CS is low cardiac output due to intrinsic cardiac dysfunction. It is often a complication of myocardial infarction, but it may also be due to arrhythmia, congestive heart failure, or a primary valvular disorder. CS is estimated to complicate 3–8% of cases of acute myocardial infarction [1–3]. The mortality rate associated with CS in patients with acute myocardial infarction historically approached 80% [1]. Due to contemporary strategies in the management of acute coronary syndrome, the adjusted mortality rate ranges from 45 to 66% [4].

Hypovolemic shock is shock due to hemorrhagic or nonhemorrhagic volume loss. It is often related to traumatic injury, gastrointestinal bleeding, vomiting, or diarrhea. The incidence of hypovolemic shock varies depending on its underlying etiology.

The most common etiology of *distributive shock* is sepsis, a dysfunctional systemic response to an infection. Every year, 1.6 million patients are diagnosed with sepsis in the United States [5]. In its severe form, septic shock may ensue. The mortality associated with severe sepsis and septic shock is estimated to be 14.7–29.9% [6]. These figures are likely underestimated as severe sepsis and septic shock often occur concomitantly with other leading causes of mortality including pneumonia, urinary tract infection, gastrointestinal tract infection, skin infection, and malignancy.

The primary disorder in *obstructive shock* is low cardiac output due to extrinsic cardiac dysfunction. The incidence of obstructive shock varies according to its etiology which includes pulmonary embolism, cardiac tamponade, and tension pneumothorax.

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Clinical Presentation of Shock: A Case-Based Approach

Understanding the pathophysiology associated with each category of shock is essential in performing an appropriate clinical assessment and guiding appropriate therapy. Examples of different conditions associated with each major category of shock are listed in Table 21.1. The following cases are examples from each category of shock.

Case 1: Cardiogenic Shock

A 55-year-old male with hypertension, dyslipidemia, and a family history of coronary artery disease presents to the emergency department

Table 21.1 Etiologies of shock

Cardiogenic[a]	**Distributive**
Myopathic	Sepsis; pancreatitis
Acute myocardial infarction	Pancreatitis
Left ventricular failure	Anaphylaxis
Right ventricular failure	Bacterial toxins
Acute myocarditis	Acidosis
Idiopathic cardiomyopathy	Adrenal crisis
Restrictive/constrictive cardiomyopathy	Myxedema coma
Stress-induced (Takotsubo) cardiomyopathy	Iatrogenic
Acute heart transplant rejection	Neurogenic insult
Iatrogenic (negative inotropic or vasodilatory medications)	Post-resuscitation syndrome
Post-cardiac arrest	Post-cardiopulmonary bypass
Mechanical	**Hypovolemic**
Ventricular septal rupture	Hemorrhagic
Ventricular Free Wall rupture	Trauma
Hypertrophic obstructive cardiomyopathy	Gastrointestinal bleeding
Dynamic left ventricular outflow tract obstruction	Ruptured hematoma
Atrial myxoma	Hemorrhagic pancreatitis
Traumatic	Fractures
Valvular	Ruptured aortic aneurysm
Papillary muscle/chordal rupture	Acute aortic dissection
Acute mitral regurgitation	Plasma extravasation related
Prosthetic valve obstruction	Systemic inflammatory response
Severe aortic regurgitation	Sepsis
Critical aortic stenosis	Major surgery
Severe mitral stenosis	Pancreatitis
Arrhythmic	Major surgery
Tachycardia	Fluid loss related
Sustained ventricular tachycardia	Dehydration
Ventricular fibrillation	Severe burns
Bradycardia	Emesis
High-grade AV block	Diarrhea
Complete heart block	Diaphoresis
	Insensible losses
	Inadequate fluid intake
	Obstructive
	Cardiac tamponade
	Pulmonary embolism
	Pulmonary hypertension
	Tension pneumothorax

[a]*Causes that may complicate acute myocardial infarction are italicized*

with severe substernal chest pain and shortness of breath. Initial vital signs demonstrate tachycardia and hypotension. A third heart sound (S3) is auscultated. He has inspiratory rales in both lung bases, and his internal jugular vein is appreciated above the clavicle in an upright position. His extremities are cool, mottled, and cyanotic. An electrocardiogram reveals ST segment elevation across the anterior precordial leads.

This case describes a patient with CS caused by acute myocardial infarction. CS is a clinical condition in which inadequate tissue perfusion is the consequence of intrinsic cardiac dysfunction. It is characterized by a reduction in cardiac output despite adequate filling pressures. Causes of CS include myopathic, mechanical, valvular, and arrhythmic processes (Table 21.1). The criteria typically used to define CS include systolic blood pressure less than 90 mmHg for at least 30 min or the need for a vasopressor or mechanical circulatory support to maintain a systolic blood pressure greater than 90 mmHg; pulmonary capillary wedge pressure greater than 15 mmHg; and cardiac index less than 2.2 L/min/kg/m^2 [7].

Tachycardia, hypotension, oliguria, confusion, cyanosis, and cold extremities typically characterize the clinical presentation of CS. Tachycardia occurs in an effort to maintain cardiac output when the stroke volume is reduced. Oliguria and confusion are the result of poor tissue perfusion. Cool, mottled, and cyanotic extremities are manifestations of peripheral vasoconstriction.

Peripheral pulses are often diminished in CS due to decreased pulse pressure (*pulsus parvus*). In a failing left ventricle, the strength of every other beat may alternate, a phenomenon known as *pulses alternans*. Delayed pulses (*pulsus tardus*) may be seen in cardiogenic shock in the setting of severe aortic stenosis.

CS due to left ventricular failure may present with pulmonary congestion; patients may complain of orthopnea and paroxysmal nocturnal dyspnea (PND). Examination may demonstrate bilateral inspiratory rales, S3 gallop, and laterally displaced apical impulse due to left ventricular dilatation. Chest radiography may demonstrate cardiomegaly, pulmonary vessel cephalization, Kerley B lines, and parenchymal edema. If right ventricular failure is also present, evidence of venous congestion may be observed including jugular vein distention, hepatojugular reflux, and bilateral lower extremity edema.

Of note, a significant proportion of patients in the SHOCK (SHould we emergently revascularize Occluded Coronaries in cardiogenic shocK?) trial had no pulmonary congestion [8]. Neither auscultation nor chest radiograph detected pulmonary edema in 28% of patients.

Right ventricular infarction complicates up to half of all transmural inferior-posterior myocardial infarctions [9]. Patients with hemodynamically significant right ventricular infarction classically present with hypotension, clear lung fields, and jugular venous distention. Right ventricular failure may be associated with a holosystolic tricuspid regurgitation murmur at the left lower sternal border, jugular venous distension, liver engorgement, pulsatile liver, and peripheral edema. Patients with patent foramen ovale and acute right ventricular infarction may present with profound hypoxia due to decreased compliance in the infarcted right ventricle and right-to-left shunting.

Mechanical complications of acute myocardial infarction may result in CS and can sometimes be evident on physical exam. Acute mitral regurgitation, tricuspid regurgitation, and ventricular septal rupture are associated with holosystolic murmurs. However, if there is rapid equalization of pressure in the atria and ventricles, acute regurgitant lesions may not cause a significant murmur. Prominent jugular *v*-waves suggest severe tricuspid regurgitation. Jugular cannon *a*-waves suggest complete heart block. Ventricular free wall rupture will frequently result in fulminant cardiac tamponade (see section "Case 4: Obstructive Shock").

In CS, a compensatory increase in systemic vascular resistance (SVR) typically occurs through peripheral vasoconstriction in an effort to maintain tissue perfusion. However, this classic paradigm has been challenged. Data from the SHOCK trial demonstrated that many patients with CS instead have low systemic resistance, similar to patients with septic shock [10]

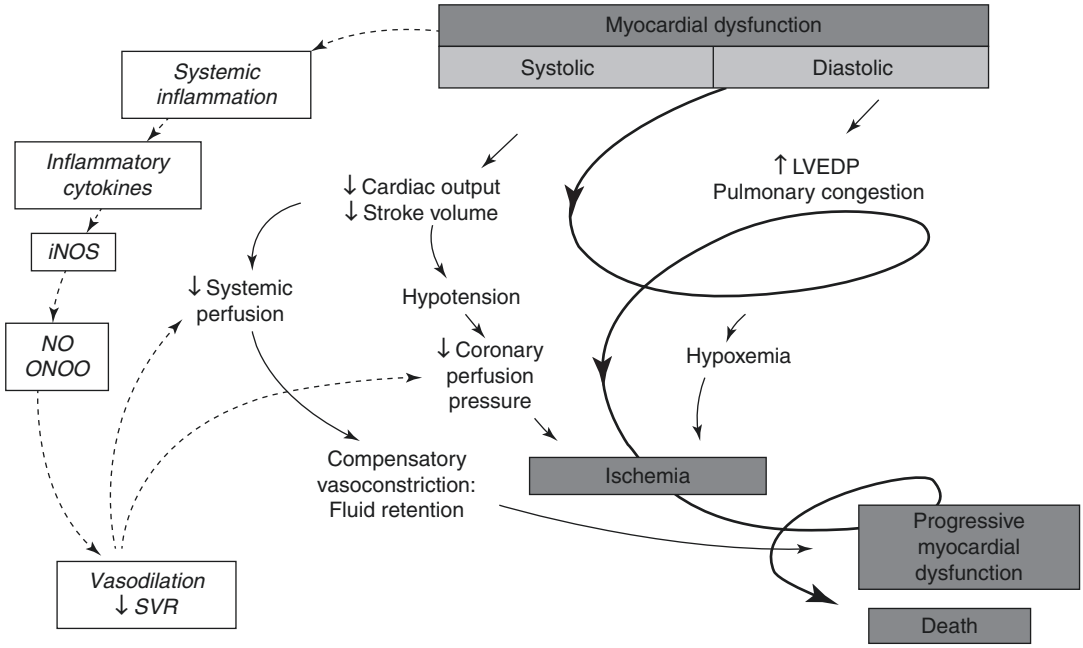

Fig. 21.1 Expansion of the pathophysiological paradigm of cardiogenic shock to include the potential contribution of inflammatory mediators. LVEDP left ventricular end-diastolic pressure; NO nitric oxide; iNOS inducible nitric oxide synthase; ONOO− peroxynitrite; SVR systemic vascular resistance. (Reprinted with permission from Hochman and Ohman [26])

(Fig. 21.1). It has been postulated that a systemic inflammatory response-like syndrome with a low SVR may be encountered in up to one-fifth of patients with acute myocardial infarction complicated by CS [11].

The clinical presentation can be utilized to risk-stratify patients with acute myocardial infarction complicated by CS. Killip described a case series of 250 patients presenting to an academic university intensive care unit with myocardial infarction [12]. He divided the patients into four classes according to the clinical presentation. Class I had no clinical signs of heart failure; class II presented with basilar rales and/or S3 gallop and/or elevated jugular venous pressure; class III had frank pulmonary edema; and class IV had cardiogenic shock. Reported mortality was 6%, 17%, 38%, and 67% for each class, respectively.

Current mortality with reperfusion therapy is dramatically lower than that observed in the original observation. However, Killip Class II, III, and IV continue to identify a high-risk subset of patients.

Therapeutic measures for CS often need to be implemented before invasive hemodynamic data are available. The clinical presentation may guide the most effective therapy. For example, patients with a clinical presentation consistent with myocardial infarction require urgent revascularization. A profile consistent with left ventricular failure may require inotropic or vasoactive agents and/or mechanical circulatory support in the form of an intra-aortic balloon pump, percutaneous left ventricular assist device, or extracorporeal membrane oxygenation. Patients with findings consistent with right ventricular infarction may require rapid fluid resuscitation and/or percutaneous right ventricular assist device placement.

Case 2: Hypovolemic Shock

A 25-year-old male patient presents shortly after a gunshot wound to the abdomen. He has experienced profound bleeding. He is tachycardic, hypotensive, and confused. Neck veins are

flat and lung fields are clear. The mucus membranes and skin are dry. Extremities are cool and clammy. He has a large drop in blood pressure and is more tachycardic when changing from supine to standing position.

The second case describes a patient with hypovolemic shock related to traumatic hemorrhage. Hypovolemic shock is characterized by inadequate intravascular volume. Etiologies are generally related to hemorrhage, plasma extravasation, or fluid loss (see Table 21.1). Hemorrhagic etiologies include trauma, gastrointestinal bleeding, ruptured hematoma, hemorrhagic pancreatitis, fracture, and ruptured abdominal aortic aneurysm. Etiologies related to plasma extravasation include systemic inflammatory response, major surgery, and severe pancreatitis. Fluid loss may be due to severe burns, emesis, diarrhea, diaphoresis, other insensible losses, and inadequate oral intake.

Hypotension, tachycardia, confusion, oliguria, and cold extremities typically characterize the clinical presentation of hypovolemic shock. Cardiac output generally falls as a result of decreased ventricular preload. Compensatory tachycardia and an increase in SVR, mediated by peripheral vasoconstriction, occur in an effort to improve tissue perfusion. Increased sympathetic activity may cause narrow pulse pressure and diaphoresis. Peripheral vasoconstriction results in cool, mottled, and cyanotic extremities.

The clinical history will generally suggest the underlying etiology. However, clinical findings may overlap with cardiogenic shock. A key difference is that the intracardiac filling pressure is adequate or elevated (PCWP greater than 15 mmHg) in CS, whereas it is generally low in hypovolemic shock due to inadequate intravascular volume. In hypovolemic shock, the lung fields are generally clear, and there is no evidence of jugular venous distension or peripheral edema.

The physical diagnosis of hypovolemia has been systematically reviewed in adults [13]. The most helpful physical findings include severe postural dizziness or a postural pulse increment of at least 30 beats/min, a systolic pressure decrement of at least 20 mmHg, or a diastolic pressure decrement of at least 10 mmHg for measurements obtained 2 min after assuming an upright posture (orthostasis). Supine hypotension and tachycardia were frequently absent, even after up to 1150 mL of blood loss [13]. In patients with vomiting, diarrhea, or decreased oral intake, the presence of a dry axilla supports the diagnosis of hypovolemia. In adults, the capillary refill time and poor skin turgor have no proven diagnostic value.

Case 3: Distributive Shock

An 80-year-old female with recurrent urinary tract infection presents with fever, tachycardia, hypotension, decreased urine output, and confusion. She is diaphoretic and has foul smelling urine. Her neck veins are flat and the lung fields are clear. The extremities are warm and hyperemic. Capillary refill is brisk. Her hypotension does not improve despite aggressive fluid resuscitation.

This third case describes a patient with distributive shock related to severe sepsis. Inappropriate vasodilation, decreased SVR, hypotension, and poor tissue oxygenation characterize distributive shock. Etiologies of distributive shock include systemic inflammatory response syndrome, severe sepsis, bacterial toxins (e.g., staphylococcal toxic shock), anaphylaxis, adrenal insufficiency, myxedema coma, neurogenic insult, post-resuscitation syndrome, and post-cardiopulmonary bypass (see Table 21.1).

Septic shock is the most common presentation of distributive shock. Septic shock is defined by sepsis-induced hypotension that persists despite adequate fluid resuscitation (20–30 mL/kg starch or 40–60 mL/kg saline or PCWP 12–20 mmHg) [14].

Clinical presentations of distributive shock vary by etiology but are generally characterized by tachycardia, hypotension, oliguria, confusion, and warm, well-perfused extremities. Compensatory tachycardia, oliguria, and confusion are manifestations of poor tissue perfusion. Warm and hyperemic extremities with brisk capillary refill time are the result of inappropriate

vasodilatation and decreased SVR. The cardiac output is typically elevated in distributive shock and may manifest as bounding pulses. The lungs are typically clear, and there is no jugular venous distension or peripheral edema.

Patients with severe sepsis and shock may have fever or hypothermia, diaphoresis, and rigors. Findings associated with an infectious process (e.g., pneumonia) may be present. Tachypnea is common and occurs in an effort to compensate for severe metabolic acidosis caused by elevated lactate. Hoarseness, stridor, wheezing, pruritus, flushing, hives, and abdominal pain may accompany anaphylactic shock. Hypothermia, hypoventilation, and somnolence are associated with myxedema coma (severe hypothyroidism). Profound orthostatic hypotension and skin hyperpigmentation may be presenting signs of adrenal insufficiency. Although most patients with distributive shock present with tachycardia, patients with neurogenic shock may have bradycardia due to sympathetic denervation.

Case 4: Obstructive Shock

A 65-year-old female patient with metastatic breast cancer presents with dyspnea and palpitations. Although she is tachycardic, her heart sounds seem distant. Her initial blood pressure is 80/50 mmHg. Her internal jugular vein is distended, but her breath sounds are clear to auscultation. Careful examination of her blood pressure demonstrates an exaggerated dissipation of Korotkoff sounds during inspiration. She has no leg edema and has not observed any recent fever, bleeding, vomiting, or diarrhea. Chest radiography revealed cardiomegaly with no significant parenchymal disease.

The fourth case is representative of obstructive shock due to cardiac tamponade. In the setting of breast cancer, pericardial metastasis is suspected. Classic findings of cardiac tamponade include distant, muffled heart sounds, jugular venous distension, and hypotension. Ventricular interdependence, manifested by an inspiratory drop in blood pressure (*pulses paradoxus*) greater than 10 mmHg, may also be observed.

Other causes of obstructive shock include tension pneumothorax and pulmonary embolism. Tension pneumothorax may be accompanied by dyspnea with tympany and decreased breath sounds in the affected hemithorax. Pulmonary embolism may be associated with cough, hemoptysis, and limb asymmetry in the context of prolonged immobilization. Both may present with desaturation.

Diagnostic Evaluation of Shock

The etiology of shock can often be determined using data acquired from the medical history, physical examination, basic laboratory evaluation, and radiographic findings. However, additional diagnostic tests may be needed in the optimal assessment and management of shock. Intra-arterial pressure monitoring, echocardiography, and pulmonary artery catheterization are frequently utilized diagnostic modalities.

Brachial cuff measurements are often inaccurate in states of shock. Intra-arterial pressure monitoring provides a continuous assessment of the blood pressure and heart rate and allows for safe and effective titration of vasoactive medications.. Additional data may also be obtained by assessing the arterial waveform. Further, the pulse pressure (systolic blood pressure − diastolic blood pressure) may be helpful in differentiating various shock states.

The 2004 American College of Cardiology/American Heart Association (ACC/AHA) guidelines for the management of patients with ST segment elevation myocardial infarction state that intra-arterial pressure monitoring should be performed (class I indication) for severe hypotension (systemic arterial pressure less than 80 mmHg), during the administration of vasopressor and/or inotropic agents, and for cardiogenic shock [15]. Although the 2016 ACC/AHA guideline does not make any reference to intra-arterial pressure monitoring, it continues to be employed frequently for the same indications. Potential complications of intra-arterial pressure monitoring include pain, infection, hematoma, arterial obstruction, and arterial embolus.

Echocardiography is an invaluable tool in the assessment of shock. It may help determine the etiology of shock and guide management. Echocardiography can be utilized to assess left and right ventricular function and can detect tamponade, restrictive/constrictive physiology, severe valvular regurgitation or valvular stenosis, ventricular septal or free wall rupture, and proximal aortic dissection. Echocardiographic findings of a clot-in-transit and right ventricular dysfunction may suggest the presence of a hemodynamically significant pulmonary embolism.

Echocardiography using agitated saline is a sensitive diagnostic modality for detecting intracardiac and pulmonary vascular shunting. Hemodynamic parameters such as central venous pressure, pulmonary artery pressure, and left ventricular end-diastolic pressure can be estimated using conventional echocardiographic methods.

The pulmonary artery catheter can be valuable in determining the etiology of shock and may help in guiding management. Data obtained from the pulmonary artery catheter includes central venous pressure, right atrial pressure, right ventricular pressure, pulmonary artery pressure, and pulmonary capillary wedge pressure. Obtaining a mixed venous oxygen saturation permits the calculation of the cardiac output, cardiac index, and systemic venous resistance. Furthermore, important diagnostic information may also be determined by analyzing the pressure waveforms.

The hemodynamic data gathered with a pulmonary artery catheter can be utilized to titrate vasopressor therapy, assess hemodynamic effects of changes in mechanical ventilation (e.g., positive end expiratory pressure), and guide fluid resuscitation. In addition, the data may help differentiate between cardiogenic and noncardiogenic pulmonary edema when a trial of diuretic and/or vasodilator therapy has failed.

Pulmonary artery catheterization may aid in determining if pericardial tamponade is present when clinical assessment is inconclusive and echocardiography is not available. Findings of cardiac tamponade include diastolic equalization of pressures and blunted y descent of the arterial waveform (see Chap. 15). Other uses of pulmonary artery catheterization include assessment of valvular heart disease severity and reversibility of pulmonary vasoconstriction in patients being considered for heart transplant. An oximetry run may also be performed using a pulmonary artery catheter. This is important in the investigation of cardiac shunts, which may occur as a complication of myocardial infarction.

Despite its potential advantages, pulmonary artery catheterization has not been shown to broadly improve patient outcomes. Its use is therefore controversial and is not favored in some centers. Whether the lack of benefit on important outcomes is a result of the severity of illness in the patients for whom the use of this tool is contemplated, or a result of incorrect interpretation and use of the data obtained, is a debated topic. It is important to note that the Evaluation Study of Congestive Heart Failure and Pulmonary Artery Catheterization Effectiveness (ESCAPE) trial showed no significant difference in endpoints of mortality and days out of hospital in the management of congestive heart failure refractory to standard medical therapy [16]. However, this trial demonstrated that the use of the pulmonary artery catheter for this group of patients was safe.

In ESCAPE, the addition of pulmonary artery catheterization to careful clinical assessment was associated with a higher frequency of adverse events but did not affect overall mortality and hospitalization. Adverse events included implantable cardioverter-defibrillator firing, cardiogenic shock, ischemia/angina, pulmonary artery catheter infection, myocardial infarction, stroke or ischemic attack, cardiac arrest, and infection. The only individual event that was statistically different (p value < 0.05) between the groups was pulmonary artery catheter infection (p value $= 0.03$).

The external validity of the findings of the ESCAPE trial has been debated [17]. Many of the registry patients did not get randomized into the trial because pulmonary artery catheterization was deemed necessary for management by a study investigator. Subsequently, only patients with clinical equipoise in whom physicians were comfortable managing heart failure decompensation with or without hemodynamic monitoring were included in the study. As such, the study is not applicable to the critically ill heart failure

patients who are often considered for pulmonary artery catheterization.

The routine use of pulmonary artery catheterization in intensive care units is also controversial. A 2005 meta-analysis of 13 randomized trials including over 5000 critically ill patients showed that the use of pulmonary artery catheters was not associated with benefit or increased mortality [18]. However, the meta-analysis included patients who were critically ill from a wide variety of causes.

There have not been any randomized studies aimed at directly evaluating the utility of pulmonary artery catheters in patients presenting with CS. There were 2968 patients with cardiogenic shock enrolled in the Global Utilization of Streptokinase and Tissue Plasminogen Activator for Occluded Coronary Arteries (GUSTO-1) trial. Mortality among patients (n = 995) managed with PA catheters (45.2%) was less than that among patients (n = 1406) not managed with PA catheters (63.4%) [19].

A potential limitation of pulmonary artery catheter hemodynamic monitoring is that many physicians do not know how to correctly interpret findings from the device [20]. A 31-question multiple-choice exam was administered to 496 medical doctors at 13 different institutions to assess their knowledge and understanding of the use of the pulmonary artery catheter and interpretation of data derived from it. The examination was given unannounced at general meetings in the departments of medicine, anesthesiology, and surgery. The mean score was 67%; almost half of the responders (47%) could not read a pulmonary capillary wedge pressure from a clear tracing.

The 2013 ACCF/AHA guidelines for the management of heart failure provide guidance for appropriate pulmonary catheter use in hemodynamic assessment [21]. It is a class I indication to use pulmonary artery catheter monitoring to guide therapy in patients with respiratory distress or clinical evidence of impaired perfusion when intracardiac filling pressures cannot be determined by clinical assessment. The guidelines also state that pulmonary artery catheters "can be" useful (class IIa indication) in acute heart failure with persistent symptoms despite seemingly appropriate adjustments of standard therapies if fluid status, perfusion, systemic vascular resistance, or pulmonary vascular resistance is uncertain. Similarly, pulmonary artery catheters can be useful in the setting of renal dysfunction, vasoactive agent use, or mechanical circulatory support. The 2017 ACC/AHA/HFSA Focused Update of the aforementioned guideline did not provide additional guidance on the use of pulmonary artery catheterization.

Routine invasive monitoring of hemodynamics is not recommended in normotensive patients with acute decompensated heart failure responding appropriately to therapy [21]. Potential complications of pulmonary artery catheter use include infection, right bundle branch block, ventricular tachycardia, pulmonary artery rupture, and pulmonary infarction. Over time, pulmonary artery catheters tend to soften and migrate distally, leading to spontaneous wedging even when the balloon tip is not inflated.

Hemodynamic Assessment of Shock

A fundamental understanding of the hemodynamics of shock is critically important. The etiology of shock may not be evident despite the data acquired from the medical history, physical examination, basic laboratory evaluation, and radiographic findings. Hemodynamic data can help diagnose the correct etiology of shock and guide appropriate management.

Systemic tissue perfusion (blood pressure) is determined by the cardiac output (CO) and SVR. Similar to Ohm's law, whereas electrical current through a circuit is directly proportional to the potential difference across the circuit and inversely proportional to the resistance, cardiac output (CO) is directly proportional to the blood pressure difference across the systemic circulation (mean arterial pressure [MAP] − mean right atrial pressure [mean RAP]) and inversely proportional to SVR. Therefore [CO = (MAP − mean RAP)/SVR].

Cardiac output can be measured with a pulmonary artery catheter by utilizing either the Fick method or thermodilution technique (see

Table 21.2 Hemodynamic patterns classically associated with different categories of shock

	RA (mmHg)	RV (mmHg)	PA (mmHg)	PCWP (mmHg)	CI (L/min/kg/m^2)	PP (mmHg)	HR (bpm)	SVR (dynes)	SVO_2 (%)
Normal values	<6	<25/0–12	<25/0–12	<6–12	>2.5	40–50	60–100	800–1600	70
Cardiogenic	↑	↑	↑	↑ (>15)[a]	↓ (<2.2)	↑↓	↑	↑[b]	↓
Distributive	↑↓	↑↓	↑↓	↑↓	↑[c]	↑	↑[d]	↓	↑↓[e]
Hypovolemic	↓	↓	↓	↓	↓	↓	↑	↑	↓

RA right atrium, *RV* right ventricle, *PA* pulmonary artery, *PCWP* pulmonary capillary wedge pressure, *CI* cardiac index, *PP* pulse pressure, *HR* heart rate, *SVR* systemic vascular resistance, *SVO_2* mixed venous oxygen saturation

[a]In the SHOCK trial, 28% of patients had no auscultatory or radiographic evidence of pulmonary edema suggestive of elevated PCWP

[b]A systemic inflammatory response-like syndrome with a low SVR may be encountered in up to one-fifth of patients with acute myocardial infarction complicated by cardiogenic shock

[c]Cardiac output can be reduced in distributive shock due to myopathic processes such as severe acidosis or when preload is decreased because of inadequate intravascular volume

[d]Patients with neurogenic shock may have bradycardia due to sympathetic denervation

[e]SVO_2 is generally increased in sepsis due to poor oxygen utilization

Chap. 4). Different categories of shock can be discriminated using the calculated cardiac output. Whereas cardiac output is low in cardiogenic shock and hypovolemic shock, it is generally elevated in distributive shock (Table 21.2). However, cardiac output can also be reduced in distributive shock due to myopathic processes such as severe acidosis or when preload is decreased because of inadequate intravascular volume.

Systemic vascular resistance (SVR) can be calculated if the blood pressure, right atrial pressure, and cardiac output are known. The drop in arterial pressure across the systemic circulation divided by cardiac output is equal to SVR. [SVR = [MAP − mean RAP]/CO]. The units for SVR are mmHg/mL/m^2 (Woods units) and are typically multiplied by 80 to convert to dynes/cm^5 (dyn). Pulmonary vascular resistance can be calculated by substituting the drop in pressure across the systemic circulation with that of the pulmonary circulation (mean pulmonary artery pressure − mean pulmonary capillary wedge pressure). SVR is reduced in distributive shock but is generally elevated in CS and hypovolemic shock [22].

Mixed venous oxygen concentration (SVO_2) may help differentiate shock from its different categories. In distributive shock related to sepsis, SVO_2 is generally elevated because the mitochondria are unable to utilize oxygen appropriately. As a result, there is a higher than expected venous oxygen saturation. In other states of shock, SVO_2 is generally low.

Elevated pulmonary capillary wedge pressure (greater than 15 mmHg) has classically been used to distinguish CS from noncardiogenic causes of shock. However, in the SHOCK trial, 28% of patients had no auscultatory or radiographic evidence of pulmonary edema to suggest elevated PCWP [8]. Severe sepsis can cause myocardial depression and may elevate left-sided pressures as well. CS related to right ventricular infarction may be associated with marked hypotension, low cardiac output, and shock despite a normal pulmonary capillary wedge pressure.

The intra-arterial pressure waveform can help differentiate various shock etiologies. Pulse pressure equals the difference between systolic and diastolic blood pressure and is normally 40–50 mmHg (Fig. 21.2). The pulse pressure is a reflection of the stroke volume and the strength of each ventricular contraction.

In patients with CS related to left ventricular failure, the pulse pressure is reduced (see Fig. 21.3). A narrow pulse pressure, defined as a pulse pressure <25% of the systolic blood pressure, has a sensitivity and specificity of 91% and 83% for a cardiac index of <2.2 L/min/m^2 [23]. Other causes of narrow pulse pressure include profound intravascular volume loss, cardiac tamponade, and aortic stenosis.

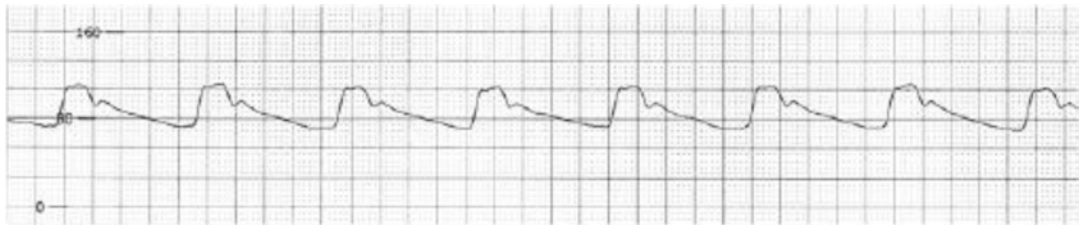

Fig. 21.2 Normal aortic waveform

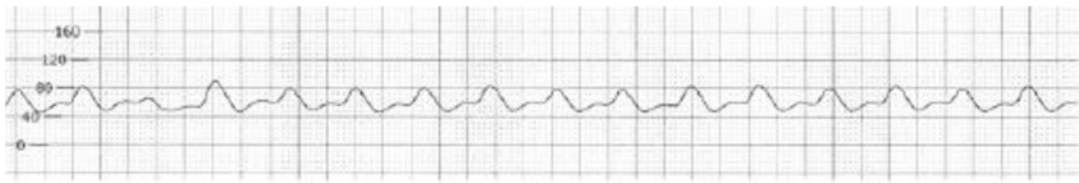

Fig. 21.3 Example of narrow pulse pressure in a patient with cardiogenic shock

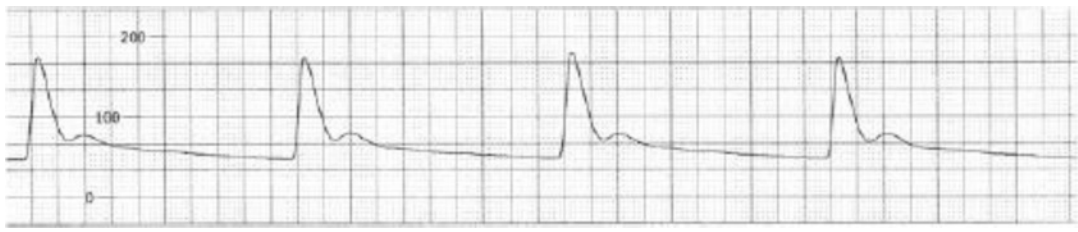

Fig. 21.4 Example of wide pulse pressure in a patient with complete heart block

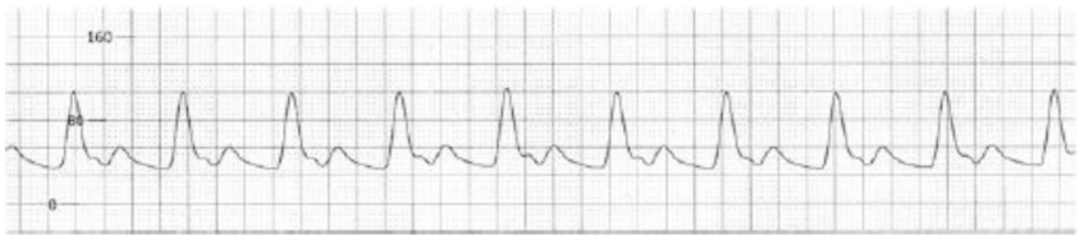

Fig. 21.5 Example of narrow pulse width in a patient with acute aortic dissection

Widened pulse pressure is a physiological response to exercise and can be seen pathologically with atherosclerosis, aortic insufficiency, complete heart block, aortic dissection, arteriovenous fistula, fever, anemia, thyrotoxicosis, pregnancy, and elevated intracranial pressure (see Fig. 21.4).

The pulse width is also a reflection of stroke volume. Patients in shock with normal or hypercontractile ventricles often have aortic waveforms with narrow pulse widths but normal pulse pressures. Both noncardiac (e.g., anaphylaxis and severe sepsis) and cardiac (e.g., tamponade, acute mitral regurgitation, post-myocardial infarction ventricular septal defect, and aortic dissection) etiologies of shock may be associated with narrow pulse widths. A narrow pulse has a spike appearance with a dicrotic notch that appears low (see Fig. 21.5).

Pulsus alternans occurs when there is an alternating rise and fall in systolic pressure

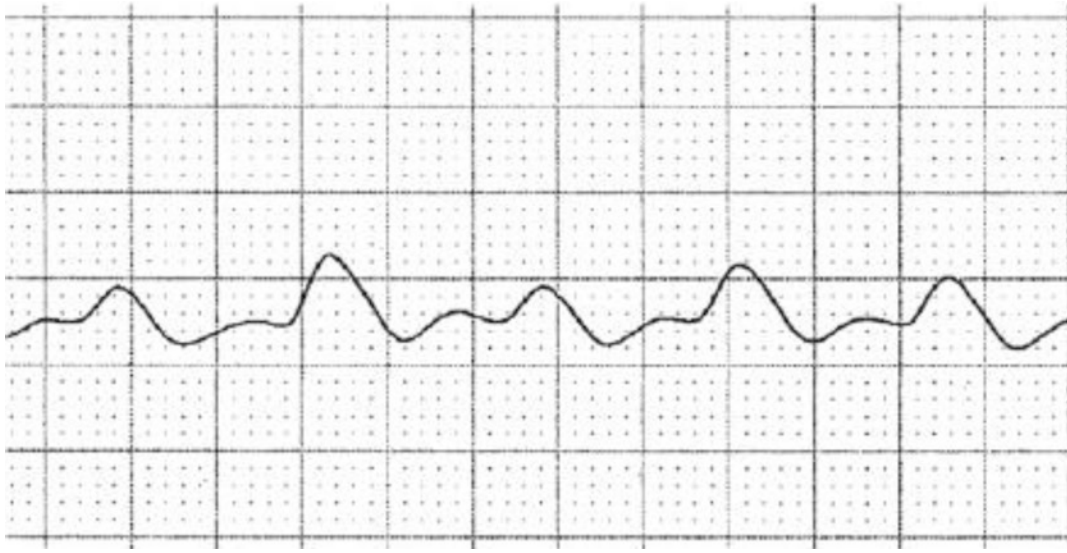

Fig. 21.6 Example of pulses alternans in a patient with advanced left ventricular failure

from beat to beat despite a regular rhythm (see Fig. 21.6). *Pulsus alternans* implies severe myocardial dysfunction and is most often seen in left ventricular failure. It may also occur in association with severe aortic stenosis and severe coronary artery disease. *Pulsus alternans* is thought to be due to variability in myocardial contractility on a beat-to-beat basis because of abnormal intracellular calcium cycling [24].

Pulmonary artery catheter intravascular waveform tracings can also help differentiate various etiologies of shock. Large *v*-waves on the pulmonary capillary wedge pressure tracing suggest shock from acute, severe mitral regurgitation. Likewise, large *v*-waves may be seen on the RAP tracing with severe tricuspid regurgitation. Cannon *a*-waves suggest complete heart block. Elevated and equalized diastolic pressures with loss of y descent suggest cardiac tamponade. Markedly elevated right atrial and right ventricular diastolic pressures and a normal pulmonary capillary wedge pressure suggest CS related to right ventricular infarction. An increase in right-sided pressures with inspiration (Kussmaul's sign) may be observed in right ventricular infarction due to decreased ventricular compliance.

Data obtained from the pulmonary artery catheter may reveal complications of acute myocardial infarction that occur in association with CS. For example, an oxygen step-up upon advancing the catheter from the right atrium to the right ventricle suggests left to right shunting related to ventricular septal rupture.

Arterial hypoxia may complicate shock. Evaluation of hypoxia begins with calculation of the alveolar-arterial (A-a) oxygen gradient. A normal A-a gradient equals 4 + (age/4) or 2.5 + (0.21 × age). Alveolar oxygen is calculated using the alveolar air equation: partial pressure alveolar oxygen (P_AO_2) = oxygen concentration (F_iO_2) × (barometric pressure at sea level (760 mmHg) − partial pressure water vapor (43 mmHg)) − (partial pressure carbon dioxide (P_ACO_2)/respiratory exchange ratio (0.8)). [$P_AO_2 = (F_iO_2 \times (760 - 43)) - (P_ACO_2/0.8)$]. Arterial partial pressure of oxygen is measured using conventional blood gas analysis.

If the A-a gradient is normal and P_aCO_2 is increased, hypoventilation is the suggested cause of hypoxia. With an elevated A-a gradient, proceed to check the mixed venous oxygen saturation. A low mixed venous oxygen saturation suggests hypermetabolism, anemia, or decreased cardiac output. If mixed venous oxygen is normal, administer 100% oxygen. If hypoxia corrects, ventilation/perfusion mismatch is suggested. Ventilation/perfusion mismatch

is caused by airway (e.g., asthma and chronic obstructive pulmonary disease), alveolar (e.g., pneumonia and congestive heart failure), and vascular (e.g., pulmonary embolism) phenomenon. Shunting is suggested if hypoxia does not correct with oxygen supplementation and may be physiological or vascular. Physiological shunting occurs with alveolar collapse (e.g., atelectasis) or decreased alveolar filling (e.g., pneumonia and congestive heart failure). Vascular shunting occurs with right-to-left intracardiac shunts (e.g., atrial or ventricular septal defect) and intrapulmonary shunts (e.g., arteriovenous malformation and hepatopulmonary syndrome).

Hemodynamic data can also be utilized to risk-stratify patients with acute myocardial infarction complicated by CS. In the SHOCK trial, cardiac power (cardiac power = MAP × CO/451) was the strongest independent correlate of in-hospital mortality in patients with CS [25].

Pearls of Assessment

There can be considerable overlap in the clinical presentation of the various categories of shock. Although classically associated with vasoconstriction and cold extremities, patients with CS can present with peripheral vasodilation and warm extremities. Indeed, in the SHOCK trial, the average SVR was not elevated, and the range of values was wide, suggesting that compensatory vasoconstriction is not universal [8].

Patients with distributive shock typically have increased cardiac output. However, in sepsis, depressed myocardial function may occur due to metabolic acidosis and other factors. Acidemia is detrimental to LV contractility and may result from decreased clearance of lactate by the liver, kidneys, and skeletal muscle, further compounded by an anaerobic metabolic state. Inadequate intravascular volume may complicate distributive shock and result in decreased cardiac preload and cardiac output.

Review Questions

1. Which of the following types of shock is typically associated with a high mixed venous oxygenation saturation?
   (a) Septic
   (b) Cardiogenic
   (c) Hypovolemic
   (d) Anaphylactic
   - Answer: (a) In severe sepsis, the mitochondrial respiratory chain does not utilize oxygen effectively. As a result there is more oxygen than expected in the venous blood.
2. Which of the following hemodynamic profiles is most consistent with cardiogenic shock?
   (a) Systolic blood pressure 120 mmHg, pulmonary capillary wedge pressure 10 mmHg, cardiac index 3.5 L/min/m^2, systemic vascular resistance (SVR) 1000 dyn, and mixed venous oxygen 75%
   (b) Systolic blood pressure 80 mmHg, pulmonary capillary wedge pressure 5 mmHg, cardiac index 1.3 L/min/m^2, SVR 1800 dyn, and mixed venous oxygen 60%
   (c) Systolic blood pressure 80 mmHg, pulmonary capillary wedge pressure 10 mmHg, cardiac index 3.5 L/min/m^2, SVR 600 dyn, and mixed venous oxygen 85%
   (d) Systolic blood pressure 80 mmHg, pulmonary capillary wedge pressure 20 mmHg, cardiac index 1.3 L/min/m^2, SVR 1800 dyn, and mixed venous oxygen 60%
   - Answer: (d) Hemodynamic criteria typically associated with cardiogenic shock include systolic blood pressure less than 90 mmHg for at least 30 min or need for vasopressor or intra-aortic balloon support to maintain systolic blood pressure greater than 90 mmHg, pulmonary capillary wedge pressure greater than 15 mmHg, and cardiac index less than 2.2 L/min/kg/m^2 [7].
3. An 85-year-old male presents with hypotension, oliguria, and confusion. An electrocardiogram reveals inferior ST segment elevation. A pulmonary artery catheter is inserted, and the hemodynamic parameters are consistent with cardiogenic shock. His lung fields are clear, and the jugular venous pulse is elevated.

A transthoracic echocardiogram reveals normal left ventricular function. What is the cause of this patient's clinical presentation?
(a) Right ventricular infarct
(b) Ventricular septal rupture
(c) Acute mitral regurgitation
(d) Tamponade
- Answer: (a) The classic findings of right ventricular infarction are hypotension, jugular venous distension, and clear lung fields.

4. An 85-year-old female presents 1 week after developing severe chest pain. She has dyspnea with minimal activity. She is hypotensive and has pulmonary rales. An electrocardiogram reveals anterior *q*-waves. Troponin is elevated but CK-MB is within normal limits. Other laboratory analysis reveals renal and liver injury. Pulmonary artery catheter hemodynamic findings are consistent with cardiogenic shock. An oxygen saturation run reveals an increased oxygen gradient upon advancing from the right atria to the right ventricle. Which complication of acute myocardial infarction is the cause of this patient's shock?
(a) Ventricular free wall rupture
(b) Ventricular septal rupture
(c) Acute mitral regurgitation
(d) Tamponade
- Answer: (b) Ventricular septal rupture may complicate myocardial infarction. An oxygen step-up upon advancing from the right atria to the right ventricle is typical.

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