CHAPTER 16

Pericardiocentesis

Haney A. Mallemat and Semhar Z. Tewelde

DEFINITION

Pericardial effusion, the presence of fluid within the pericardial space, has a number of causes. As fluid accumulates, a critical point is reached at which pericardial pressure negatively affects cardiac filling and causes circulatory insufficiency. This is called pericardial tamponade. Once compensatory mechanisms begin to fail, obstructive shock ensues and a failure to restore hemodynamics eventually leads to cardiac arrest. Only removal of fluid can stabilize hemodynamics at this point.

ANATOMY AND PHYSIOLOGY

Pericardium and Pericardial Space

The pericardium is a two-layered fibroelastic sac surrounding the heart.[1] The pericardium is avascular but well innervated, so inflammation induces pain. The visceral pericardium is a single-cell layer that adheres to the epicardium. The outer parietal pericardium consists mostly of collagen with some elastin. These two layers create the pericardial space, which normally contains 15 to 50 mL of serous fluid and is under negative pressure to promote filling of the right ventricle (RV) during diastole.[2] Pericardial fluid provides lubrication for cardiac contractility and acts as a "shock absorber" for deceleration forces.

The pericardium is a tense structure, but it also has some elasticity. These properties limit the amount of cardiac dilation that is possible during diastole and enhance mechanical interactions between the atria and ventricles during systole.[3] This semi-elastic property can also tolerate an acute (i.e., over a period of hours to days) accumulation of pericardial fluid (80 to 120 mL) without significantly increasing intrapericardial pressure, which is the flat portion of the pressure-volume curve for pericardial pressure (Fig. 16.1).[4,5] Once a critical volume is reached, adding as little as 20 to 40 mL can double intrapericardial pressure (the steep portion of the pressure-volume curve [see Fig. 16.1]) and cause clinical decompensation from cardiac tamponade. Cardiac tamponade typically occurs with an intrapericardial pressure of 15 to 20 mm Hg.[6]

Slow and chronic accumulation of pericardial fluid (over a period of weeks to months) causes the pericardium to expand circumferentially, and it can accommodate several liters of fluid with minimal alteration in intrapericardial pressure. Patients with this condition may be asymptomatic despite large effusions. No specific pericardial volume predicts the hemodynamic consequences of an effusion; such consequences depend on the acuity of the accumulation of the fluid.

Pericardiocentesis

Indications

Diagnostic
- Determining the cause of pericardial effusion

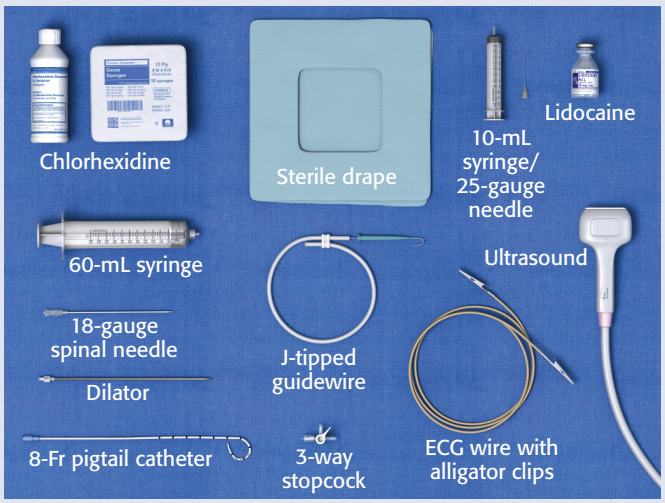
Therapeutic
- Shock/hemodynamic instability
- Cardiac arrest, pulseless electrical activity

Contraindications

Absolute
- None (if hypotension or hypoperfusion is evident or if the patient is in cardiac arrest)

Relative
- Coagulopathy
- Prosthetic heart valve
- Pacemakers and cardiac devices
- Lack of direct visualization (e.g., ultrasound) during the procedure
- Traumatic pericardium (thoracotomy preferred)

Equipment



Complications

- Dysrhythmias
- Intra-abdominal injury (e.g., liver injury)
- Air embolism
- Fluid reaccumulation
- Hemothorax
- Pneumothorax
- Pneumopericardium
- Costochondritis
- Suppurative pericarditis
- Coronary artery injury (puncture or laceration)
- Internal mammary artery injury
- Intercostal vessel or nerve injury
- Atrial or ventricular puncture or laceration

Review Box 16.1 Pericardiocentesis: indications, contraindications, complications, and equipment.

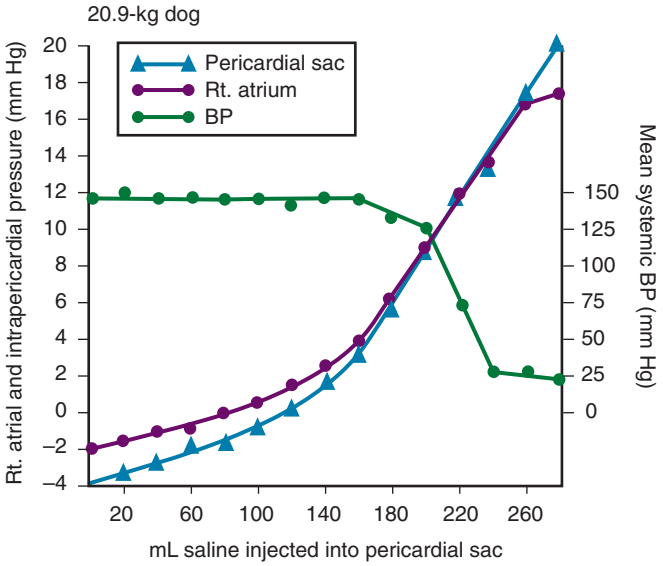


Figure 16.1 Production of cardiac tamponade by injection of saline into the pericardial sac. The pericardial space can accommodate the acute introduction of 80 to 120 mL of fluid without a significant increase in pericardial pressure, but with approximately 200 mL of saline, pressure increases steeply and rapidly (within minutes) and blood pressure (BP) drops. Once critical volumes are reached, very small increases cause significant hemodynamic compromise. (From Fowler NO: Physiology of cardiac tamponade and pulsus paradoxus. II: physiological, circulatory, and pharmacological responses in cardiac tamponade. *Mod Concepts Cardiovasc Dis*. 47:116, 1978. Reproduced by permission of the American Heart Association, Inc.)

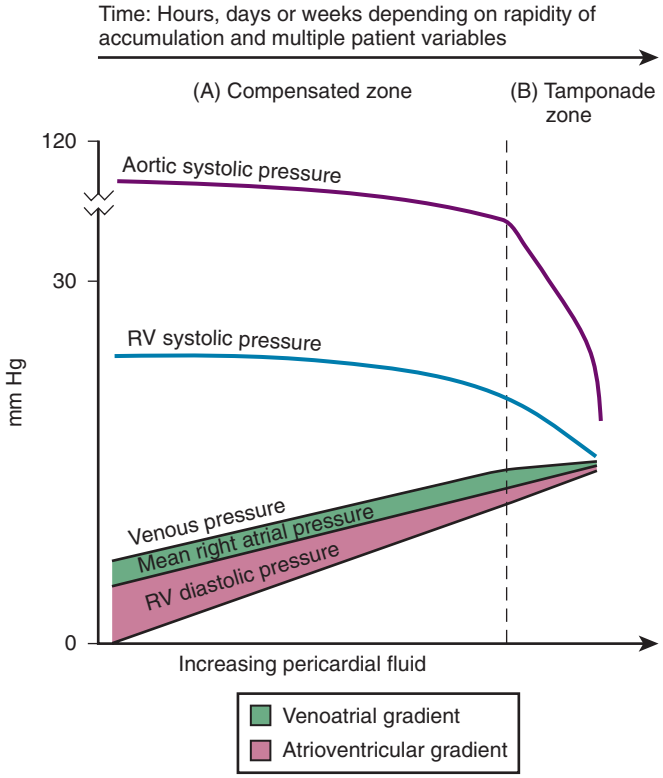


Figure 16.2 Summary of physiologic changes in tamponade. *RV,* Right ventricle. Note the initial slow changes, then the rapid decrease in systolic pressures once critical pericardial volume and pressure are reached. (From Shoemaker WC, Carey JS, Yao ST, et al: Hemodynamic monitoring for physiological evaluation, diagnosis, and therapy of acute hemopericardial tamponade from penetrating wounds, *J Trauma* 13:363, 1973; and Spodick D: Acute cardiac tamponade: pathologic physiology, diagnosis, and management, *Prog Cardiovasc Dis* 10:65, 1967. Reproduced by permission.)

Pathophysiology of Pericardial Tamponade

To generate an effective stroke volume, the left ventricle (LV) must be filled during diastole, and this process relies primarily on adequate filling of the RV. Normal inspiration makes the intrathoracic space more negative, which promotes RV filling by increasing venous return and causing dilation of the RV chamber secondary to a reduction in the tension in the RV free wall (Video 16.1).

Elevated intrapericardial pressure (i.e., early tamponade) first results in abnormal RV filling followed by abnormal LV filling. In this situation, the free wall of the RV cannot expand against the pericardial fluid during inspiration. To accommodate RV filling, the interventricular septum bows abnormally into the LV, which reduces its volume; this is also known as ventricular interdependence. LV filling, stroke volume, and ultimately distal tissue perfusion is reduced.[7] This phenomenon is responsible for pulsus paradoxus (PP; described later in this chapter), which is sometimes observed with tamponade.[8] LV filling is also reduced by the collapse of right-sided structures. After a critical volume is reached on the pressure-volume curve (see Fig. 16.1), the intrapericardial pressure is transmitted to the inferior vena cava (IVC) and right atrium. These thin-walled structures then become compressed and reduce filling of the RV.

The atria and pulmonary circulation are at much lower pressure than systemic arterial pressure and are also vulnerable to rising intrapericardial pressure (Fig. 16.2). Late in tamponade a "pressure plateau" occurs in which right atrial pressure, RV diastolic pressure, pulmonary artery diastolic pressure, and pulmonary capillary wedge pressure are virtually identical. This equalization of chamber pressure leads to a reduction in venous return and the echocardiographic hallmark of tamponade: diastolic collapse of the RV (discussed later in this chapter). At this point, hemodynamic collapse is imminent, with severe hypotension, and potentially pulseless electrical activity (PEA) developing. Unless intrapericardial pressure is decreased immediately, cardiac arrest will ensue.[9]

Compensatory Mechanisms and Pericardiocentesis

To maintain a physiologic cardiac output early in tamponade, the sympathetic nervous system increases the heart rate, arterial vasoconstriction (to maintain mean arterial blood pressure), and venoconstriction (to maintain normal venous-atrial and atrioventricular filling gradients). Early in tamponade, these compensatory mechanisms are usually effective in maintaining adequate cardiac output.[7]

Compensatory mechanisms also preserve normal cardiac contractility and myocardial perfusion.[10,11] However, when the pericardial pressure overwhelms the compensatory mechanisms, coronary perfusion pressure is reduced, which leads to myocardial ischemia, systolic dysfunction, and ultimately a reduction in cardiac output. Experimental induction of severe tamponade

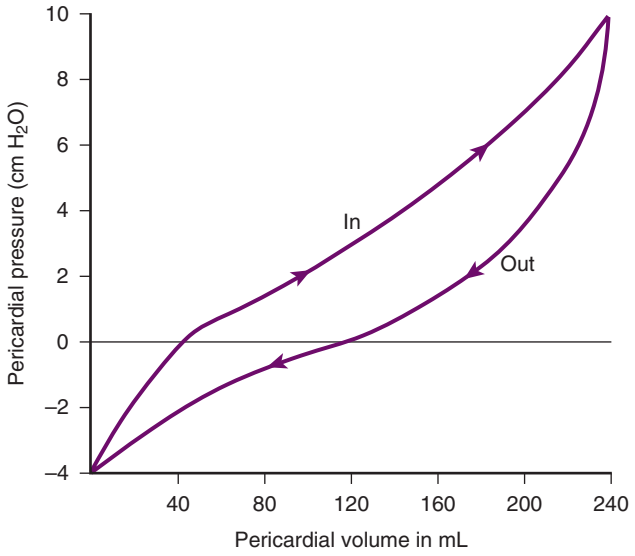


Figure 16.3 Relationship of intrapericardial pressure to volume of pericardial fluid. Pressure drops rapidly when a small amount of fluid is removed, hence the initial significant benefit of pericardiocentesis. (From Pories W, Gaudiani V. Cardiac tamponade: *Surg Clin North Am* 55:573, 1975. Reproduced by permission.)

demonstrated microscopic ischemic cardiac injury.[12] Lactic acidosis (resulting from reduced cardiac output and systemic hypoperfusion) also leads to further reduction in cardiac contractility and, ultimately, cardiac output.[9]

Removal of pericardial fluid (i.e., pericardiocentesis) reverses the pathophysiologic processes just described by improving coronary filling, cardiac filling, and hence cardiac output. Interestingly, the pressure-volume relationship of the pericardial space demonstrates hysteresis; that is, withdrawing a certain quantity of fluid reduces intrapericardial pressure more than addition of the same amount of fluid increases intrapericardial pressure. This effect, however, is not universal and may vary among patients and in various disease states (Fig. 16.3).[2]

Special Considerations in Patients With Pericardial Effusion and Tamponade

Under normal circumstances, positive pressure ventilation (e.g., mechanical ventilation) reduces venous return to the right side of the heart by increasing intrathoracic pressure. This could be detrimental for patients with tamponade because right-sided filling is already compromised and further reductions can lead to severe hemodynamic instability.[13] Therefore positive pressure ventilation (i.e., noninvasive or invasive) should be avoided in patients with known or suspected tamponade unless it is absolutely necessary (e.g., respiratory failure).

Low-pressure pericardial tamponade is defined as a hemodynamically significant effusion with an intrapericardial pressure that is lower than expected.[14] This category of tamponade occurs in patients with subacute or chronic effusions, but present with a superimposed hypovolemia (e.g., associated with long-term diuretic use, dehydration, or excessive dialysis).[15] The diagnosis may be challenging because the classic symptoms and findings on physical examination (e.g., distended neck veins) may be absent.[16] Ultrasound is most helpful in making the diagnosis in these patients. Fluid boluses may temporize the hemodynamic compromise while pericardial decompression is being arranged.

EPIDEMIOLOGY

The major categories of pericardial effusion include infection, inflammation, malignancy, trauma, and metabolic abnormalities. Effusion may also be associated with aortic disease, connective tissue disease, or idiopathic causes. It is often difficult to report the exact incidence of each type of pericardial effusion because of variations in patient populations, local epidemiology, and the diagnostic protocols used during evaluation. The prevalence of a chronic effusion is also difficult to ascertain because it is often asymptomatic and underreported. General autopsy studies demonstrate an overall prevalence of 3.4%.[17]

CAUSES OF PERICARDIAL EFFUSION (BOX 16.1)

Acute Hemopericardium

Acute hemopericardium, or rapid accumulation of blood in the pericardial space, can have a traumatic or nontraumatic etiology. It is one of the most feared causes of tamponade because the semi-elastic pericardium cannot accommodate acute increases in pericardial fluid and clinical deterioration can be rapid. This diagnosis can be challenging to make because there may be little or no evidence during the initial evaluation (e.g., pericardial size might be normal on a chest radiograph). Common causes include trauma and aortic dissection retrograde into the pericardial sac.

Traumatic Hemopericardium

Penetrating Trauma

Penetrating cardiac trauma can cause acute hemopericardium by either external forces (e.g., a stab wound to the heart) or internal forces (e.g., iatrogenic injury during placement of a pacemaker). Cardiac perforation can lead to rapid clinical deterioration and PEA.

External cardiac puncture is associated with stab wounds or projectile injuries (e.g., gunshot wounds). Tamponade develops in 80% to 90% of patients with cardiac stab wounds as opposed to 20% of those with gunshot wounds.[18,19] Stab wounds cause tamponade more frequently because if the pericardial injury is small, it can reseal and trap blood within the pericardial space.[20] On the other hand, a gunshot typically produces both large myocardial and pericardial wounds that allow continuous drainage into the mediastinal and pleural space.[21] Clinical deterioration is usually secondary to hypovolemia.[21] Any penetrating injury to the chest, back, or upper part of the abdomen may injure the pericardium and cause tamponade.

Internal penetrating trauma is typically caused by invasive diagnostic or therapeutic procedures. The procedures most often associated with this injury are cardiac catheterization (angioplasty or valvuloplasty) and pacemaker insertion.[22–24] Hemopericardium results from puncturing a cardiac chamber, a coronary artery, or a great vessel (e.g., the superior vena cava). Ironically, pericardiocentesis itself (treatment of a pericardial effusion) can cause hemopericardium if a coronary vessel or the myocardium is injured during the procedure.[25,26]

BOX 16.1 Causes of Pericardial Effusion

NEOPLASM
Mesothelioma
Lung
Breast
Melanoma
Lymphoma

PERICARDITIS
Radiation related (especially after Hodgkin's disease)
Viral
Bacterial
- *Staphylococcus*
- *Pneumococcus*
- *Haemophilus*

Fungal
Tuberculosis
Amebiasis
Toxoplasmosis
Idiopathic

CONNECTIVE TISSUE DISEASE
Systemic lupus erythematosus
Scleroderma
Rheumatoid arthritis
Acute rheumatic fever

METABOLIC DISORDERS
Myxedema
Uremia
Cholesterol pericarditis
Bleeding diatheses

CARDIAC DISEASE
Acute myocardial infarction
Dissecting aortic aneurysm
Congestive heart failure
Coronary aneurysm

DRUGS
Hydralazine
Phenytoin
Anticoagulants
Procainamide
Minoxidil
Doxorubicin
Daunorubicin
Bleomycin

TRAUMA
Blunt
- Major trauma
- Closed-chest cardiopulmonary resuscitation

Penetrating
- Major penetrating trauma

Intracardiac injections
Transthoracic and transvenous pacing wires
Pericardiocentesis
Cardiac catheterization
Central venous catheters

MISCELLANEOUS
Serum sickness
Chylous effusion
Löffler's syndrome
Reiter's syndrome
Behçet's syndrome
Pancreatitis
Postpericardiotomy
Amyloidosis
Ascites

Data from Guberman BA, Fowler NO, Engel PJ, et al: Cardiac tamponade in medical patients, Circulation 64:633, 1981; and Pories WJ, Caudiani VA: Cardiac tamponade, Surg Clin North Am *55:573, 1975.*

Internal jugular and subclavian venous catheters (e.g., central venous or hemodialysis catheters) are commonly inserted in the emergency department (ED). During such procedures, hemopericardium results from perforation of the superior vena cava, right atrium, or RV. Hemopericardium can occur immediately or can be delayed for days subsequent to erosion of the catheter through myocardial or vascular tissue.[27,28] Although this complication seldom occurs, it should always be considered when a patient experiences sudden hemodynamic deterioration following an invasive procedure.

Blunt Trauma

Major blunt chest trauma can cause hemopericardium with or without obvious signs of injury, from rupture of a cardiac chamber or, less commonly, damage to a coronary artery.[29] Myocardial rupture can be uncontained or contained.[30,31] Patients with uncontained rupture do not typically survive long enough to reach the hospital.[32] Contained rupture may be found soon after injury or may be a late finding.[33] Tamponade can also be caused by a deceleration mechanism of injury that induces either aortic or vena caval disruption.[34] In one case series the incidence of tamponade following deceleration injury was found to be 2.3% (1 in 43 patients).[35]

Miscellaneous Trauma

Chest compressions during cardiopulmonary resuscitation (CPR) can also cause hemopericardium from broken ribs or bleeding intercostal vessels.[36] Hemopericardium following CPR has been described in case reports[37,38] but is unlikely to be significant, much less to cause tamponade.

Atraumatic Hemopericardium

Atraumatic hemopericardium is difficult to diagnose. Diagnosis is often delayed because it occurs spontaneously and the clinical findings can be less obvious than those of hemopericardium from traumatic causes. Maintain a high index of suspicion for this condition in patients with risk factors, such as certain malignancies (e.g., lung cancer) or tuberculosis within endemic areas. Common causes of atraumatic hemopericardium are discussed later.

Bleeding diathesis is an important cause of spontaneous hemopericardium and may be associated with the use of anticoagulants (reported incidence of 2.5% to 11%)[22] or thrombolytic therapy (incidence <1%).[39] Patients who have undergone cardiac surgery are at increased risk because of the anticoagulative effects of the cardiopulmonary bypass machine and medications started postoperatively (e.g., clopidogrel, warfarin).[40] Fortunately, tamponade has a low incidence and is generally detected in the postoperative period before discharge.[41,42] This complication is usually prevented by the intraoperative placement of mediastinal or pericardial drains.[22,43]

Hemopericardium can develop following myocardial infarction (MI). Early after a transmural MI (1 to 3 days), the necrotic myocardium causes inflammation of the overlying pericardium and then effusions can form. Late-developing effusions (weeks after an MI) are caused by an autoimmune pericarditis called Dressler's syndrome.[1] Improved reperfusion techniques have drastically reduced the incidence of post-MI pericarditis and effusion.[44]

Ascending aortic dissection causes rapid and usually fatal hemopericardium. The dissection may expand in a retrograde fashion by extending to the base of the aorta and into the pericardial sac. This is a very difficult diagnosis, and best visualized and comfirmed by bedside ultrasound. Risk factors for aortic dissection include hypertension, atherosclerosis, vasculitis (e.g., giant cell arteritis, syphilis), collagen vascular disease (e.g., Marfan's syndrome), and the use of sympathomimetics (e.g., cocaine).[45–47]

Ventricular free-wall rupture is a rapidly fatal cause of acute hemopericardium that can occur after MI leading to cardiac failure and shock. This complication is less common today than in the past (<1%)[39] secondary to improved revascularization techniques, better therapeutic medications, and faster intervention times (shorter door-to-balloon times) for coronary ischemia. Despite a reduction in its overall incidence, 7% of all deaths related to MI are caused by this complication.[48,49] Survival is theoretically possible with prompt recognition and treatment, but the prognosis is grim once tamponade occurs.[50,51]

Nonhemorrhagic Effusions

Nonhemorrhagic pericardial effusions usually accumulate slower than acute hemopericardium does (over a period of weeks to months). Chronic fluid accumulation allows the pericardium to stretch circumferentially and accommodate up to 2000 mL of fluid without any hemodynamic compromise.[52] Effusions that grow slowly allow the circulatory system to adapt to increased intrapericardial pressure, thereby further maintaining hemodynamic stability. Thus, asymptomatic patients with moderate to large effusions may not need emergency pericardiocentesis, in contrast to patients with acute hemopericardium.[53,54]

Nonhemorrhagic effusions have several causes (see Box 16.1), and the exact one may not be obvious during the initial evaluation without diagnostic pericardiocentesis. Common causes of nonhemorrhagic effusions are discussed in the following sections.

Idiopathic Effusions

Most idiopathic effusions are believed to be viral in origin and most commonly caused by infection with coxsackievirus, echovirus, cytomegalovirus, or human immunodeficiency virus (HIV) (discussed later in the chapter). Idiopathic pericardial effusions may be asymptomatic or have an associated component of pericarditis (e.g., positional pain or diffuse ST-segment changes on the electrocardiogram [ECG]).[55] These effusions are often labeled *idiopathic* because the diagnosis cannot be made noninvasively (i.e., based on the history, physical examination, or serum testing) and the risk associated with diagnostic pericardiocentesis often outweighs the risk of observation in asymptomatic adults who appear to be well.[56] Diagnostic pericardiocentesis may be recommended for idiopathic effusions that are persistent or symptomatic without a known cause.[57]

Neoplastic Effusion

Tumors of the pericardium or myocardium may cause nonhemorrhagic effusions.[54,58] Primary cardiac tumors are less common (0.001% to 0.003%) than metastases from another site (2% to 18%), but either may cause a malignant effusion.[59] Although no malignancy preferentially metastasizes to the heart, certain tumors commonly involve the heart when they metastasize; frequently implicated are lung cancer, breast cancer, mediastinal tumors, malignant melanoma, leukemia, and lymphoma.[46]

Cardiac metastasis is usually a late finding in cancer, as other foci are generally evident first.[47] The classic signs and symptoms of tamponade (e.g., chest pain and dyspnea) may not be obvious with malignant tamponade. When present, they may be mistakenly attributed to the underlying malignancy.[46] Thus, in the relevant clinical scenario, consider screening patients with malignancy for pericardial effusion (e.g., ultrasound) before the clinical findings of tamponade appear.

Radiation

Pericardial effusions (secondary to radiation-induced pericarditis) can develop acutely during radiation therapy or may be delayed for years. Risk factors include the radiation dose, duration of exposure, and age of the patient. Patients treated with radiation for Hodgkin's disease have the highest association of radiation-induced pericarditis and subsequent effusions.[22] These effusions can be serous, hemorrhagic, or fibrinous.[57]

Congestive Heart Failure

Congestive heart failure (CHF) is a cause of pericardial effusion. Diagnosis may be difficult because of overlapping signs and symptoms with exacerbations of CHF (e.g., chest pain or dyspnea). Adding to the diagnostic complexity is that 12% to 20% of patients with CHF have a coexisting pericardial effusion.[60] Fortunately, treatment of CHF-associated pericardial effusion does not differ from that for an effusion from other causes: treat the underlying cause unless the patient has evidence of hemodynamic compromise.

HIV-Associated Effusions

HIV can cause nonhemorrhagic pericardial effusion and tamponade.[61,62] The incidence has been reported to be approximately 11% in patients with HIV infection or acquired immunodeficiency syndrome, and 13% of cases are classified as moderate to severe. It is unclear whether antiretroviral therapy has affected these data.[17]

HIV-related effusions have been attributed to bacterial (e.g., *Staphylococcus aureus*), viral (e.g., cytomegalovirus), fungal (*Cryptococcus neoformans*), and mycobacterial causes (e.g., tuberculosis, which is the most common cause of HIV-related effusions worldwide).[63] Kaposi's sarcoma and lymphoma can cause noninfectious pericardial effusions in HIV patients.[64,65]

Renal Failure and Uremia

Pericardial effusion develops in approximately 15% to 20% of dialysis patients, and tamponade may eventually occur in as many as 35% of those people.[66,67] Up to 7% of chronic dialysis patients have effusions with volumes of 1000 mL or greater.[68] In many cases, effusions secondary to renal failure can be managed solely with aggressive dialysis without pericardiocentesis. Any sign of hemodynamic compromise, however, warrants strong consideration of pericardiocentesis.

Hypothyroidism

Hypothyroid patients are at risk for pericardial effusions (up to 30%), but the fluid accumulates gradually, so tamponade develops in only a few patients.[54] If a pericardial effusion is present, other areas of the body usually demonstrate serositis (e.g., pleural effusions). Medically managing the underlying hypothyroidism often reverses the effusion without the need for pericardiocentesis.

Special Considerations in Pericardial Disease

Pericardial tamponade is classically described as being secondary to circumferential effusion, which causes a generalized increase in pericardial pressure and compression of multiple cardiac chambers. Loculated effusions (caused by a local hematoma or an infectious process) or pericardial adhesions (from previous inflammation) can lead to tamponade by compressing one or more cardiac chambers and thus reducing both cardiac filling and cardiac output.[69,70]

Constrictive pericarditis occurs following chronic pericardial inflammation, infection, or mediastinal irradiation. These processes cause scarring, fibrosis, or calcification, and the pericardium eventually becomes a nonelastic and "constrictive" sac around the heart. Myocardial relaxation and cardiac filling are impaired, and diastolic dysfunction ensues. Without echocardiography, constrictive pericarditis can be difficult to distinguish from pericardial tamponade.[1]

Effusive-constrictive pericarditis is defined by the presence of both pericardial effusion and pericardial constriction. It may be quite difficult to differentiate between effusive-constrictive pericarditis and pericardial tamponade in stable patients because both are associated with effusions.[71] Fortunately, distinguishing between these diagnoses is less important in hemodynamically unstable patients because they are treated identically (i.e., with pericardiocentesis).[72]

Pneumopericardium is an interesting, though rare cause of cardiac tamponade. It is most commonly associated with pneumothorax caused by barotrauma (e.g., mechanical ventilation).[73] It also occurs spontaneously during acute asthma exacerbations,[74] and it can follow blunt chest injury.[75,76] Although typically benign, tension pneumopericardium has been reported as a cause of life-threatening tamponade after blunt[77,78] and penetrating chest trauma.[79,80]

DIAGNOSING CARDIAC TAMPONADE

Diagnosis of pericardial effusions requires careful integration of the patient's history, findings on physical examination, and diagnostic testing. Unfortunately, even experienced clinicians may not initially consider pericardial effusion because the clinical findings are often vague and nonspecific. Nonspecific symptoms, such as chest pain and dyspnea, can be ascribed to more common conditions (e.g., CHF or pulmonary pathology), so the diagnosis might be delayed until diagnostic testing is performed (e.g., computed tomography [CT] of the chest for pulmonary embolism),[81] or until hypotension develops and bedside ultrasound is performed.[82]

Acute pericardial tamponade (e.g., secondary to blunt chest wall trauma) is usually challenging to diagnose because the findings on physical examination may resemble those of other life-threatening conditions (e.g., tension pneumothorax, hemothorax, hypovolemia, pulmonary edema, severe contusion of the RV, aortic dissection, or pulmonary embolism).[67] In hemodynamically unstable patients, diagnostic (e.g., bedside ultrasound) and therapeutic (e.g., pericardiocentesis) interventions must be performed even with a paucity of findings on physical examination because rapid clinical deterioration and cardiac arrest can occur before a definitive diagnosis can be made.

Once a pericardial effusion is suspected (or diagnosed), the next step is to determine its size and hemodynamic significance and presence of underlying or associated diseases.[83] Specific therapies will hinge on this information and is discussed in the following sections.

History: Patient Profile and Symptoms

The historical features of pericardial effusions are nonspecific and the diagnosis may easily be overlooked. An astute clinician might be suspicious based on comorbid conditions (e.g., warfarin therapy or a history of myxedema) and the time course of the symptoms (e.g., free-wall rupture several days after MI, dyspnea in a patient with uremic pericarditis). Patients are likely to present with symptoms relating to the underlying disease rather than the pericardial effusion itself. Box 16.2 lists important details to be ascertained from the history when pericardial effusion is suspected.[84]

Physical Examination

Physical examination of patients with pericardial effusion (e.g., displaced point of maximal impulse, muffled heart sounds) lacks sensitivity and specificity. If the history suggests pericardial effusion, the physical examination should focus on determining the underlying cause (e.g., stigmata of hypothyroidism) to guide definitive diagnostic testing (e.g., echocardiography). Ironically, many pericardial effusions are not diagnosed from the history or findings on physical examination but are found incidentally during the evaluation for other diseases.

In 1935, Beck characterized the physical manifestations of tamponade with two triads, one for chronic and one for acute tamponade.[85] Beck's chronic triad consists of increased central venous pressure (CVP) (i.e., distended neck veins), ascites, and a small, quiet heart. *Beck's triad* is a classic description of acute cardiac compression, which includes increased CVP, decreased arterial pressure, and muffled heart sounds. Almost 90% of patients have one or more of these "acute" signs,[86] but only approximately 33% demonstrate the complete triad.[9,87] The utility of this triad is further limited because all three signs are usually observed shortly before cardiac arrest.

It would be clinically desirable to identify patients in early tamponade, before hemodynamic collapse. Unfortunately, findings on physical examination in early tamponade are nonspecific and may be indistinguishable from those of other critical diseases (e.g., septic shock, right heart failure).[57] Patients

BOX 16.2 Information to Obtain from Patients When Pericardial Effusion Is Suspected

ONSET AND DURATION OF SYMPTOMS

Acute

Trauma
Recent cardiac surgery
Recent myocardial infarction
Acute aortic dissection
Recent diagnostic or therapeutic intervention (e.g., catheterization)
Recent upper thoracic vascular procedure (e.g., hemodialysis catheter, central line, peripherally inserted central catheter line, Mediport)
Recent placement of a pacemaker or automatic implantable cardioverter-defibrillator

Subacute/Chronic

Metabolic (e.g., uremia)
Endocrine (e.g., hypothyroidism)
Infectious
- Viral (e.g., human immunodeficiency virus)
- Bacterial (e.g., *Staphylococcus*)
- Fungal (e.g., *Aspergillus*)

Neoplastic
- Primary cardiac
- Metastatic

Autoimmune disorders (e.g., lupus)
Inflammatory
- Vasculitis

MEDICAL/SURGICAL HISTORY

Autoimmune disorders
- Lupus
- Mixed connective tissue disease
- Vasculitis

Endocrine disease
- Hypothyroidism
- Ovarian hyperstimulation syndrome

Metabolic diseases
- End-stage renal disease and uremia
- Thyroid disorders
- Coagulopathies

Artificial cardiac valves
- Anticoagulation medications
- Risk for myocarditis

Cardiac disease
- Anticoagulation medications
- Pericarditis
- Aneurysm

Recent placement of a vascular catheter
Recent cardiac surgery
Recent cardiac intervention
Recent thoracic radiation

MEDICATIONS

Anticoagulants
- Aspirin
- Warfarin
- Clopidogrel

Antiarrhythmic
- Procainamide (drug-induced lupus)

Tuberculosis therapy
- Isoniazid (drug-induced lupus)

COMMON SYMPTOMS

Altered mental status, confusion
Fatigue
Dizziness, light-headedness
Orthostatic changes
Exercise intolerance
Hoarseness
Hiccups
Fever
Chills
Chest pain
- Substernal
- Pleuritic
- Positional
- Scapula (phrenic nerve irritation)
- Palpitations
- Cough
- Dyspnea
- Myalgia
- Arthralgia

initially seen in late tamponade also have nonspecific findings. They may be agitated, panic-stricken, confused, uncooperative, restless, cyanotic, diaphoretic, acutely dyspneic, or hemodynamically unstable. Such patients should undergo a brief and focused physical examination followed by a rapid hemodynamic assessment with bedside ultrasound because the time between initial evaluation and full arrest may be brief.

Some of the findings on physical examination associated with tamponade are described later. A more comprehensive list is presented in Box 16.3.

Vital Sign Abnormalities

There are three sequential stages that are typically described to reflect the natural history of acute tamponade (Table 16.1).[88] The time course within each stage varies from patient to patient. Some patients are stable within a given stage for hours, whereas others proceed through all three stages and develop cardiac arrest within minutes.[9,88] Grade I tamponade is characterized by normal blood pressure and cardiac output with an increase in the heart rate and CVP (measured invasively with a central venous catheter). Grade II tamponade is defined by normal or slightly reduced blood pressure; CVP and the heart rate remain increased. Grade III tamponade is identified on the basis of Beck's triad: hypotension, tachycardia, and elevated CVP.

Nearly all patients with tamponade present with sinus tachycardia, although its specificity is low.[89] The physiologic purpose of tachycardia is to maintain normal cardiac output

BOX 16.3 Physical Examination Findings Suggestive of Pericardial Effusion or Tamponade

VITAL SIGNS
Normal vital signs
Tachycardia (tamponade)
Hypotension (tamponade)
Narrow pulse pressure
Pulsus paradoxus
Fever (if the cause is infectious or neoplastic)

APPEARANCE
Normal appearance
Anxiety
Sense of impending doom
Diaphoretic
Cold and clammy
Pallor
Altered mental status, confusion

NECK
Supraclavicular retractions
Distended neck veins (may be flat with hypotension)

CARDIOVASCULAR
Normal findings on examination
Tachycardia
Increased pain with a supine position, relieved by leaning forward
Muted, distant heart sounds
Pericardial friction rub (inflammatory pericarditis)
Displaced point of maximal impulse
Increased cardiac borders with percussion
Distended neck veins
Auscultatory dullness along the left scapular area

PULMONARY
Telegraphic speech
Respiratory distress
Supraclavicular retractions, abdominal breathing
Cough
Hoarseness
Clear breath sounds (may distinguish tamponade from congestive heart failure)

ABDOMINAL
Hepatomegaly
Splenomegaly

SKIN
Cool extremities
Clammy extremities
Dilated head and scalp veins
Peripheral edema
Anasarca

TABLE 16.1 Shoemaker System of Grading Cardiac Tamponade

GRADE	PERICARDIAL VOLUME (mL)	CARDIAC INDEX	STROKE INDEX	MEAN ARTERIAL PRESSURE	CENTRAL VENOUS PRESSURE	HEART RATE	BECK'S TRIAD
I	<200	Normal or ↑	Normal or ↓	Normal	↑	↑	Venous distention, hypotension, muffled heart sounds usually not present
II	≥200	↓	↓	Normal or ↓	↑ (≤12 cm H_2O)	↑	May or may not be present
III	>200	↓↓	↓↓	↓↓	↑↑ (30–40 cm H_2O)	↑	Usually present

From Shoemaker WC, Carey SJ, Yao ST, et al: Hemodynamic monitoring for physiologic evaluation, diagnosis, and therapy of acute hemopericardial tamponade from penetrating wounds, *J Trauma* 13:36, 1973.

despite reductions in stroke volume from worsening tamponade. Exceptions to the pairing of tachycardia with tamponade usually relate to the underlying cause of the effusion (e.g., myxedema) or the concomitant use of certain medications (e.g., β-blockers).

Adding to the diagnostic complexity, not all patients in tamponade have a reduction in blood pressure. In fact, Brown and co-workers[90] described several tamponade patients with elevated blood pressure. These patients were previously hypertensive and paradoxically had reduced systolic blood pressure following pericardiocentesis.

Pulsus Paradoxus

PP is an exaggerated decrease in systolic blood pressure (>12 mm Hg) during inspiration secondary to reduced stroke volume (Fig. 16.4*A*).[30,91,92] Patients with moderate to severe tamponade typically demonstrate PP greater than 20 mm Hg.[9,86,93] Unfortunately, PP is not pathognomonic for tamponade. It is observed in other conditions, such as hypotension associated with labored breathing (secondary to extreme reductions in intrathoracic pressure) (see Fig. 16.4*B*), severe emphysema, severe asthma, obesity, cardiac failure, constrictive pericarditis, pulmonary embolism, and cardiogenic shock.[9,86,93]

The absence of PP does not rule out tamponade because it can occur with several conditions: atrial septal defects, aortic insufficiency, positive pressure ventilation, loculated pericardial effusions, and elevated left ventricular diastolic pressure (e.g., poor left ventricular compliance secondary to chronic hypertension).[14] Finally, PP should be interpreted with caution in patients

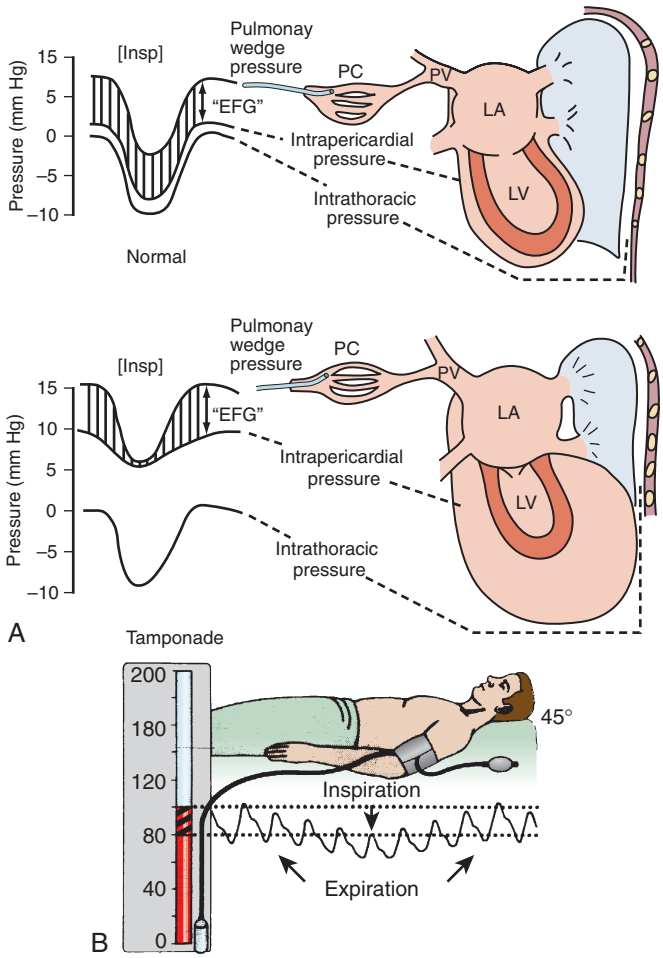


Figure 16.4 Pulsus paradoxus to diagnose pericardial tamponade. **A,** *Top*, The normal situation in which changes in intrathoracic pressure are transmitted to both the pericardial sac and the pulmonary veins. The effective filling gradient (EFG) changes only slightly during respiration. *Bottom*, Cardiac tamponade in which changes in intrathoracic pressure are transmitted to the pulmonary veins but not to the pericardial sac. The EFG falls during inspiration (Insp). *LA*, Left atrium; *LV*, left ventricle; *PC*, pulmonary capillaries; *PV*, pulmonary veins. **B,** Normally, systolic blood pressure drops slightly during inspiration. To assess for pulsus paradoxus, have the patient breathe normally while lying at a 45-degree angle. Inflate the blood pressure cuff well above systolic pressure and slowly deflate it. When the pulse is first heard only during expiration, this is the upper value. Deflate the cuff until the pulse is heard during both inspiration and expiration; this is the lower value. A difference of more than 12 mm Hg between the two values indicates pulsus paradoxus. (**A,** Adapted from Sharp JT, Bunnell IL, Holand JF, et al: Hemodynamics during induced cardiac tamponade in man, *Am J Med* 25:640, 1960.)

with traumatic tamponade because it may not be present.[94–96] In a study of 197 patients with traumatic tamponade, only 8.6% had PP.[97]

Measuring PP is useful only occasionally because it is difficult to perform, time-consuming, and not specific or sensitive for tamponade. Its description here is for historical value and for use in hemodynamically stable patients. When managing unstable patients, especially those in extremis, assessment for PP should not replace more definitive testing, such as bedside ultrasound.

Neck Vein Distention and Elevated CVP

Neck vein distention (a surrogate for measuring CVP) occurs late in tamponade, when right-sided chambers (e.g., the RV) collapse. Neck vein distention may be obvious on examination (Fig. 16.5*A*), but visualization of such distention is less accurate than measuring CVP by central venous catheter or evaluation of the IVC with ultrasound (Table 16.2). Patients with significant tamponade typically have a CVP of 12 cm H_2O or higher.[95] Finally, although initial CVP readings are useful and diagnostic when grossly elevated (e.g., 20 to 30 cm H_2O),[95,98] upward trends in CVP can be a more sensitive diagnostic tool.[95]

Overreliance on increased CVP and venous distention should be avoided because they do not always indicate tamponade. For example, increased intrathoracic pressure (as induced by positive pressure ventilation or Valsalva maneuvers) increases CVP and causes neck vein distention even without pericardial effusion. Conversely, hypovolemic patients may have reduced CVP and no neck vein distention despite having clinical tamponade. The absence of distended neck veins may also result from severe venoconstriction secondary to intrinsic sympathetic discharge, vasopressor use, or severe hypovolemia.[9,88,93,95]

Diagnostic Testing

Diagnostic testing should be initiated when pericardial effusion or tamponade is suspected. Definitive diagnosis requires imaging, which may be done by CT but preferably is done by cardiac ultrasound (see Fig. 16.5*B*). Bedside ultrasound is the fastest and most reliable diagnostic tool because it is noninvasive, does not emit radiation, and can be performed at the bedside without transporting unstable patients outside the ED.

As discussed previously, pericardial effusions are occasionally discovered incidentally during evaluation for other disorders. For example, a chest x-ray and ECG performed on a patient being assessed for acute coronary syndrome (ACS) may suggest tamponade. If the clinical context supports pericardial effusion as the primary diagnosis rather than ACS, further workup for the effusion (e.g., bedside echocardiogram) should be ordered. If neither diagnosis is more likely than the other, dual workups may be necessary.

Chest Radiography

Chest radiographs are not diagnostically useful in patients with acute traumatic tamponade because the pericardium does not have sufficient time to change size or shape (see section on Pathophysiology of Pericardial Tamponade). Radiographs, however, may reveal other associated findings such as hemothorax, bullets in the thorax, or even pneumopericardium. Chest radiography may also be helpful when other diagnoses (e.g., CHF) have clinical findings similar to those of pericardial effusion. For example, a dyspneic patient with a clear chest film is less likely to have decompensated CHF than tamponade.

In patients with chronic pericardial effusions, chest films often demonstrate an enlarged, saclike, "water-bottle" cardiac shadow or a pleural effusion (see Fig. 16.5*C*). Unfortunately, it is difficult to differentiate a large effusion from myocardial enlargement (e.g., dilated cardiomyopathy) because radiographs demonstrate only the cardiac silhouette and do not reveal the physiologic differences between these two diagnoses.

Electrocardiography

Pericardial effusion secondary to acute pericarditis is suspected on the basis of typical changes on the ECG. Pericarditis has

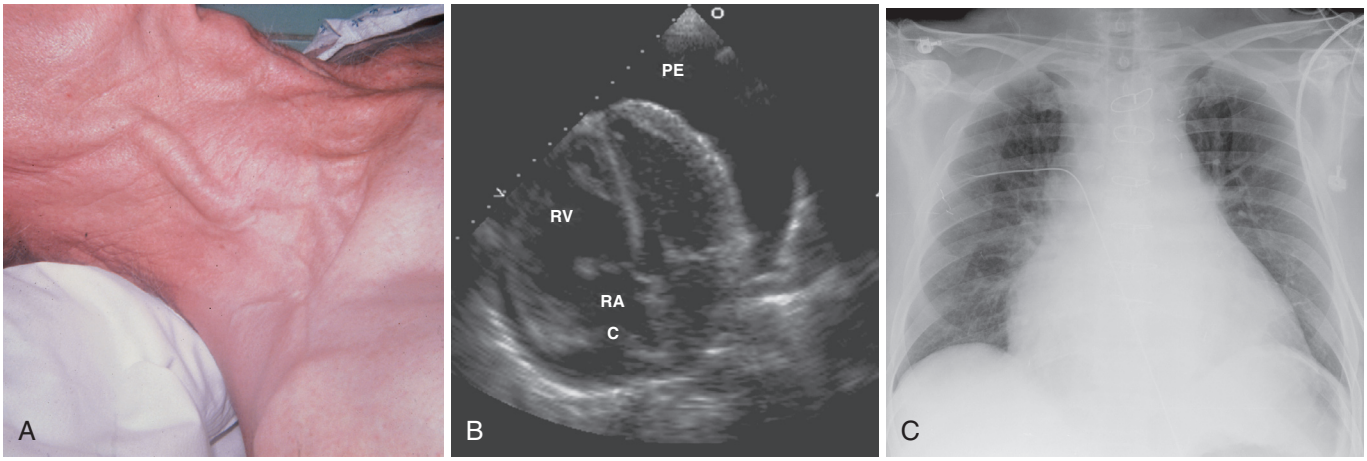


Figure 16.5 **A,** The neck veins might be markedly distended with cardiac tamponade, but this finding is not universal, especially in patients with hypovolemic trauma. **B,** Ultrasound. An apical view of a large pericardial effusion in early ventricular diastole reveals marked right atrial collapse. **C,** Chest radiograph showing an enlarged, globular cardiac silhouette (water-bottle heart) in a patient with tamponade caused by a malignant effusion. The chest film has minimal value in diagnosing tamponade but is usually abnormal when significant *chronic* effusions are present. *C,* Collapsed segment of the right atrial wall; *PE,* pericardial effusion; *RA,* right atrium; *RV,* right ventricle.

TABLE 16.2 Noninvasive Estimation of Right Atrial Pressure With Ultrasound

DIAMETER (cm) OF INFERIOR VENA CAVA	CHANGE IN DIAMETER WITH RESPIRATION	ESTIMATED RIGHT ATRIAL PRESSURE (mm Hg)
Normal (<2.1)	Decrease >50%	~3 (normal, 0–5)
Dilated (<2.1)	Decrease <50%	~8 (normal, 5–10)
Dilated (>2.1)	Decrease >50%	~8 (normal, 5–10)
Dilated (>2.1)	Decrease <50%	>15 (10–20)

Based on Rudski LG, Lai WW, Afilalo J, et al: Guidelines for the echocardiographic assessment of the right heart in adults: a report from the American Society of Echocardiography endorsed by the European Association of Echocardiography, a registered branch of the European Society of Cardiology, and the Canadian Society of Echocardiography. *J Am Soc Echocardiogr* 23:685, 2010.

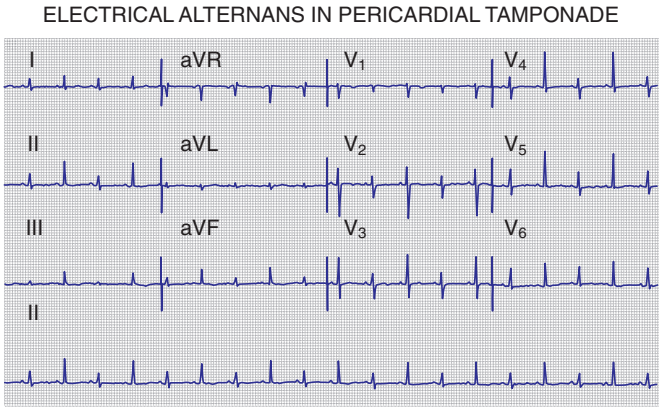


Figure 16.6 Electrical alternans may develop in patients with pericardial effusion and cardiac tamponade. Notice the beat-to-beat alternation in the voltage in the P-QRs-T axis; this is caused by the periodic swinging motion of the heart in a large pericardial effusion. Relatively low QRS voltage and sinus tachycardia are also present. Overall, the electrocardiogram has low sensitivity for pericardial effusion and tamponade. Note that electrical alternans may be more evident in the V leads.

four stages: (1) diffuse ST-segment elevation with PR depression, (2) ST- and PR-segment normalization, (3) diffuse T-wave inversion, and (4) normalization of T waves.[99]

Electrocardiography has acceptable specificity but poor sensitivity[93,100,101] in diagnosing pericardial effusion and tamponade. In a study of patients with pericardial effusion, electrocardiography had an overall sensitivity of 1% to 17% and a specificity of 89% to 100%.[93] Therefore an ECG may suggest but should never be the only means of diagnosing a pericardial effusion (Fig. 16.6). Furthermore, electrocardiography cannot reliably differentiate tamponade from effusion.[102]

The four most commonly described electrocardiographic findings in pericardial effusion are sinus tachycardia, PR depression, low-voltage QRS complexes, and electrical alternans. PR-segment depression is defined as depression of 1 mV or greater in at least one lead other than aVR. Low-voltage QRS complexes (most frequently associated with moderate to large effusions) are defined by a QRS complex with an amplitude of 5 mm or less across all limb leads. Alternatively, low-voltage QRS complexes can be identified by a sum of 10 mm or less for all precordial lead QRS amplitudes. Electrical alternans is a beat-to-beat alternation in QRS axis caused by the pendulum motion of the heart within the fluid-filled pericardial sac.[103] Alternans has been observed in 22% of medical patients with tamponade[104] and in 5% of patients with tamponade secondary to cancer.[105] Electrical alternans of P waves and QRS complexes (i.e., total electrical alternans) is a rare finding but, when seen, is pathognomonic of tamponade (see Fig. 16.6).[93,106]

Echocardiography

Echocardiography is the best tool for diagnosing pericardial effusion or tamponade (Figs. 16.7–16.9 and the Ultrasound Box). It not only demonstrates the presence of a pericardial

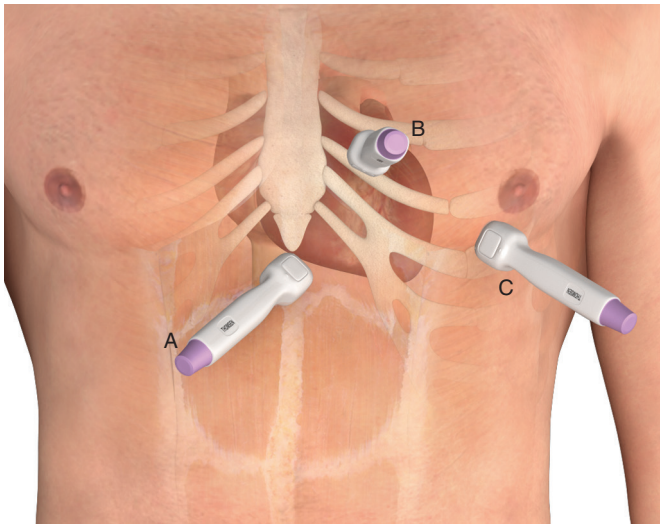


Figure 16.7 Areas of the chest to obtain basic echocardiographic windows: *A*, subxiphoid (subcostal) view; *B*, parasternal view; *C*, apical four-chamber view.

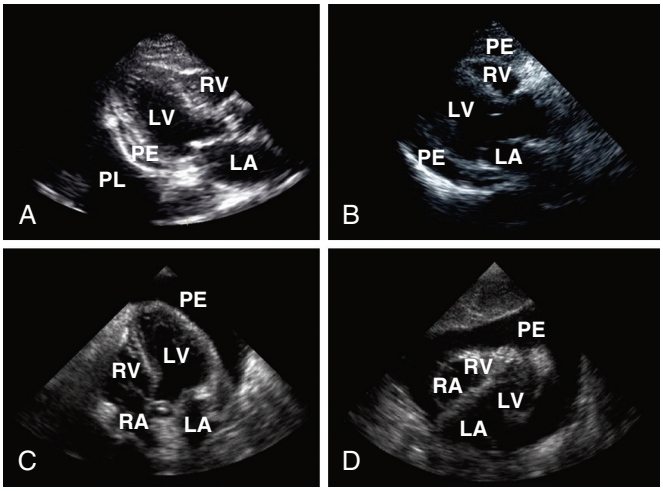


Figure 16.9 Examples of pericardial effusion. **A,** Small pericardial effusion (parasternal long-axis view). **B,** Moderate pericardial effusion (parasternal long-axis view). **C,** Large pericardial effusion (apical four-chamber view). **D,** Large pericardial effusion (subcostal/subxiphoid view). *LA*, Left atrium; *LV*, left ventricle; *PE*, pericardial effusion; *PL*, pleural effusion; *RA*, right atrium; *RV*, right ventricle.

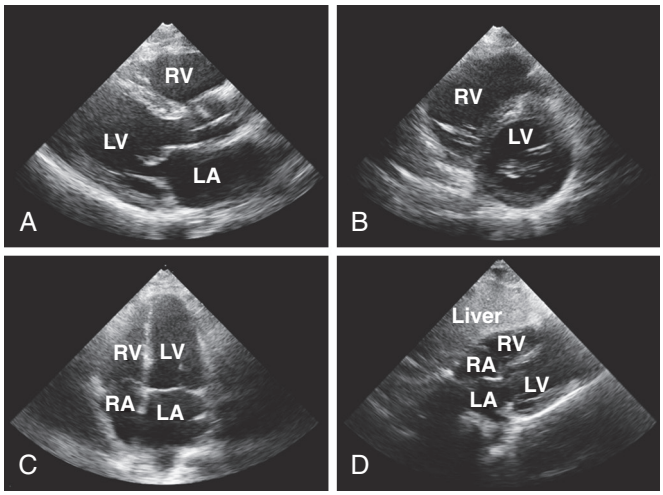


Figure 16.8 Normal echocardiographic views: **A,** Parasternal long-axis view. **B,** Parasternal short-axis view. **C,** Apical four-chamber view. **D,** Subxiphoid (subcostal) view. *LA*, Left atrium; *LV*, left ventricle; *RA*, right atrium; *RV*, right ventricle.

effusion but can also detect hemodynamic abnormalities. Ultrasonography (used interchangeably with *echocardiography* from here on) has the advantage that it is noninvasive, portable for use at the patient's bedside, and involves no ionizing radiation.[9] Echocardiography is a very sensitive and specific tool for the diagnosis of pericardial effusion and tamponade,[57,87,89] and its use in diagnosing pericardial effusions has been endorsed by several academic societies.[107–110]

Diagnosing Pericardial Effusions and Tamponade

Fluid found within the pericardial space is not always pathologic as it normally holds 15 to 50 mL of fluid. Small effusions may be clinically insignificant. The sonographer/clinician should exercise caution to not over-read an effusion, particularly when the patient is hemodynamically stable.[111] Small effusions typically contain 50 to 100 mL with an echo-free space less than 10 mm in thickness between the visceral and parietal pericardium. Moderate effusions typically contain 100 to 500 mL with 10 to 20 mm of echo-free space. Large effusions contain more than 500 mL and have an echo-free space greater than 20 mm. Large effusions tend to have an echo-free space seen circumferentially around the heart.[110] A loculated pericardial effusion can present in an atypical location and may be more difficult to appreciate compared to the non-loculated types described previously.

Once a pericardial effusion is discovered, the next step is to evaluate the patient for evidence of hemodynamic compromise. Echocardiographic signs include (1) diastolic collapse of the right atrium (highly specific and sensitive for tamponade, especially when the collapse occurs for more than a third of the cardiac cycle)[112]; (2) early diastolic collapse of the right ventricular free wall (less sensitive than right atrial collapse but specific for tamponade)[113]; (3) left atrial collapse (a very specific sign of tamponade)[114]; (4) a small, slit-like, hyperkinetic left ventricle; (5) dilation of the hepatic veins and IVC; (6) respiratory variations in the velocity of blood flow through the tricuspid and mitral valves; and (7) visualizing the heart swinging to and fro within the pericardial sac.[115] The presence of any of these signs should alert the clinician to the possibility of hemodynamic instability. Obtain expert consultation if there is any uncertainty about the ultrasound findings.

Limitations of Ultrasound

Ultrasound is the best diagnostic tool for pericardial effusion and tamponade, but great care must be taken when it is used as the only diagnostic modality (Video 16.2). In a postoperative series of cardiac surgery patients, 60% of loculated effusions causing tamponade were missed on transthoracic echocardiography but were visualized with transesophageal echocardiography.[116]

There are several false positive findings for a pericardial effusion on ultrasound. Examples include pericardial thickening, large pleural effusions, atelectasis, and mediastinal lesions.[17] Epicardial (or anterior) fat pads can also be misinterpreted as

ULTRASOUND BOX 16.1: Pericardiocentesis

by Christine Butts, MD

Pericardiocentesis has traditionally been performed blindly. This approach was associated with a low success rate and a high rate of complications, such as inadvertent puncture of the lung, ventricle, or epicardial vessels.[1] Using ultrasound to both diagnose and guide pericardiocentesis has resulted in increased success rates, as well as a lower rate of complications.[2,3]

Bedside ultrasound may additionally allow the emergency physician to make a rapid diagnosis of pericardial effusion. Evidence of impending cardiac tamponade, including right ventricular collapse and distention of the inferior vena cava, can also be identified.

Equipment

The pericardium should be imaged with a low-frequency (2 to 4 mHz) transducer to achieve adequate depth. A phased-array or microconvex transducer is preferred for its smaller footprint, which enables the sonographer to image between the ribs. However, a curvilinear transducer may be used if that is what is available.

Image Interpretation

The initial step in the procedure is to evaluate the pericardium in multiple windows. This will allow identification and characterization of the effusion, as well as planning of the best approach for drainage. This chapter focuses primarily on the views most commonly used in this procedure, the subxiphoid and parasternal.

The subxiphoid view is best known to most emergency physicians as part of the focused abdominal sonography in trauma (FAST) examination. This view provides a four-chamber view of the heart and uses the left lobe of the liver as an acoustic window. To obtain this view, the transducer is placed just inferior to the xiphoid process in the midline. The indicator faces the patient's right side (Fig. 16.US1). To obtain the best image possible it is best to place the hand over the transducer and press down into the epigastric area. The transducer can then be aimed toward the left side of the chest until the heart comes into view. The depth may need to be adjusted to view all four chambers of the heart, as well as the pericardium.

In this view the left lobe of the liver can be seen at the top of the image. Deep to the liver, a four-chambered view of the heart should be seen, surrounded by the brightly echogenic (white) border of the pericardium (Fig. 16.US2). The right ventricle will abut the liver, with the left ventricle located deeper into the body. A pericardial effusion can be identified as an anechoic (black) or hypoechoic (dark gray) collection between the heart and pericardium (Fig. 16.US3). Although fluid will typically collect at the most gravity-dependent area, loculated collections may not follow this rule.

The parasternal view is obtained by placing the transducer to the left of the patient's sternum in the fourth to fifth intercostal space. The indicator should be pointing toward the patient's right shoulder (Fig. 16.US4). Slight adjustments in angle may be needed to obtain the best image of the heart. If the patient's hemodynamic status allows, placing the patient in a left lateral decubitus position may improve this view by moving the heart closer to the anterior chest wall and displacing the air-filled lungs.

The parasternal view will demonstrate the left atrium, left ventricle, and a small portion of the right ventricle (Fig. 16.US5). The pericardium can be seen as an echogenic (white) border surrounding the heart. As in the subxiphoid view, an effusion will appear as an anechoic (black) or hypoechoic (dark gray) collection between the heart and pericardium (Fig. 16.US6).

Procedure and Technique

Once the views have been evaluated and an effusion has been identified, evaluate the pericardium in multiple views to determine the

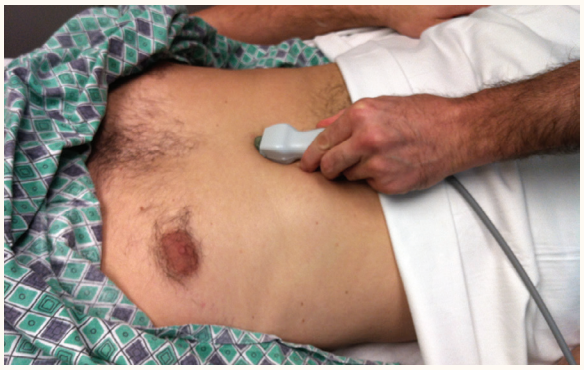


Figure 16.US1 Placement of the ultrasound transducer to obtain a subxiphoid image of the heart.

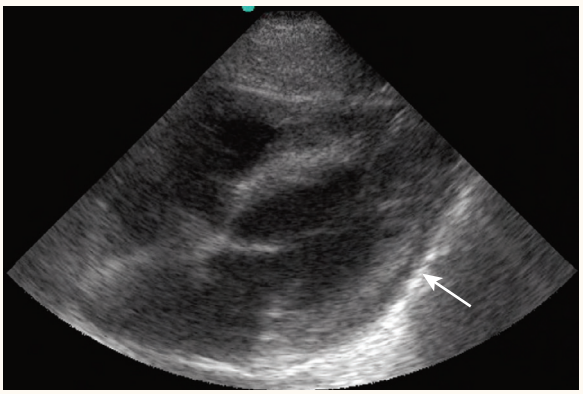


Figure 16.US2 Normal subxiphoid view of the heart seen with ultrasound. The pericardium *(arrow)* can be identified as a bright white (hyperechoic) outline, typically best seen at the inferior aspect of the heart. The pericardium should directly abut the heart, as seen in this image.

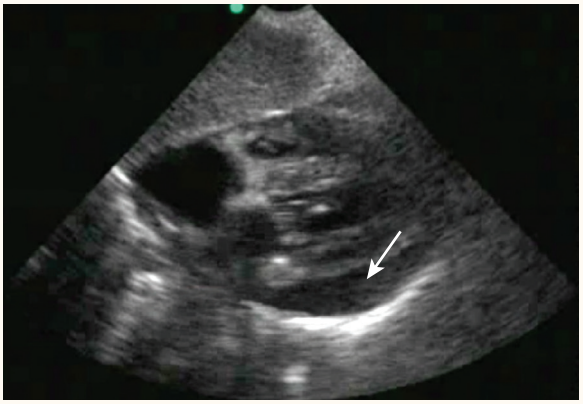


Figure 16.US3 Subxiphoid view demonstrating a pericardial effusion. When comparing this image with the normal view, a black (anechoic) fluid collection *(arrow)* can be seen between the pericardium and the left ventricle.

ULTRASOUND BOX 16.1: Pericardiocentesis—cont'd

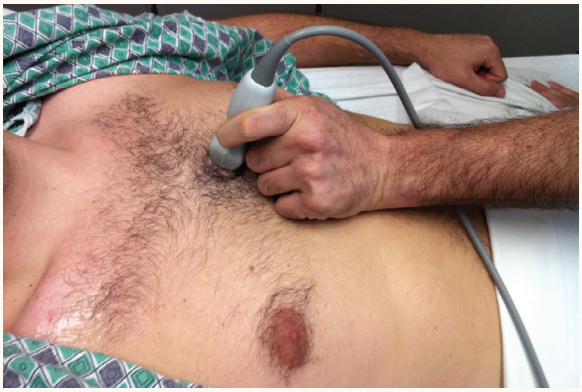


Figure 16.US4 Placement of the ultrasound transducer to obtain a parasternal long-axis view of the heart.

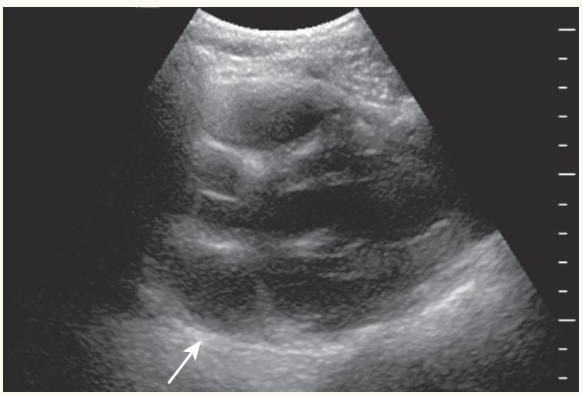


Figure 16.US5 Normal parasternal long-axis view of the heart. The pericardium can be seen as a bright white (hyperechoic) outline surrounding the heart *(arrow)*. As with the subxiphoid image, the pericardium should directly abut the heart with no intervening fluid.

best area to attempt pericardiocentesis. Tsang and colleagues described the procedure for echocardiographically guided pericardiocentesis in detail in 1998.[4] Ideally, the procedure should be attempted at the site at which the largest fluid collection is closest to the skin surface. Typically, the anterior chest wall is preferred because of its proximity to the pericardium and the absence of vital interfering structures such as the liver. The air-filled lung creates a scatter artifact that does not allow the ultrasound beam to pass. Therefore if the heart and pericardium can be viewed clearly, avoidance of the lung can be ensured. The sonographer should also attempt to approximate and avoid the location of the internal mammary artery, which lies 3 to 5 cm lateral to the sternum. Once the optimum site is identified, the practitioner should note the trajectory of the ultrasound beam. This is the trajectory that should be followed by the needle.

Sterilize the field. Anesthetize the area to be traversed with local anesthetic. Place the ultrasound transducer in a sterile covering. Take care to not reposition the patient after initial assessment because this will alter the position and trajectory. Use a 16-gauge catheter with a retractable needle to minimize potential injury to the underlying structures. After the field is prepared, attach the catheter to an attached syringe. Advance the needle in the predetermined location

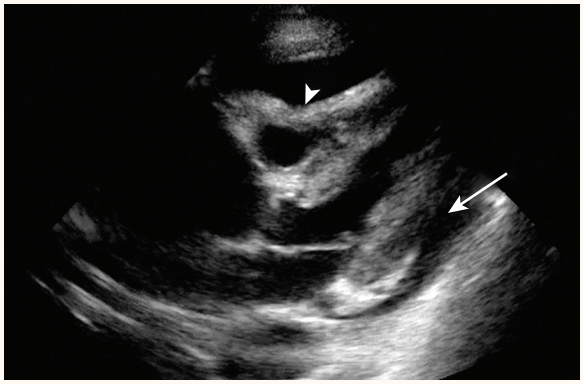


Figure 16.US6 Parasternal long-axis view demonstrating a pericardial effusion. An anechoic *(black)* fluid collection can be seen on the right of the image *(arrow)*, between the left ventricle and the pericardium. The right ventricle, at the top of the image *(arrowhead)*, can be seen to "bow" inward from the pressure exerted by the effusion. This finding, right ventricular collapse, indicates that the effusion is causing hemodynamic compromise.

along the predetermined trajectory. This can be done blindly or can be guided directly by the transducer. Apply gentle continuous pressure to the syringe until the pericardial space is entered and fluid is obtained.

If there is any question of whether the needle tip is in the pericardial space, inject agitated saline through the catheter under direct ultrasound guidance. This will allow analysis of the location of the needle tip. Prepare saline echocardiographic contrast medium by using two 5-mL syringes, one with saline and the other air, connected via a three-way stopcock to the needle catheter sheath. Saline in one syringe is rapidly injected between the syringes and then injected into the sheath once it is agitated. Entrance of the agitated saline into the pericardial space will appear sonographically as a brightly echogenic area.

Complications

Complications may occur when a pericardial effusion is misdiagnosed. The most common factor causing misdiagnosis is the presence of a fat pad anterior to the heart. Unless the effusion is loculated, it should lie within the most dependent portion of the heart and should be circumferential, depending on its size. Considering these factors and evaluating the pericardium in multiple views will aid in decreasing this misdiagnosis. Additionally, take care when performing pericardiocentesis in stable patients, particularly when the effusion is small. Smaller effusions may be more difficult to access and therefore lead to increased complications.

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pericardial effusions, although several details help distinguish between the two entities. First, epicardial fat pads tend to occur anteriorly, unlike circumferential effusions, which occur posteriorly. If a fat pad is suspected, multiple ultrasound views should be obtained to rule out a posterior effusion. Second, the echocardiographic appearance of an epicardial fat pad is isoechoic and homogeneous. This differs from blood in the pericardial space, which may look like fronds of clot waving within an anechoic (black) pericardial space. Third, an epicardial fat pad does not alter hemodynamics like tamponade does; it should not cause diastolic collapse of the right ventricular free wall, dilation of the IVC, or any signs indicating hemodynamic compromise. After careful examination, if doubt still exists regarding the presence of an effusion, hemodynamically stable patients should have a formal echocardiogram; consider CT scan if an echocardiogram is not available.

CT Scan

CT may be the only diagnostic option at institutions where ultrasound or formal echocardiography is not available in the ED. It is also helpful when concomitant diseases of the lung or mediastinum are considered. CT can demonstrate dilated hepatic veins, a plethoric IVC, and interventricular septal bowing.[117] This modality is less desirable than bedside ultrasound because it requires patients to be transported to the radiology suite. Thus, patient safety and cardiopulmonary stability must be considered before the decision is made to transport the patient. Unstable patients should never be transported until they are fully stabilized.

For stable patients, CT is effective in defining the presence, severity, and extent of pericardial effusions (i.e., circumferential versus loculated). In certain circumstances, it even provides a more definitive diagnosis than echocardiography does because it may reveal the type of pericardial fluid (by differences in tissue density) and pericardial disease (e.g., constrictive pericarditis).[118] In one series, eight equivocal echocardiograms were evaluated by follow-up CT.[119] Two patients thought to have pericardial effusion by ultrasonography were found to have pleural effusions. Another patient in whom a pericardial effusion was diagnosed on ultrasonography was found by CT to have an epicardial lipoma. CT defined three loculated pericardial effusions not identified by ultrasonography. Finally, two patients had hemopericardium visualized by CT but not by ultrasonography.

Treating Pericardial Effusions and Tamponade

Treatment of pericardial effusions in the ED depends on the degree of hemodynamic compromise. Patients with stable effusions should be treated supportively while the underlying cause (known or suspected) is addressed. For example, stable patients with pericardial effusions secondary to uremia may best be treated with dialysis, observation, and serial echocardiograms.[120] Even when the diagnosis is unknown there may be no need to perform emergency pericardiocentesis if the effusion is small to moderate.[121] Deferring diagnostic pericardiocentesis to the inpatient setting may be preferred because the cardiac catheterization laboratory or operating room is a more sterile and controlled environment than the ED.[57]

Patients with evidence of tamponade need urgent pericardiocentesis (discussed in the next section). Even those with early tamponade who are stable can decompensate quickly with little warning. Fluid boluses may improve hemodynamics temporarily, especially in patients with concomitant hypovolemia.[89] Administration of a vasopressor (e.g., norepinephrine) is a temporizing measure and should be initiated while preparing for emergency pericardiocentesis. Finally, positive pressure ventilation (e.g., mechanical ventilation) should be avoided if possible because, as discussed in the section on physiology, it can lead to hemodynamic collapse secondary to changes in intrathoracic pressure.

INDICATIONS FOR PERICARDIOCENTESIS

There are two indications for pericardiocentesis: (1) to diagnose the presence and cause of a pericardial effusion (diagnostic pericardiocentesis) and (2) to relieve tamponade (therapeutic pericardiocentesis). Diagnostic pericardiocentesis is an elective procedure. It is ideally performed under visual guidance (e.g., ultrasound or fluoroscopy). Therapeutic pericardiocentesis may be an urgent or emergency procedure based on patient hemodynamics. In the semi-stable patient, ultrasound guidance should be utilized for pericardial fluid aspiration; however, in the case of cardiovascular collapse or cardiac arrest, a blind approach may be warranted.

Diagnostic Pericardiocentesis

The use of pericardiocentesis to determine the cause of nonhemorrhagic effusions is common practice despite varying opinions on its utility.[52,122,123] Recovery of pericardial fluid to assess for tumor, autoimmune, and biochemical markers in addition to bacterial and viral cultures with Gram stain can be valuable in making the diagnosis. Measurement of pericardial fluid pH can also be helpful because inflammatory fluid is significantly more acidotic than noninflammatory fluid.[124] When a specific cause is suspected, additional diagnostic testing may be useful (e.g., adenosine deaminase in patients with tuberculosis and carcinoembryonic antigen in those with suspected malignancy).[125]

The diagnostic accuracy of pericardiocentesis is variable, and certain diagnoses are unlikely to be made from pericardial fluid. In one large series, fluid samples were obtained in 90% of aspirations, but the specific cause was determined from only 24% of those specimens.[126] Another series demonstrated false-negative cytologic results in certain cases of lymphoma and mesothelioma.[126] In some HIV patients, effusions secondary to Kaposi's sarcoma and cytomegalovirus infection have been diagnosed by pericardial biopsy following nondiagnostic fluid analysis.[127,128] Nevertheless, pericardial biopsy is not routinely indicated and is considered only in cases of recurrent effusions, such as a neoplasm or granulomatous disease.[129] When a biopsy is obtained it is often during implementation of a subxiphoid pericardiotomy (pericardial window).

A pericardial window is typically employed to obtain both pericardial fluid and a pericardial biopsy specimen, because once a tissue sample is obtained, a definitive diagnosis is much more likely.[130] It is reserved for those with recurring episodes of tamponade.[130] This procedure can be performed safely in the operating suite without general anesthesia.[131] In a prospective series of 57 patients who underwent subxiphoid pericardiotomy, a definitive diagnosis was obtained in 36%, a probable diagnosis in 40%, a possible diagnosis in 16%, and no diagnosis in 7%. It is uncertain whether this technique is safer than

ultrasound-guided pericardiocentesis, but published reports show low rates of complications in experienced hands.[58]

The most valuable clinical predictors in diagnosis are size of the effusion (larger effusions and tamponade are more likely to yield a diagnosis) and signs of inflammation (e.g., fever, pericardial friction rub, ST-segment elevation).[58] Unfortunately, the only methods of making definitive diagnosis are pericardial fluid aspiration with analysis and pericardial biopsy.

Diagnostic pericardiocentesis has limited utility for hemopericardium secondary to traumatic causes. When used diagnostically after trauma to assess for the presence of pericardial bleeding, the procedure has a false-negative rate (i.e., no blood aspirated) of between 20% and 40%.[95,132–134] The high false-negative rate is due to the fact that posttraumatic blood tends to clot within the pericardial space and therefore cannot be aspirated.[19] Furthermore, pericardiocentesis should not delay emergency thoracotomy if cardiac tamponade is suspected.[135] If there is uncertainty about the presence of tamponade, the focused abdominal sonography in trauma (FAST) examination rapidly and noninvasively identifies pericardial fluid.[136–138]

Therapeutic Pericardiocentesis

The ultrasonographic finding of a large pericardial effusion (>20 mm) in a stable patient should lead to early cardiology or cardiothoracic surgery consultation for percutaneous drainage or placement of a pericardial window. Patients with a pericardial effusion who remain hypotensive despite fluid resuscitation require urgent therapeutic drainage. The decision to wait for consultants is best made by the emergency physician at the bedside and should be based on clinical judgment. Until recently, few algorithms existed to assist in the risk stratification of patients with pericardial effusions and who should undergo emergency versus delayed pericardiocentesis. In 2012, Halpern and colleagues introduced a scoring system based on effusion size, echocardiography derangements, and clinical criteria.[139] Similarly, Ristic and colleagues in 2014 created a system incorporating etiology, clinical factors, and diagnostic imaging.[140] Such tools can aid the emergency physician in identifying who would benefit most from ED pericardiocentesis and who could await consultant recommendations. (Fig. 16.10)

For patients in extremis, pericardiocentesis should be performed immediately even if consultation is unavailable. The clinician who performs the procedure should be the one who is most experienced in both sonography and pericardial fluid aspiration.

Tamponade of Uncertain Cause: Pulseless Electrical Activity

A major indication for emergency pericardiocentesis is a patient in cardiac arrest with PEA. Always consider cardiac tamponade in the differential diagnosis for PEA, especially if jugular venous pressure is elevated or a pericardial effusion is demonstrated on ultrasound. In a series of 20 patients with PEA, 3 had tamponade and another 5 had some degree of pericardial effusion.[141] In this setting, blind (i.e., landmark method), ECG-guided, or ultrasound-guided pericardiocentesis can be lifesaving.

Tamponade Caused by Nonhemorrhagic Effusions

Most nonhemorrhagic effusions are liquid. They can be drained by pericardiocentesis with a small needle or by a catheter left in the pericardial space. Removal of even a small amount of fluid can cause immediate and dramatic improvement in blood pressure and cardiac output. Pericardiocentesis relieves tamponade caused by nonhemorrhagic effusions in 60% to 90% of cases.[54,126,142]

If aspiration with small-needle pericardiocentesis fails, the patient may have a purulent, malignant, or loculated effusion. Placement of a pericardial catheter is more useful in the long-term management of these patients. In Krikorian and Hancock's series,[126] 24% of patients were managed successfully with a single pericardiocentesis procedure, but 37% needed multiple aspirations or an indwelling catheter (39% required surgical drainage and 55% of these patients had traumatic hemopericardium). Therefore if time and patient stability permit, consider pericardiocentesis with a catheter to reduce the necessity for repeated aspirations.

Patients with renal failure and tamponade require urgent pericardiocentesis. Without signs of tamponade, however, these patients may be better managed by dialysis. In one series, 63% of renal failure patients were managed successfully with only dialysis.[126] There is some evidence that needle pericardiocentesis is a poor choice in patients who need pericardial drainage; in one series, 9 of 10 patients had serious complications following this procedure.[66] Consultation with specialists is advised when there is no evidence of hemodynamic collapse but pericardial drainage is still being considered.

An algorithm for the urgent management of nonhemorrhagic cardiac tamponade is presented in Fig. 16.11.

Pericardiocentesis in Patients With Hemorrhagic Tamponade

For hemorrhagic tamponade, pericardiocentesis is never the definitive treatment because this strategy has several drawbacks.[143,144] Aspiration of a small quantity of fluid may cause a dramatic improvement in hemodynamics, but pericardial clots can prevent adequate drainage, so blood usually reaccumulates.[93,106] Patients with traumatic pericardial hemorrhage ultimately require thoracotomy to explore and repair the cardiac injury. Pericardiocentesis simply delays this definitive procedure. A study investigating traumatic cardiac injury found that all patients who underwent surgery within 2 hours of the injury survived but the mortality rate was higher in patients who experienced longer operating room delays.[143]

Sugg and associates documented a 43% mortality rate when pericardiocentesis was the sole treatment of traumatic tamponade, as compared with 16% in those who also underwent surgical intervention.[133] All patients managed by pericardiocentesis had repairable wounds at autopsy, thus suggesting that in this population, operative repair is preferable. The number of deaths from stab wounds has been decreasing over time in response to a shift in trauma philosophy in which early thoracotomy is supported rather than repeated pericardiocentesis.[95,134,144,145]

In the austere setting where no surgeon is immediately available and hemopericardium is identified on FAST exam, pericardiocentesis can serve as a life-saving bridge until definitive surgical intervention is arranged (Fig. 16.12). A study of penetrating trauma patients with tamponade found that preoperative pericardiocentesis decreased the mortality rate from 25% to 11%.[146]

CONTRAINDICATIONS

There are no absolute contraindications to pericardiocentesis in hemodynamically unstable patients. Relative contraindications

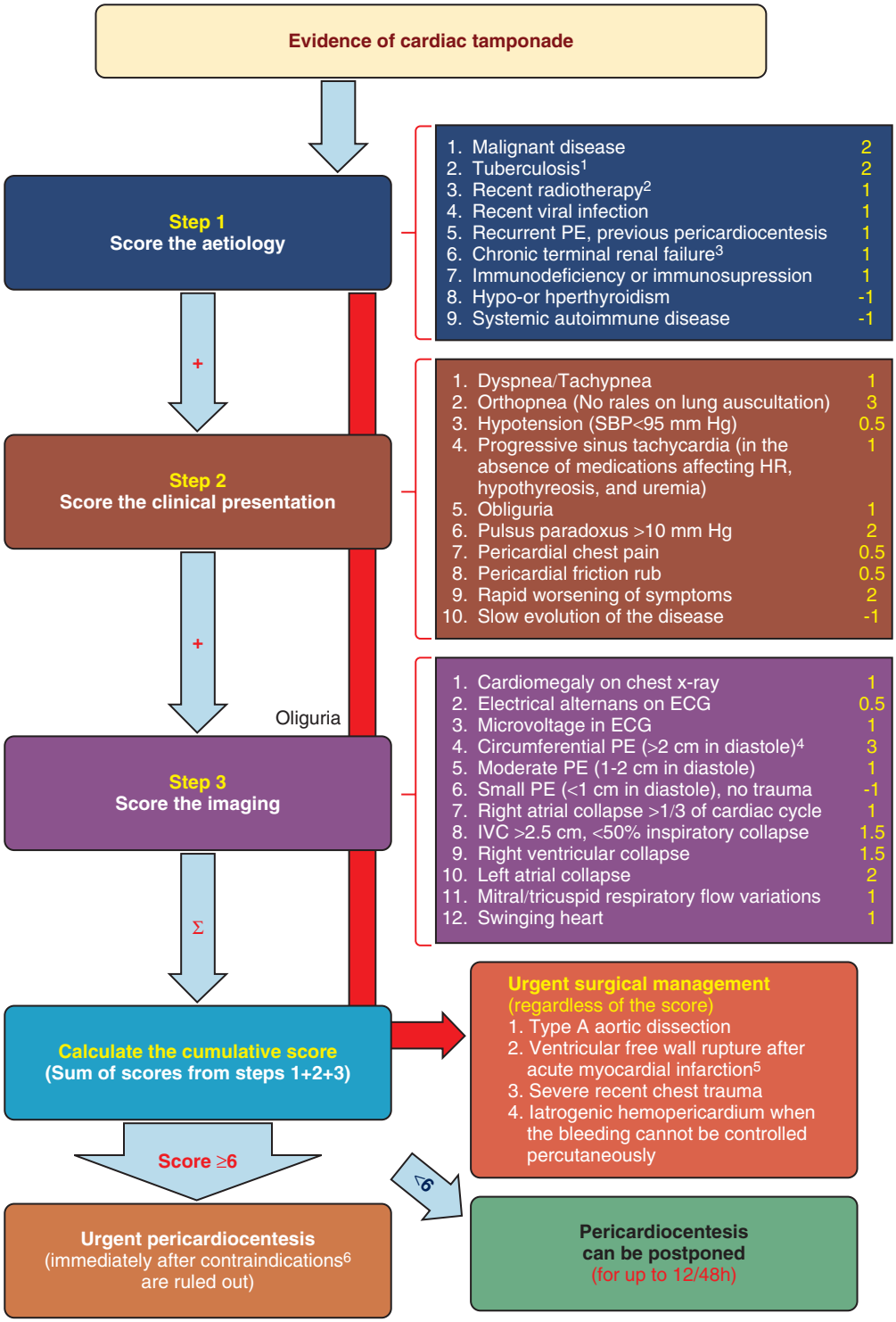


Figure 16.10 Three-step scoring system triage patients requiring urgent percutaneous or surgical drainage of pericardial effusion. Diagnosis of cardiac tamponade is based on clinical symptoms, signs, and echo findings. Total score ≥6 indicates urgent pericardiocentesis. *HR*, Heart rate; *IVC*, inferior vena cava; *PE*, pulmonary embolism; *SBP*, systolic blood pressure. (From Ristic A, Imazio M, Adler Y, et al: Triage strategy for urgent management of cardiac tamponade: a position statement of the European Society of Cardiology Working Group on Myocardial and Pericardial Diseases, *Eur Heart J* 35:2279, 2014. Oxford University Press.)

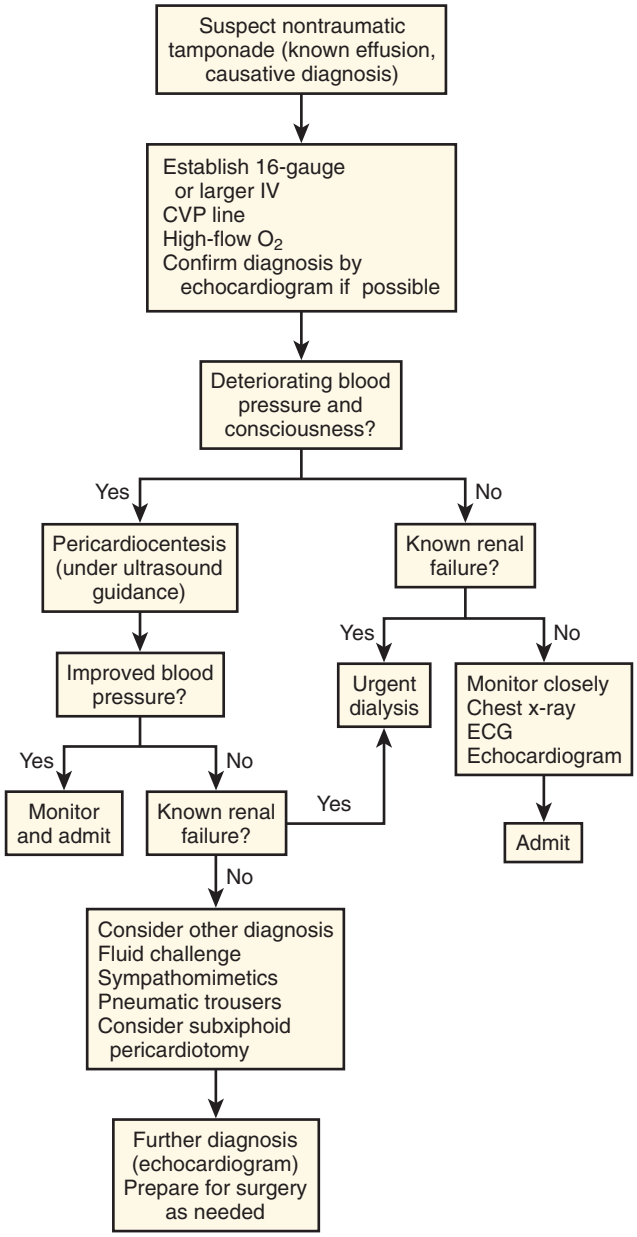


Figure 16.11 Management of nontraumatic cardiac tamponade. *CVP*, Central venous pressure; *ECG*, electrocardiogram; *IV*, intravenous line.

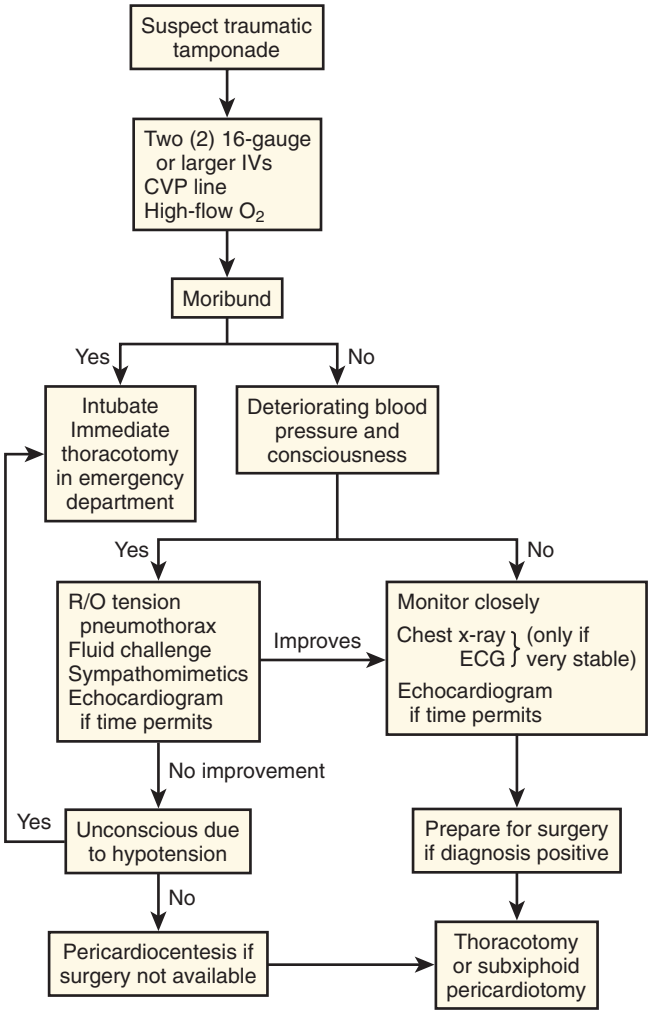


Figure 16.12 Management of traumatic cardiac tamponade. *CVP*, Central venous pressure; *ECG*, electrocardiogram; *IVs*, intravenous lines; *R/O*, rule out.

to pericardiocentesis include coagulopathy; previous thoracoabdominal surgery; the presence of prosthetic heart valves, pacemakers, or cardiac devices; inability to visualize the effusion with ultrasound during the procedure; and situations in which better treatment modalities are immediately available (e.g., thoracotomy for trauma patients).

Ideally, pericardiocentesis is performed in the cardiac catheterization laboratory under fluoroscopic or echocardiographic guidance. With the advent of bedside ultrasound and immediate visualization of large pericardial effusions, pericardiocentesis is being performed in the ED more frequently. Ultrasound can accurately identify the area of the heart with the greatest fluid accumulation and clarify its relationship to the body wall.[40,147,148] This allows the physician to choose an entry site and angle of penetration with the greatest likelihood of obtaining fluid while avoiding vital structures.

OVERVIEW OF TECHNIQUES AND EQUIPMENT

In an urgent situation (e.g., PEA arrest) with no adjunctive equipment available, pericardiocentesis can be performed with minimal equipment. Use a long 18-gauge spinal needle attached to a 10-mL syringe to withdraw fluid from the pericardial sac. Although the procedure can be done with these two simple devices, access to several others would be beneficial during the procedure. Of all the components, the most essential is an ultrasound machine, which first is used to determine whether a pericardial effusion is present and then assists in accurate needle placement. Ideally, use a probe with a small-footprint and a frequency of 2 to 4 MHz. If this type of probe is not available, use a 2- to 3.5-MHz curvilinear probe in the subxiphoid view, which will also provide excellent images of the heart.

Before the introduction of real-time sonography to guide needle placement, electrocardiographic monitoring was used to indicate appropriate needle placement. This is done by connecting the electrocardiographic machine to one of the precordial leads (e.g., V_1). That precordial lead is then attached to the distal end of a spinal needle with an alligator clamp

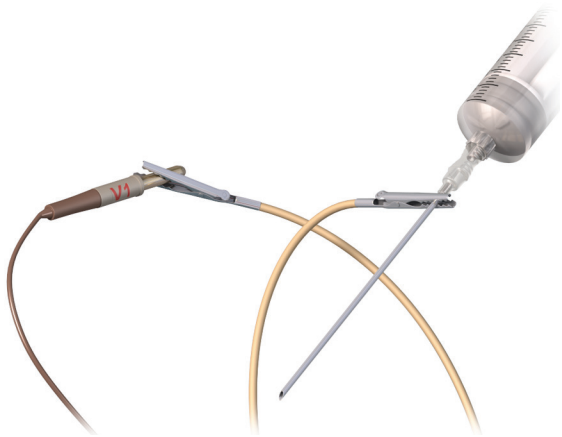


Figure 16.13 For emergency pericardiocentesis, a long 18-gauge spinal needle is connected to a V lead of an electrocardiographic machine via a cable with alligator clips.

(Fig. 16.13). The precordial lead can then be used as a rhythm strip to monitor the needle tip continuously.

Other tools that are desirable for urgent pericardiocentesis can be found easily in the ED or in a pericardiocentesis kit (see Review Box 16.1): a finder needle; Seldinger wire; dilator; flexible catheter guide; 6- to 8-Fr pigtail catheter; plastic drainage tube; extra syringes; sterile hat, gown, gloves, and drape; and local anesthetic.

PROCEDURE

Temporizing Measures

Cardiac tamponade is an emergency that requires urgent therapy. Therapy typically consists of either pericardial drainage by needle aspiration or placement of a pericardial window. These procedures are not classically performed in an ED, so temporizing methods are the mainstay of therapy unless the patient is unstable (e.g., in PEA arrest). The most common therapeutic procedures used as temporizing measures in the setting of tamponade are intravascular volume expansion with crystalloids and administration of vasopressors or inotropes.[5,14,149] Although most textbooks and protocols encourage the use of these temporizing methods, they are backed by only sparse scientific evidence.[150,151] Studies in animals have shown an increase in cardiac output and improvement in blood pressure with expansion of central blood volume; the validity of this in humans with cardiac tamponade is uncertain. Fluid resuscitation in a trauma patient with penetrating cardiac injury might cause deterioration. Animal experiments indicate that the response depends on whether fluid boluses produce recurrent bleeding from the cardiac wound.[152] Despite the lack of evidence, judicious volume expansion with or without an adjunctive vasopressor before definitive therapy may be the only option for patients with tamponade.

Norepinephrine, isoproterenol, dopamine, and dobutamine have all been evaluated as the vasopressor or inotropes of choice in patients with cardiac tamponade. Norepinephrine and isoproterenol increased cardiac output in animal models of tamponade, but failed to increase it in humans.[153,154] Dopamine and dobutamine increased cardiac output and improved hemodynamics in the setting of tamponade.[33,151] Any of these agents may be beneficial as a temporizing agent only, but theoretically, dobutamine is preferable because of its greater β-adrenergic activity.[154]

Preparation

Before preparing for pericardiocentesis, place all resuscitation equipment at the bedside in anticipation of clinical deterioration. Most patients undergoing pericardiocentesis in the ED have already experienced hemodynamic collapse and are lying supine. If the patient is able to cooperate, elevate the chest 30 to 45 degrees to bring the heart closer to the chest wall. Sedation of stuporous patients is typically forgone because of the risk for further hemodynamic collapse. If the patient is awake and undergoing the procedure without obvious hemodynamic compromise, short-acting medications (e.g., ketamine, midazolam, or fentanyl) are preferred.

Every effort should be made to ensure aseptic technique. Prepare the chest and upper part of the abdomen with a chlorhexidine-based solution. Drape the patient and ensure that all care providers involved in the procedure are wearing a sterile hat, mask, gown, and gloves. If the patient is awake, anesthetize the skin and the proposed route with 1% lidocaine (see Review Box 16.1). Because the pericardium is extremely sensitive, it should be anesthetized.[155]

The approach to pericardiocentesis depends on the clinical status of the patient, the availability of ultrasound, and the distribution of pericardial fluid. Pericardial fluid is not always distributed circumferentially in the pericardial sac, so ultrasound can quickly identify the maximal effusion pocket and demarcate the appropriate site for needle placement. Pericardiocentesis with ultrasound guidance is currently the safest and most reliable method for the diagnosis and treatment of pericardial effusion and tamponade.[156] Studies of echocardiography-directed pericardiocentesis have found that the apical approach is the best site for puncture.[148,157,158] Cadaver studies have corroborated this finding and have demonstrated greater safety with an apical approach. However, these studies also revealed that an apical approach is associated with a greater incidence of pneumothorax than a traditional subcostal approach is. Before the advent of ultrasound guidance, the subxiphoid approach (discussed later in this chapter) was the preferred method of pericardiocentesis. It is still used frequently during cardiac arrest and when ultrasound is not readily available.

ECG Monitoring

If ultrasound is not readily available to guide needle placement, electrocardiographic monitoring can serve as a useful adjunct. Electrocardiographic monitoring is used to prevent puncture of the ventricle. When using this method, an assistant is essential to ensure sterile technique, observe for dysrhythmias, and make sure that the electrocardiographic machine is functioning properly.

After all equipment is sterile, attach an alligator clip from one of the precordial leads (e.g., V_1) to the distal end of the spinal needle. Record a rhythm strip of this lead (the "exploring electrode") continuously. Advance the needle through the skin while remembering that any contact with the epicardium will cause a current-of-injury pattern that can be seen on the ECG. Typically, this is represented as a wide-complex premature ventricular contraction with an elevated ST segment (Fig. 16.14). When a current-of-injury pattern is seen, the needle

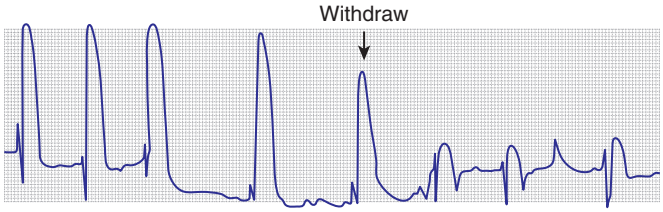


Figure 16.14 Current-of-injury. There is an obvious change in the electrocardiogram when the pericardiocentesis needle touches the epicardium. Following slight withdrawal *(arrow)*, the ST-segment elevation diminishes. This is best seen when the needle is directly attached to the electrocardiographic V lead.

is probably touching the epicardium. Withdraw it several millimeters to prevent laceration of the myocardium or coronary vessels. After slight withdrawal, the needle should be within the pericardial space. Aspirate any fluid, but watch for changes on the ECG. Electrocardiographic monitoring is not infallible: if the patient has an abnormal myocardium from conditions such as a previous MI or the formation of scar tissue, no current-of-injury pattern will be generated on the rhythm strip.

Ultrasound-Guided Pericardiocentesis

Pericardiocentesis has traditionally been performed blindly. This approach was associated with a low success rate and a high rate of complications, such as inadvertent puncture of the lung, ventricle, or epicardial vessels. Using ultrasound to both diagnose and guide pericardiocentesis has resulted in increased success rates, as well as a lower rate of complications. The techniques are described in the Ultrasound Box.

Subxiphoid/Subcostal Approach

As mentioned earlier, the traditional blind subxiphoid approach can still be used for pericardiocentesis in the ED (e.g., for patients in cardiac arrest and when ultrasound is not available). The technique is performed as follows: introduce the needle 1 cm inferior to the left xiphocostal angle at a 30-degree angle to the skin (Fig. 16.15). Because the heart is an anterior structure, angles greater than 45 degrees may lacerate the liver or stomach. Aim toward the left shoulder and advance the needle slowly while continuously maintaining negative pressure on the syringe to aspirate any fluid. Aspirate with an "in-and-out" vector only, not "side-to-side," which may lacerate tissue. If no fluid is aspirated, withdraw the needle completely and redirect it in a deeper posterior trajectory. If no fluid is aspirated after redirecting the needle, withdraw the needle and redirect it, working from the patient's left to right, until it is aimed at the right shoulder. Recommendations regarding needle trajectory vary widely, including toward the right shoulder, sternal notch, and left shoulder.[149,155]

Apical Approach

The apical approach is occasionally used as an alternative to the subcostal approach to drain a pericardial effusion when ultrasound is available. Use ultrasound to identify the largest area of the apical effusion (Fig. 16.16*A*) or simply feel for the apex. If the apex cannot be palpated, it typically lies within the area of cardiac dullness, often between the fifth, sixth, or seventh intercostal space, between the midclavicular and midaxillary lines. Introduce the needle 1 cm lateral to and into the intercostal space below the apical heartbeat. Advance the needle over the cephalad border of the rib and aim it toward the right shoulder to avoid the neurovascular bundle located caudal to the rib space. This area is close to the lingula and the left pleural space, thus making pneumothorax a frequent complication. Theoretically, this technique is used because the coronary vessels are small at the apex; therefore if a ventricle is entered, it is the thick-walled LV, which is more likely to seal off after ventricular injury. With echocardiographic guidance, the apical approach is used more commonly.[159]

Parasternal Approach

The parasternal approach is an alternative approach to the previously described techniques. First, identify the largest area of the parasternal effusion on ultrasound if possible. If ultrasound is not available or if the effusion is not clearly identified on the ultrasound image, proceed by introducing the needle 1 cm lateral to the sternal border at the left fifth or sixth intercostal interspace. Advance the needle over the cephalad border of the rib to avoid the neurovascular bundle on the caudal aspect of the rib. Avoid going too far laterally from the sternal border because of potential injury to the internal mammary artery.[160] Occasionally, a right parasternal approach may be used when ultrasound predicts superior access to an effusion from this direction.

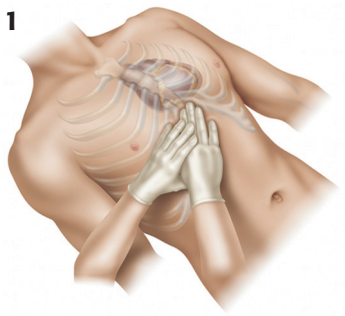
Tsang and co-workers[161] described this technique for ultrasound-guided pericardiocentesis in 1998. The ideal site for skin puncture is where the largest area of fluid accumulation is closest to the skin surface. On ultrasound, this is indicated by a large anechoic (black) area at the top of the screen, usually corresponding to the left anterior chest wall (rather than the subcostal region). This approach also avoids injury to the liver (common with the subcostal approach). Inadvertent puncture of the lung is also prevented with this approach because air in the lung will not conduct sound waves and will prevent visualization of the heart when located immediately beneath the probe. Avoid choosing a site that could puncture the internal mammary artery, which lies 3 to 5 cm from either parasternal border, or the neurovascular bundle, which is located at the inferior border of the rib. Mark the best site with a sterile pen.

Procedure and Technique

Confirm the trajectory and depth of the needle before puncturing the skin. Be aware that repositioning the patient alters the position of the heart and pericardial sac within the chest, so reassessment will be necessary. Prepare the skin antiseptically and place a sterile cover over the ultrasound probe. If time permits, anesthetize the selected area with 1% lidocaine, with the superior border of the adjacent rib being used as a landmark. Select an 18-gauge spinal needle. Ideally, the needle should have a sheath that allows it to be withdrawn after the pericardial space is entered. This helps avoid injury to the heart and other vital structures. Attach a saline-filled syringe to the needle, and gently aspirate while slowly advancing the needle. Keep the ultrasound probe on the chest wall, immediately adjacent to the aspiration site.

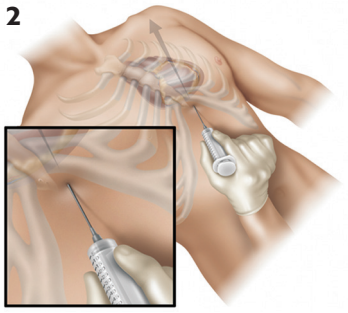
Once the pericardial space is entered, inject agitated saline to confirm needle placement, particularly if the pericardial fluid is grossly bloody or if there is any question about needle

PERICARDIOCENTESIS (SUBXIPHOID APPROACH)



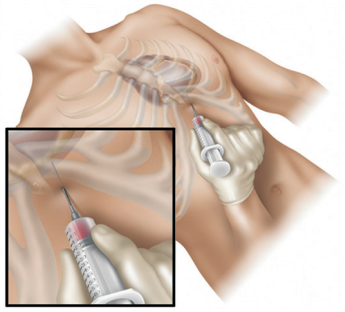
1 Examine the patient and identify the xiphoid process and the costal margin.

Prepare the area with antiseptic and administer local anesthetic.



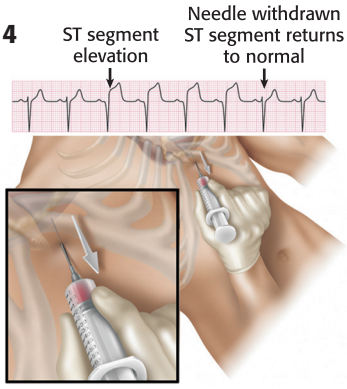
2 Introduce the needle 1 cm inferior to the left xiphocostal angle at a 30-degree angle to the skin.

Aim toward the left shoulder.

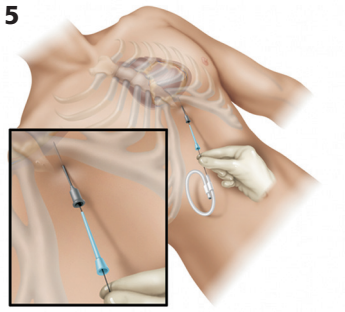


3 Aspirate during needle advancement and monitor for fluid return.

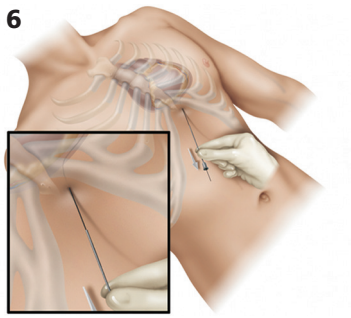
Stop advancing once fluid is returned.



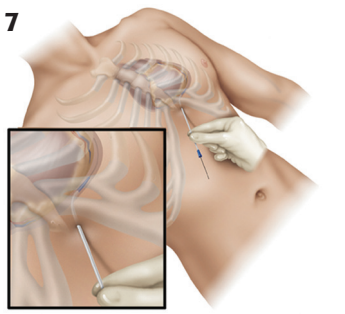
4 If using electrocardiographic monitoring, observe for current of injury during needle advancement, which indicates epicardial contact. If this occurs, withdraw the needle slightly.



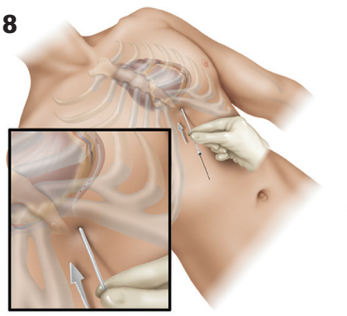
5 Advance a J-tipped guidewire through the needle and into the pericardium.



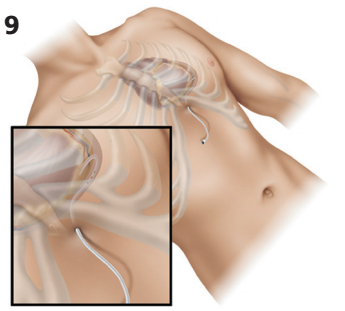
6 Remove the needle, while leaving the guidewire in place in the pericardium.



7 Advance a 6- to 8-Fr dilator over the wire and then remove the dilator.



8 Advance a 6- to 8-Fr pigtail catheter over the wire and into the pericardium.



9 Remove the wire and drain the pericardial fluid.

Figure 16.15 Pericardiocentesis (preferred subxiphoid approach). (From Custalow CB: *Color atlas of emergency department Procedures*, Philadelphia, 2005, Saunders, p 123.)

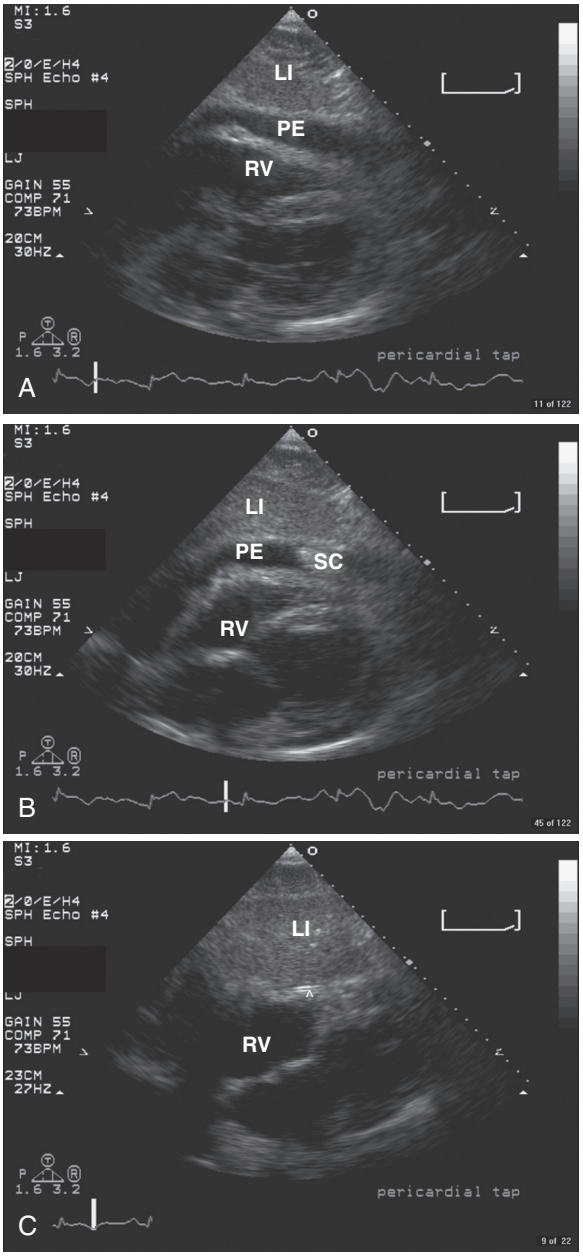


Figure 16.16 Placement of a pigtail catheter in the pericardial space under ultrasound assistance: subcostal view of a small but hemodynamically significant pericardial effusion during pericardiocentesis. **A,** The effusion. **B,** After the injection of approximately 0.1 mL of agitated saline through the pericardiocentesis needle to confirm its position in the pericardial space. **C,** The shaft of the pigtail catheter (*arrowhead*, two discrete parallel echogenic lines reflect the catheter walls; the echo-free area represents the catheter lumen) lying in the pericardial space after the majority of fluid has been drained. *LI*, Liver; *PE*, pericardial effusion; *RV*, right ventricle; *SC*, saline contrast.

position (see Fig. 16.16*B*). Prepare a saline echocardiographic contrast medium by using two 5-mL syringes, one with saline and the other with air. Connect them via a three-way stopcock to the needle and catheter. Rapidly inject saline between the syringes and then inject it into the sheath. Monitor the entrance of the agitated saline into the pericardial space sonographically—it appears as a brightly echogenic stream. If the use of agitated saline proves to be inconclusive or suboptimal, use an echocardiographic contrast agent (e.g., Definity) as a safe and successful alternative.[162,163] Contrast agents contain gas microbubbles, which markedly enhance the fluid echo by introducing multiple liquid-gas interfaces. Inject this solution as a bolus. If the contrast material clears immediately after administration (as occurs with agitated saline) or persists temporarily within the cardiac chambers, an intracardiac location is suggested.

Fluid Aspiration and Evaluation

Removal of even a small amount of pericardial fluid (e.g., 30 to 50 mL) usually results in either return of spontaneous circulation or hemodynamic improvement. After any approach used for pericardiocentesis, place a temporary drain not only to ensure rapid access into the pericardial sac but also to allow more fluid to be removed quickly if hemodynamic collapse recurs. After needle placement is confirmed, a temporary drain can be placed by the Seldinger technique, described in Chapter 22.

Remove the syringe from the needle, advance a guidewire through the needle, and then remove the needle (see Fig. 16.15). Position a dilator (6 to 8 Fr Cordis) over the wire. If a dilator is not used, particularly with the subxiphoid approach, the pigtail catheter tip may get caught in the subcutaneous tissue and make placement of the catheter difficult. Remove the dilator and slide an introducer sheath dilator (6 to 8 Fr Cordis) over the wire. Remove the wire and the dilator while leaving the introducer sheath in place. Insert the pigtail angiocatheter through the introducer sheath, and aspirate fluid to confirm placement.[157,161] After the catheter is advanced into place, secure it with a suture to ensure that it does not migrate after the procedure. Apply an appropriate catheter dressing. Attach the catheter to a three-way stopcock and connect it to a water seal to drain by gravity. The pigtail catheter allows prolonged drainage and safe access into the pericardial sac without requiring the introduction of another needle.[164,165] If drainage of pericardial fluid becomes sluggish, flush the catheter with a heparinized saline solution to ensure patency of the lumen.[155]

Aspiration of blood during pericardiocentesis raises the possibility of cardiac puncture. Blood retrieved from the ventricle usually clots faster than bloody fluid aspirated from the pericardium. In general, hemorrhagic pericardial effusions have local fibrinolytic activity, which prevents clot formation. If the bleeding is brisk enough, however, blood may still clot and does not necessarily point toward ventricular puncture. The hematocrit of pericardial fluid should always be lower than that of a sample from the systemic vascular system, except in patients with aortic dissection or acute myocardial rupture. These circumstances aside, a hematocrit value similar to that for systemic blood should raise concern for an intracardiac needle location. Several other simple laboratory tests can differentiate normal from abnormal pericardial fluid, but they require the availability of a centrifuge system and time. Under normal conditions, pericardial fluid is less than 50 mL in volume and clear to pale yellow in color with no red or white blood cells, inflammatory markers, bacteria, or cancer cells and with a glucose concentration similar to that of blood.

Immediately following the procedure, obtain a chest film to ensure the absence of pneumothorax and free air under the diaphragm. Place the patient on continuous cardiac monitoring for 24 hours and watch for signs of reaccumulating fluid or

iatrogenic complications. Repeating the ultrasound examination in 24 hours is recommended. Diagnostic evaluation of nonhemorrhagic fluid is similar to that for pleural fluid (see Chapter 9).

Suture the pigtail catheter to the skin, but be careful not to occlude the catheter by tying it too tightly. Wrap the catheter in gauze at the skin and cover it with a sterile dressing. Attach the catheter to suction tubing and a drainage system.

COMPLICATIONS

Emergency physicians often perform pericardiocentesis under duress on a patient in PEA arrest. Many also perform the technique blindly because they have little or no time to gather adjunctive assistance or tools. It is critical for the emergency physician to be aware of both the traditional and contemporary methods of performing the procedure and the complications that can be associated with these methods (see Review Box 16.1).

With the advent of ultrasound- and CT-guided pericardiocentesis, the complication rate has been greatly reduced. Complication rates as low as 4% have been reported in large observational studies. Earlier studies of blind pericardiocentesis documented morbidity rates of 20% to 40% and mortality rates as high as 6%.[166] Because pericardiocentesis is performed in moribund patients, the likelihood of cardiac arrest and death is high. However, they are not usually a direct complication of pericardiocentesis but of poor cardiopulmonary reserve. Cardiac arrest and death are rarely associated with echocardiographically guided pericardiocentesis. When blind or electrocardiographically guided pericardiocentesis is performed, the patient is usually already in full arrest and attributing the cause of death to the procedure is nearly impossible. In a series of 52 patients the only death occurred in a patient in cardiogenic shock in whom pericardiocentesis was nonproductive and who was found to have severe arteriosclerotic heart disease, not tamponade, on postmortem examination.[167] In a series of 352 fluoroscopically guided pericardiocenteses, two deaths were documented.[168] Ultrasonographic or CT confirmation of effusion was used in all but 15 cases. The two deaths occurred during or after the procedure, but whether they could be attributed to the procedure is unclear. One patient with aortic rupture that penetrated into the pericardial space died of cardiac arrest immediately after the puncture. The other death, in a post-MI patient with a left ventricular aneurysm, was caused by ventricular fibrillation that occurred approximately 15 minutes after the procedure.

One of the most frequent complications is a dry tap, especially when a blind approach is used. A dry tap is often caused by blockage of the needle with clotted blood or a skin plug. With the parasternal approach, the needle can become blocked by vigorous probing of the anterior costal cartilage. The problem can be solved by repositioning or irrigating the needle, which allows the effusion to be aspirated unless it is loculated.

Preventricular contractions are frequently noted after the needle enters the pericardial sac; however, no serious dysrhythmias resulting in hemodynamic compromise have been mentioned in the literature. Several case series reported no dysrhythmias.[54,67,157] Krikorian and Hancock[126] reported one episode of ventricular tachycardia and several "hypotensive vasovagal reactions" that were associated with bradycardia and responded to atropine and fluid loading. Duvernoy and associates[168] reported 1 case of ventricular tachycardia and 1 case of atrial fibrillation in 352 procedures. Maggiolini and co-workers reported transient third-degree heart block in a single patient.[169] The traditional subxiphoid approach carries a risk for liver laceration. Fortunately, inadvertent needle passage into the liver has not been reported to cause significant hemorrhage or death.[170]

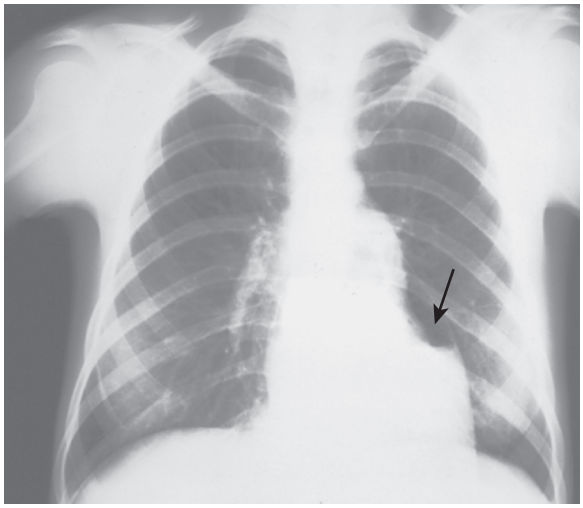


Figure 16.17 Air-fluid level *(arrow)* in the pericardial space immediately after pericardiocentesis. A minor pneumopericardium is inconsequential; a larger collection may cause tamponade.

The parasternal and apical approaches have been documented as causing pneumothorax and pneumopericardium in several case series, but without any clinical consequence (Fig. 16.17). The pneumothoraces were treated with 100% oxygen or thoracostomy. There have also been infrequent reports of pneumopericardium after removal of a pericardiocentesis catheter. The cause of the pneumopericardium is thought to be the formation of a bronchopericardial fistula, but the exact mechanism is unclear. The mortality rate associated with tension pneumopericardium is approximately 50%, so consider pneumopericardium when patients complain of dyspnea and hypotension after removal of their catheter.[171–173]

Very few studies have reported ventricular or coronary vessel laceration during pericardiocentesis. These complications occur more frequently during blind or electrocardiographically guided procedures. Most cardiac perforations occur in the RV, but punctures in the LV and atria have also been reported.[25] When these perforations occur, they tend to be silent and result in hemopericardium and death. In patients taking anticoagulants, it is important to check coagulation factors and monitor them closely after a seemingly insignificant pericardiocentesis because hemopericardium could develop just from the procedure itself.

In the series compiled by Krikorian and Hancock,[126] hemopericardium developed in 13 of 123 patients as a result of pericardiocentesis, one as a result of a lacerated coronary artery. One patient died of a punctured ventricle. Surgical control was necessary in four patients in whom tamponade developed, whereas it did not develop in eight patients with hemopericardium, and they were managed conservatively. Guberman and colleagues[54] reported three lacerations of the RV in 46 patients; one was fatal. Wong and colleagues[167] found five punctures of the RV, four in patients with nonproductive

pericardiocentesis, but none caused any adverse sequelae. In their series of 352 procedures, Duvernoy and associates[168] reported 23 penetrations. In two cases both the RV and LV had been perforated, and in all other cases the RV had been entered.

Researchers differ in their opinions regarding the adverse effects of ventricular puncture. Most ventricular punctures involve the lower aspect of the RV. The wall of the RV is thin and therefore vulnerable to laceration. However, pressure in the RV is low,[2] so a puncture should cause little bleeding. In a series of patients who underwent ultrasound-directed pericardiocentesis, ventricular puncture occurred in 1.5% but was without consequence.[157] In another study, laceration of the RV occurred in 1 patient despite the use of echocardiography; it resulted in tamponade and necessitated emergency surgery.[123] Of the 23 perforations in the series by Duvernoy and associates, 3 were considered major complications (2 patients required thoracotomy). Left ventricular pseudoaneurysm typically occurs as a complication of MI. It is rarely seen after surgery, trauma, or infection. Rare cases of severe left ventricular pseudoaneurysm after pericardiocentesis have been reported.[174,175]

Even when pericardiocentesis has induced no physical injury, adverse events have been documented. Most have to do with the fact that during pericardiocentesis the stroke volume of the previously collapsed RV increases 75% after the first 200 mL of fluid is removed.[7] In general, this increase in stroke volume is greater initially than that demonstrated by the LV. This imbalance can cause significant consequences for both right and left ventricular function. Three of six patients in whom large effusions were removed by pericardiocentesis experienced right ventricular dilation and overload, abnormal septal motion, and either no increase or a decrease in the right ventricular ejection fraction.[176] These patients subsequently and slowly returned to normal hemodynamic status.

Pulmonary edema following pericardiocentesis has also been reported, presumably caused by a sudden increase in venous return to the LV when peripheral vascular resistance is still high from compensatory catecholamine secretion.[177–181] Supporting evidence for this explanation is that right ventricular stroke volume increases more than left ventricular stroke volume after relief of tamponade.[10] Circulatory collapse with persistently low arterial blood pressure has been reported in a patient from whom 700 mL of clear fluid was drained at a rate of 100 mL/min.[182] Thus, many authors recommend that the pericardial drainage rate not exceed 50 mL/min.

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