

121 Pericardial Effusion and Cardiac Tamponade

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NORMAL ANATOMY OF THE PERICARDIUM

The pericardium is a two-layered fibroelastic membrane or sac that surrounds the heart and proximal segments of the aorta, pulmonary artery, pulmonary veins, and vena cava. A small physiologic amount of fluid is present between the fibrous parietal layer and the single-cell-thickness visceral layer. The small volume of normal pericardial fluid plasma ultrafiltrate is usually less than 50 mL^{1,2} and is believed to come from the visceral pericardium. Normal pericardial pressure is 0 to 3 mm Hg, varying with respiration. The pericardial space drains via lymphatics through both the thoracic duct and the right lymphatic duct. The pericardial space in normal health is more potential than actual, containing only enough fluid to allow for smooth sliding of the serosal layers over one another during cardiac motion. However, a wide variety of diseases affecting the pericardium can lead to an abnormal increase in pericardial fluid.

PERICARDIAL EFFUSION

Accumulations of fluid within the pericardial space may be caused by transudate, exudate, hemopericardium, and pyopericardium.³ Table 121.1 describes some of the most common causes of pericardial effusions.⁴ The presence or absence of symptoms or signs of pericardial effusion depend on many factors, including the cause of the fluid, the size of the effusion, and most important from a hemodynamic standpoint, the rate of fluid accumulation. Typically, a small noninflammatory pericardial effusion is asymptomatic and may be discovered only incidentally when an imaging study (usually either echocardiography or chest computed tomography [CT]) is ordered for other reasons. Large pericardial effusions may also be asymptomatic if they develop slowly; however, large effusions can cause symptoms if associated with increased intrapericardial pressure (discussed later under Cardiac Tamponade) or if they cause mechanical compression of structures adjacent to the heart. Specifically, esophageal compression can cause dysphagia, bronchial or tracheal compression can cause cough, phrenic nerve compression can cause hiccups, recurrent laryngeal nerve compression can result in hoarseness, and lung compression or atelectasis can cause dyspnea. Various arbitrary definitions have been promulgated⁵ to define pericardial effusion size into small, moderate, and large categories. Small effusions are usually defined as 50 to 100 mL,

moderate as 100 to 500 mL, and large as greater than 500 mL. It is important to note that there is often a poor correlation between the volume of pericardial fluid and hemodynamic effects.

Echocardiography in Pericardial Effusion

Echocardiography has long been considered the first-line imaging test to diagnose pericardial effusion. Echocardiography takes advantage of the different acoustic properties of pericardial fluid as compared with the myocardium and parietal pericardium because pericardial fluid is typically echolucent, and the myocardium and pericardial membrane are echo dense. The detection of pericardial effusion was one of the first clinically useful applications of echocardiography,⁶ which continues being the most practical way to diagnose, assess the severity of, and follow pericardial effusions.

Pericardial fluid, when trivial or small, tends to first accumulate posteriorly behind the left ventricle in the oblique sinus and is best imaged in the parasternal long-axis view. It is seen (when trivial) as an echolucent space between the posterior left ventricular (LV) myocardium and pericardial reflection only in systole. As the fluid volume increases, the echolucent posterior separation occurs in both systole and diastole. As the fluid accumulates to greater than 100 mL, it tends to become circumferential, and an echolucent space appears both anteriorly and posteriorly. Measuring the depth in millimeters of the echolucent separation of pericardial reflection from the myocardium or epicardium on two-dimensional (2D) echocardiography is an accepted semiquantitative way to size pericardial effusion (Table 121.2).

There are both normal anatomic variants (epicardial fat) and nonpericardial processes that can sometimes confuse and potentially mimic the echocardiographic appearance of pericardial effusion (Table 121.3). As noted in Table 121.3, left pleural effusion may also appear as an echolucent space posterior to the heart. However, the 2D parasternal long-axis view shows this fluid as posterior to the descending thoracic aorta, whereas pericardial fluid tracks anterior to the descending aorta (Fig. 121.1). Pericardial cysts and LV pseudoaneurysms can also occasionally be mistaken for loculated pericardial effusions. Epicardial fat can appear as a relatively echolucent space most often anteriorly within the pericardial reflection. The stippled appearance of epicardial fat is often seen to move in unison with cardiac motion (Fig. 121.2). Increased epicardial fat is typically seen in older adults, particularly in obese women.⁶

TABLE 121.1 Causes of Pericardial Effusion

Inflammatory
• Infectious
• Autoimmune
NEOPLASTIC
Endocrine or Metabolic
• Myxedema
• Uremia
Trauma, iatrogenic, or Surgery
Radiation Treatment
Volume Overload States
• Congestive heart failure
• Cirrhosis
Myocardial Infarction
Idiopathic

TABLE 121.2 Echocardiographic Quantification of Pericardial Effusion

Trivial	Echolucent space <10 mm Seen only in systole
Small	Echolucent space <10 mm Seen in systole and diastole
Moderate	Echolucent space 10–20 mm
Large	Echolucent space >20 mm

TABLE 121.3 Mimics of Pericardial Effusion

Pleural effusion
Epicardial fat
Left ventricular pseudoaneurysm
Pericardial cyst

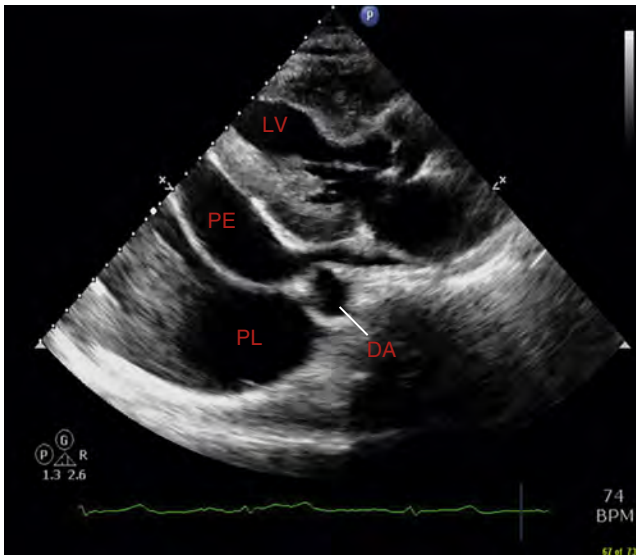


Figure 121.1. Parasternal long-axis image in a patient with both pericardial (PE) and pleural (PL) effusions. Note that the pericardial fluid lies anterior to the descending thoracic aorta (DA). The left ventricle (LV) is marked for reference.

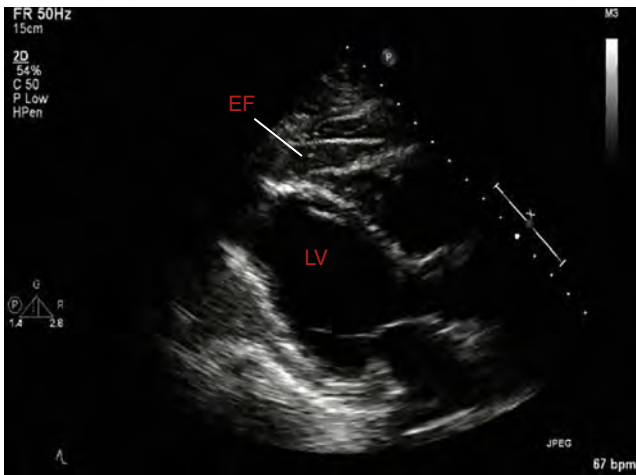


Figure 121.2. Parasternal long-axis image demonstrating anterior epicardial fat (EF). Note the typical stippled appearance of the tissue. The left ventricle (LV) is marked for reference. (See accompanying Video 121.2.)

Pericardial effusions are not always completely echolucent on echocardiography and may have varying degrees of echogenicity related to the presence of fibrin or clot, protein content, chyle, tumor cells, bacteria, and so on. The specific fluid density can be better defined sometimes with other imaging modalities such as CT and magnetic resonance imaging (MRI). However, the relative echogenicity on echocardiography should always be noted and characterized in the report because the fluid appearance (echolucent vs fibrin stranding and organization) can sometimes provide clues to the cause of the pericardial effusion and can also help decide whether drainage (if indicated) would be better accomplished with needle pericardiocentesis or surgically, with highly organized effusions most often requiring operative intervention because of loculation.

CARDIAC TAMPONADE

Cardiac tamponade is an abnormal accumulation of fluid or blood (or both) in the pericardial space that both compresses the cardiac

TABLE 121.4 Clinical and Echocardiographic Findings in Cardiac Tamponade

Clinical Findings	Echocardiographic Findings
Hypotension	Pericardial effusion
Elevated JVP	Chamber collapse
Pulsus paradoxus	IVC plethora
Tachycardia or tachypnea	Hepatic venous flow pattern change
Diminished heart sounds	Exaggerated respiratory-related changes in RV and LV size and Doppler inflow velocities
	Swinging heart

IVC, Inferior vena cava; JVP, jugular venous pressure; LV, left ventricular; RV, right ventricular.

chambers and inhibits chamber filling, leading to varying degrees of decreased cardiac output. When severe, it is characterized by elevated and equalized diastolic chamber and pericardial pressures, an exaggerated drop in blood pressure during inspiration (pulsus paradoxus: a >10-mm drop in systolic blood pressure with inspiration), and a reduced blood pressure (Table 121.4). It is important to remember that cardiac tamponade occurs along a continuum ranging from mild (in which clinical or bedside signs may be absent) to severe (cardiogenic shock and ultimately death). The amount of fluid or blood needed to cause cardiac tamponade is determined primarily by two interacting factors: rate of fluid accumulation (ranging from slow to very fast) and pericardial compliance or elasticity.⁷

Acute Tamponade

Rapid accumulation of as little as 150 mL of fluid or blood can cause profound tamponade physiology. This situation, known as acute tamponade, involves abrupt bleeding into a relatively stiff pericardial space, resulting in rapidly rising intrapericardial pressure (>15 mm Hg), chamber compression, and hypotension, leading to cardiogenic shock. Acute tamponade can occur with penetrating chest wounds, cardiac contusion, complication of diagnostic or therapeutic invasive cardiac procedures (coronary angiography, pacemaker implantation, cardiac ablation, and so on), free wall LV rupture as a late complication of acute myocardial infarction, and proximal aortic dissection rupturing into the pericardial space.

Subacute Tamponade

When pericardial effusion develops gradually, the pericardium stretches and can allow for the accumulation of large amounts of pericardial fluid (>1000 mL) without any significant increase in intrapericardial pressure (<10 mm Hg). Eventually, increasing pericardial fluid volume reaches a critical point at which the pericardium can no longer stretch, and even small additional pericardial fluid may cause a marked increase in intrapericardial and intracardiac pressures, leading to cardiac tamponade. Importantly, these more chronic effusions can result in tamponade if there is a decrease in intracardiac pressure (e.g., with diuresis, acute blood loss, and hemodialysis), resulting in so-called “low-pressure tamponade.”^{8,9}

Echocardiography in Cardiac Tamponade

As noted earlier, cardiac tamponade can occur with relatively small pericardial collections, whereas large pericardial effusions sometimes do not have hemodynamic import. Thus, the echocardiography and Doppler examination needs to assess the hemodynamic significance of the pericardial effusion. Many echocardiographic and Doppler signs of cardiac tamponade have been described; these are described in the following paragraphs. In any individual patient case, the number of abnormal echocardiography and Doppler signs

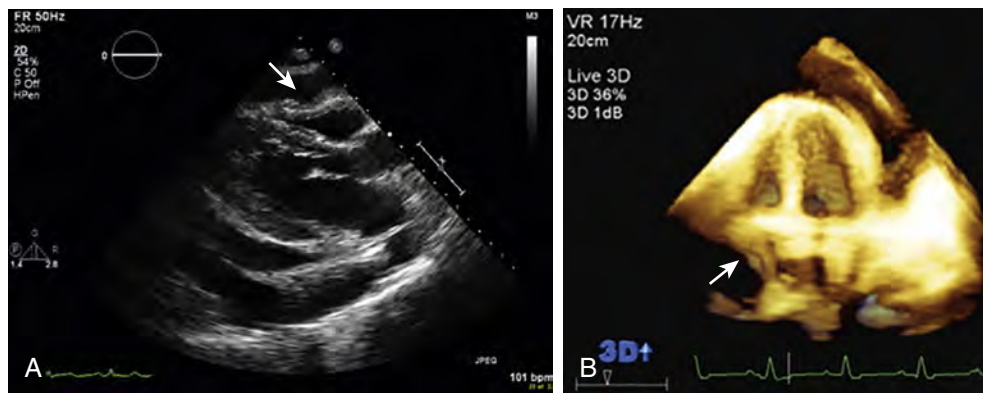


Figure 121.3. Chamber collapse. **A**, Parasternal long-axis view shows right ventricle (RV) collapse (arrow) in end-diastole. **B**, Three-dimensional apical four-chamber view demonstrating right atrial collapse. **C**, Note the open mitral valve and indented RV cavity during diastole. RV collapse may also be seen on parasternal short-axis views. (See accompanying Video 121.3.)

of cardiac tamponade present increases as the hemodynamic or clinical severity of the pericardial effusion progresses.

The reported echocardiography and Doppler signs of cardiac tamponade are given in Table 121.4.

Chamber Collapse

As intrapericardial pressure rises with increasing pericardial fluid, intrapericardial pressure begins to exceed diastolic intracardiac pressure and cause partial chamber wall collapse. This usually occurs with the right heart chambers because they operate at lower diastolic pressure and have thin or compliant walls (Fig. 121.3).

1. *Right atrial (RA) chamber collapse (inversion).*¹⁰ As the intrapericardial pressure rises, it will exceed RA intracardiac pressure first in late diastole, when RA pressure is lowest, at the onset of atrial relaxation. RA chamber collapse is often seen early in the course of tamponade physiology, commonly preceding typical clinical or bedside signs of tamponade, such as hypotension or pulsus paradoxus. Thus, RA collapse is sensitive but not a specific sign of cardiac tamponade. However, the specificity of this sign improves if the duration of RA collapse exceeds 30% of the cardiac cycle.¹¹ RA collapse is often best imaged either in the apical or subcostal four-chamber 2D views.
2. *Right ventricular (RV) chamber collapse (inversion).*¹² RV wall inversion occurs typically in early diastole, when intracavitary RV pressure and volume is at its nadir. Again, as in RA wall collapse, the RV wall inversion extends further into diastole (longer duration) as the hemodynamics of tamponade worsen. This echocardiography finding is often best seen in the parasternal long-axis view, with transient “dimpling” of the RV outflow tract anterior wall noted when the mitral valve opens. If the patient is tachycardic, the timing of this early diastolic inward motion of the RV wall can sometimes be better appreciated using M-mode recording.

Both of these right heart chamber signs of compression depend on the intrapericardial pressure rising above the intracardiac chamber pressure. Thus, any preexisting condition associated with elevated right heart pressure may potentially mask these signs.¹² Conditions including RV hypertrophy and significant pulmonary hypertension are examples. In addition, cardiac tamponade physiology without right heart collapse has also been described¹³ in the setting of infected or organized pericardial collections (tuberculosis and pyogenic), in which it is hypothesized that intrapericardial adhesions prevent the development of collapse.

3. *Left-sided chamber collapse.* Left atrial (LA) and LV chamber compression has almost exclusively been described^{14–16} as related to loculated or regional collections occurring

postoperatively in the cardiac or thoracic surgical patients and is discussed further later. However, circumferential pericardial effusion leading to cardiac tamponade from LV diastolic compression has rarely been reported in the setting of severe pulmonary hypertension.⁷

Inferior Vena Cava Plethora

A dilated (≥ 2.1 cm) inferior vena cava (IVC) with associated minimal ($<50\%$) respiratory change in size is considered a sensitive sign of cardiac tamponade physiology, reflecting the elevated intrapericardial pressure transmitted to the right heart chambers. Studies have found a sensitivity of greater than 90% for cardiac tamponade in patients with both pulsus paradoxus and dilated IVC, with improvement after pericardial drainage.¹⁸ Because a dilated IVC is seen in many other conditions, IVC plethora is considered a nonspecific sign of cardiac tamponade. However, its presence in the setting of a patient with a moderate or large pericardial effusion is helpful in implying that the pericardial fluid has a hemodynamic burden. This echocardiographic sign, often obvious to experienced readers with 2D imaging subcostally, can be better visualized with M-mode recording of the IVC, allowing measurement of size and greater than 50% variation in size with respiration or sniff (Fig. 121.4). Of note, IVC plethora is typically absent with low-pressure tamponade because patients have decreased right heart filling pressure.

Echocardiography and Doppler Signs of Increased Ventricular Interdependence

As increasing pericardial fluid leads to increasing intrapericardial pressure, the cardiac chambers “compete” for increasingly more limited space. Thus, an increase in filling of one ventricle causes a decrease in filling of the other. This is a respiration-related finding, which occurs for two reasons:

- Inspiration-related drop in intrathoracic pressure (and pulmonary venous pressure) is not transmitted to the heart and left heart chambers (shielded by high intrapericardial pressure or volume); thus, the pulmonary venous to LA pressure gradient is reduced in inspiration, lessening flow during inspiration into the left heart.¹⁹
- The systemic venous to RA pressure gradient is less affected with inspiration, favoring flow into the right heart during inspiration. The only RV wall with any “give” is the interventricular septum, which shifts leftward during inspiration. This septal shift increases RV size and reduces LV size and filling.

Doppler mitral and tricuspid diastolic inflow velocities are the most useful and practical way to quantify the exaggerated changes

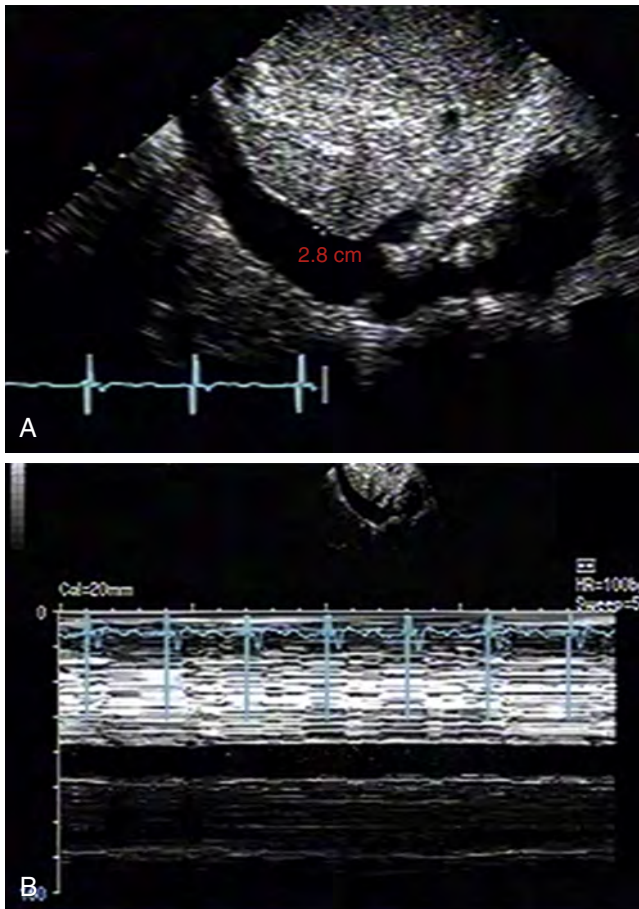


Figure 121.4. **A**, Subcostal view shows engorgement of the inferior vena cava. **B**, M-mode echocardiography shows an absence of inspiratory collapse.

in right and LV filling related to breathing.²⁰ Specifically, mitral inflow E-velocity decreases with inspiration: a drop of greater than 30% compared with expiration during regular breathing is considered consistent with significant tamponade physiology. Conversely, tricuspid inflow E velocities are significantly increased with inspiration, and a greater than 60% increase is seen compared with expiration. These changes are usually best demonstrated with pulsed-wave Doppler recording (and superimposed respirometer) from the apical four-chamber view at a slow sweep speed (Fig. 121.5). Prolongation of the isovolumetric relaxation time, measured at the mitral valve annulus on tissue Doppler images, is also increased with inspiration, reflecting increased LV filling pressure.²¹ The 2D echo correlate to these Doppler findings is the presence of a noticeable sudden leftward shift of the interventricular septum toward the left ventricle with inspiration. This finding is best appreciated in the apical four-chamber view.

These echocardiography and Doppler signs of increased ventricular interdependence, along with their clinical correlate of pulsus paradoxus (Fig. 121.6), are not specific for cardiac tamponade²² (Table 121.5). Marked dyspnea, severe chronic obstructive pulmonary disease, and pulmonary embolism have all been associated with both pulsus paradoxus and the above-described echocardiography and Doppler findings. Thus, clinical history together with presence or absence of pericardial effusion is often required to clarify the cause.

Certain conditions, when present in a patient with other signs of cardiac tamponade, may prevent or lessen this enhanced interventricular interaction (see Table 121.5). Pulsus paradoxus and the echocardiography and Doppler signs of exaggerated ventricular

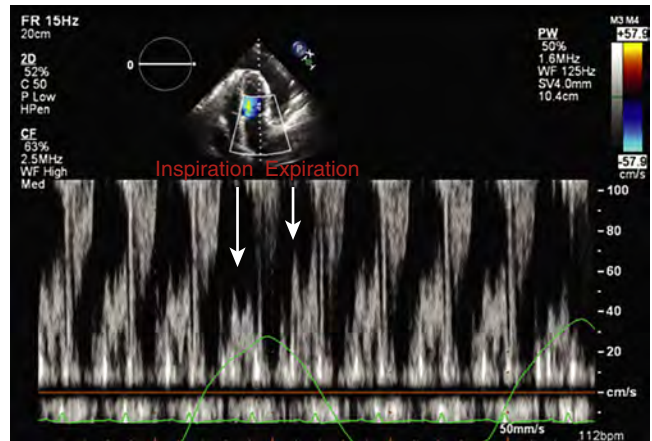


Figure 121.5. Respiratory variation in flow across the mitral valve as measured by Doppler echocardiography. Note the inspiratory decrease in left ventricular inflow.

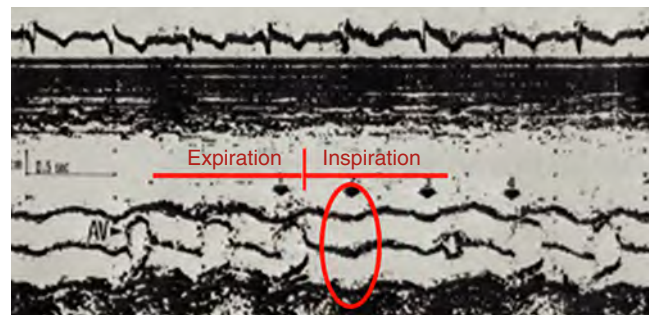


Figure 121.6. Absolute pulsus paradoxus in a patient with pericardial tamponade. Note that the aortic valve fails to open during inspiration—a dramatic M-mode representation of decreased left ventricular stroke volume.

TABLE 121.5 Presence of Pulsus Paradoxus in the Presence of Tamponade

Pulsus Paradoxus+, Tamponade-	Pulsus Paradoxus+, Tamponade-
Constrictive pericarditis	Positive-pressure ventilation
Pulmonary embolus	Atrial septal defect
COPD	Aortic insufficiency
Rapid, deep breathing	High LV filling pressure

COPD, Chronic obstructive pulmonary disease; *LV*, left ventricular.

interaction may not occur if the LV diastolic pressure is markedly elevated. Also, atrial septal defect, in which the increased inspiratory systemic venous return is shared between both sides of the heart, may prevent these findings. Significant aortic regurgitation and positive-pressure ventilation are two additional conditions in which pulsus paradoxus may be absent in cardiac tamponade.^{23,24}

Caveats

- Loculated pericardial collections causing “regional” cardiac tamponade. This scenario is most commonly seen in early post-operative cardiac surgery patients^{14,25} and often is related to a collection of blood clot compressing an individual chamber, most commonly the atria. Given the often limited transthoracic windows available in these patients, together with the localized or loculated nature of these collections, both a high index of

suspicion together with other imaging modalities (transesophageal echocardiography, CT, cardiac MRI [CMRI]) are often necessary to diagnose these situations of regional cardiac tamponade, particularly as typical compressive right heart findings may be absent.

- Large left pleural effusions²⁶ have occasionally been described as causing tamponade physiology, sometimes with echocardiography signs such as RV diastolic collapse. In these situations, there is often pericardial effusion present, and it can be difficult to decide which collection is more significant. Clinical experience usually favors first draining the more accessible pleural fluid and then reassessing both clinically and by echocardiography.
- Hepatic venous Doppler recording,^{4,13} although not always easy to obtain, can show characteristic findings in cardiac tamponade. Specifically, reduced velocities (decrease from the normal 50-cm/s range to 20 to 40 cm/s in tamponade) and a marked predominance of flow in systole is seen. In addition, the most characteristic sign of tamponade is noted on the first beat of expiration, showing absent or reversed diastolic flow. When these findings can be obtained, they are believed to be highly specific for cardiac tamponade. However, in addition to the technical difficulties in obtaining these recordings, the presence of either atrial fibrillation or significant tricuspid regurgitation may confound these hepatic venous changes.
- Swinging of the heart sometimes occurs with large pericardial effusions and cardiac tamponade as the heart swings toward and away from the chest wall and is easily appreciated with 2D echocardiographic imaging. This excessive cardiac motion is the basis for the classic ECG finding of electrical alternans (Fig. 121.7). This phenomenon is caused by beat-to-beat reciprocal variation in ventricular size. Removal of even small amounts of fluid may relieve the tamponade and this excess cardiac motion.

ECHOCARDIOGRAPHY-GUIDED PERICARDIOCENTESIS

Most hospitals now perform pericardiocentesis under echocardiographic guidance. This technique, championed by the Mayo Clinic,²⁷ involves transthoracic preprocedural imaging to select the optimal location on the chest with the most direct route to the largest collection of pericardial fluid, preferably at a minimal distance from the skin to fluid, and avoiding unintended structures such as the left lung or internal thoracic arteries. The most common approach with echo guidance is from the paraapical cardiac region²⁸ and has been performed in experienced hands with more than 99% success, no deaths, and a low complication rate. Imaging during the procedure, sometimes with injection of agitated saline to document proper needle and catheter position in the pericardial space, allows for assessment of success in eliminating the fluid.

SUMMARY

Echocardiography is particularly well-suited for the assessment of pericardial effusion presence, size, and hemodynamic significance. Coupled with careful clinical assessment, echocardiography and Doppler are often essential in helping to decide when and whether pericardial effusion should be drained and even how (surgical or pericardiocentesis) to treat it. When a pericardial effusion is large, circumferential, and not organized, most centers now favor needle pericardiocentesis as the procedure of choice. Other imaging modalities (CT and CMRI) are of significant value in complex pericardial effusions (but not life-threatening tamponade) for both tissue characterization of fluid and to define the feasibility of percutaneous versus surgical intervention.

Please access ExpertConsult to view the corresponding videos for this chapter.

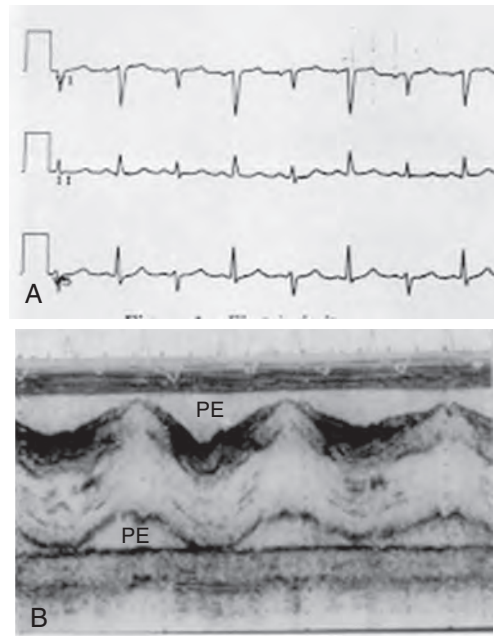


Figure 121.7. Electrical alternans on electrocardiography (A) and M-mode echocardiography (B). Swinging of the heart in the pericardial fluid (PE) alters the vectors on electrocardiography in every other beat.

Acknowledgments

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