# Pericardial effusion: Approach to diagnosis

**AUTHOR:** Brian D Hoit, MD
**SECTION EDITORS:** Martin M LeWinter, MD, Daniel J Sexton, MD
**DEPUTY EDITOR:** Susan B Yeon, MD, JD, FACC

All topics are updated as new evidence becomes available and our peer review process is complete.

Literature review current through: **Oct 2023.**
This topic last updated: **Nov 10, 2023.**

## INTRODUCTION

The pericardium is a fibroelastic sac surrounding the heart that contains a thin layer of fluid. A pericardial effusion is considered to be present when accumulated fluid within the sac exceeds the small physiologic amount (15 to 50 mL). An approach to pericardial effusion, including identification and management, is presented here.

Related issues are discussed separately:

- (See "Etiology of pericardial disease" and "Acute pericarditis: Clinical presentation and diagnosis" and "Pericardial disease associated with cancer: Clinical presentation and diagnosis" and "Purulent pericarditis" and "Tuberculous pericarditis" and "Post-cardiac injury syndromes" and "Pericardial complications of myocardial infarction".)
- (See "Management of pericardial effusion and acute pericarditis during pregnancy" and "Pericardial disease associated with cancer: Management".)

## WHEN TO SUSPECT PERICARDIAL EFFUSION

Pericardial effusion should be suspected in the following clinical settings, particularly in patients with any of the disorders known to involve the pericardium ( table 1). (See "Etiology of pericardial disease", section on 'Pericardial effusion'.)

**Urgent conditions** — Diagnosis and management of pericardial effusion is most urgent for patients with cardiac tamponade. Purulent pericarditis also requires urgent identification and treatment.

- **Cardiac tamponade** – Cardiac tamponade is caused by pericardial fluid accumulating under pressure, impairing cardiac filling and reducing stroke volume. Tamponade is suspected based upon the patient's history (which may include a cause of pericardial disease or effusion ( table 1 and table 2)), symptoms (eg, fatigue, dyspnea, syncope, presyncope), and signs. Nearly all patients with cardiac tamponade have one or more of the following physical findings (see "Cardiac tamponade", section on 'Clinical presentation'):

  - **Sinus tachycardia** – This nonspecific sign is seen in nearly all patients with cardiac tamponade, while hypotension is more variably present. In the setting of cardiac tamponade, sinus tachycardia is a sign of hemodynamic compromise, which should be urgently treated with pericardial fluid drainage.

  - **Elevated jugular venous pressure** – This finding is seen in nearly all patients with cardiac tamponade. Peripheral edema may also be present. However, patients who are severely hypovolemic may present with low pressure tamponade, which is associated with low jugular venous pressure. (See "Cardiac tamponade", section on 'Low-pressure cardiac tamponade'.)

  - **Pulsus paradoxus** – This finding is defined as an abnormally large decrease in systolic blood pressure (>10 mmHg) on inspiration. It is a common finding in moderate to severe cardiac tamponade and is a direct consequence of ventricular interdependence ( waveform 1 and waveform 2 and waveform 3). (See "Pulsus paradoxus in pericardial disease".)

  - **Hemodynamic deterioration** – Cardiac tamponade is one of the causes of hemodynamic instability and should be suspected, particularly in patients with a condition that may involve the pericardium (eg, ascending aortic dissection). (See "Clinical features and diagnosis of acute aortic dissection", section on 'Hypotension'.)

  The diagnosis and management of cardiac tamponade, including urgent pericardial fluid removal, are discussed separately. (See "Cardiac tamponade".)

- **Purulent pericarditis** – This condition typically presents with high fever and tachycardia, with or without chest pain. A high index of suspicion is required, particularly in patients with a known infection that may seed or spread to the pericardium or prior perforating injury of the pericardium. Urgent diagnosis and management is required, as discussed separately. (See "Purulent pericarditis".)

**Other clinical settings** — An evaluation for pericardial effusion is also indicated in patients with the following findings or conditions:

- All cases of acute pericarditis. (See "Acute pericarditis: Clinical presentation and diagnosis", section on 'Diagnostic evaluation'.)
- Unexplained low voltage on the electrocardiogram (ECG). (See 'ECG findings' below.)
- Unexplained new radiographic cardiomegaly, particularly when present without pulmonary congestion. (See 'Chest radiograph' below.)
- Presence of isolated left (or left larger than right) pleural effusion [1].

Additional clinical presentations that may suggest pericardial effusion are discussed below (see 'Clinical presentation' below). Also, a pericardial effusion may be detected as an incidental finding on imaging (eg, echocardiography or computed tomography [CT]) performed to evaluate another condition.

**Clinical presentation** — The following clinical findings occur in patients with pericardial effusion with or without cardiac tamponade.

**Symptoms and signs** — Patients with pericardial effusion may experience chest pain or fullness. However, many patients with pericardial effusion without tamponade have no symptoms or signs directly caused by the pericardial effusion, although they may have symptoms related to the underlying cause of pericardial disease (eg, fever in the setting of infectious or immune-mediated pericarditis). Thus, pericardial effusions are often discovered incidentally during evaluation for other cardiopulmonary diseases.

In the absence of cardiac tamponade, physical signs of pericardial effusion are so insensitive and nonspecific that they are of historic rather than practical interest. As an example, the ability to percuss cardiac dullness beyond the apical point of maximal impulse is consistent with pericardial effusion, but it depends upon the absence of disease of the lower lobe of the left lung or left pleura and the expertise of the examiner. Ewart sign (which includes a triangular area of dullness at the tip of the left scapula along with tubular breath sounds and egophony at the same location) suffers from similar limitations [2].

**Initial test findings** — The presence of a pericardial effusion may be suggested by the ECG or chest radiograph.

**ECG findings** — An ECG should be performed in patients with suspected pericardial disease. The most common ECG findings in patients with a pericardial effusion are sinus tachycardia, low QRS voltage, and electrical alternans. If pericarditis is present, the ECG findings typical of that disorder (such as ST elevation and PR depression) are also seen ( waveform 4). (See "Acute pericarditis: Clinical presentation and diagnosis", section on 'Electrocardiogram'.)

- **Sinus tachycardia** – Sinus tachycardia (sinus rhythm with rate >100 beats per minute) is a nonspecific sign that is common in patients with pericardial effusion, particularly those with cardiac tamponade. In a study of ECG findings in 241 patients with moderate or large pericardial effusion, sinus tachycardia was present in 43 of 64 patients (67 percent) with cardiac tamponade and 41 of 160 patients (26 percent) without tamponade [3].

- **Low QRS voltage** – Low QRS voltage is common in patients with pericardial effusion. Low QRS voltage may be observed in the limb leads and/or precordial leads. Low QRS voltage in the limb leads ( waveform 5) is present when the amplitudes (nadir to peak) of the QRS complexes in each of the six limb leads is less than 5 mm (0.5 mV). Low voltage in the precordial leads is present when the amplitude in each these leads is less than 10 mm (1 mV).

  While the frequency of low voltage appears to be higher among patients with cardiac tamponade than among other patients with pericardial effusion, the presence of low voltage is not specific for cardiac tamponade. In the above-cited study of ECG findings in 241 patients with moderate or large pericardial effusion, low voltage in limb and/or precordial leads was present in 66 percent of patients with cardiac tamponade and in 34 percent of patients without cardiac tamponade [3]. Low voltage is more common with moderate or large pericardial effusions than with small effusions (eg, 83 versus 13 percent in one series [4]).

  The combination of low voltage and sinus tachycardia should raise concern about pericardial effusion with cardiac tamponade. However, the combination of low voltage and sinus tachycardia is not specific to patients with pericardial effusion, as this combination of findings may also be seen in patients in a variety of clinical settings, such as chronic obstructive pulmonary disease, pleural effusion, cardiomyopathy, and prior cardiac surgery ( table 3).

- **Electrical alternans** – Electrical alternans is an ECG finding that is observed in some patients with pericardial effusion, particularly those with cardiac tamponade. Electrical alternans is characterized by beat-to-beat alternating amplitude of the QRS complexes (and more subtly of other waveforms) that reflects swinging of the heart in the pericardial fluid ( waveform 6 and movie 1). The pattern is usually most apparent in one or more of the precordial leads. (See "ECG tutorial: Miscellaneous diagnoses", section on 'Electrical alternans'.)

  Electrical alternans ( waveform 6) with sinus tachycardia is a highly specific sign of pericardial effusion, usually with cardiac tamponade, but is only modestly sensitive. As a result, its absence does not exclude cardiac tamponade. In the above-cited study of ECG findings in 241 patients with moderate or large pericardial effusion, electrical

alternans was observed in 35 percent of patients with cardiac tamponade and in only 7 percent of patients without tamponade [3].

In contrast, QRS alternans with other tachycardias (eg, paroxysmal supraventricular tachycardias or ventricular tachycardia) is a nonspecific finding resulting from alternation in conduction on a beat-to-beat basis, not from mechanical movement of the heart.

**Chest radiograph** — A chest radiograph performed in patients with suspected pericardial effusion may identify findings (such as pleural effusions, lung disease, hilar masses, or mediastinal widening) related to the cause of pericardial effusion [5].

However, an enlarged cardiac silhouette on chest radiograph is neither a sensitive nor specific sign of pericardial effusion or cardiac tamponade. The findings on chest radiograph in patients with pericardial effusion are variable, depending on the etiology and size of the pericardial effusion and underlying comorbidities. Small to moderate effusions (less than 200 to 300 mL) are generally not associated with significant findings on the chest radiograph [5], while larger pericardial effusions typically present with an enlarged cardiac silhouette with clear lung fields ( image 1).

**Cardiac biomarkers** — Among patients with pericardial effusion, cardiac biomarkers (eg, serum troponin) may be elevated in those in whom the effusion occurs in association with acute myopericarditis or myocardial infarction. Troponin elevations, typically modest, may occur in association with a variety of other acute illnesses. (See "Elevated cardiac troponin concentration in the absence of an acute coronary syndrome" and "Acute pericarditis: Clinical presentation and diagnosis", section on 'Cardiac biomarkers' and "Myopericarditis".)

## DIAGNOSTIC APPROACH

Once a pericardial effusion is suspected, the diagnostic approach includes the following three steps:

- **Identify and assess the pericardial effusion** – A pericardial effusion is generally diagnosed by echocardiography, with additional cardiac imaging performed only in selected cases, as discussed below. (See 'Cardiac imaging' below.)
- **Assessing its hemodynamic impact** – Once it is determined that a pericardial effusion is present, the next step is to determine if cardiac tamponade is present, as this is a cause of hemodynamic instability and requires urgent pericardial drainage. (See 'Urgent conditions' above and "Cardiac tamponade".)

- **Diagnostic evaluation** – After a pericardial effusion has been identified, evaluation is performed to determine its cause. (See 'Clinical presentation' above.)

## CARDIAC IMAGING

An echocardiogram is the key diagnostic test for pericardial effusion [5]. Additional cardiac imaging with cardiac computed tomography (CT), cardiovascular magnetic resonance (CMR) imaging, and cardiac catheterization is reserved for patients in selected clinical settings, as discussed below.

**Effusion types** — A pericardial effusion may be free-flowing or loculated:

- **Free-flowing effusion** – (See 'Effusion size' below.)
- **Loculated** – When a pericardial effusion becomes loculated (regional or compartmentalized) it may not be visible in standard echocardiographic views. In some cases, it may be best appreciated in subcostal or transesophageal echocardiographic views, or by cardiac CT or CMR. In other cases (eg, cardiac tamponade early after cardiac surgery), a loculated hematoma may be identified by surgical exploration [6].

### Echocardiography

**Identifying the effusion** — Most pericardial effusions are diagnosed on transthoracic echocardiogram (TTE). Two-dimensional (2D) echocardiography is both sensitive and specific for the detection of pericardial effusion and also provides information regarding the hemodynamic significance of the effusion (ie, identification of cardiac tamponade). (See 'Hemodynamic effects' below and "Echocardiographic evaluation of the pericardium".)

Pericardial fluid appears on an echocardiogram as an echolucent space between the pericardium and the epicardium. Physiologic pericardial fluid (25 to 50 mL) is generally visible only during ventricular systole. A significant pericardial effusion (exceeding 25 to 50 mL) is identified as an echo-free space throughout the cardiac cycle. (See "Echocardiographic evaluation of the pericardium", section on 'Normal appearance on echocardiographic imaging' and 'Effusion size' below.)

While M-mode echocardiography can identify an effusion ( image 2), 2D echocardiography permits a better estimate of the size of the effusion, its distribution, and its distance from potential pericardiocentesis sites.

**Effusion size** — Pericardial effusion size is assessed and monitored in patients with pericardial effusion, although pericardial size alone does not determine its hemodynamic significance. (See "Pericardial effusion: Approach to management", section on 'General management' and 'Hemodynamic effects' below.)

Although 2D echocardiography cannot precisely quantify pericardial effusion size (which is better quantified by cardiac CT or CMR), a common approach is to grade free-flowing effusions as small, medium, or large as determined by the size of the echo-free space surrounding the heart. The site of fluid accumulation for a free-flowing effusion is positional due to gravity dependence.

- Small effusions (50 to 100 mL) are typically only seen posteriorly and are generally less than 10 mm wide, causing minimal separation between the epicardial (visceral) pericardium and the thicker parietal pericardial sac. A small free-flowing pericardial effusion is typically identified on echocardiography above the right atrium in the apical four-chamber view with the patient in the left lateral decubitus position ( image 3).

- Moderate effusions (100 to 500 mL) are generally seen along the length of the posterior wall but not anteriorly; the echo-free space is 10 to 20 mm at its greatest width.

- Large effusions (>500 mL) are generally seen circumferentially ( movie 2 and image 4 and image 5); the echo-free space is greater than 20 mm at its greatest width.

**Hemodynamic effects** — The diagnosis of cardiac tamponade is based largely upon clinical and imaging (primarily echocardiographic) findings, although definitive confirmation of the diagnosis is made by hemodynamic assessment via cardiac catheterization during pericardial fluid drainage. (See "Cardiac tamponade", section on 'Diagnosis'.)

With cardiac tamponade, the pericardial effusion is commonly moderate or large ( movie 1), but may be small if the effusion has accumulated rapidly. The speed of pericardial fluid accumulation affects the size of pericardial effusion that causes cardiac tamponade ( figure 1). The normal pericardium can stretch to accommodate increases in pericardial volume, with the amount of stretch related to the period of time over which the pericardial effusion accumulates, as discussed separately. (See "Cardiac tamponade", section on 'Pathophysiology'.)

Echocardiographic features of cardiac tamponade include (see "Cardiac tamponade", section on 'Echocardiogram'):

- Collapse of the right atrium at end-diastole and the right ventricle in early diastole ( movie 3 and movie 4).

- Reciprocal changes in left and right ventricular volumes with respiration, which are important in the pathogenesis of pulsus paradoxus ( figure 2). (See "Pulsus paradoxus in pericardial disease".)

- Increased respiratory variation of mitral and tricuspid valve inflow velocities (increase in mitral flow by 30 percent and decrease in tricuspid valve flow by 60 percent on the first beat of expiration).

- Dilation (plethora) of the inferior vena cava and less than a 50 percent reduction in its diameter during inspiration, reflecting systemic congestion ( image 6 and image 7).

The diagnosis and management of cardiac tamponade are discussed in detail separately. (See "Cardiac tamponade".)

**Cardiac CT and CMR** — Imaging with cardiac CT or CMR is reserved for patients with nondiagnostic echocardiographic evaluation who require further assessment to identify and examine a pericardial effusion (eg, suspected loculated effusion) or to identify other suspected pericardial pathology (eg, constrictive pericarditis or a pericardial mass). (See "Constrictive pericarditis: Diagnostic evaluation", section on 'Additional testing' and "Pericardial disease associated with cancer: Clinical presentation and diagnosis", section on 'Further cardiac imaging'.)

Cardiac CT or CMR is helpful in identifying, characterizing, and quantifying a pericardial effusion and also identifying other pericardial findings such as pericardial thickening and pericardial tethering (which are associated with constrictive pericarditis) [7,8]. In patients who are hemodynamically unstable, cardiac CT is generally preferred to CMR. (See 'Differential diagnosis' below and "Constrictive pericarditis: Diagnostic evaluation", section on 'Additional testing'.)

In some cases, pericardial effusion may be first detected by CT, especially when CT is used to detect pulmonary embolus, a more common cause of shortness of breath and hypotension.

## CARDIAC CATHETERIZATION

Hemodynamic assessment via cardiac catheterization is generally reserved for patients (usually with suspected cardiac tamponade) undergoing pericardial fluid drainage. When feasible, the diagnosis of cardiac tamponade is confirmed by identifying typical hemodynamic findings prior to pericardial fluid removal and resolution of hemodynamic abnormalities after drainage. (See "Cardiac tamponade", section on 'Cardiac catheterization'.)

## DIFFERENTIAL DIAGNOSIS

The differential diagnosis of pericardial effusion suspected on echocardiography includes epicardial fat (commonly visualized anterior to the right ventricular free wall and in the

interventricular and atrioventricular grooves) and pleural effusion. These entities can usually be distinguished from pericardial effusion by careful review of the echocardiogram. Epicardial fat may be identified by the presence of faint linear striations within it. A key landmark for posterior pericardial effusion is that it tracks anterior to the descending aorta. If echocardiographic findings are nondiagnostic, cardiac CT or CMR can distinguish these entities. (See "Echocardiographic evaluation of the pericardium", section on 'Normal anatomy' and "Echocardiographic evaluation of the pericardium", section on 'Differentiating between pleural and pericardial effusions'.)

## IDENTIFYING THE ETIOLOGY

The cause of the effusion is investigated by clinical evaluation (history, physical examination, blood tests) and in some cases by evaluation of pericardial fluid with or without pathologic examination of the pericardium ( algorithm 1).

The cause of a pericardial effusion may be apparent or suspected based upon the clinical setting in which it occurs. Examples include suspected cardiac perforation complicating an invasive procedure, breast or lung cancer, a recent myocardial infarction, severe hypothyroidism, or end-stage kidney disease. (See "Invasive diagnostic cardiac electrophysiology studies", section on 'Complications of invasive cardiac electrophysiology studies' and "Pericardial disease associated with cancer: Clinical presentation and diagnosis" and "Post-cardiac injury syndromes" and "Overview of the management of chronic kidney disease in adults", section on 'Pericarditis' and "Clinical manifestations of hypothyroidism", section on 'Cardiovascular system' and "Clinical manifestations of hypothyroidism".)

When the cause of pericardial effusion is not apparent, evaluation starts with a clinical assessment but may require sampling of the pericardial effusion and/or the pericardial tissue for laboratory analysis. The frequency of different causes of pericardial effusion varies in published reports, depending in part upon the patient population (location and clinical features), as well as the aggressiveness with which an underlying diagnosis was sought ( table 2) [9-12]. (See "Etiology of pericardial disease".)

In many cases, the specific etiology of a pericardial effusion cannot be established even when fluid and/or pericardial tissue is analyzed. The likelihood of establishing a specific cause appears to be greater with larger effusions. In series of patients with acute pericarditis (including those with small or undetectable effusions), the underlying diagnosis was established in only approximately 15 to 20 percent [13,14]. In contrast, studies confined to moderate to large pericardial effusions have revealed diagnoses in up to 90 percent of cases when very aggressive diagnostic approaches are taken [10]. Such aggressive approaches are often not warranted in clinical practice, however, given the benign natural history of many of

these effusions. (See "Acute pericarditis: Clinical presentation and diagnosis", section on 'Establishing a definite etiology'.)

**Causes** — Pericardial effusion can develop in patients with virtually any condition that affects the pericardium, including acute pericarditis and a variety of systemic disorders. The development of a pericardial effusion may have important implications for prognosis (as in patients with intrathoracic neoplasm), diagnosis (as in myopericarditis or acute pericarditis), or both (as in dissection of the ascending aorta).

Pericardial effusions can occur as a component of almost any pericardial disorder ( table 1), but the majority result from one of the following conditions (see "Etiology of pericardial disease"):

- Acute pericarditis (viral, bacterial, tuberculous, or idiopathic in origin)
- Autoimmune disease
- Post-myocardial infarction or cardiac surgery
- Sharp or blunt chest trauma, including a cardiac diagnostic or interventional procedure
- Malignancy, particularly metastatic spread of noncardiac primary tumors
- Mediastinal radiation
- Renal failure with uremia
- Myxedema
- Aortic dissection extending into the pericardium
- Selected drugs

The frequency of the different causes of pericardial effusion varies in published reports, depending primarily upon geography and the patient population ( table 2). Patients with hemorrhagic pericardial effusions have a somewhat different distribution of causes, although the overlap with serous effusions is significant. (See "Etiology of pericardial disease", section on 'Spectrum of clinical presentation'.)

**Evaluation** — Selected clinical information may be helpful for establishing the etiology of a pericardial effusion. This was illustrated in a review of 322 patients with a moderate or large pericardial effusion [9]. In 60 percent of patients, the cause of the effusion was a known medical condition. In the remaining patients, the cause of the effusion was assessed based upon the following features:

- The size of the effusion.
- The presence or absence of cardiac tamponade.
- Inflammatory signs (defined as two or more of the following features: characteristic chest pain, pericardial friction rub, fever >37°C, and diffuse ST-segment elevation).

The presence or absence of these features suggested, but did not confirm, a specific diagnosis [9]:

- The presence of inflammatory signs was associated with acute idiopathic pericarditis (likelihood ratio 5.4).
- A large effusion without inflammatory signs or cardiac tamponade was associated with chronic idiopathic pericardial effusion (likelihood ratio 20).
- Cardiac tamponade without inflammatory signs was associated with a malignant effusion (likelihood ratio 2.9).

**Laboratory tests** — If the initial history and physical examination do not suggest a specific diagnosis, extensive laboratory testing seeking an etiology is unlikely to be helpful. In such cases, we limit routine testing to:

- Complete blood count
- Chemistry profile and renal function
- Thyroid function
- Chest radiography

A serum antinuclear antibody (ANA) test should be considered in a young female with an effusion and associated acute pericarditis since, rarely, this can be the initial presentation of systemic lupus erythematosus.

A careful history directed toward the presence of a coexisting rheumatologic disorder should also be obtained. It is important to keep in mind that a positive ANA is a nonspecific finding. In cases where a rheumatologic diagnosis is being considered in a patient with pericarditis and an effusion, a rheumatology consultation should be sought. (See "Pericardial involvement in systemic autoimmune diseases".)

**Pericardial fluid analysis and biopsy** — Drainage of a pericardial effusion is often performed for therapeutic benefit (ie, in cardiac tamponade). However, pericardial fluid drainage and analysis should be considered for diagnostic purposes in patients without cardiac tamponade if there is significant uncertainty regarding the diagnosis or if knowledge of a particular diagnosis would change the management of the patient (ie, the presence of metastatic malignancy). In addition, pericardial biopsy can be an important part of the diagnostic approach.

The invasive nature of pericardiocentesis and pericardial biopsy must be weighed against two factors: a relatively low diagnostic yield from the procedure, and the clinical relevance of a specific diagnosis, as many low-risk patients can be managed empirically without making a specific diagnosis. The yield of diagnostic pericardiocentesis has varied in different series,

but in most, a definitive diagnosis has been made following pericardiocentesis in less than 40 percent of cases [10,11,13]. In a more contemporary series of 269 patients who had undergone pericardiocentesis, an etiology was determined in 74 percent of cases; the most common etiologies were idiopathic in 26 percent, malignant in 25 percent, and iatrogenic in 20 percent [15]. When an etiology for the pericardial effusion was confirmed, malignancy, purulent infection, and tuberculosis were the most frequently identified causes.

We consider pericardial fluid analysis and pericardial biopsy separately since they are frequently helpful, but many patients are successfully managed without either procedure. Our approach is in general agreement with the 2015 European Society of Cardiology guidelines for the diagnosis and management of pericardial disease [5].

We perform pericardial fluid analysis in the following settings:

- In any patient undergoing therapeutic pericardiocentesis for treatment of cardiac tamponade.
- When there is a clinical suspicion of purulent, tuberculous, or neoplastic pericarditis. (See "Purulent pericarditis" and "Tuberculous pericarditis" and "Pericardial disease associated with cancer: Clinical presentation and diagnosis".)
- In patients with moderate-to-large pericardial effusions of unknown etiology that do not respond rapidly to antiinflammatory therapy.

In patients with chronic pericardial effusions, we do not recommend repeated diagnostic pericardiocentesis due to its low yield.

The biochemical, immunologic, cytologic, and bacteriologic characteristics of an effusion can be established by laboratory analysis of the pericardial fluid. In some cases this analysis can also determine the etiology of the effusion [16,17]. Among the most helpful diagnostic studies are:

- Gram stain and bacterial and fungal culture.
- Cytology.
- Acid-fast bacillus (AFB) stain and mycobacterial culture along with adenosine deaminase, interferon-gamma, or lysozyme (for tuberculous pericarditis) [18].
- Polymerase chain reaction (PCR) testing is ordered if a specific organism (eg, *Mycobacterium tuberculosis*) is suspected.

Parameters such as protein, lactate dehydrogenase (LDH), glucose, red cell count, and white cell count are rarely helpful in establishing an etiology; specifically, Light criteria, used for

differentiating transudative versus exudative pleural effusions, do not reliably distinguish exudative from transudative pericardial effusions and should not be applied to pericardial fluid analysis [19,20]. However, a PCR test for specific viruses can be ordered when management may be affected by the results, such as identifying cytomegalovirus (CMV) in a transplant patient, although the yield of PCR remains fairly low in many instances [21]. (See "Etiology of pericardial disease" and "Tuberculous pericarditis" and "Approach to the diagnosis of cytomegalovirus infection".)

If patient characteristics and local expertise favor pericardial drainage via percutaneous pericardiocentesis, analysis is limited to the pericardial fluid. However, whenever surgical drainage is performed, we also recommend pericardial biopsy. In addition, pericardial biopsy should be performed in any patient in whom there is ongoing diagnostic uncertainty as to the etiology if a specific diagnosis would significantly change the management.

Pericardioscopy enables endoscopic inspection and targeted biopsy of the parietal and visceral pericardium [2]. This approach has been used in patients with unexplained pericardial effusions or to diagnose recurrent idiopathic effusion [22-25]. When performed in one of the few centers with a special interest and expertise in using this approach, a specific diagnosis was established in 49 to 65 percent of patients, a value higher than generally obtained by pericardial biopsy performed under fluoroscopic guidance [22-24].

## SOCIETY GUIDELINE LINKS

Links to society and government-sponsored guidelines from selected countries and regions around the world are provided separately. (See "Society guideline links: Pericardial disease".)

## INFORMATION FOR PATIENTS

UpToDate offers two types of patient education materials, "The Basics" and "Beyond the Basics." The Basics patient education pieces are written in plain language, at the 5$^{th}$ to 6$^{th}$ grade reading level, and they answer the four or five key questions a patient might have about a given condition. These articles are best for patients who want a general overview and who prefer short, easy-to-read materials. Beyond the Basics patient education pieces are longer, more sophisticated, and more detailed. These articles are written at the 10$^{th}$ to 12$^{th}$ grade reading level and are best for patients who want in-depth information and are comfortable with some medical jargon.

Here are the patient education articles that are relevant to this topic. We encourage you to print or e-mail these topics to your patients. (You can also locate patient education articles on a variety of subjects by searching on "patient info" and the keyword(s) of interest.)

- Basics topic (see "Patient education: Cardiac tamponade (The Basics)")
- Beyond the Basics topics (see "Patient education: Pericarditis (Beyond the Basics)")

## SUMMARY AND RECOMMENDATIONS

- **Etiology** – Pericardial effusion can develop in patients with virtually any condition that affects the pericardium ( table 1), including acute pericarditis and a variety of systemic disorders. The frequency of different causes of pericardial effusion varies in published reports, depending on factors including geography and patient demographics, as well as the aggressiveness with which an underlying diagnosis is sought ( table 2).
- **Clinical presentation** – The presence of pericardial effusion may be suspected from the history, physical examination, ECG, and chest radiograph. However, in the absence of cardiac tamponade, physical signs are not sensitive or specific for pericardial effusion. (See 'Diagnostic approach' above.)
- **Diagnosis and evaluation** – Once a pericardial effusion is suspected, the diagnostic approach consists of three steps: confirming the presence of a pericardial effusion (generally by echocardiography), assessing its hemodynamic impact (ie, determining if cardiac tamponade is present), and identifying the cause of the pericardial effusion.
  - **Diagnosis** – The presence of pericardial effusion is usually established with echocardiography. (See 'Cardiac imaging' above.)
  - **Assess the hemodynamic impact** – Both clinical and echocardiographic findings can be helpful in assessing the hemodynamic impact of a pericardial effusion. Cardiac tamponade occurs when the intrapericardial pressure from an accumulating pericardial effusion impedes filling of one or both ventricles. (See 'Urgent conditions' above.)

    Patients with cardiac tamponade require urgent pericardial fluid drainage, as discussed separately. (See "Cardiac tamponade" and "Pericardial effusion: Approach to management", section on 'Indications for pericardial fluid removal'.)
  - **Identification of the cause** – In some cases, the most likely cause of the pericardial effusion will be clear due to the presence of another illness or systemic disorder known to involve the pericardium. If the cause is not apparent, the routine use of a broad panel of screening laboratory tests is not usually helpful. In such cases, evaluation of the pericardial fluid and pericardium itself may suggest an etiology, particularly if the effusion is large.

Patients with suspected purulent pericarditis require urgent diagnosis and management, including pericardial drainage, as discussed separately. (See "Purulent pericarditis".)



**REFERENCES**


1. Weiss JM, Spodick DH. Association of left pleural effusion with pericardial disease. N Engl J Med 1983; 308:696.
2. Ewart W. Practical Aids in the Diagnosis of Pericardial Effusion, in Connection with the Question as to Surgical Treatment. Br Med J 1896; 1:717.
3. Mathur AP, Saini A, Lucas BP, et al. Diagnostic accuracy retrospectively of electrocardiographic findings and cancer history for tamponade in patients determined to have pericardial effusion by transthoracic echocardiogram. Am J Cardiol 2013; 111:1062.
4. Kudo Y, Yamasaki F, Doi T, et al. Clinical significance of low voltage in asymptomatic patients with pericardial effusion free of heart disease. Chest 2003; 124:2064.
5. Adler Y, Charron P, Imazio M, et al. 2015 ESC Guidelines for the diagnosis and management of pericardial diseases: The Task Force for the Diagnosis and Management of Pericardial Diseases of the European Society of Cardiology (ESC)Endorsed by: The European Association for Cardio-Thoracic Surgery (EACTS). Eur Heart J 2015; 36:2921.
6. Ellenbroek DFJ, van Kessel L, Compagner W, et al. Diagnostic performance of echocardiography to predict cardiac tamponade after cardiac surgery. Eur J Cardiothorac Surg 2022; 62.
7. Klein AL, Abbara S, Agler DA, et al. American Society of Echocardiography clinical recommendations for multimodality cardiovascular imaging of patients with pericardial disease: endorsed by the Society for Cardiovascular Magnetic Resonance and Society of Cardiovascular Computed Tomography. J Am Soc Echocardiogr 2013; 26:965.
8. Rosmini S, Seraphim A, Knott K, et al. Non-invasive characterization of pleural and pericardial effusions using T1 mapping by magnetic resonance imaging. Eur Heart J Cardiovasc Imaging 2022; 23:1117.
9. Sagristà-Sauleda J, Mercé J, Permanyer-Miralda G, Soler-Soler J. Clinical clues to the causes of large pericardial effusions. Am J Med 2000; 109:95.
10. Corey GR, Campbell PT, Van Trigt P, et al. Etiology of large pericardial effusions. Am J Med 1993; 95:209.
11. Levy PY, Corey R, Berger P, et al. Etiologic diagnosis of 204 pericardial effusions. Medicine (Baltimore) 2003; 82:385.

12. Ma W, Liu J, Zeng Y, et al. Causes of moderate to large pericardial effusion requiring pericardiocentesis in 140 Han Chinese patients. Herz 2012; 37:183.
13. Permanyer-Miralda G, Sagristá-Sauleda J, Soler-Soler J. Primary acute pericardial disease: a prospective series of 231 consecutive patients. Am J Cardiol 1985; 56:623.
14. Zayas R, Anguita M, Torres F, et al. Incidence of specific etiology and role of methods for specific etiologic diagnosis of primary acute pericarditis. Am J Cardiol 1995; 75:378.
15. Strobbe A, Adriaenssens T, Bennett J, et al. Etiology and Long-Term Outcome of Patients Undergoing Pericardiocentesis. J Am Heart Assoc 2017; 6.
16. Tuon FF, Litvoc MN, Lopes MI. Adenosine deaminase and tuberculous pericarditis--a systematic review with meta-analysis. Acta Trop 2006; 99:67.
17. Levy PY, Fournier PE, Charrel R, et al. Molecular analysis of pericardial fluid: a 7-year experience. Eur Heart J 2006; 27:1942.
18. Chau E, Sarkarati M, Spellberg B. Adenosine Deaminase Diagnostic Testing in Pericardial Fluid. JAMA 2019; 322:163.
19. Ben-Horin S, Bank I, Shinfeld A, et al. Diagnostic value of the biochemical composition of pericardial effusions in patients undergoing pericardiocentesis. Am J Cardiol 2007; 99:1294.
20. Imazio M, Biondo A, Ricci D, et al. Contemporary biochemical analysis of normal pericardial fluid. Heart 2020; 106:541.
21. Ntsekhe M, Mayosi BM. Tuberculous pericarditis with and without HIV. Heart Fail Rev 2013; 18:367.
22. Nugue O, Millaire A, Porte H, et al. Pericardioscopy in the etiologic diagnosis of pericardial effusion in 141 consecutive patients. Circulation 1996; 94:1635.
23. Pankuweit S, Wädlich A, Meyer E, et al. Cytokine activation in pericardial fluids in different forms of pericarditis. Herz 2000; 25:748.
24. Seferović PM, Ristić AD, Maksimović R, et al. Diagnostic value of pericardial biopsy: improvement with extensive sampling enabled by pericardioscopy. Circulation 2003; 107:978.
25. Maisch B, Ristić AD, Pankuweit S. Intrapericardial treatment of autoreactive pericardial effusion with triamcinolone; the way to avoid side effects of systemic corticosteroid therapy. Eur Heart J 2002; 23:1503.

Topic 4941 Version 36.0

## GRAPHICS

### Causes of pericardial disease

| |
|---|
| **Idiopathic (presumed to be viral, postviral, or immune-mediated)** |
| In most case series, the majority of patients are not found to have an identifiable cause of pericardial disease. Frequently such cases are presumed to have a viral or autoimmune etiology. |
| **Infectious** |
| Viral – Coxsackievirus, echovirus, adenovirus, Epstein-Barr virus, cytomegalovirus, influenza, varicella, rubella, HIV, hepatitis B, mumps, parvovirus B19, vaccina (smallpox vaccine), severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2) |
| Bacterial – *Mycobacterium tuberculosis* (most common cause in countries where tuberculosis is endemic), *Staphylococcus*, *Streptococcus*, *Haemophilus*, *Neisseria* (*N. gonorrhoeae* or *N. meningitidis*), *Chlamydia* (*C. psittaci* or *C. trachomatis*), *Legionella*, *Salmonella*, *Borrelia burgdorferi* (the cause of Lyme disease), *Mycoplasma*, *Actinomyces*, *Nocardia*, *Tropheryma whippelii*, *Treponema*, *Rickettsia* |
| Fungal – *Histoplasma*, *Aspergillus*, *Blastomyces*, *Coccidioides*, *Candida* |
| Parasitic – *Echinococcus*, amebic, *Toxoplasma* |
| Infective endocarditis with valve ring abscess |
| **Noninfectious** |
| **Autoimmune and autoinflammatory** |
| Systemic inflammatory diseases, especially lupus, rheumatoid arthritis, scleroderma, Sjögren syndrome, vasculitis, mixed connective disease |
| Autoinflammatory diseases (especially familial Mediterranean fever and tumor necrosis factor associated periodic syndrome [TRAPS], IgG4-related disease) |
| Postcardiac injury syndromes (immune-mediated after cardiac trauma in predisposed individuals) |
| Other – Granulomatosis with polyangiitis (Wegener), polyarteritis nodosa, sarcoidosis, inflammatory bowel disease (Crohn, ulcerative colitis), Whipple, giant cell arteritis, Behçet syndrome, rheumatic fever |
| **Neoplasm** |
| Metastatic – Lung or breast cancer, Hodgkin disease, leukemia, melanoma |
| Primary – Rhabdomyosarcoma, teratoma, fibroma, lipoma, leiomyoma, angioma |
| Paraneoplastic |
| **Cardiac** |
| Early infarction pericarditis |

| |
|---|
| Late postcardiac injury syndrome (Dressler syndrome), also seen in other settings (eg, post-myocardial infarction and post-cardiac surgery) |
| Myocarditis |
| Dissecting aortic aneurysm |
| **Trauma** |
| Blunt |
| Penetrating |
| Iatrogenic – Catheter and pacemaker perforations, cardiopulmonary resuscitation, post-thoracic surgery |
| **Metabolic** |
| Hypothyroidism – Primarily pericardial effusion |
| Uremia |
| Ovarian hyperstimulation syndrome |
| **Radiation** |
| **Drugs (rare)** |
| Procainamide, isoniazid, or hydralazine as part of drug-induced lupus |
| Other – Cromolyn sodium, dantrolene, methysergide, anticoagulants, thrombolytics, phenytoin, penicillin, phenylbutazone, doxorubicin |

*References:*

*1. LeWinter M. Clinical practice. Acute pericarditis. N Engl J Med 2014; 371:2410.*

*2. Imazio M, Gaita F. Diagnosis and treatment of pericarditis. Heart 2015; 101:1159.*

*3. Imazio M. Contemporary management of pericardial diseases. Curr Opin Cardiol 2012; 27:308.*

Graphic 67851 Version 10.0

## Case series of moderate-large pericardial effusions

| | Sagrista-Sauleda, 2000 (n = 322) | Corey, 1993 (n = 57) | Levy, 2003 (n = 204) | Ma, 2012 (n = 140) | Strobbe, 2017 (n = 269) |
|---|---|---|---|---|---|
| **Size of effusion, mm** | >10 | >10 | NR | >10 | >10 (98%) |
| **Tamponade, percent** | 37 | NR | NR | 100 | 88 |
| **Etiologies, percent** | | | | | |
| Idiopathic* | 29 (9% chronic) | 7 | 48 | 0 | 26 |

| | | | | | |
|---|---|---|---|---|---|
| Malignancy | 13 | 23 | 15 | 38 | 25 |
| Uremia | 6 | 12 | 2 | 6 | 3 |
| Iatrogenic | 16 | 0 | 0 | 9 | 21 |
| Post-acute myocardial infarction | 8 | 0 | 0 | 5 | 1 |
| Infection | 6 | 27 | 16 | 28 | 7 |
| Collagen vascular disease | 5 | 12 | 10 | 6 | 3 |
| Hypothyroidism | 2 | 0 | 10 | 5 | 0 |
| Other | 15 | 23 | 0 | 3 | 14 |

NR: not reported.

* Includes both acute and chronic pericardial effusions.

---

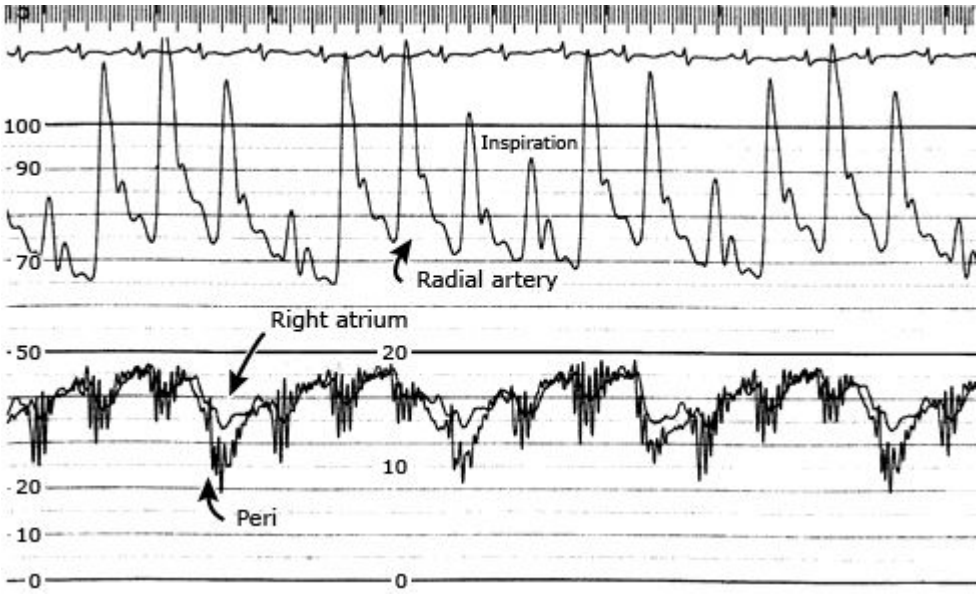
*Adapted from:*


1. *Sagrista-Sauleda J, Merce J, Permanyer-Maralda G, et al. Clinical clues to the causes of large pericardial effusions. Am J Med 2000; 109:95.*
2. *Corey GR, Campbell PT, VanTrigt P, et al. Etiology of large pericardial effusion. Am J Med 1993; 95:209.*
3. *Levy PY, Corey R, Berger P, et al. Etiologic diagnosis of 204 pericardial effusion. Medicine (Baltimore) 2003; 82:385.*
4. *Ma W, Liu J, Zeng Y, et al. Causes of moderate to large pericardial effusion requiring pericardiocentesis in 140 Han Chinese patients. Herz 2012; 37: 183.*
5. *Strobbe A, Adriaenssens T, Bennett J, et al. Etiology and long-term outcome of patients undergoing pericardiocentesis. J Am Heart Assoc 2017; 6: e007598.*


---

Graphic 78847 Version 9.0

## Intracardiac hemodynamic tracings showing acute cardiac tamponade



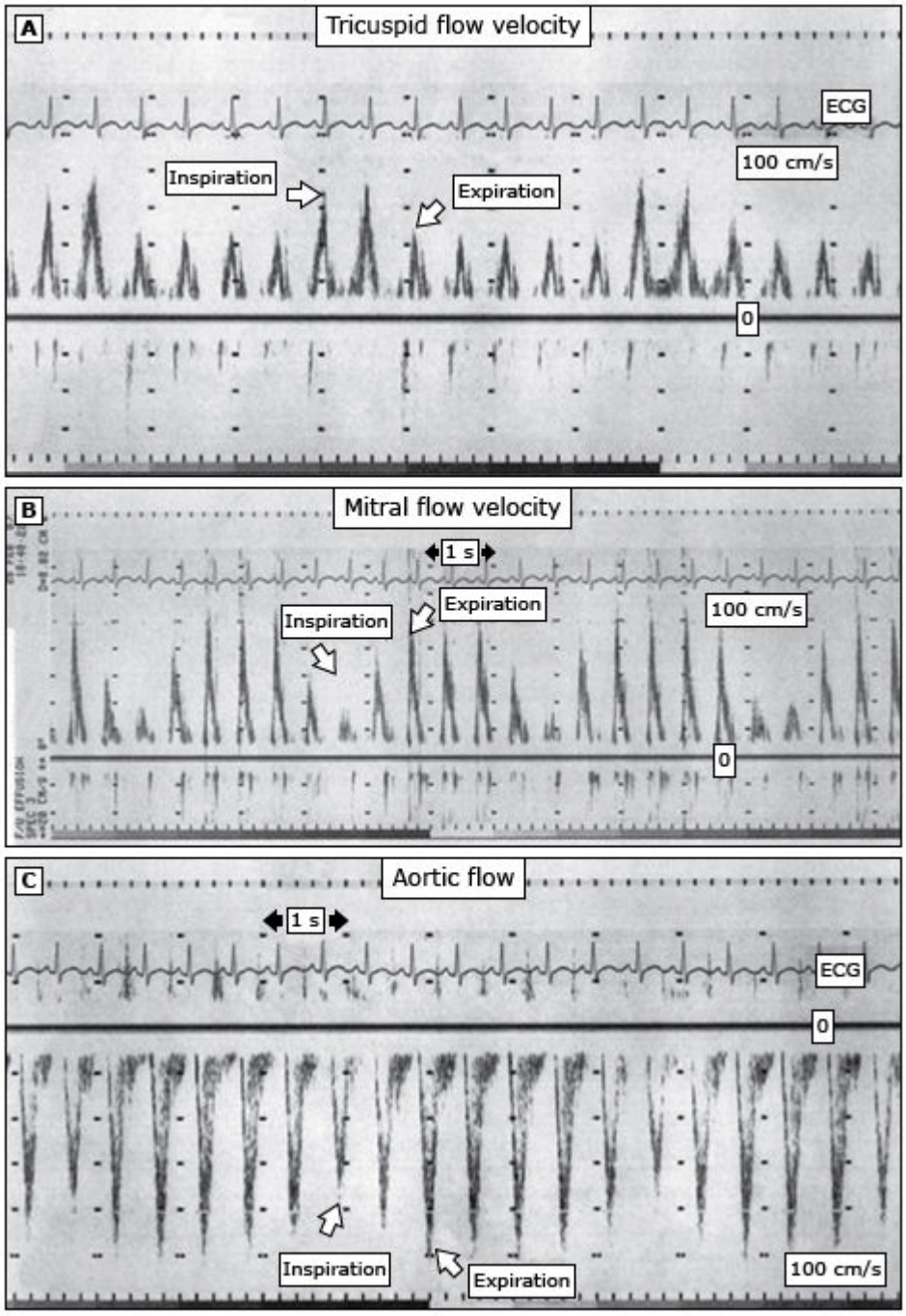

The radial arterial tracing shows tachycardia and extreme pulsus paradoxus. The right atrial and pericardial pressures are equally elevated, but the amount of effusion was very small. Note deeper y descent in the pericardial pressure compared with the right atrial pressure. ECG shows low voltage. Heavy time markings = 1 second.

ECG: electrocardiogram.



Graphic 55709 Version 5.0

## Measurement and mechanism of pulsus paradoxus



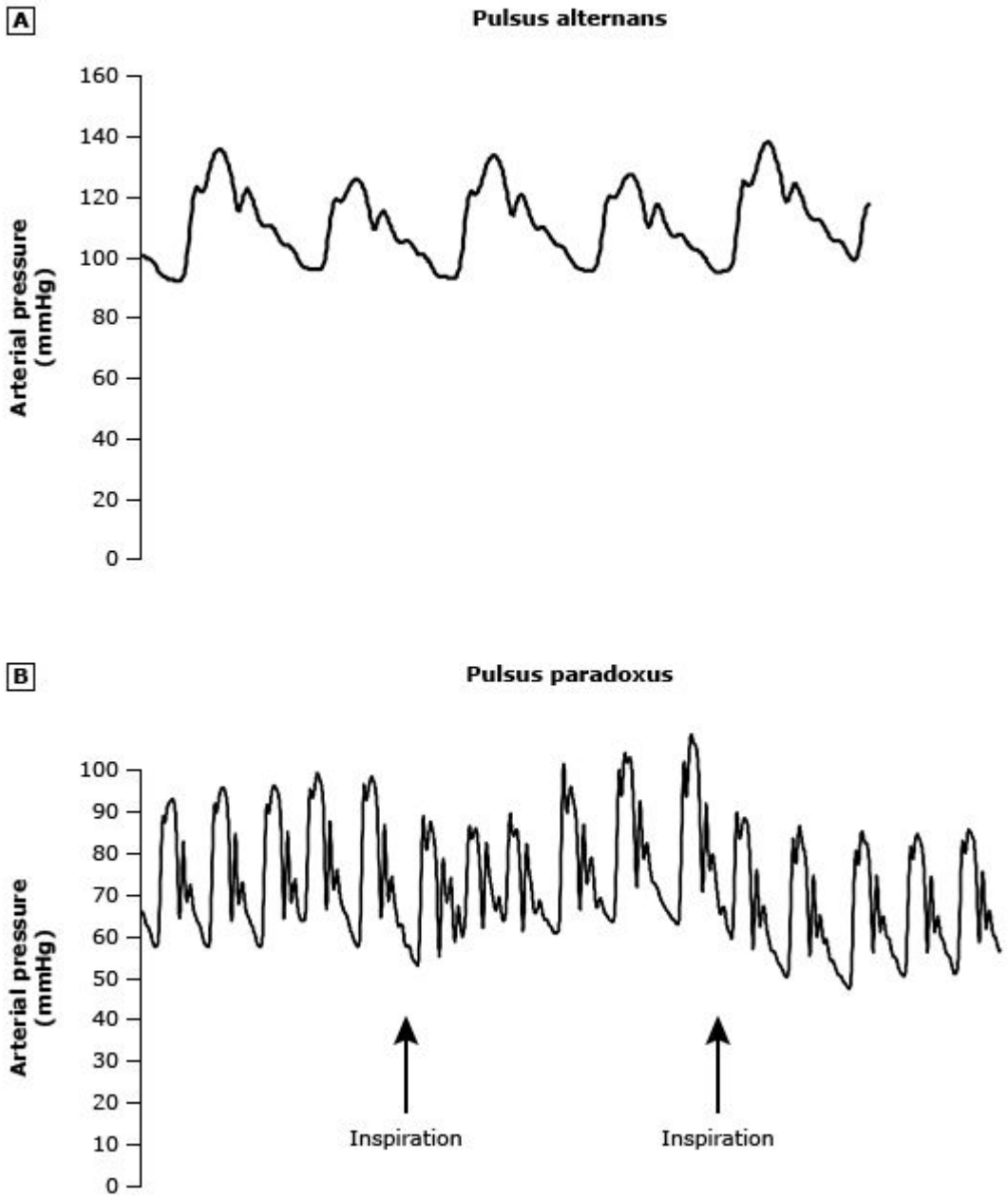

Doppler echocardiogram in a patient with cardiac tamponade. Note the inspiratory increase of tricuspid flow velocities (A) and the expiratory increase of mitral (B) and aortic (C) flow velocities.

ECG: electrocardiogram.



Graphic 63439 Version 7.0

## Pulsus alternans and pulsus paradoxus arterial waveforms




In pulsus alternans, which is associated with severe left ventricular systolic dysfunction, there is variation in the amplitude of the systolic arterial pressure with every other beat. In contrast, pulsus paradoxus varies with the respiratory cycle, such that the duration of arterial pressure reduction is sustained during the inspiratory portion of the respiratory cycle.

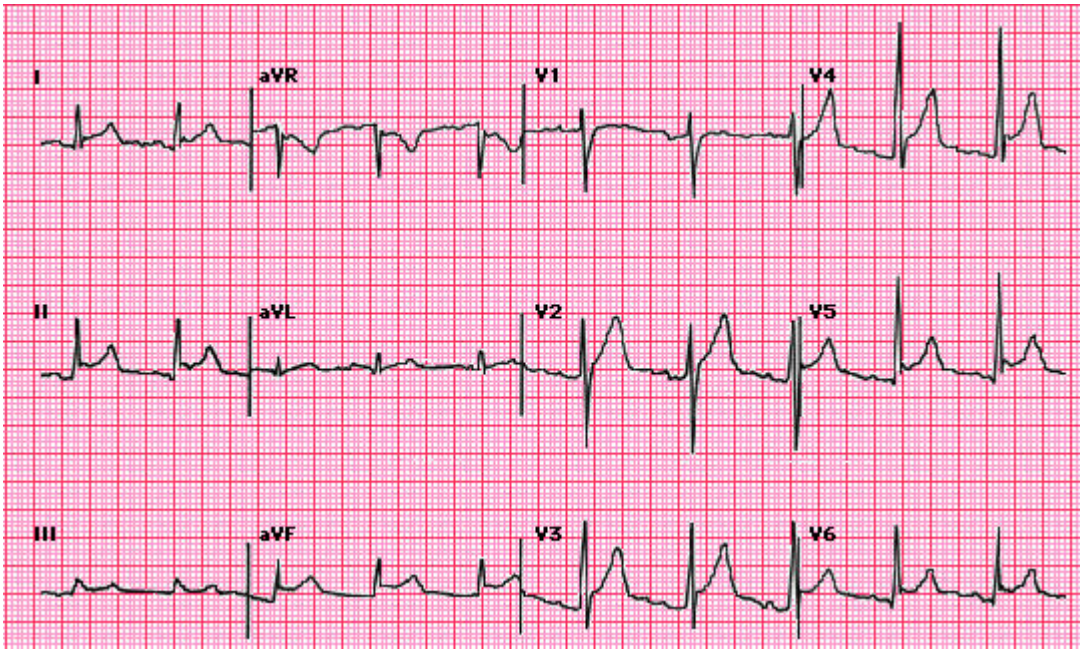
---

*Courtesy of Barry A Borlaug, MD.*

---

Graphic 70731 Version 5.0

## Electrocardiogram (ECG) in pericarditis




Electrocardiogram in acute pericarditis showing diffuse upsloping ST-segment elevations seen best here in leads II, III, aVF, and V2 to V6. There is also subtle PR-segment deviation (positive in aVR, negative in most other leads). ST-segment elevation is due to a ventricular current of injury associated with epicardial inflammation; similarly, the PR-segment changes are due to an atrial current of injury which, in pericarditis, typically displaces the PR segment upward in lead aVR and downward in most other leads.

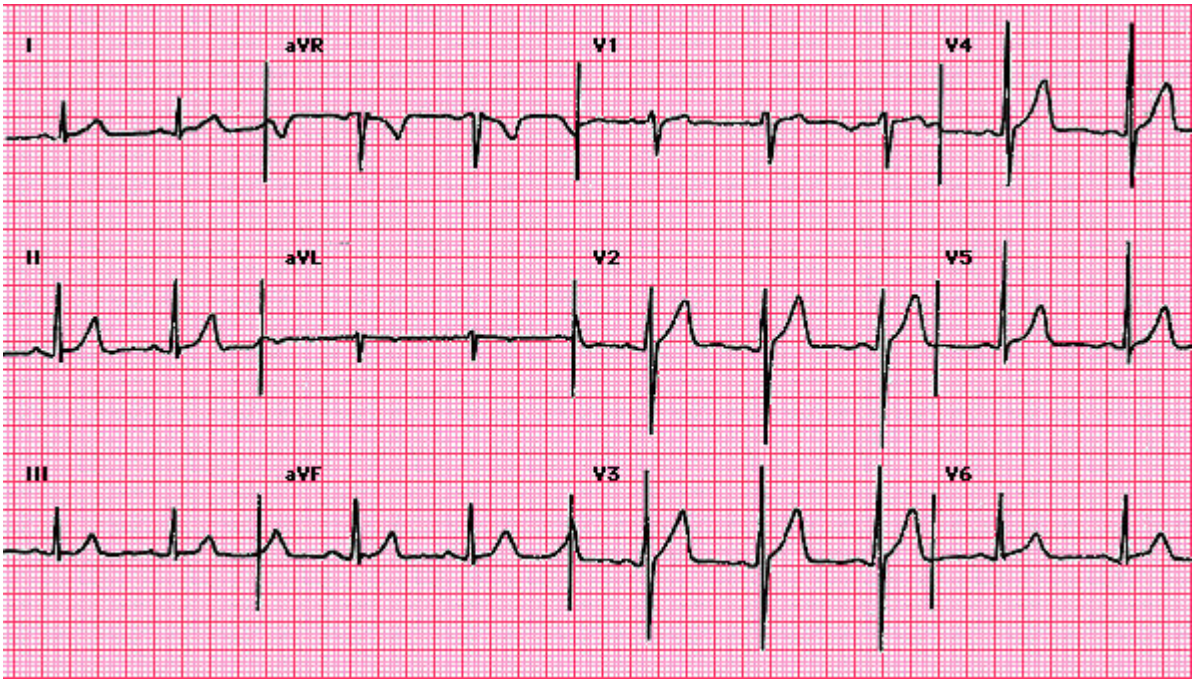
---

*Courtesy of Ary Goldberger, MD.*

---

Graphic 77572 Version 4.0

## Normal ECG



Normal electrocardiogram showing normal sinus rhythm at a rate of 75 beats/minute, a PR interval of 0.14 seconds, a QRS interval of 0.10 seconds, and a QRS axis of approximately 75°.

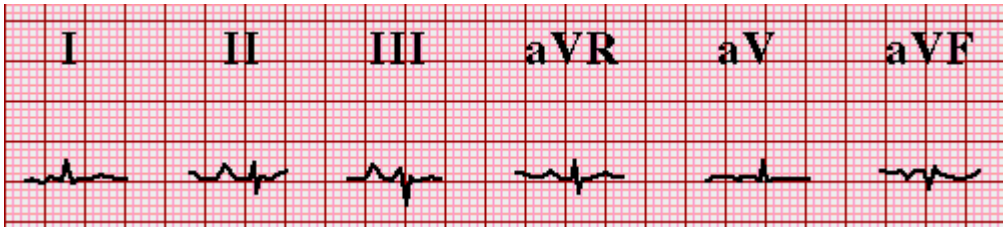
---

*Courtesy of Ary Goldberger, MD.*

---

Graphic 76183 Version 4.0

## Low voltage in the limb leads




Low voltage of the limb leads is present when the amplitude of the QRS complex in each of the three standard limb leads (1, 2, and 3) is <5 mm. Each large box represents 5 mm (calibration: 1 mV = 10 mm).

---

Graphic 74856 Version 2.0

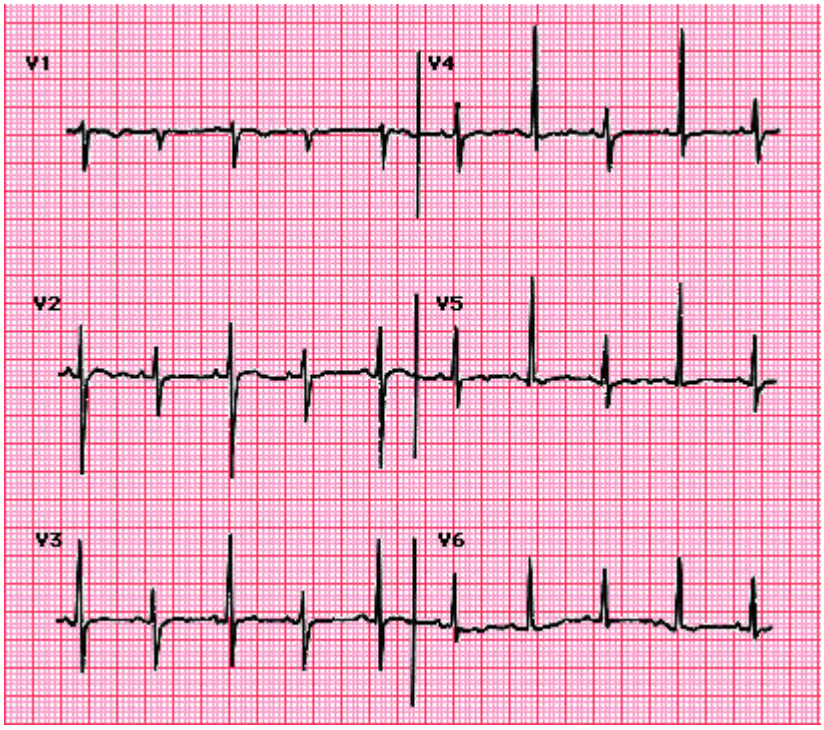
## Causes of low voltage QRS complexes

| |
|---|
| Adrenal insufficiency |
| Anasarca |
| Artifactual or spurious, eg, unrecognized standardization of ECG at one-half the usual gain (ie, 5 mm/mV) |
| Cardiac infiltration or replacement (eg, amyloidosis, tumor) |
| Cardiac transplantation, especially with acute or chronic rejection |
| Cardiomyopathy, idiopathic or secondary* |
| Chronic obstructive pulmonary disease |
| Constrictive pericarditis |
| Hypothyroidism, usually with sinus bradycardia |
| Left pneumothorax (mid-left chest leads) |
| Myocardial infarction, extensive |
| Myocarditis, acute or chronic |
| Normal variant |
| Obesity |
| Pericardial effusion |
| Pericardial tamponade, usually with sinus tachycardia |
| Pleural effusions |

* Dilated cardiomyopathy may be associated with the combination of relatively low limb lead voltages and prominent precordial voltages.

Graphic 80629 Version 1.0

## Electrical alternans




Sinus tachycardia with electrical alternans which is characterized by beat-to-beat alternation in the QRS appearance (best seen in leads V2 to V4). These findings are strongly suggestive of pericardial effusion, usually with cardiac tamponade. The alternating ECG pattern is related to back-and-forth swinging motion of the heart in the pericardial fluid.

---

ECG: electrocardiogram.

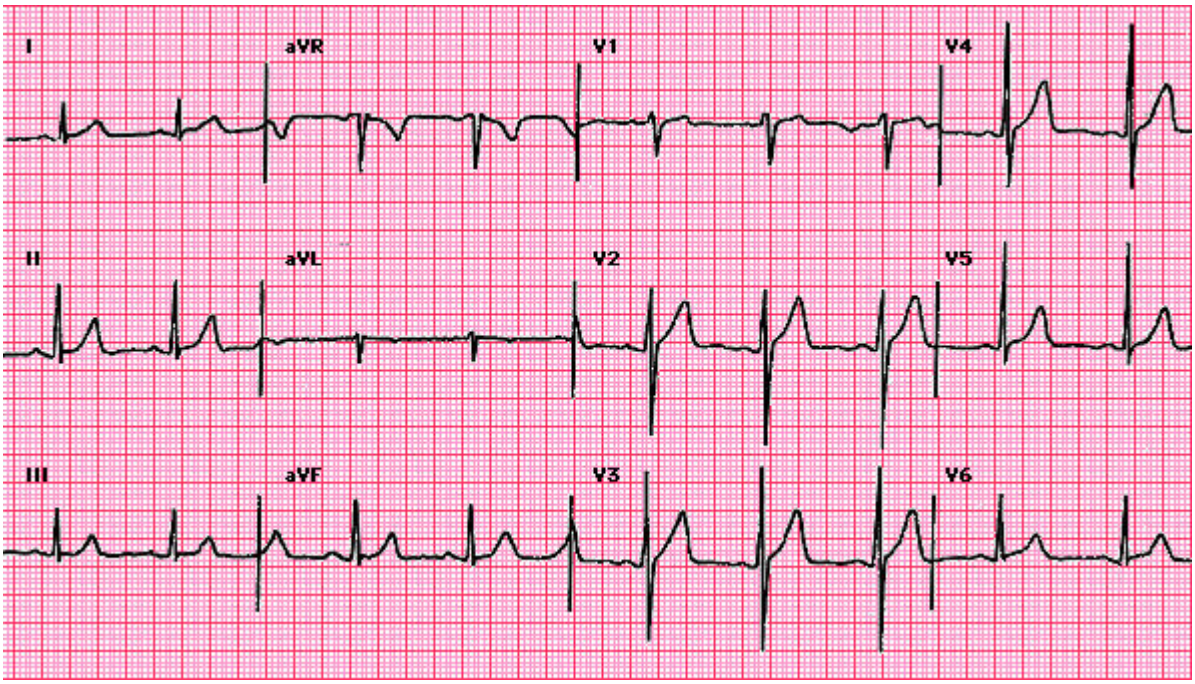
---

*Courtesy of Ary Goldberger, MD.*

---

Graphic 72525 Version 5.0

## Normal ECG




Normal electrocardiogram showing normal sinus rhythm at a rate of 75 beats/minute, a PR interval of 0.14 seconds, a QRS interval of 0.10 seconds, and a QRS axis of approximately 75°.

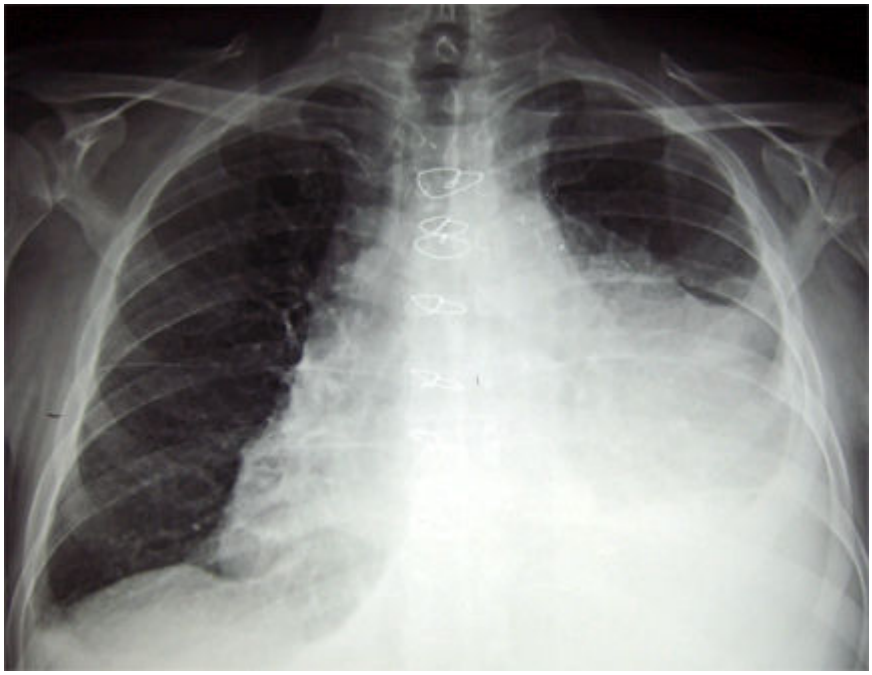
---

*Courtesy of Ary Goldberger, MD.*

---

Graphic 76183 Version 4.0

## Chest radiograph of a pericardial effusion



Cardiomegaly due to a massive pericardial effusion. At least 200 mL of pericardial fluid must accumulate before the cardiac silhouette enlarges.

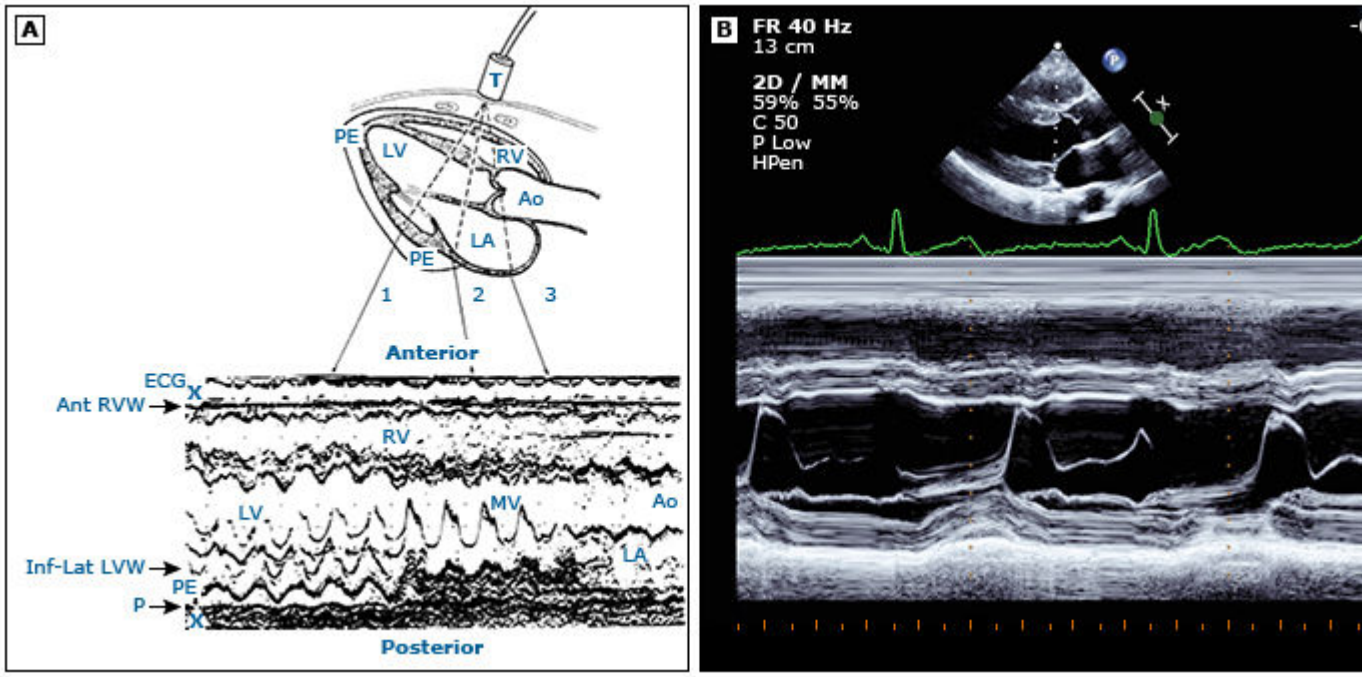
---

*Courtesy of Massimo Imazio, MD, FESC.*

---

Graphic 57640 Version 4.0

## Doppler M-mode parasternal long axis




In panel A, a diagrammatic representation of the long axis of the heart is seen on top; the transducer (T) anterior chest wall in the precordial parasternal window and the beam is swept from apex to base. The a echocardiogram at 3 beam positions on the diagram is designated by the arrows connecting the diagran echocardiogram which displays the cardiac structures that are imaged by the beam (bottom panel). A pe (PE) is seen as an echo-free space posterior to the left ventricle. This space diminishes and finally disappe the heart is approached because the pericardium reflects at mid atrium.

Panel B shows a still frame 2-dimensional and M-mode Doppler from the parasternal long-axis view at th mitral valve (corresponding to arrow 2 in panel A).

---

Ant RVW: anterior RV wall; Ao: aorta; Inf-Lat LVW: inferolateral LV wall; LA: left atrium; LV: left ventricle; M pericardium; PE: pericardial effusion; RV: right ventricle.

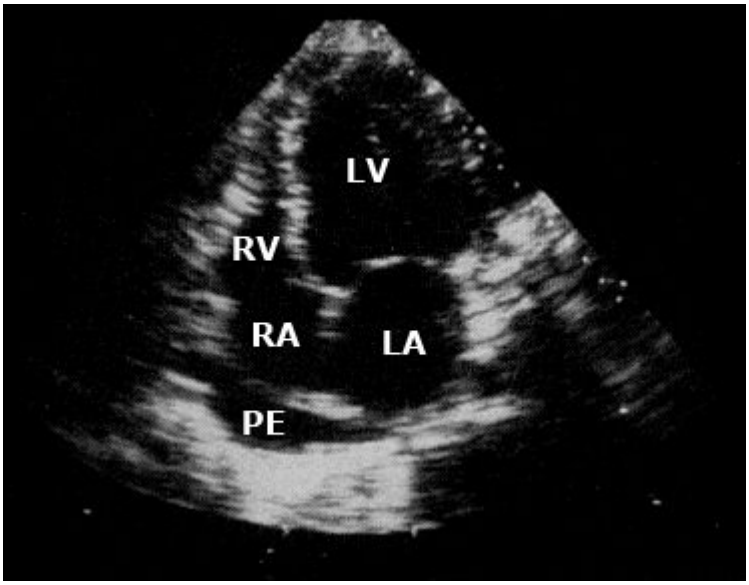
---

*Panel B reproduced with permission from: Aadhavi Sridharan, MD.*

---

Graphic 61898 Version 7.0

## Pericardial effusion




In the apical 4-chamber view from a patient with a small pericardial effusion (pe), the pe is seen best behind and above the RA, which is the most sensitive area for detecting small effusions. In larger pe, the RA collapses at this point, providing a highly sensitive, but minimally specific sign of cardiac tamponade.

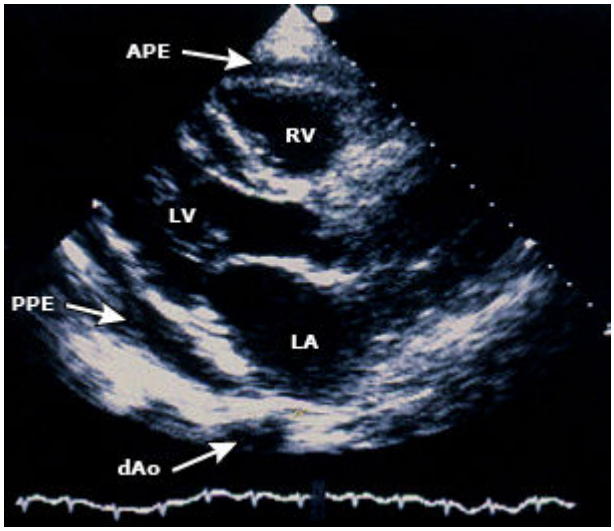
---

LA: left atrium; LV: left ventricle; PE: pericardial effusion; RA: right atrium; RV: right ventricle.

---

Graphic 82562 Version 4.0

## Large pericardial effusion




The long-axis precordial view shows a large anterior effusion (APE) and posterior pericardial effusion (PPE). This distinction between this large effusion and a moderate sized one is the easily seen anterior accumulation. Since the volume of the effusion is a cube function of its dimensions, its presence circumferentially makes a large difference in its volume without a major qualitative change in appearance.

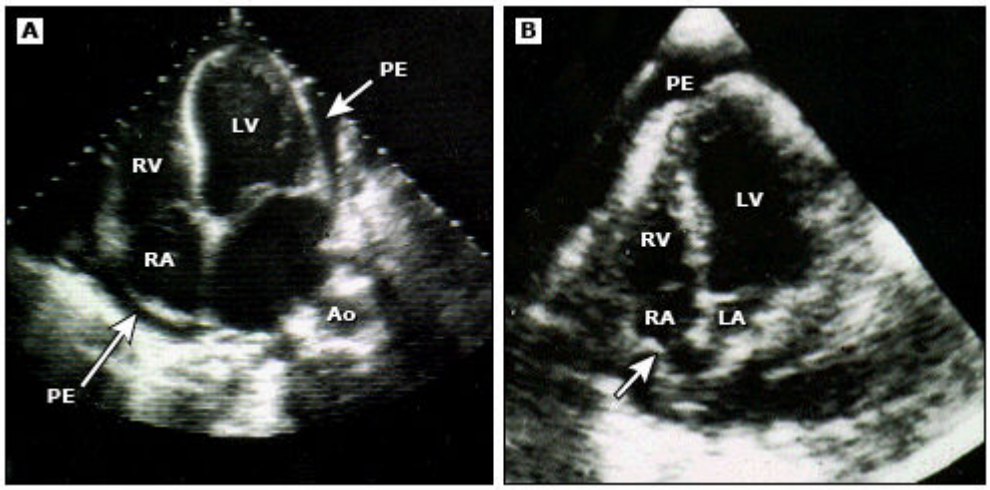
---

APE: anterior pericardial effusion; dAo: descending aorta; LA: left atrium; LV: left ventricle; PPE: posterior pericardial effusion; RV: right ventricle.

---

Graphic 56462 Version 4.0

## 4-chamber view showing circumferential pericardial effusion




The apical 4-chamber view shows a moderate to large anterior and posterior pericardial effusion (PE), but the curvature of the RA is normal. In panel B there is diastolic expiratory invagination, compression, or collapse of the RA. Collapse of the RA is a highly sensitive, but relatively nonspecific, sign of hemodynamically important tamponade. More importantly, the LA also appears compressed. This latter sign is felt to be more specific for tamponade.

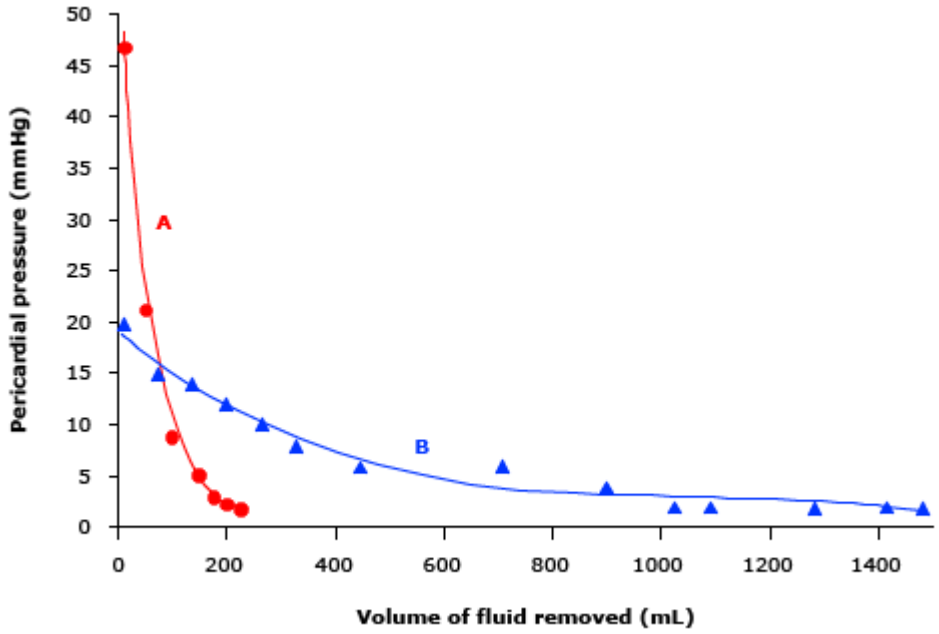
---

LA: left atrium; LV: left ventricle; PE: pericardial effusion; RA: right atrium; RV: right ventricle.

---

Graphic 57021 Version 5.0

## Pericardial pressure in acute and chronic tamponade




Volume curves recorded from data acquired during pericardiocentesis. Curve A (in red) plots data from a patient with hyperacute tamponade that followed laceration of a coronary artery during an angioplasty-stenting procedure. Note the extreme elevation of pericardial pressure and that withdrawal of only 100 mL, half the volume we could aspirate, lowered the pressure to 10 mmHg. Curve B (in blue) plots data from a patient who had a history of prior pericarditis, assumed to be of viral etiology. Subsequently, he developed a chronic pericardial effusion that reached at least 1500 mL in volume. At the time of presentation to our service, the jugular venous pressure was 22 mmHg. Aspiration of 300 mL of pericardial fluid reduced the pericardial pressure to 10 mmHg, and removing another 600 mL achieved a nearly normal pericardial pressure. Aspiration of the remaining large effusion did not affect pericardial pressure. The curves of cases of intermediate acuity or chronicity would fall between these two extremes.

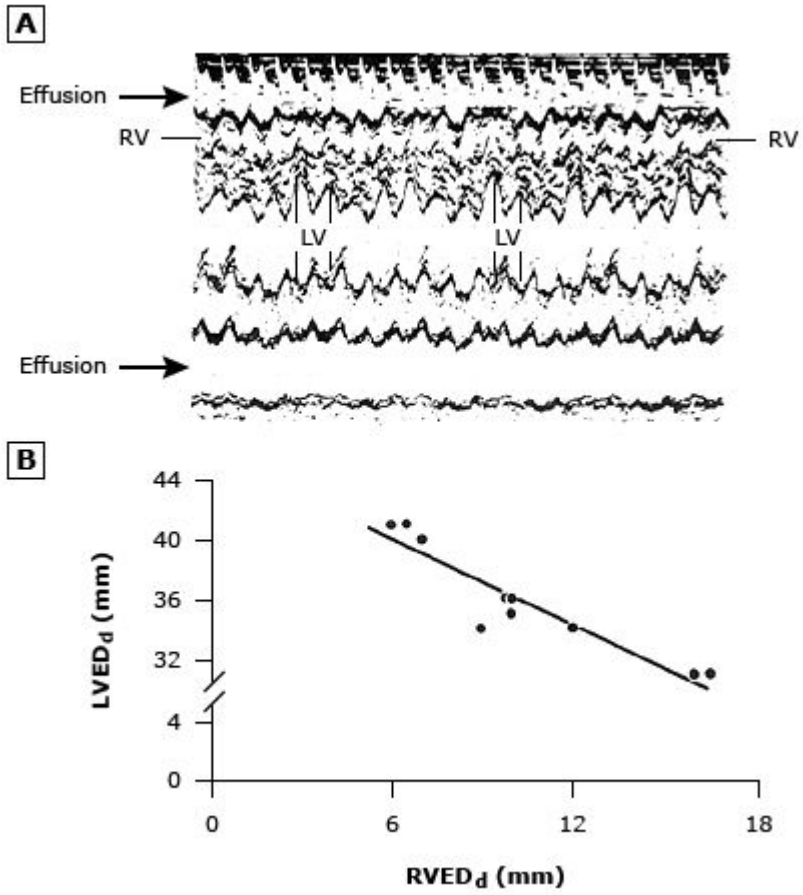
---

*Courtesy of Ralph Shabetai, MD.*

---

Graphic 75068 Version 3.0

## Hemodynamics in cardiac tamponade




The M-mode through the minor axis in a patient with an anterior and posterior pericardial effusion and tamponade is seen in panel A; during inspiration, the RV fills and the LV becomes smaller; during expiration, the opposite occurs. In the graph in panel B, $RVED_d$ and $LVED_d$ are plotted against one another and demonstrate a negative correlation, a result of reciprocation of the chambers within the pericardium. Since the pericardium is a rigid box, as respiration brings more blood into the RV, there is less room in the LV; blood pools in the inflating lungs during inspiration. This blood plus the increased stroke volume sent to the lungs during RV inspiratory expansion reaches the LV during expiration. As the LV expands, the RV is compressed. During RV expansion, underfilling of the LV results in a drop in pulse pressure perceived as the paradoxical pulse.

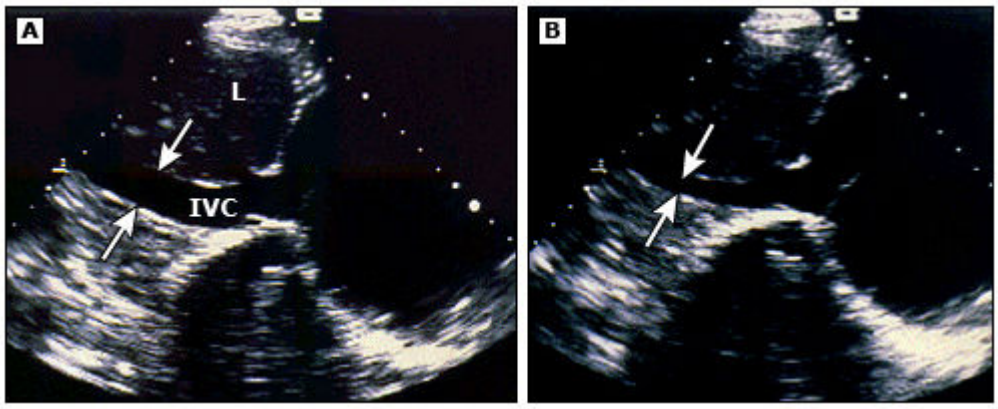
---

LV: left ventricle; $LVED_d$: LV end-diastolic diameter; RV: right ventricle; $RVED_d$: RV end-diastolic diameter.

---

Graphic 75980 Version 2.0

## 2-dimensional transthoracic echocardiogram (2D TTE) from the subcostal view showing a normal inferior vena cava (IVC) during respiration

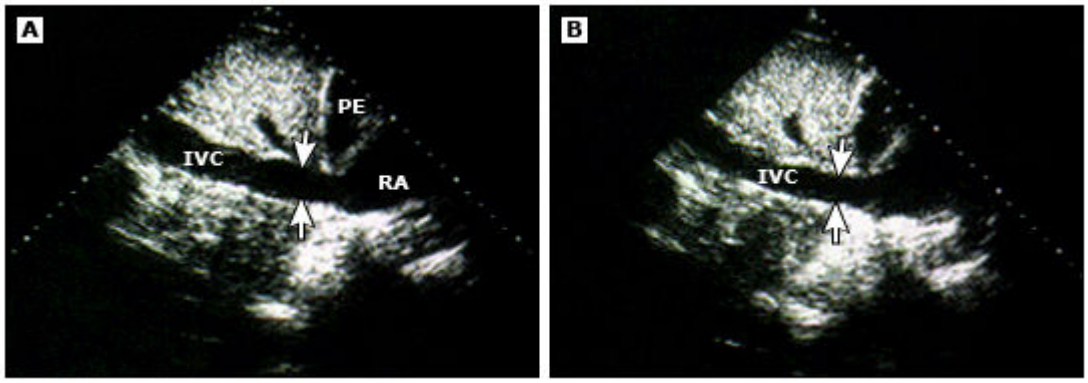



The subcostal view in a normal subject shows the IVC. (A) Prior to inspiration, the normal diastolic IVC diameter (arrows) is less than 20 mm. (B) During inspiration, the IVC collapses to less than 50% of its original diameter.

IVC: inferior vena cava; L: liver.

Graphic 58804 Version 5.0

## Subcostal view IVC during cardiac tamponade




(A) The subcostal view of the IVC in a patient with tamponade. The IVC is plethoric measuring over 20 mm in diameter.

(B) During inspiration, the IVC diameter fails to decrease. There is a large pericardial effusion (PE) surrounding the RA.

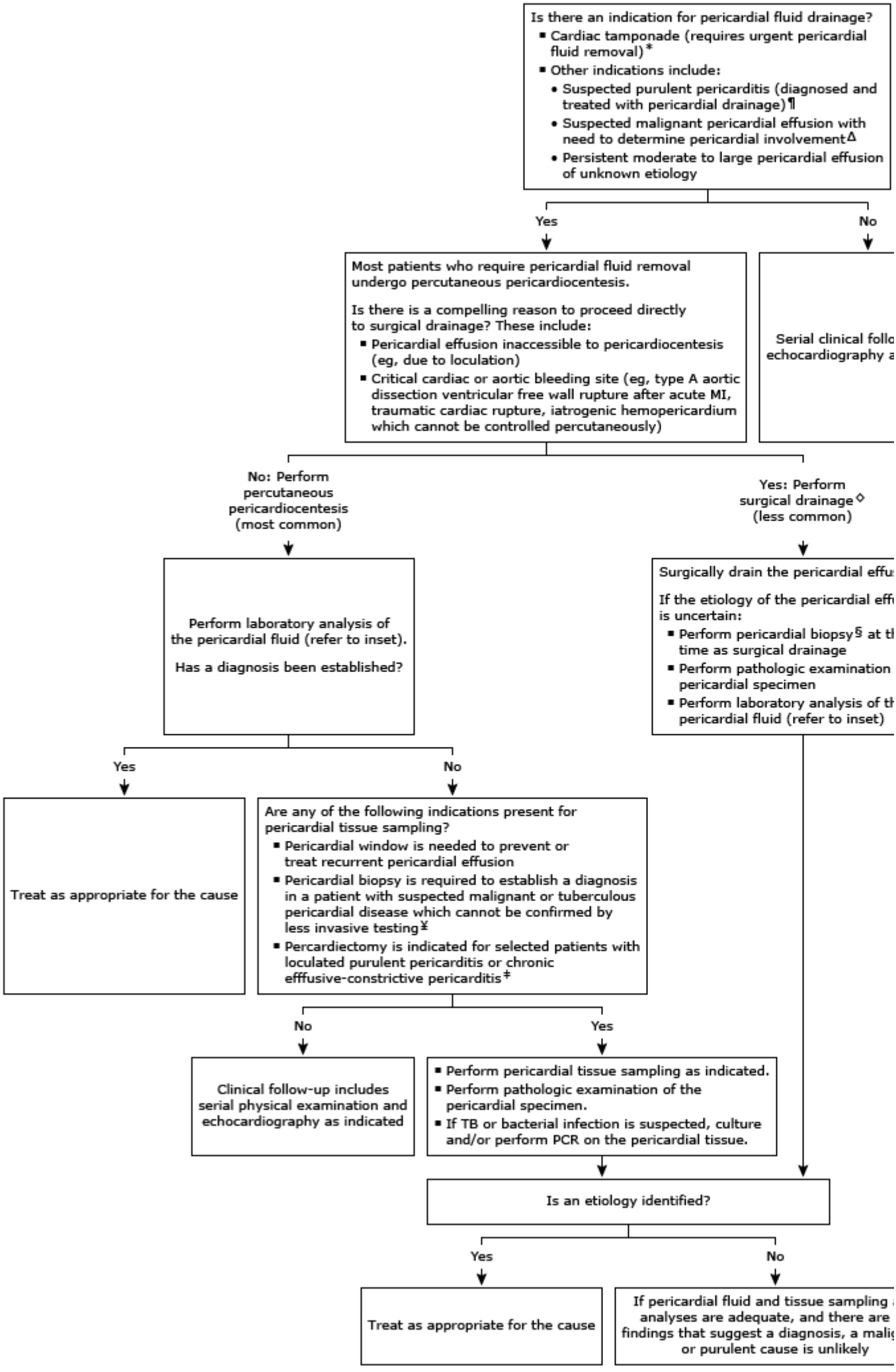
---

IVC: inferior vena cava; PE: pericardial effusion; RA: right atrial.

---

Graphic 79134 Version 6.0

## Pericardial effusion: Deciding when to perform pericardial drainage and peri


Is there an indication for pericardial fluid drainage?
Cardiac tamponade (requires urgent pericardial fluid removal)*
Other indications include:
Suspected purulent pericarditis (diagnosed and treated with pericardial drainage)¶
Suspected malignant pericardial effusion with need to determine pericardial involvementΔ
Persistent moderate to large pericardial effusion of unknown etiology
Yes
No
Most patients who require pericardial fluid removal undergo percutaneous pericardiocentesis.
Is there is a compelling reason to proceed directly to surgical drainage? These include:
Pericardial effusion inaccessible to pericardiocentesis (eg, due to loculation)
Critical cardiac or aortic bleeding site (eg, type A aortic dissection ventricular free wall rupture after acute MI, traumatic cardiac rupture, iatrogenic hemopericardium which cannot be controlled percutaneously)
Serial clinical follo
echocardiography a
No: Perform percutaneous pericardiocentesis (most common)
Yes: Perform surgical drainage◇ (less common)
Perform laboratory analysis of the pericardial fluid (refer to inset).
Has a diagnosis been established?
Surgically drain the pericardial effu
If the etiology of the pericardial eff
is uncertain:
Perform pericardial biopsy§ at th
time as surgical drainage
Perform pathologic examination
pericardial specimen
Perform laboratory analysis of th
pericardial fluid (refer to inset)
Yes
No
Treat as appropriate for the cause
Are any of the following indications present for pericardial tissue sampling?
Pericardial window is needed to prevent or treat recurrent pericardial effusion
Pericardial biopsy is required to establish a diagnosis in a patient with suspected malignant or tuberculous pericardial disease which cannot be confirmed by less invasive testing¥
Percardiectomy is indicated for selected patients with loculated purulent pericarditis or chronic efffusive-constrictive pericarditis‡
No
Yes
Clinical follow-up includes serial physical examination and echocardiography as indicated
Perform pericardial tissue sampling as indicated.
Perform pathologic examination of the pericardial specimen.
If TB or bacterial infection is suspected, culture and/or perform PCR on the pericardial tissue.
Is an etiology identified?
Yes
No
Treat as appropriate for the cause
If pericardial fluid and tissue sampling
analyses are adequate, and there are
findings that suggest a diagnosis, a malig
or purulent cause is unlikely

This algorithm presents a general approach to determining when to drain pericardial fluid and when to c
effusion do not require pericardial fluid drainage or pericardial biopsy. For further details, refer to UpToD
effusion, purulent pericarditis, tuberculous pericarditis, and pericardial disease associated with cancer.

---

AFB: acid-fast bacilli; CMV: cytomegalovirus; MI: myocardial infarction; PCR: polymerase chain reaction; T

* Refer to UpToDate content on diagnosis of cardiac tamponade.

¶ Purulent pericarditis is rare life-threatening illness and is generally manifested by fever, often with evic

Δ In a patient with known malignancy and pericardial effusion, pericardial sampling and drainage is not r
cardiac tamponade or for cancer staging.

◇ In selected cases with accessible pericardial fluid, percutaneous pericardiocentesis may be performed
surgical drainage.

§ When performing pericardial biopsy, pericardioscopic guidance with extensive pericardial sampling ma
malignancy, purulent pericarditis, or radiation-induced pericarditis.

¥ The decision to obtain a pericardial biopsy is made in the context of other clinical assessments, includir
suspected malignancy, this includes determining access to, safety, and anticipated yield of potential biop
and/or in a TB-endemic area, a pericardial biopsy is not generally required.

‡ Refer to UpToDate content on purulent pericarditis and effusive-constrictive pericarditis.

---

## Contributor Disclosures

**Brian D Hoit, MD** Speaker's Bureau: Philips Medical [Heart valve disease]. All of the relevant financial relationships listed have been mitigated. **Martin M LeWinter, MD** Grant/Research/Clinical Trial Support: Kiniksa Pharmaceuticals [Pericarditis]. Consultant/Advisory Boards: Kiniksa Pharmaceuticals [Pericarditis]. All of the relevant financial relationships listed have been mitigated. **Daniel J Sexton, MD** Equity Ownership/Stock Options: Magnolia Medical Technologies [Medical diagnostics – Ended August 2022]. Consultant/Advisory Boards: Magnolia Medical Technologies [Medical diagnostics – Ended August 2022]. All of the relevant financial relationships listed have been mitigated. **Susan B Yeon, MD, JD, FACC** No relevant financial relationship(s) with ineligible companies to disclose.

Contributor disclosures are reviewed for conflicts of interest by the editorial group. When found, these are addressed by vetting through a multi-level review process, and through requirements for references to be provided to support the content. Appropriately referenced content is required of all authors and must conform to UpToDate standards of evidence.

Conflict of interest policy

→