Pericardial Diseases

A. Normal Pericardial Anatomy and Physiology

The pericardium consists of two layers: a serous visceral layer, which is intimately adherent to the heart and epicardial fat, and a fibrous parietal layer. The pericardium encloses the greater part of the surface of the heart, the juxtacardial portions of the pulmonary and systemic veins, and the proximal segments of the great vessels. A significant portion of the left atrium, however, is not enclosed within the pericardium. The pericardium is attached by ligaments to the manubrium sterni, the xiphoid process, the vertebral column, and the central tendon of the diaphragm. The pericardium is not essential for sustaining life or health, as evidenced by preservation of cardiac function even if the pericardium is congenitally absent or surgically removed. The pericardium does play a role in normal cardiovascular function, however, and can be involved in a number of important disease states. The normal functions of the pericardium include maintaining an optimal cardiac shape, promoting cardiac chamber interaction, preventing the overfilling of the heart, reducing friction between the beating heart and adjacent structures, providing a physical barrier to infection, and limiting displacement during the cardiac cycle.

B. Pericardial Pressure and Normal Function

The true pressure in the normal pericardial space is a matter of some controversy. When measured with fluid-filled catheters, pericardial pressure is very similar to intrapleural pressure: from −1 to −2 mm Hg on average, falling to about −5 mm Hg with normal inspiration. There is considerable evidence, however, that the pressure in the normal pericardial space is best considered as a contact force between visceral and parietal pericardium and therefore is more appropriately measured by specially-designed flat balloons. When measured in this way, the pericardial pressure is clearly higher than the intrapleural pressure, although its true magnitude remains somewhat uncertain. The bulk of current evidence indicates that with normal cardiac volumes, the effective pericardial pressure ranges from 0–1 mm Hg to (at most) 3–4 mm Hg. The pericardial space between the parietal and visceral layers normally contains 15–50 mL of fluid, and the reserve volume of the pericardium is relatively small. Once this modest reserve is exceeded, intrapericardial pressure rises significantly. This can occur if the cardiac volume increases rapidly, for example, due to acute right ventricular myocardial infarction, or if additional fluid accumulates. With significant fluid in the pericardial space, fluid-filled catheters do provide accurate intrapericardial pressure measurements.

► Pathogenesis

A. Infectious Pathogens

1. Viral Pericarditis – The most common clinical manifestation of viral involvement is acute pericarditis. An unidentified virus almost certainly underlies most cases of acute idiopathic pericarditis. The possibility of a
viral cause is suggested when pericarditis occurs in the absence of other factors; it is supported by a more than fourfold rise in serial viral antibody titers during the initial weeks of illness. (Such measurement, however, is not a routine part of the management of viral pericarditis.) Frequently, a prodromal syndrome consistent with a viral infection is present.

The viral pathogens most commonly associated with pericarditis include coxsackievirus B (most common), coxsackievirus A, echovirus 8, and HIV. Although a wide range of viral agents have been implicated, no specific antiviral therapy has been shown to be effective; management and outcome are described in the section on acute pericarditis.

2. Bacterial Pericarditis – Bacterial infection of the pericardium can occur following thoracic surgery, as a result of a contiguous pleural, mediastinal, or pulmonary infection, as a complication of bacterial endocarditis, or as a result of systemic bacteremia. Direct extension from pneumonia or empyema with staphylococci, pneumococci, and streptococci accounts for most cases. The incidence of hospital-acquired penicillin-resistant staphylococcal pericarditis after thoracic surgery has increased during the past decade. Preexisting pericardial effusions and immunosuppressed states are important predisposing factors.

Common clinical manifestations include fever, chills, night sweats, and dyspnea; pleuritic chest pain and pericardial friction rubs are present in only a minority of patients. Leukocytosis with a shift to the left is generally present, and chest radiography usually reveals an increase in the cardiac silhouette. Although electrocardiograms (ECGs) are frequently normal, they can show typical changes of acute pericarditis.

Although high intrapericardial antibiotic levels are achievable, medical therapy alone is usually insufficient, and prompt percutaneous or surgical drainage is essential. Cardiac tamponade may occur very rapidly with hemodynamic deterioration that can be confused with septic shock. In view of the continuing high mortality rates of 65–77%, bacterial pericarditis should be considered a medical emergency.

3. Tuberculous Pericarditis – Although several decades of effective antituberculosis therapy and public health measures have brought about a declining rate of tuberculous pericarditis, this condition remains a major problem in immunocompromised persons and in the non-industrialized world. Thus, HIV-associated tuberculosis is a common cause of symptomatic pericardial effusion.

Tuberculous pericarditis typically occurs with no demonstrable pulmonary or extrapulmonary tuberculosis. Symptoms may be insidious and nonspecific. Findings are predominantly systemic, and pericardial friction rubs are unusual. Large effusions (Figure 17–1) and resulting tamponade are common, and constriction occurs as a late complication. Demonstration of tubercle bacillus by stain or culture is possible in only one-third to one-half of the patients, and the diagnosis is often presumptively established through a history of contact or a positive purified protein derivative (PPD) skin test. Alternatively, finding characteristic granulomata on pericardial biopsy specimens confirms the diagnosis, but even these are often falsely negative. High levels of
adenosine deaminase (ADA), an enzyme produced by white blood cells in pericardial fluid, is a sensitive and specific test for tuberculous pericarditis. Increased interferon-gamma in pericardial fluid is an additional marker. Combined with ADA it provides even greater diagnostic accuracy. Most recently, polymerase chain reaction (PCR) to detect Mycobacterium tuberculosis DNA has been used and can be performed in minute amounts of pericardial fluid. Use of one or more of these modern tests should be routine whenever tuberculous pericarditis is suspected. Often the diagnosis of tuberculous pericarditis is made retrospectively in patients with constrictive pericarditis and a history of tuberculosis. Such cases are managed like any case of constrictive pericarditis.

Untreated tuberculous pericarditis is associated with mortality rates in excess of 80%. Management consists of triple-drug antituberculous therapy for at least 9 months. Some authorities advocate the use of corticosteroids to prevent the development of constrictive pericarditis, but data to support this approach are inconclusive. Pericardiocentesis is indicated for patients with large or compromising effusions. As much as one-third of patients will require pericardial resection despite antibiotic therapy.

4. AIDS – The most common pericardial abnormality encountered in AIDS is an asymptomatic pericardial effusion. Small effusions can occur in HIV-positive individuals who do not have full-blown AIDS, although they often herald the transition to AIDS. Pericardial effusion can be considered part of a generalized effusive process that can involve the peritoneum and pleura as well. Asymptomatic effusions do not mandate invasive diagnostic studies or treatment, and many will resolve spontaneously without specific treatment.

In AIDS patients, symptomatic pericardial effusion with or without chest pain, friction rub, and ECG changes is caused by a variety of opportunistic infections and neoplasms. The most common infectious pathogens identified in symptomatic pericardial effusion are M tuberculosis, and *Mycobacterium avium-intracellulare*. The HIV virus itself can cause an effusion. Lymphomas and Kaposi sarcoma are the most common neoplasms associated with effusion. Pericarditis or symptomatic pericardial effusion in a patient with AIDS should therefore prompt an immediate search for infection or neoplasm. Pericardial effusion in HIV disease usually occurs in the context of full-blown AIDS and is strongly associated with a shortened survival time independent of the CD4 count. The mortality rate at 6 months for patients with effusion was nine times greater than for patients without effusion. There is no published information describing the effects of modern, highly active antiretroviral therapy on HIV-associated pericardial disease.

B. Iatrogenic Causes

1. Surgery-Related Syndromes – Several distinct pericardial syndromes may occur after heart surgery.

Cardiac tamponade may occur during in-hospital recuperation, most commonly in the first 24 hours. It is identified by the hemodynamic perturbations typical of tamponade. The sudden cessation of previously brisk bleeding from drains should alert the physician to the possibility of clogging. Therapy consists of prompt
surgical exploration and evacuation. Cardiac tamponade is less common after the first 24 hours, with fewer typical clinical manifestations, and symptoms may consist largely of nonspecific generalized complaints. Two-dimensional echocardiography establishes the presence of a significant effusion and may delineate its anatomic distribution. Pericardial effusions in this setting are often loculated and may compress only one cardiac chamber. The approach to drainage is largely dictated by the location of the effusion.

**Early pericarditis**, consisting of fever, chest pain, pericardial friction rubs, and typical ECG features, is common. In most cases, the syndrome resolves spontaneously, and nonsteroidal antiinflammatory drugs (NSAIDs) are effective treatment.

Postpericardiotomy syndrome is reported in as many as 30–40% of patients. This syndrome, which usually occurs during the first several postoperative weeks, consists of fever, pleuritis, and pericarditis. Diagnosis proceeds by exclusion, and treatment consists of administering NSAIDs; the need for corticosteroids is rare.

**Constrictive pericarditis** occurs rarely as a complication of cardiac surgery. Its incidence is estimated to only be 0.2–0.3% of cardiac operations. However, cardiac surgery is emerging as an important cause of constrictive pericarditis because so many cardiac surgeries are performed in the United States annually.

Constrictive pericarditis has been reported to occur at times ranging from 2 weeks to 21 years after the surgery. Because of the relative rarity of this complication, it has been difficult to identify specific predisposing procedural factors. In the cases of constriction reported in the literature, 95% have been associated with an open pericardium, which may serve as a reservoir for the collection of blood. Occasionally, constrictive pericarditis appears within days or weeks after surgery. These cases appear to respond well to a course of corticosteroids. With this exception, the mainstay of therapy for postsurgical constrictive pericarditis is pericardiectomy.

2. **Trauma** – Traumatic hemorrhagic pericardial effusions, which can result from blunt or penetrating injuries of the chest, can also be caused by a variety of iatrogenic causes such as cardiac catheterization and coronary interventional procedures, pacemaker insertion, arrhythmia ablation procedures, endoscopy, and closed chest cardiac massage. The rapidity with which pericardial fluid can accumulate can quickly cause hemodynamic compromise. Hypotension in this setting should prompt both an immediate echocardiographic search for pericardial fluid and swift evacuation of any significant effusions. Delayed manifestations may include recurrent pericardial effusions and, in rare cases, constrictive pericarditis.

3. **Radiation Therapy** – The incidence of pericardial injury from therapeutic radiation is related to dose, duration, and technical features. Pericardial damage from radiation may appear during the course of therapy or following it. The syndrome that appears during radiation therapy is acute pericarditis. The onset of clinical manifestations in the delayed form is usually within 12 months but may take many years. The clinical features of the late form range from asymptomatic pericardial effusions to acute pericarditis or constrictive
pericarditis. Radiation therapy is now one of the leading causes of constrictive pericarditis. Diagnosis and management are discussed later in the sections on pericardial effusion and constrictive pericarditis.

C. Connective Tissue Disorders

1. Rheumatoid Arthritis – Pericarditis has been found during postmortem examination in up to 50% of patients with rheumatoid arthritis. The clinical recognition of pericarditis during life, however, is far less frequent. Tamponade may occur rarely as a complication of pericardial effusion in rheumatoid arthritis and should be treated appropriately. Patients with nodular rheumatoid arthritis are much more likely to develop pericarditis than are those without nodules, and pericarditis commonly occurs in association with arthritis and pleuritis. Symptomatic pericarditis can be treated with NSAIDs. There is no evidence to support a role for corticosteroids in preventing constrictive pericarditis; as in other conditions, the presence of constrictive pericarditis warrants pericardiectomy.

2. Systemic Lupus Erythematosus – Pericarditis is the most common cardiac manifestation of systemic lupus erythematosus (SLE) and tends to occur during periods of active disease. Clinical and ECG features tend to be typical of acute pericarditis. Although tamponade can complicate pericarditis, progression to constrictive pericarditis is rare. Pericarditis generally subsides as systemic manifestations improve in response to corticosteroid or immunosuppressive therapy.

3. Progressive Systemic Sclerosis – As in rheumatoid arthritis, pericarditis is found during autopsy more frequently than during life. Asymptomatic pericardial effusions can be found in up to 40% of patients. Patients with scleroderma may have typical symptoms of pericarditis and nonspecific ECG changes, and tamponade may occur. The prognosis is generally poor for patients with scleroderma in whom pericarditis develops. Pericardial involvement can also occur in association with myocardial involvement and differentiating constrictive pericarditis from restrictive cardiomyopathy may be difficult.

D. Other Causes

1. Myocardial Infarction – Clinical evidence of pericarditis can be found in 7–20% of patients within the first week after myocardial infarction, although autopsy series suggest a significantly higher incidence of clinically silent, localized fibrinous pericarditis. The incidence is greatest with large, ST-segment elevation myocardial infarctions. Anticoagulant therapy and antiplatelet therapy administered in conjunction with percutaneous revascularization procedures do not appear to increase the incidence of pericardial effusions after myocardial infarction. While it is theoretically possible that these drugs may increase the chance of hemorrhage into the pericardial space if an effusion does occur, they are not generally considered to be contraindicated in the setting of early postmyocardial infarction pericarditis. Thrombolytic therapy has been associated with a decreased incidence of pericarditis in placebo-controlled studies. Because these agents can contribute to the
development of tamponade, however, their use may be relatively contraindicated when concurrent pericarditis is identified.

**Dressler syndrome (postmyocardial infarction syndrome)** occurs from 1 to 6 weeks after myocardial infarction and consists of fever, pleuropericardial pain, malaise, and evidence of pleural and pericardial effusions. The syndrome is a contraindication to anticoagulant therapy because pericardial hemorrhage can occur, increasing the likelihood of tamponade. The incidence of this syndrome has been decreasing in recent years. It is believed to have an autoimmune cause due to sensitization to myocardial cells at the time of necrosis. Antimyocardial antibodies have been demonstrated in patients with Dressler syndrome. Management is as outlined in the section on pericarditis.

**2. Malignancy** – A variety of hematologic and solid malignancies can cause pericardial metastases that are more frequently revealed during autopsy than during life. Typically, the diagnosis of malignancy has already been established in the patient with a malignant pericardial effusion, and other sites of metastatic spread are evident. On rare occasions, tamponade from the malignant effusion is the first manifestation of tumor. It is important to distinguish malignant effusions from other causes of effusion, such as radiation, infection, and uremia, because the management and prognosis of malignant and nonmalignant effusions in cancer patients differ substantially. The diagnosis is established through echocardiography and cytologic examination; the fluid will be positive in approximately 80% of malignant effusions, with the balance usually positively identified through surgical biopsy of the pericardium. There may be a role for routine measurement of selected tumor markers as a general screen for malignant effusion and an adjunct to direct detection of malignant cells.

Patients may survive for a number of months or occasionally longer after a diagnosis of malignant effusion; therapy to alleviate symptoms and prevent reaccumulation of the effusion is therefore indicated (treatment of nonmalignant effusions is discussed in the section on Pericardial Effusion). Pericardiocentesis usually provides immediate relief. Subsequent management to control the effusion may include radiation or chemotherapy for tumors, such as breast cancer, lymphoma, and leukemia, that are sensitive to these modalities. Instillation of chemicals, most commonly tetracycline, into the pericardium is effective in most cases but can be associated with unpleasant side effects such as pain and fever. Surgical approaches include subxiphoid pericardiotomy or thoracotomy with either pleuropericardiotomy or pericardiectomy. The subxiphoid approach, which can frequently be carried out under local anesthetic, has the advantage of high success rates and low morbidity and mortality. Malignancies, most commonly lymphomas, can also involve the pericardium through enlargement of anterior mediastinal lymph nodes and obstruction of lymphatic drainage. Shrinking the lymph nodes by chemotherapy or radiation therapy may dramatically improve the resulting effusions.

**3. Renal Failure** – Pericardial involvement in patients with renal failure can take several forms. Pericardial effusions can be found on an echocardiogram in many patients with chronic kidney disease who are asymptomatic. These effusions, which are typically small, are related more closely to the patient's volume status than other variables and usually warrant no intervention beyond clinical vigilance.
The incidence of uremic pericarditis has been decreasing for years, a trend attributable to earlier and more intensive dialysis. Uremic pericarditis typically occurs before the initiation of long-term dialysis; its development is related, in part, to the elevation of absolute levels of blood urea nitrogen (BUN) and serum creatinine, and it almost always responds to dialysis. Patients in this setting can have symptoms and signs typical of acute pericarditis, or they may have few symptoms despite large pericardial effusions, with or without a pericardial rub. Although uremic pericarditis can lead to tamponade, it is more commonly associated with the slow accumulation of large, low-pressure pericardial effusions. As in any clinical setting, tamponade warrants prompt evacuation of the pericardial fluid.

Classic ECG changes of acute pericarditis are present in less than half the patients; abnormalities are more often nonspecific and may include atrial fibrillation. Complications from pericardiocentesis in this patient population may be more frequent than in other groups of patients with pericardial effusions.

Pericardial involvement can also occur in patients who are already undergoing dialysis. The development of dialysis-related pericardial disease is not strongly related to serum BUN or creatinine levels. Chest pain, typically pleuritic, is the most common symptom. Pericarditis in a patient undergoing dialysis presents complicated management issues. In a significant fraction of these patients, the pericarditis may have an identifiable, remediable cause that should be promptly identified. Although most patients in whom pericarditis develops while undergoing dialysis respond to intensification of the dialysis regimen, a significant subset do not improve. Clinical predictors of failure include fever higher than 102 °F evidence of heart failure, a white blood cell count greater than 15,000/mcL, a large effusion observed echocardiographically, and the use of peritoneal dialysis as a sole treatment modality. NSAIDs may be tried, but a randomized, prospective study found indomethacin effective only in ameliorating fever. It did not affect the duration of chest pain and pericardial rub or the subsequent development of tamponade. When intensive dialysis and NSAIDs have failed to remedy symptomatic pericarditis, colchicine therapy (see below) should be considered. Effusions resulting in tamponade should be treated in the usual fashion. If tamponade recurs following an initial episode, creation of a pericardial window is recommended.

4. Drug-Related Causes – A number of pharmacologic agents have been implicated in pericardial disease. Pericarditis can occur as a feature of drug-induced SLE syndrome caused by procainamide, hydralazine, diphenylhydantoin, reserpine, methyldopa, and isoniazid. In addition to their propensity for causing myocardial inflammation, anthracycline antineoplastic agents can cause acute pericarditis. Methysergide can cause pericardial constriction as part of the syndrome of mediastinal fibrosis, and pericarditis can be part of a hypersensitivity reaction to penicillin. Minoxidil has been reported to cause pericarditis and tamponade; the mechanism is unknown.

5. Hypothyroidism – Pericardial effusion can be found in one-third of patients with myxedema. The frequency of pericardial involvement is related to both the severity and duration of hypothyroidism. The accumulation of
pericardial fluid in this condition appears to be a result of a combination of increased capillary permeability and retarded lymphatic drainage. Because the pericardial fluid accumulates slowly, tamponade is rare. The ECG demonstrates low QRS. If pericardiocentesis is required before the diagnosis of hypothyroidism is made, the diagnosis can be suspected if the fluid is yellow and contains a high level of cholesterol. Pericardial disease in hypothyroidism reliably responds to thyroid hormone replacement therapy.


ACUTE PERICARDITIS

► Essentials of Diagnosis

► Central chest pain aggravated by coughing, inspiration, or recumbency.
► Pericardial friction rub on auscultation.
► Characteristic ECG changes.
► General Considerations
Acute pericarditis is an inflammatory condition of the pericardium that can be caused by virtually any of the conditions just discussed. As discussed above, a viral infection is the most common cause.

**Clinical Findings**

**A. Symptoms and Signs**

The primary symptom of acute pericarditis is chest pain whose location, intensity, and nature are variable. The pain may be described as sharp or dull. Most often it is precordial or retrosternal in location and may be referred to the trapezius ridge, which is almost pathognomonic for pericarditis. It is characteristically aggravated by inspiration, coughing, or recumbency and lessened by sitting upright and leaning forward. Although it typically takes an hour or two to develop fully, the pain can sometimes appear remarkably abruptly. Many patients relate prodromal symptoms suggestive of a viral infection.

**B. Physical Examination**

Patients with pericarditis may be febrile and tachycardic. The pericardial friction rub—the characteristic auscultatory finding—is typically scratchy and has three components corresponding to atrial contraction, ventricular systole, and early diastole. It is not unusual for only one or two components to be audible; the systolic component is most consistently present. Exercise may facilitate the identification of all three components. Because the friction rub may be evanescent, varying widely in intensity even in the course of a single day, repeated auscultation is important. Furthermore, because posture can affect the pericardial rub, auscultation with the patient in several positions, (supine, sitting, etc) is often helpful. When the intensity of the rub is modulated significantly by respiration, it is termed a "pleuropericardial friction rub."

**C. Diagnostic Studies**

Evaluation of a patient with suspected pericarditis should routinely include an ECG, complete blood count, chest radiograph, creatine kinase with MB fraction, troponin I, and an echocardiogram. Additional diagnostic laboratory tests should be tailored to the clinical presentation. Echocardiography is a sensitive test for detecting pericardial effusion; however, pericardial effusion can occur in the absence of pericardial inflammation.

1. **Electrocardiography** – Serial ECGs are valuable in diagnosing pericarditis. Four stages of ECG changes have been described (Table 17–1). In stage I, the changes accompany the onset of chest pain and consist of widespread ST-segment elevation (Figure 17–2). The ST segment is concave upward (in distinction to the elevation in myocardial infarction). ST-segment elevation is typically present in all leads except aVR and V1, where ST-segment depression is often present. The T waves are upright in the leads with ST elevation. The
stage I pattern of pericarditis may be difficult to distinguish from the normal variant of early repolarization. A differentiating point that may be useful is the ST:T ratio in $V_6$. A T-wave apex four times (or greater) higher than the height of the ST segment is more likely to indicate early repolarization; if this ratio is less than 4, pericarditis is more likely. In addition, pericarditis causes changes in the ECG that distinguish it from early repolarization. In stage II, typically occurring several days later, the ST segments return to baseline, and the initially upright T waves flatten. In stage III, the T waves invert, and the ST segments may become depressed—changes that may persist indefinitely. Finally, in stage IV, which may occur weeks or months later, the T waves revert to normal. All four stages can be serially identified in about 50% of patients.

**Table 17–1. Serial Electrocardiographic Changes in Pericarditis.**

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<tr>
<th>Stage</th>
<th>ST Segment</th>
<th>T Waves</th>
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<tr>
<td>I</td>
<td>Elevated</td>
<td>Upright</td>
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<tr>
<td>II</td>
<td>Isoelectric</td>
<td>Upright flat</td>
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<tr>
<td>III</td>
<td>Isoelectric</td>
<td>Inverted</td>
</tr>
<tr>
<td>IV</td>
<td>Isoelectric</td>
<td>Upright</td>
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2. **Other Tests** — Laboratory evidence of inflammation, such as mild leukocytosis and a modestly elevated erythrocyte sedimentation rate, is common in viral or idiopathic pericarditis. These findings are less consistent in pericarditis associated with uremia or connective tissue disorders. Cardiac enzymes may be slightly elevated when the inflammatory process involves subepicardial myocardium. Alternatively, some cases occur in conjunction with a true viral myocarditis with more substantial elevations in creatine kinase and troponin I. Although the chest radiograph most often reveals no abnormalities in uncomplicated pericarditis, it may occasionally show evidence of pericardial effusion (discussed in the following section).

► **Treatment**
The management of pericarditis associated with an identifiable etiology is directed primarily to the underlying cause. In the usual case of idiopathic acute pericarditis, treatment with any of the NSAIDs usually suppresses the clinical manifestations within 24 hours—and frequently more rapidly. Because of its excellent side-effect profile, ibuprofen, 800 mg orally three times a day for 10–14 days, is recommended. Colchicine, which has an excellent track record in the prophylaxis of recurrent pericarditis (see below), may be added if there is a slow or inadequate response to initial NSAID treatment. A 2–3 mg loading dose followed by 1 mg daily is recommended. A short course of corticosteroids is very effective in ameliorating the pain of acute pericarditis. However, their use is rarely required and recent evidence suggests they may increase the chance of recurrence. Accordingly, corticosteroids should be avoided unless absolutely necessary to control severe symptoms that do not respond to other measures.

In about 85–90% of patients, a single course of NSAID therapy will effectively control the illness, and pericarditis will resolve without sequelae. In a small number of patients, a recurrence may develop over a period of weeks or months after the initial episode. Recurrences can be managed with repeated courses of an NSAID or colchicine, or both. In difficult cases of recurrent pericarditis, prolonged colchicine therapy has demonstrated efficacy in prophylaxis and should be strongly considered. While corticosteroids in general should also be avoided in chronic, recurrent pericarditis, occasional patients can best be managed with very brief, intensive courses of prednisone at the first sign of symptoms. Rarely, immunosuppressive drugs such as azathioprine and cyclophosphamide may be helpful in these patients, but there is virtually no organized experience with their use.

In rare cases, frequent and severe recurrences despite aggressive medical therapy have prompted pericardiectomy. However, the efficacy of pericardiectomy in this circumstance is unknown and in one small series was reported to only rarely be effective.

Pericardial effusion can develop as a result of pericarditis or an injury of any kind to the parietal pericardium. It can be encountered in the absence of pericarditis in many clinical settings, such as, uremia, cardiac trauma or chamber rupture, malignancy, AIDS, and hypothyroidism.

►Clinical Findings

A. Symptoms and Signs

Clinical manifestations of pericardial effusion are directly related to the absolute volume of the effusion and the rapidity of accumulation. Small, incidental effusions rarely, if ever, cause symptoms or complications, and patients with slowly developing pericardial effusions can accumulate large volumes of fluid without symptoms. With a very slowly accumulating effusion, the pericardium can accommodate 1–2 L or more of fluid without a clinically significant elevation of intrapericardial pressure. Many of these large effusions are discovered incidentally when a chest radiograph is performed. Some may become clinically manifest by compression of adjacent structures or by causing dysphagia, cough, dyspnea, hiccups, hoarseness, nausea, or a sense of abdominal fullness. It is exceedingly rare for a specific cause of large, incidentally discovered effusions to be identified. In contrast, more rapid accumulation of even modest fluid volumes can be associated with increased intrapericardial pressures and life-threatening hemodynamic compromise.

B. Physical Examination

On examination, signs of pericardial effusion are absent in patients who have small effusions without increased pressure. Large effusions may muffle the heart sounds or cause left lower lobe lung dullness to percussion of the chest (Ewart sign) as a result of compression of lung parenchyma.

C. Diagnostic Studies

1. Electrocardiography – The ECG may be entirely normal. Large effusions can cause both reduced voltage and electrical alternans, alternating QRS voltage as a result of a swinging motion of the heart that characteristically occurs at a frequency of half the heart rate. If pericarditis coexists with effusion, the usual findings may be present.

2. Chest Radiography – An increase in the cardiac silhouette combined with clear or oligemic lung fields suggests the presence of a significant pericardial effusion, although the chest radiograph can appear entirely normal in the presence of a small to moderate effusion. Very rapidly accumulating fluid may result in only the subtlest of changes in the cardiac silhouette. With slowly accumulating fluid, the cardiac silhouette may assume a globular shape that has been likened to a water bottle. Radiographic differentiation of pericardial
effusion and cardiac enlargement may not be possible. Occasionally, the presence of an effusion may cause increased separation of the pericardial fat-pad layers.

3. Echocardiography – Transthoracic echocardiography is the fastest and most accurate means of diagnosing and estimating the size of a pericardial effusion, but it is not accurate in assessing pericardial thickness. The effusion appears as an echo-free space between the moving epicardium and the stationary pericardium. Although M-mode echocardiography can identify effusions as small as 20 mL, two-dimensional echocardiography has the advantage of demonstrating the full distribution of the effusion and identifying a loculated effusion. Quantification of the volume of effusion by echocardiography is not always precise. Small effusions tend to be imaged only posteriorly; a posterior echo-free space, however, may reflect subepicardial fat rather than pericardial effusion. Larger effusions are usually distributed both anteriorly and posteriorly. On occasion, large effusions are associated with an excessive swinging motion of the heart within the fluid-filled pericardium. Transesophageal echocardiography is particularly useful in quantifying the anatomic distribution of effusions, especially when loculated, and is superior to transthoracic echocardiography in imaging the thickness of the pericardium.

4. Cardiac Magnetic Resonance Imaging and Computed Tomography – Both of these modalities provide highly accurate imaging of pericardial effusions but, in most cases, are not necessary if echocardiographic images are technically satisfactory. However, they are the most accurate methods for delineating the size of effusions, their anatomic distribution, and the thickness of the pericardium. Thus, they are often valuable adjuncts to echocardiography.

► Treatment

The management of a pericardial effusion is largely dictated by its size, the presence or absence of hemodynamic compromise from increased intrapericardial pressure (see next section), and the nature of the underlying disorder (discussed earlier). In most cases, a small or incidentally discovered effusion warrants no specific intervention. At the same time it should be recalled that once an effusion reaches a certain magnitude, even small additional amounts of fluid can cause a marked increase in intrapericardial pressure and rapid clinical deterioration; these patients must be monitored closely. Very large, chronic effusions discovered accidentally usually do not progress. However, some of these will eventually result in cardiac tamponade. Therefore, elective, closed pericardiocentesis may be recommended in these cases. Interestingly, these effusions typically do not recur following removal of the fluid.

CARDIAC TAMPO NADE

► Essentials of Diagnosis

► Increased jugular venous pressure with an obliterated y descent.
► Pulsus paradoxus.
► Echocardiographic evidence of right atrial and ventricular collapse.
► Equal diastolic pressures in all four cardiac chambers.

► General Considerations

Cardiac tamponade exists when increased intrapericardial pressure from accumulation of fluid compromises the filling of the heart, thereby impairing cardiac output. Whether the intrapericardial pressure rises to a level that impedes filling depends on both the rapidity of accumulation and the volume of the effusion. Severe tamponade may thus ensue in the setting of a traumatic effusion where a modest volume of blood fills the pericardial space in a brief time. Conversely, in settings such as myxedema or chronic idiopathic effusions, a slowly accumulating effusion may reach a remarkably large volume without raising the intrapericardial pressure.

► Clinical Findings

A. Symptoms and Signs

Patients with cardiac tamponade may complain of dyspnea and chest discomfort. In more severe cases, consciousness may be impaired, and there may be signs of reduced cardiac output and shock. The systemic arterial pressure is typically low, although it may be surprisingly well-preserved on occasion; pulse pressure is usually diminished. The patient with tamponade is typically tachycardiac and tachypneic although bradycardia may ensue in terminal stages. Patients with tamponade are almost always more comfortable sitting upright. If pericarditis coexists, typical pain and a friction rub may be present.

1. Pulsus Paradoxus – Pulsus paradoxus, defined as an abnormally large decline in systolic arterial pressure during inspiration, is present in most cases. The term is actually something of a misnomer because the paradoxical pulse represents an exaggeration of the normal small decline in systolic arterial pressure that occurs during inspiration. Pulsus paradoxus is evaluated through careful auscultation of the Korotkoff sounds as the cuff pressure is slowly released. It is measured as the difference in cuff pressure from the point at which sounds are initially heard intermittently during expiration and the point at which the sounds are audible throughout the respiratory cycle and with each ventricular systole. A pulsus paradoxus greater than 10 mm Hg
is considered abnormal. In severe tamponade, the peripheral pulse reveals an obvious decrease in the stroke volume and may even disappear on inspiration. The presence of an abnormal pulsus paradoxus is not essential to the diagnosis of cardiac tamponade, and it may be absent in clinical situations where tamponade coexists with cardiac volume overload lesions, such as atrial septal defect or aortic insufficiency. The absence of a pulsus paradoxus in these settings should not dissuade the physician from the correct diagnosis. In addition, pulsus paradoxus can be present when the inspiratory decrease in intrapleural pressure is exaggerated, as in obstructive airway disease. Reversed pulsus paradoxus—a fall in pressure with expiration—can be seen in patients on positive pressure respirators.

2. Jugular Venous Pressure – The venous pressure is usually markedly elevated, and examination of the jugular venous pulse wave reveals obliteration of the normal y descent. In patients with low-pressure tamponade (see section on Cardiac Catheterization), the venous pressure may actually be normal or only mildly elevated.

3. Other Findings – Diminished heart sounds can be heard in one-third of cases; a pericardial friction rub may be heard but is absent in most patients. The concept of an inverse relation between the intensity of a pericardial rub (when present) and the size of the effusion cannot be used in assessing individual patients; the relationship is too variable to be reliable.

B. Diagnostic Studies

1. Electrocardiography – Electrocardiography may offer no specific diagnostic clues, although the ECG abnormalities described in pericarditis and pericardial effusion may be seen. The development of electrical alternans almost always indicates a hemodynamically significant effusion.

2. Chest Radiography – Chest radiography offers no specific diagnostic signs of tamponade. As mentioned earlier, the cardiac silhouette may be remarkably normal in size in cases where a modestly sized effusion accumulates rapidly. A pericardial fat pad sign is diagnostic of pericardial effusion but does not necessarily indicate tamponade. The lung fields are frequently oligemic. Occasionally, the chest radiograph offers clues to important coexisting conditions, such as aortic dissection or malignancy.

3. Echocardiography – Echocardiography is an invaluable adjunctive tool. It confirms the presence of pericardial fluid and can provide evidence of increased intrapericardial pressure. The most useful echocardiographic sign is diastolic collapse of the right atrium and right ventricle. Though these changes are neither completely sensitive nor specific, they first occur when the pericardial pressure transiently exceeds the intracardiac chamber pressure. They can therefore be useful in identifying patients whose pericardial pressure level should be of concern. Echocardiography is also extremely useful as a guide in pericardiocentesis. The cardiac chambers are small in tamponade and, as discussed above, in extreme cases the heart swings
anteroposteriorly within the effusion. Distention of the caval vessels that does not diminish with inspiration is another useful sign.

Doppler velocity recordings demonstrate exaggerated respiratory variation in right- and left-sided venous and valvular flow, with marked inspiratory increases on the right and decreases on the left. Loss of the y descent in the jugular venous pressure is caused by reduced systemic venous inflow during early diastole. Correspondingly, Doppler evaluation reveals that most caval and pulmonary venous inflow occurs during ventricular systole. These flow patterns were found to have a sensitivity of 75% and a specificity of 91% for diagnosing tamponade. The absence of chamber collapse is especially useful in excluding tamponade in patients with effusions but its presence is less well-correlated with tamponade than abnormal venous flow patterns. Newer techniques such as tissue Doppler do not yet have a well-defined, additive role in cardiac tamponade.

4. Cardiac Catheterization – In the patient with tamponade, cardiac catheterization reveals a depressed cardiac output as well as elevated and equal or near-equal filling pressures in all four chambers. Examination of the atrial pressure waveforms reveals the loss of the normal y descent. The initial presentation and hemodynamic profile of tamponade may, however, be altered by a concomitant state of intravascular volume depletion, a scenario that has been called low-pressure cardiac tamponade. This term underscores an important feature of the pathophysiology of pericardial effusions; the hemodynamic effect is a function of both the intrapericardial pressure and the intravascular volume. Although this syndrome typically occurs in patients undergoing dialysis for chronic renal failure, it can be encountered in any setting of increased intrapericardial fluid and intravascular volume depletion. In these patients, the effusions are ordinarily insufficient to cause major hemodynamic embarrassment; they become significant when intravascular volume is depleted. The diagnosis should be considered in patients who become unusually hypotensive during dialysis. In some cases, volume expansion will result in more typical hemodynamic findings.

► Treatment

Drainage of pericardial fluid is the cornerstone of therapy; reflecting the small pericardial reserve volume, draining even modest amounts (100–200 mL) of fluid may result in striking improvement. Drainage is most commonly achieved by subxiphoid percutaneous pericardiocentesis. Although the procedure is effective and safe, there may be complications; the most common serious complication is laceration or puncture of the heart, typically the right ventricle, because of its anterior location. Echocardiography, by confirming the presence of a sufficiently large volume of fluid in an anterior location, can decrease the risk of cardiac puncture. The presence of at least 1 cm of echo-free space anterior to the heart has been recommended as a guideline for the minimum volume of fluid that should be present before percutaneous pericardiocentesis is undertaken. In addition, the patient should be positioned in a semiupright position to allow inferior pooling of the effusion.
Several aspects of the performance of pericardiocentesis also contribute to a safe and successful outcome. The procedure is ideally carried out in the cardiac catheterization laboratory with fluoroscopic guidance and concomitant right-heart catheterization. The latter allows for hemodynamic confirmation of the diagnosis and assessment of the response to therapy. Occasionally, performance of emergency pericardiocentesis may be required at the bedside; however, it is rare for circumstances to be so critical as to preclude confirming the diagnosis with echocardiography.

Intravenous fluids and pressors can be administered as temporizing measures until the procedure can be performed. These modalities usually will not significantly improve the clinical status, however, and should never be used in place of or allowed to interfere with prompt evacuation of the fluid.

Pericardial fluid can also be evacuated through a subxiphoid surgical pericardiotomy performed under local anesthesia; this procedure also permits pericardial biopsy in cases of suspected malignant effusion. The pericardial fluid should be sent for cultures and cytologic examination except when the tamponade is clearly traumatic. The gross appearance of the fluid is not helpful in establishing the cause, and cell counts and chemistries are also of limited value. The risks of pericardiocentesis must therefore be weighed against the likely benefits before performing the procedure solely for diagnostic purposes. Usually a pericardial biopsy is most useful when the diagnosis is unclear.

In some cases, a single pericardiocentesis alleviates the effusion fully, but in most cases, a pericardial catheter should be left in place for continued drainage, typically for 24–48 hours. The catheter can be removed when the rate of drainage decreases and plans for definitive management of the pericardial disease are in place. Subsequent management is largely dictated by the specific cause of the effusion (discussed earlier).

Definitive management of pericardial fluid accumulation in some conditions may require surgical removal of the pericardium or the surgical creation of an opening between the pericardium and left pleura (a pericardial window). A percutaneous balloon technique for creating a pleuropericardial opening has also been described. If tissue is not required for diagnostic purposes, this may be the preferred technique for draining a chronically recurring effusion and preventing tamponade. However, at present, few centers have sufficient experience with this method. Pericardial windows can close in patients with intense inflammation, and pericardial stripping may be required.

CONSTRUCTIVE PERICARDITIS

ESSENTIALS OF DIAGNOSIS

Markedly elevated jugular venous pressure with accentuated x and y descents and Kussmaul sign.
Pericardial knock on auscultation.
Magnetic resonance, computed tomography, or echocardiographic imaging showing a thickened pericardium.

► General Considerations

Constrictive pericarditis can develop as the aftermath of virtually any pericardial injury or inflammation. Cardiac surgery, radiation therapy, and idiopathic causes are currently the most common. Tuberculous constriction, a leading cause in previous decades, is now rare in most of the industrialized world, but it remains significant in underdeveloped countries and may reappear in developed countries if tuberculosis continues to increase. A highly variable length of time—sometimes many years—can elapse between the initial insult and the development of constriction and its clinical manifestations.

The major physiologic perturbation of constrictive pericarditis is thickening, fibrosis, and (especially with tuberculosis) calcification of the pericardium, causing it to encase the heart in a solid, noncompliant envelope that impairs diastolic filling. In early diastole, the ventricles fill normally until the volume limit of the noncompliant pericardium is attained. At that point, diastolic filling halts abruptly. At the same time, the rigid pericardium markedly increases the intracardiac filling pressures. Because contractile function is usually normal, constrictive pericarditis can be considered an extreme example of heart failure that is caused by diastolic dysfunction.

► Clinical Findings

A. Symptoms and Signs

Many symptoms of constrictive pericarditis are nonspecific and are related to chronically elevated cardiac filling pressures and chronically depressed cardiac output; symptoms secondary to venous congestion are most common. Ascites, peripheral edema, and symptoms referable to congestion of the gastrointestinal tract and liver (eg, dyspepsia, anorexia, and postprandial fullness) usually develop. Cardiac cirrhosis may be present in extreme cases. Symptoms of left-sided congestion, such as exertional dyspnea, orthopnea, and cough, may occur but are much less frequent. The chronically low cardiac output results in fatigue and, in conjunction with the effects of visceral congestion, wasting.
B. Physical Examination

The patient may have a striking body habitus with a marked contrast between a massively swollen abdomen and edematous lower extremities and a cachectic, wasted upper torso. Ascites, hepatomegaly with prominent hepatic pulsations, and other signs of hepatic failure are common.

Patients with constrictive pericarditis have marked elevation of the jugular venous pressure. In contrast with cardiac tamponade, the $x$ and $y$ descents are prominent, typically resulting in an M or W shape of the venous waves. Kussmaul sign—the loss of normal inspiratory decrease in the jugular venous pressure or even a frank increase with inspiration—may be present. (Kussmaul sign may be seen in other disorders, however, including restrictive cardiomyopathy.) The arterial pulse pressure may be diminished or normal. A pulsus paradoxus is present in perhaps one-third of cases.

Auscultation of the heart can reveal a characteristic early diastolic sound, the pericardial knock. The knock occurs slightly earlier in diastole than a third heart sound and has a higher acoustic frequency.

C. Diagnostic Studies

1. Electrocardiography – Characteristic ECG abnormalities in constrictive pericarditis are nonspecific and include low voltage, T-wave inversions, and P mitrale or atrial fibrillation. Atrioventricular and intraventricular conduction delays or the development of Q waves are related to the extension of calcification into the myocardium and surrounding the coronary arteries.

2. Chest Radiography – The cardiac silhouette on chest radiograph can be small, normal, or enlarged. The presence of pericardial calcification is helpful in confirming the diagnosis and suggests tuberculosis as the cause. However, only a small number of patients with constriction have pericardial calcification. Conversely, a calcified pericardium does not always indicate constriction.

3. Echocardiography – Echocardiography demonstrates pericardial thickening in most cases of constriction. As with chest radiography, however, the presence or absence of echocardiographic pericardial thickening neither establishes nor excludes the diagnosis with certainty, and imaging of the pericardium by echocardiography can sometimes be misleading. (Transesophageal echocardiography is more reliable than transthoracic echocardiography for imaging the pericardium.) Echocardiography may be useful in first raising the suspicion of constrictive pericarditis in a patient with heart failure, preserved left ventricular ejection fraction, and normal or small cardiac chamber sizes. In cases with extreme limitation of cardiac filling or when pericardial calcification extends into the myocardium, left ventricular ejection may be impaired. Thus, preserved systolic function is not a prerequisite for diagnosis. A characteristic echocardiographic feature of constrictive pericarditis is the septal "bounce," a stuttering motion of the septum during diastole. A variety of indices using Doppler echocardiography to assess ventricular filling in various stages of diastole have been proposed to
distinguish between constrictive pericarditis and restrictive cardiomyopathy. Those that appear most useful are exaggerated respiratory variations of transmitral and hepatic vein flow and isovolumetric relaxation time. Both of these are present in constriction but absent in restrictive cardiomyopathy. Tissue Doppler examination reveals increased E’ velocity of the mitral annulus as well as septal abnormalities analogous to the "bounce." Tissue Doppler appears to be at least as sensitive as conventional echocardiography-Doppler for diagnosing constriction.

4. Magnetic Resonance Imaging and Computed Tomography – Both of these methods are especially useful for imaging the pericardium. They are more accurate than echocardiography in this regard and provide a much more complete assessment of the appearance of the entire pericardium. Figure 17–3 is an example of a computed tomographic scan from the same patient whose chest radiograph is shown in Figure 17–4. Patients being considered for pericardiectomy should always be evaluated with one of these techniques. Magnetic resonance imaging in particular can be used to discern features of constrictive pericarditis such as the septal bounce and abnormal respiratory variations in mitral and tricuspid flow. Although the presence of pericardial thickening ordinarily is a key point in distinguishing constrictive pericarditis, occasional cases of constriction in the absence of thickening have been reported.

5. Cardiac Catheterization – Cardiac catheterization can help establish the correct diagnosis. It confirms elevated—and usually virtually equal—diastolic pressures in both ventricles. The diastolic pressure waveform has been described as a square root sign, or dip and plateau: an exaggerated early diastolic downward deflection (the dip) followed by a rapid early pressure rise and plateau. This waveform, however, is not pathognomonic for constriction; it can also be found in restrictive cardiomyopathy. Examination of the right and left atrial waveforms reveals prominent x and y descents. As with the jugular venous waveform, the appearance of the atrial pressure recording has been likened to a W or an M shape. Several hemodynamic criteria are useful in distinguishing constrictive pericarditis from restrictive cardiomyopathy (Table 17–2). Although the accuracy of any one of these criteria is far from perfect, the concordance of all three criteria favoring constriction renders the diagnosis 91% certain. Similarly, if none or only one hemodynamic criterion in favor of constriction is met, the patient has restriction with 94% certainty. One-fourth of patients with the appropriate physiology will meet two criteria, making their chances of either diagnosis approximately equal.

Table 17–2. Hemodynamic Criteria Differentiating Constrictive Pericarditis from Restrictive Cardiomyopathy

<table>
<thead>
<tr>
<th>Criteria Favoring Constriction</th>
<th>Predictive Accuracy (%)</th>
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</thead>
<tbody>
<tr>
<td>Difference between LVEDP and RVEDP &lt; 5 mm Hg</td>
<td>85</td>
</tr>
<tr>
<td>RV systolic pressure &gt; 50 mm Hg</td>
<td>70</td>
</tr>
</tbody>
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LVEDP, left-ventricular end-diastolic pressure; RVEDP, right ventricular end-diastolic pressure; RV, right ventricle.

6. Endomyocardial Biopsy – At the time of cardiac catheterization, endomyocardial biopsy can be performed to search for evidence of infiltrative cardiomyopathy. The finding of amyloidosis, sarcoidosis, or hemochromatosis eliminates the need for further investigation. The finding of myocarditis, however, is not specific, and further studies may be needed.

► Differential Diagnosis

With the combined use of Doppler echocardiography, magnetic resonance imaging or computed tomography to image the pericardium, careful hemodynamic studies, and endomyocardial biopsy, it should be possible in the great majority of cases to distinguish constrictive pericarditis from restrictive cardiomyopathy. This distinction is not always possible, however, and such differentiation can sometimes be a major diagnostic challenge. Restrictive cardiomyopathy is a condition that is most commonly caused by infiltrative diseases of the myocardium such as amyloidosis, sarcoidosis, and hemochromatosis—or, in Africa, by endocardial fibroelastosis. Both constrictive pericarditis and restrictive cardiomyopathy are characterized by impaired diastolic filling of the ventricles. The two differ, however, in the degree of impairment of various phases of diastole. Unlike the rapid early diastolic filling and abrupt halt of constrictive pericarditis, ventricular filling in restrictive cardiomyopathy is impaired uniformly throughout diastole. This physiologic difference underlies many of the features that help distinguish the two diagnoses. Other useful distinguishing features are biatrial enlargement, which is usually striking in restrictive cardiomyopathy and absent in constriction, and markedly increased ventricular wall thickness, which is the rule in most cases of restrictive cardiomyopathy. Last, brain natriuretic peptide levels are helpful; they are elevated in restrictive cardiomyopathy and normal in constriction. The correct differentiation of these two conditions is of paramount importance: Constrictive pericarditis is an eminently treatable disease; cardiac restriction of almost any cause carries a limited prognosis—despite therapy. When the distinction between constriction and restriction remains ambiguous, it may be necessary to proceed to thoracoscopy to permit direct inspection of the pericardium. Pulmonary hypertension causing right heart failure has several similarities with both constrictive pericarditis and restrictive cardiomyopathy. Measurement of the pulmonary artery pressure and vascular resistance establish the diagnosis of pulmonary hypertension.

At the bedside, constrictive pericarditis is sometimes difficult to distinguish from more common causes of congestive heart failure. Disproportionate right-sided failure or ascites out of proportion to peripheral edema may be clues to the presence of constriction. Because of the relative rarity of constriction, it is often not
suspected, and congestive heart failure or even noncardiac cirrhosis is diagnosed instead. Because the primary form of therapy for constriction is surgical, it is essential to be sure that the diagnosis of constriction is correct.

► Treatment

Although intensive medical management may effectively control symptoms, the long-term prognosis with medical therapy alone is limited: The natural history in most cases is one of advancing severity.

Pericardiectomy is the definitive treatment for constrictive pericarditis. The only exception is early postsurgical constriction which, as discussed earlier, responds well to a course of corticosteroids. In most cases, the procedure is straightforward, and the surgical mortality rate in recent series has ranged from 4% to 11%. Occasionally, however, the dense fibrosis and calcification extend into the epicardium, making identification of a cleavage plane impossible. The operation in this case is associated with excessive hemorrhage and an inability to relieve the compression completely. In other patients, hepatic or cardiac failure may be irreversible. The myocardium may undergo atrophy as a result of long-standing compression, and a low-cardiac-output state may persist after pericardiectomy.

In most cases, patients will exhibit dramatic and sustained improvement, although full improvement may occur only after several months. When symptoms are persistent or recurrent, three possibilities should be considered: myocardial dysfunction resulting from severe, prolonged compression; incomplete or inadequate pericardiectomy; and recurrence of the constriction. In some cases, the inflammatory and fibrotic process involves the epicardial layers and progresses after the pericardium has been removed, leading to a recurrence of constrictive physiology and symptoms.

The combination of constrictive pericarditis and occlusive coronary artery disease is especially difficult to manage. In some cases, the coronary lesions are due to the visceral pericardial process, and stripping the pericardium may damage the coronary arteries. Bypass surgery can be extremely difficult in the presence of the dense calcium. If the coronary obstructions are not corrected, however, the increased myocardial oxygen demands of increased cardiac filling following pericardiectomy may cause postoperative ischemia—which may severely complicate recovery from the surgery.

**EFFUSIVE-CONSTRUCTIVE PERICARDITIS**

**ESSENTIALS OF DIAGNOSIS**

- Echocardiographic demonstration of pericardial fluid.
- Persistance of elevated intracardiac filling pressures following pericardiocentesis.
- General Considerations

Effusive-constrictive pericarditis combines features of pericardial effusion and constrictive pericarditis. The syndrome is dynamic and may represent an intermediate stage in the development of constrictive pericarditis. The most common causes of effusive-constrictive pericarditis are uremia and metastatic pericardial disease, but any cause of pericarditis can produce this condition. Echocardiography usually shows a small-to-moderate-sized effusion with strands of solid material between the visceral and parietal pericardium. Although effusive-constrictive pericarditis may be suspected on clinical grounds, the diagnosis is established through cardiac catheterization and characterization of the hemodynamics. Effusive-constrictive pericarditis can present as frank tamponade. As the effusion is drained during pericardiocentesis, elevation of intracardiac filling pressures persists and the recorded waveforms may exhibit the classic appearance of constriction.

Although pericardiocentesis may be associated with an improved cardiac output and diminished symptoms, subsequent management is essentially that for pericardial constriction. Thoracotomy with pericardiectomy is indicated to relieve the constriction in symptomatic patients who do not have diagnoses with a very poor short-term prognosis, such as advanced metastatic cancer.