The diagnosis of pericardial fluid or thickening may be confirmed by computed tomography (CT) or magnetic resonance imaging (MRI). These techniques may be superior to echocardiography in detecting loculated pericardial effusions, pericardial thickening, and the identification of pericardial masses.

TREATMENT ACUTE PERICARDITIS

There is no specific therapy for acute idiopathic pericarditis, but bed rest and anti-inflammatory treatment with aspirin (2–4 g/d), with gastric protection (e.g., omeprazole 20 mg/d), may be given. If this is ineffective, one of the nonsteroidal anti-inflammatory drugs (NSAIDs), such as ibuprofen (400–600 mg tid) or indomethacin (25–50 mg tid), should be tried. In responsive patients, these doses should be continued for 1–2 weeks and then tapered over several weeks. In patients who are unresponsive, colchicine (0.5 mg bid, given for 4–8 weeks) has been found to be effective, not only in acute pericarditis, but also in reducing the risk of recurrent pericarditis. Colchicine is concentrated in and interferes with the migration of neutrophils, is contraindicated in patients with hepatic or renal dysfunction, and may cause diarrhea and other gastrointestinal side effects. Glucocorticoids (e.g., prednisone 1 mg/kg per day) usually suppress the clinical manifestations of acute pericarditis in patients who have failed therapy with the anti-inflammatory therapies described above, but appear to increase the risk of subsequent recurrence. Therefore, full-dose corticosteroids should be given for only 2–4 days and then tapered. Anticoagulants should be avoided because their use could cause bleeding into the pericardial cavity and tamponade.

In patients with recurrences that are multiple, frequent, disabling, continue for more than 2 years, and are not prevented by colchicine and other NSAIDs and are not controlled by glucocorticoids, pericardial stripping may be necessary to terminate the illness, and usually does so.

CARDIAC TAMPONADE

The accumulation of fluid in the pericardial space in a quantity sufficient to cause serious obstruction of the inflow of blood into the ventricles results in cardiac tamponade. This complication may be fatal if it is not recognized and treated promptly. The most common causes of tamponade are idiopathic pericarditis and pericarditis secondary to neoplastic disease. Tamponade may also result from bleeding into the pericardial space after leakage from an aortic dissection, cardiac operations, trauma, and treatment of patients with acute pericarditis with anticoagulants.

The three principal features of tamponade (*Beck's triad*) are hypotension, soft or absent heart sounds, and jugular venous distention with a prominent *x* descent but an absent *y* descent. The limitations of ventricular filling are responsible for a reduction of cardiac output. The quantity of fluid necessary to produce cardiac tamponade may be as small as 200 mL when the fluid develops rapidly to as much as >2000 mL in slowly developing effusions when the pericardium has had the opportunity to stretch and adapt to an increasing volume. Tamponade may also develop more slowly, and in these circumstances, the clinical manifestations can resemble those of heart failure, including dyspnea, orthopnea, and hepatic engorgement.

A high index of suspicion for cardiac tamponade is required because in many instances no obvious cause for pericardial disease is apparent, and this diagnosis should be considered in any patient with otherwise unexplained enlargement of the cardiac silhouette, hypotension, and elevation of jugular venous pressure. There may be reduction in amplitude of the QRS complexes, and *electrical alternans* of the P, QRS, or T waves should raise the suspicion of cardiac tamponade (Fig. 288-3).

Table 288-2 lists the features that distinguish acute cardiac tamponade from constrictive pericarditis.

TABLE 288-2 FEATURES THAT DISTINGUISH CARDIAC TAMPONADE FROM CONSTRICTIVE PERICARDITIS AND SIMILAR CLINICAL DISORDERS

Characteristic	Tamponade	Constrictive Pericarditis	Restrictive Cardiomyopathy	RVMI	Effusive Constrictive Pericarditis
Clinical					
Pulsus paradoxus	+++	+	+	+	+++
Jugular veins					
Prominent *y* descent	–	++	+	+	–
Prominent *x* descent	+++	++	+++	+	+++
Kussmaul's sign	–	+++	+	+++	++
Third heart sound	–	–	+	+	+
Pericardial knock	–	++	–	–	–
Electrocardiogram					
Low ECG voltage	++	++	++	–	++
Electrical alternans	++	–	–	–	+
Echocardiogram					
Thickened pericardium	–	+++	–	–	++
Pericardial calcification	–	++	–	–	
Pericardial effusion	+++	–	–	++	
RV size	Usually small	Usually normal	Usually normal	Enlarged	
RA and RV	+++	–	–	–	
Exaggerated respiratory variation in flow velocity	+++	+++	–	+++	
CT/MRI					
Thickened pericardium	–	+++	–	++	
Cardiac catheterization					
Equalization of diastolic pressures	+++	+++	–	++	

Abbreviations: +++, always present; ++, usually present; +, rare;–, absent; DC, diastolic collapse; ECG, electrocardiograph; RA, right atrium; RV, right ventricle; RVMI, right ventricular myocardial infarction.

Source: Adapted from GM Brockington et al: Cardiol Clin 8:645, 1990, with permission.