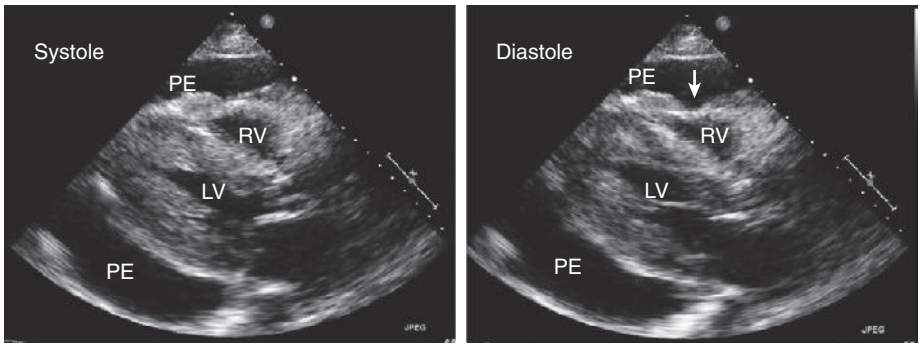

FIGURE 10-1 Parasternal long axis echocardiographic views of the right ventricle in systole and diastole show right ventricular diastolic collapse *(arrow)* in a patient with a large, circumferential pericardial effusion. LV, Left ventricle; PE, pericardial effusion; RV, right ventricle.

surrounding structures. A diagnostic pericardiocentesis is the procedure of choice for evaluating for bacterial, tuberculous, or malignant causes.

Treatment

Routine drainage of pericardial effusions is unnecessary in the absence of hemodynamic compromise. Cardiac tamponade is a life-threatening emergency requiring urgent drainage of the pericardial effusion. Fluid resuscitation should be initiated to increase preload and filling of the cardiac chambers. Inotropic and vasopressor support has limited utility. Surgical drainage is appropriate and therapeutic for loculated, purulent, and tuberculous effusions and for tissue biopsy.

Fluid should be analyzed for pH, cell count, glucose, protein, cholesterol, triglycerides, and acid-fast bacilli by Gram stain, culture, cytology, and laboratory tests. For patients with chronic, recurring effusions, the surgical creation of a pleuropericardial window provides a long-term solution.

Prognosis

The underlying cause of the pericardial effusion and the availability of effective treatment determine the prognosis.

Constrictive Pericarditis

Definition and Epidemiology

Pericardial constriction is a condition characterized by a rigid, scarred pericardium that limits diastolic filling of the ventricles, resulting in increased intracardiac pressures. It can be caused by any type of pericardial inflammation. The most common causes are infection, prior cardiac surgery, trauma, and irradiation. Less common causes include connective tissue disorders, uremia, and neoplastic involvement of the pericardium. In developing countries, tuberculous pericarditis is a common cause of pericardial constriction. Often a specific cause cannot be determined.

Pathology

Constriction is the end result of pericardial inflammation with scarring, fibrosis, calcification, and adhesion of the parietal and visceral layers of the pericardium. Although pericardial thickening is a usual pathologic finding, its absence does not exclude constriction.

Clinical Presentation

In the early stages, symptoms consist of dyspnea, fatigue, decreased exercise tolerance, and lower extremity edema. As the disease progresses, early signs and symptoms may be accompanied by ascites, anasarca, cachexia, and muscle wasting.

Physical examination reveals jugular venous distention with prominent *x* and *y* descents and an increase (or failure to decrease) of central venous pressure with inspiration (i.e., Kussmaul sign). The arterial blood pressure is usually normal, and pulsus paradoxus is absent in most patients. Ascites and hepatomegaly can be prominent with advanced disease. On cardiovascular examination, the apical impulse may be decreased and the cardiac sounds muffled. An early diastolic sound (i.e., pericardial knock) corresponding to the abrupt cessation of early ventricular diastolic filling is pathognomonic of pericardial constriction, but it is not always detected.

Diagnosis

The diagnosis of pericardial constriction may be challenging and frequently requires the use of multiple imaging modalities. The electrocardiogram may display low QRS voltage, left atrial enlargement, and nonspecific T-wave changes. Atrial fibrillation occurs in one third of cases. The chest radiograph may reveal pleural effusions and pericardial calcification, which are best appreciated in the lateral projection.

Transthoracic echocardiography shows dilation of the inferior vena cava, abnormal interventricular septal motion, and pericardial thickening. Doppler echocardiography demonstrates abnormal respirophasic variations of the pulmonary and hepatic venous flow and mitral valve inflow. CT and MRI can accurately measure pericardial thickness.

Cardiac catheterization is essential in the diagnosis of pericardial constriction and differentiation from restrictive cardiomyopathy (RCM). The right atrial pressure tracing shows prominent *x* and *y* descents with equalization of the end-diastolic atrial and ventricular pressures. The ventricular pressure tracings show a rapid early diastolic filling of the ventricles, with abrupt cessation in middle and end diastole due to the finite volume of the rigid pericardium (i.e., dip-and-plateau morphology or the square root sign) (Fig. 10-2). Enhanced ventricular interdependence demonstrated by simultaneous measurement of right and left ventricular pressures during respiration is a more specific finding of pericardial constriction.