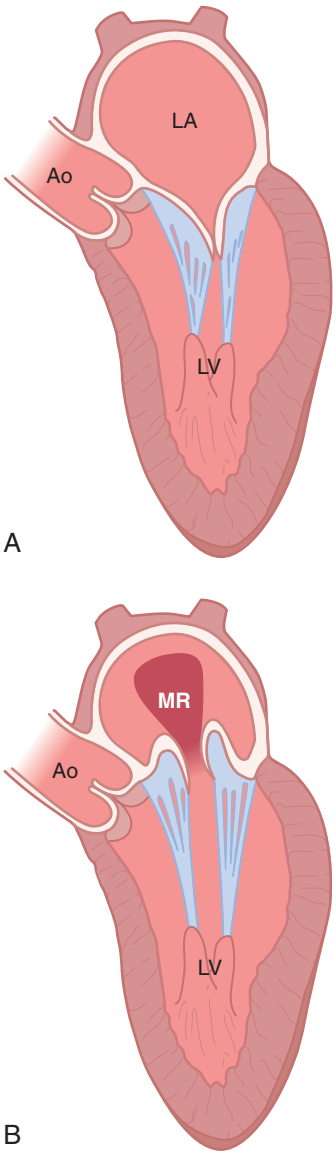

FIGURE 7-8 Degenerative mitral valve disease is an inheritable leaflet abnormality that often results in mitral valve prolapse, the most common cause of mitral regurgitation. **A,** Diagram shows normal leaflet position during closure in systole. **B,** Prolapsing motion of the mitral leaflets above the mitral annulus during systole results in mitral regurgitation (MR). Ao, Aorta; LA, left atrium; LV, left ventricle.

When the regurgitation develops slowly over months to years, the heart typically compensates with ventricular remodeling, enlargement, and increased compliance. This permits the stroke volume to be maintained at normal filling pressures for prolonged periods, preventing symptoms. Eventually, the systolic function declines or the patient develops symptoms, heralding the need for surgery.

Clinical Presentation

Patients with acute severe mitral regurgitation are acutely ill at presentation and often are in cardiogenic shock. In addition to dyspnea, hypotension, and tachycardia, they may have signs and symptoms of the primary etiology. For example, a patient who has a ruptured papillary muscle due to a myocardial infarction may also have chest pain and ECG changes at presentation.

When the heart has time to develop compensatory mechanisms with chronic severe regurgitation, patients may remain asymptomatic and not even realize the condition is present until it is identified by physical examination. Over time, exertional fatigue and shortness of breath develop. If the condition is not identified in these early stages, the patient will ultimately develop typical signs and symptoms of chronic congestive heart failure and arrhythmia.

Diagnosis

In patients with acute severe regurgitation, the examination findings typically observed with mitral regurgitation may be absent or difficult to detect. Rapid rise in left atrial pressure during systole results in an abbreviated systolic murmur. When tachypnea and tachycardia are present as additional confounders to the physical examination, the diagnosis can be easily missed at the bedside (see Table 7-2). If clinical suspicion is present, echocardiography is an essential tool in rapidly confirming the diagnosis.

A patient with chronic severe mitral regurgitation is more likely to have a classic holosystolic murmur present at the LV apex. If the cause of the regurgitation is mitral valve prolapse, there is often a mid-systolic click followed by a late-peaking systolic murmur. The cardiac enlargement that has evolved may be appreciated by a diffuse, laterally displaced PMI. An S_3 gallop may be present, even in the absence of congestive heart failure.

Diagnosis is best confirmed by TTE. This technique is important, not only for quantitatively estimating the regurgitation severity but for defining the anatomic mechanism, which has significant implications for management. If the valvular abnormality cannot be confirmed by transthoracic imaging, both two- and three-dimensional TEEs are very sensitive for identifying the anatomic mechanism of regurgitation.

Techniques in the cardiac catheterization laboratory can be useful in evaluating the severity of regurgitation, but these are less commonly used and have been largely supplanted by echocardiography.

Treatment

As in patients with acute severe aortic regurgitation, the primary objective is to stabilize the patient while urgently arranging for definitive mechanical correction—namely, repair or replacement of the mitral valve. In contrast to acute severe aortic regurgitation, an intraortic balloon pump is an option for reducing afterload and improving cardiac output in patients with mitral regurgitation. Otherwise, intravenous sodium nitroprusside or hydralazine can be attempted to lower afterload, or a diuretic can be used to reduce pulmonary edema if hypotension is not prohibitive.

In patients with chronic severe mitral regurgitation, the clinical status may remain stable, and many experts advocate a strategy of "watchful waiting." In general, the active patient may be monitored for development of exertional symptoms. However, even in the absence of symptoms, patients can develop occult LV systolic dysfunction. Therefore, periodic echocardiographic surveillance in asymptomatic patients is critical to prevent irreversible systolic dysfunction.

When a patient develops exertional symptoms or congestive heart failure, providing there has not already been a large decline