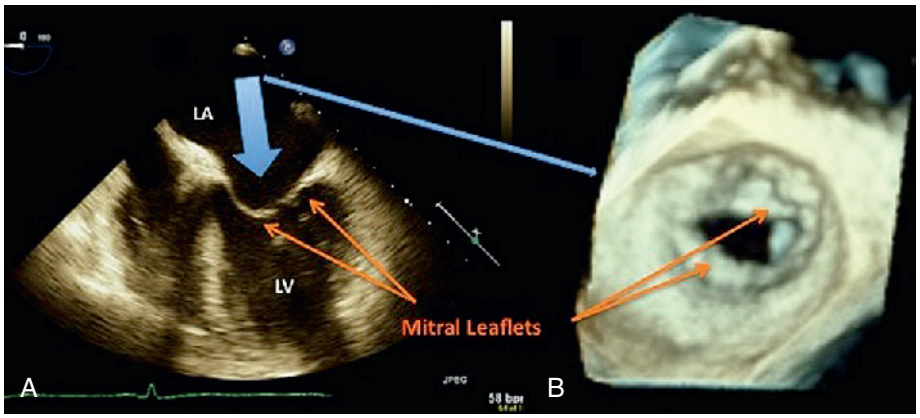

FIGURE 7-6 Abnormal motion of the mitral valve in rheumatic mitral stenosis. **A**, Transesophageal echocardiographic (TEE) image demonstrates the abnormal doming motion of the mitral leaflets in diastole. The *blue arrow* denotes the orientation of image **B**. **B,** Three-dimensional TEE image demonstrates the appearance of the mitral opening if viewed looking down at the valve from inside the left atrium (LA), a surgeon's orientation to the valve. The area of the mitral opening between anterior and posterior leaflets is well visualized, and the area measured from this view is used to estimate stenosis severity. LV, Left ventricle.

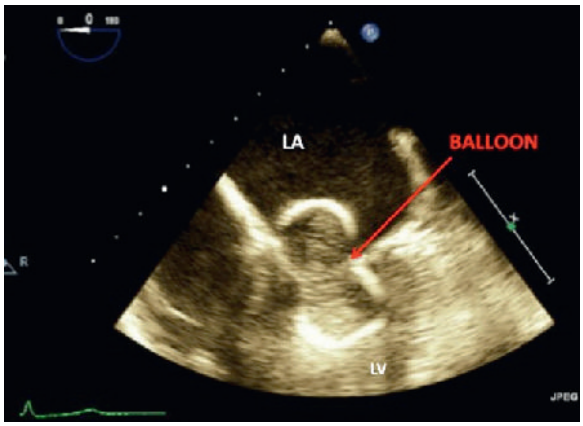

FIGURE 7-7 Balloon inflation during percutaneous balloon mitral valvuloplasty for treatment of mitral stenosis. Orientation is the same as in Figure 7-6A, and the inflated balloon is visible between the leaflets. LA, Left atrium; LV, left ventricle.

is primarily an anatomic problem, there is a limited role for medical therapy in improving survival. However, diuretics and heart rate slowing can be very helpful in controlling symptoms while a definitive mechanical solution to the problem is sought. Anticoagulation is key to prevent thromboembolism in any patient with a previous event or with onset of atrial fibrillation.

Prognosis

Although survival in asymptomatic patients is good, onset of symptoms heralds a rise in risk. Given the physiology of the disease, these risks understandably include heart failure, thromboembolism, and death. One study suggested a 5-year survival rate of only 44% in symptomatic patients who declined treatment.

MITRAL REGURGITATION

Definition

Mitral leaflet coaptation in systole should produce a tight seal to prevent regurgitation. This mechanism can fail for a variety of reasons, resulting in a problem for the left ventricle similar to that caused by aortic regurgitation—both being states of volume overload. Severe mitral regurgitation is also similar to aortic regurgitation in that its hemodynamic and clinical significance depend on the rapidity of onset.

Pathology

In considering the causes of mitral regurgitation, it is important to understand the anatomic components, which act collectively to provide valvular competence. Correct function of this system starts at the LV wall where the papillary muscles attach. A system of primary, secondary, and tertiary chordae provides the anatomic and structural attachments between the papillary muscles and the mitral leaflets. The leaflets, in turn, are attached at their circumference to the annulus. When one or more of these components are defective, regurgitation occurs. Given the complexity of this system, it is useful to divide major causes into valve leaflet abnormalities (i.e., primary or organic etiology) and causes related to structures other than the leaflets (i.e., secondary or functional etiology) (see Table 7-1).

Valvular etiologies include mitral valve prolapse (Fig. 7-8) and rheumatic valvular disease. When the LV wall fails to contract (after myocardial infarction) or the mitral annulus is enlarged, the normal valve leaflets are prevented from coapting properly, resulting in functional regurgitation.

Some causes, such as endocarditis of the valve leaflet or rupture of valve chordae, can occur suddenly and result in acute severe mitral regurgitation. Like aortic regurgitation, this condition is poorly tolerated medically and can be life-threatening without surgical intervention. In contrast, mitral valve prolapse can result in slow progression to chronic severe mitral regurgitation.

Acute severe mitral regurgitation results in a sudden increase in left atrial pressure. Given that a significant portion of the LV stroke volume then passes into the low-impedance left atrium during systole, cardiac output drops. The resulting pulmonary edema and hypotension often become life-threatening if the progression is not interrupted.