Treatment

Acute Severe Regurgitation

The mainstay of management in the patient with cardiogenic shock consists of attempts at medical stabilization through afterload reduction while preparing for urgent surgery. Death due to pulmonary edema, ventricular arrhythmias, or hemodynamic collapse is well described, and surgery is the established standard of care for these severely ill patients.

Drugs such as intravenous nitroprusside can be useful to rapidly achieve afterload reduction and improve cardiac output while the patient is prepared for urgent surgery. Diuretics may be used simultaneously to improve pulmonary edema. β-Blockers, although useful with aortic dissection, can cause further hemodynamic deterioration when acute severe regurgitation accompanies the dissection.

Chronic Severe Regurgitation

Patients may tolerate this lesion well due to compensatory mechanisms, remaining asymptomatic for many years. Although one study suggested that antihypertensive therapy with a dihydropyridine calcium blocker delays the need for surgery, a more recent study called into question the efficacy of either calcium blocker or angiotensin-converting enzyme (ACE) inhibitor therapy. These antihypertensives are considered a class IIb indication (level B evidence) for use in cases of asymptomatic severe aortic regurgitation with LV enlargement and normal systolic function. No studies of level A evidence have been completed to evaluate indications for AVR. Level B and C evidence indicates that AVR should be recommended based on the development of symptoms or asymptomatic structural changes in the heart. Specifically, a decrease in LV ejection fraction to 50% or less, even in an asymptomatic patient, is considered a class I indication for surgery. Similarly, marked diastolic (>75 mm) or systolic (>55 mm) LV enlargement, despite an absence of symptoms, is a class IIa indication for AVR. Finally, AVR has a class I indication in symptomatic patients, regardless of the status of their LV systolic function.

Options for prosthetic valve selection are similar to those for aortic stenosis, with the exception that a percutaneous method of valve replacement has not been approved by the FDA for this indication.

Prognosis

Acute Severe Regurgitation

Any cardiac surgery that has to be performed urgently entails greater surgical risk. When the cause of aortic regurgitation is infectious endocarditis, the long-term mortality rate, even with surgery, can be as high as 50%.

Chronic Severe Regurgitation

Close monitoring of patients for the evolution of surgical indications, as described earlier, leads to an excellent prognosis with acceptably low surgical mortality rates and survival curves that approach those of an otherwise normal population.

MITRAL STENOSIS

Definition

When the mitral leaflets open in diastole, they permit the entire stroke volume to pass from the atria to the left ventricle at relatively low pressure gradients. If mitral leaflet motion becomes restricted in opening, resistance to flow develops. The resulting severity of stenosis can be described by the pressure gradient that develops between the left atrium and left ventricle during diastole or by the size of the mitral valve opening.

Pathology

Both leaflet- and non–leaflet-related causes of stenosis can occur. Although restricted mitral leaflet motion due to rheumatic heart disease is by far the most common cause, immune disease affecting the valve and congenital abnormalities can also result in stenosis. Mitral inflow stenosis can also occasionally occur from severe calcification around the mitral annulus. A left atrial myxoma may lodge persistently or intermittently in the mitral annulus, resulting in obstruction to ventricular inflow. Finally, mitral stenosis can be the inadvertent outcome of surgical mitral valve repair or replacement.

Regardless of the etiology, the symptoms of mitral stenosis develop as left atrial pressure increases in response to forward flow limitation. As this increased pressure is reflected back to the lungs, pulmonary congestion evolves and, if severe enough, ultimately leads to pulmonary edema. The resulting pulmonary hypertension eventually results in right ventricular (RV) failure.

The mean gradient between the left atrium and left ventricle, measured at the valve orifice during diastole, is the most common way of describing the stenosis severity. The normal mitral valve area is 4 to 5 cm^2, and a normal gradient is less than 2 mm Hg. Although symptoms typically do not develop until the valve area is reduced to less than 2.5 cm^2, both cardiac output and heart rate can significantly affect the onset of symptoms for any particular degree of stenosis. Increased cardiac output, with a resulting increase in gradient, can lead to symptoms in a previously asymptomatic patient without change in mitral valve area (Fig. 7-5). Pregnancy is an example of a physiologic state of increased cardiac output that may produce symptoms in someone who was previously asymptomatic without any change in valve area. Shortening of the diastolic filling period after onset of a tachyarrhythmia is another reason that symptoms may suddenly develop without any anatomic change in valve size.

Rheumatic Valve Disease

In some patients who develop group A streptococcal pharyngitis, an abnormal immune response results in rheumatic fever, which can occur anywhere from 10 days to 3 weeks after the initial infection if left untreated. It typically occurs in children 6 to 15 years of age, with clinical manifestations leading to a diagnosis as summarized in the revised Jones Criteria (Table 7-4). The diagnosis is made based on the presence of two major or one major and two minor Jones criteria occurring after a recent documented group A streptococcal infection.

The pancarditis that occurs affects the pericardium and the myocardium as well as the valve tissue. The inflammation eventually leads to chordal thickening, shortening, and together with