in LV systolic function, mitral repair or replacement surgery is considered a class I indication (level B evidence). When an asymptomatic patient develops an LV ejection fraction of 60% or less, surgery is again a class I level B recommendation. When patients with chronic severe mitral regurgitation develop new-onset atrial fibrillation or confirmed significant pulmonary hypertension, surgery is advised with a class IIa recommendation (level C evidence). There are no medications that have been confirmed as able to alter the natural history of chronic severe mitral regurgitation, and no specific medical therapy in the absence of hypertension is recommended.

Prognosis

Rarely, patients with acute severe mitral regurgitation rapidly improve and enter a prolonged compensated phase in which watchful waiting is appropriate. However, most patients require urgent surgical intervention for survival. In chronic severe mitral regurgitation, it has been demonstrated that a program of watchful waiting, if diligently followed for appearance of a surgical indication as described, leads to excellent patient outcomes. However, failure to act when surgical criteria are met clearly leads to increased risk of morbidity and mortality.

TRICUSPID VALVE DISEASE

Definition

Anatomic requirements for tricuspid competence are similar to those described for the mitral valve: A complex of valve tissue and supportive structures must work in concert to maintain competence. With the tricuspid valve, however, three leaflets must come together appropriately to prevent regurgitation.

Tricuspid valve stenosis is similar to mitral stenosis in that the restriction of leaflet motion results in obstruction of right atrial emptying in diastole. The degree of stenosis determines the onset and severity of symptoms.

Pathology

Tricuspid stenosis in adults most often occurs as a result of rheumatic heart disease, which is uncommon in developed countries. Tricuspid valve involvement is less common than mitral and aortic valve involvement in rheumatic disease, and it almost never occurs without the mitral valve also being affected. Congenital abnormalities resulting in stenosis occur but are typically identified in childhood. Large leaflet vegetations and carcinoid disease may rarely cause stenosis, as may orifice obstruction from a large myxoma.

Tricuspid regurgitation can result from leaflet abnormalities (primary) or from another pathology affecting the right ventricle (functional). Functional tricuspid regurgitation is more common clinically; it occurs in patients with significant pulmonary hypertension, often caused by left-sided heart disease or lung disease. RV enlargement resulting from an atrial septal defect or anomalous pulmonary venous return can lead to severe functional tricuspid regurgitation. Primary leaflet abnormalities can occur from infectious endocarditis, carcinoid, or blunt chest trauma or iatrogenically from pacemaker or defibrillator leads (see Table 7-1).

Clinical Presentation

Because patients with tricuspid stenosis most often have mitral or aortic valve disease, or both, it is difficult to differentiate those symptoms that are solely caused by tricuspid disease. Symptoms of isolated tricuspid stenosis are similar to those of right-sided heart failure, although RV function is typically normal. These symptoms include edema and ascites as well as fatigue and dyspnea from low cardiac output.

Tricuspid regurgitation similarly results in heart failure symptoms of peripheral edema and low cardiac output that can include fatigue and exertional dyspnea. As RV systolic function deteriorates over time, symptoms progress and become more challenging to treat. Eventually, bowel edema results in decreased appetite and decreased absorption of oral diuretics, leading to malnutrition and a downward clinical spiral. As in tricuspid stenosis, the enlarging right atrium can precipitate atrial arrhythmias that further confound therapy.

Diagnosis

Physical examination for tricuspid stenosis is confounded by findings from left-sided valve disease in most cases. In normal sinus rhythm, an atrium contracting against a restricted tricuspid orifice can result in a prominent *a* wave on inspection of jugular venous pulsations. An opening snap may be audible. A soft diastolic-flow rumble may be identified by placing the bell of a stethoscope at the right parasternal border but may be inaudible. The key to distinguishing murmurs of right-sided origin is the respiratory variation in intensity, which augments with inspiration. Chest radiography may demonstrate right atrial enlargement, but there is no RV or pulmonary artery enlargement or prominence (see Table 7-2). A normal tricuspid valve has an area of 4 to 5 cm^2. Severe stenosis is thought to be present when the area is reduced to less than 1 cm^2 or the mean tricuspid valve gradient is 7 mm Hg or greater. Right heart catheterization is possible but is rarely needed to confirm the diagnosis of tricuspid stenosis.

The holosystolic murmur of tricuspid regurgitation can be soft or absent, and the diagnosis may go unsuspected. When present, the murmur is typically loudest at the left sternal border and increases with inspiration. Inspection of the jugular veins may demonstrate a characteristic *v* wave. Hepatic enlargement, with a pulsatile liver in systole, may be felt. Right atrial and ventricular enlargement may be seen on a chest radiograph.

TTE is the gold standard imaging modality for identifying the presence, cause, and severity of both tricuspid stenosis and tricuspid regurgitation. If the right heart cannot be adequately visualized by this technique, TEE or magnetic resonance imaging (MRI) can be helpful.

Treatment

There is no effective medical treatment for symptomatic isolated severe tricuspid valve stenosis. Diuretics typically reduce the cardiac output further, improving fluid status at the cost of worsening fatigue and dyspnea. Although percutaneous valvuloplasty of the tricuspid valve has been described, patients do poorly if there is resulting significant regurgitation. Although there are no studies to confirm optimal treatment of isolated symptomatic