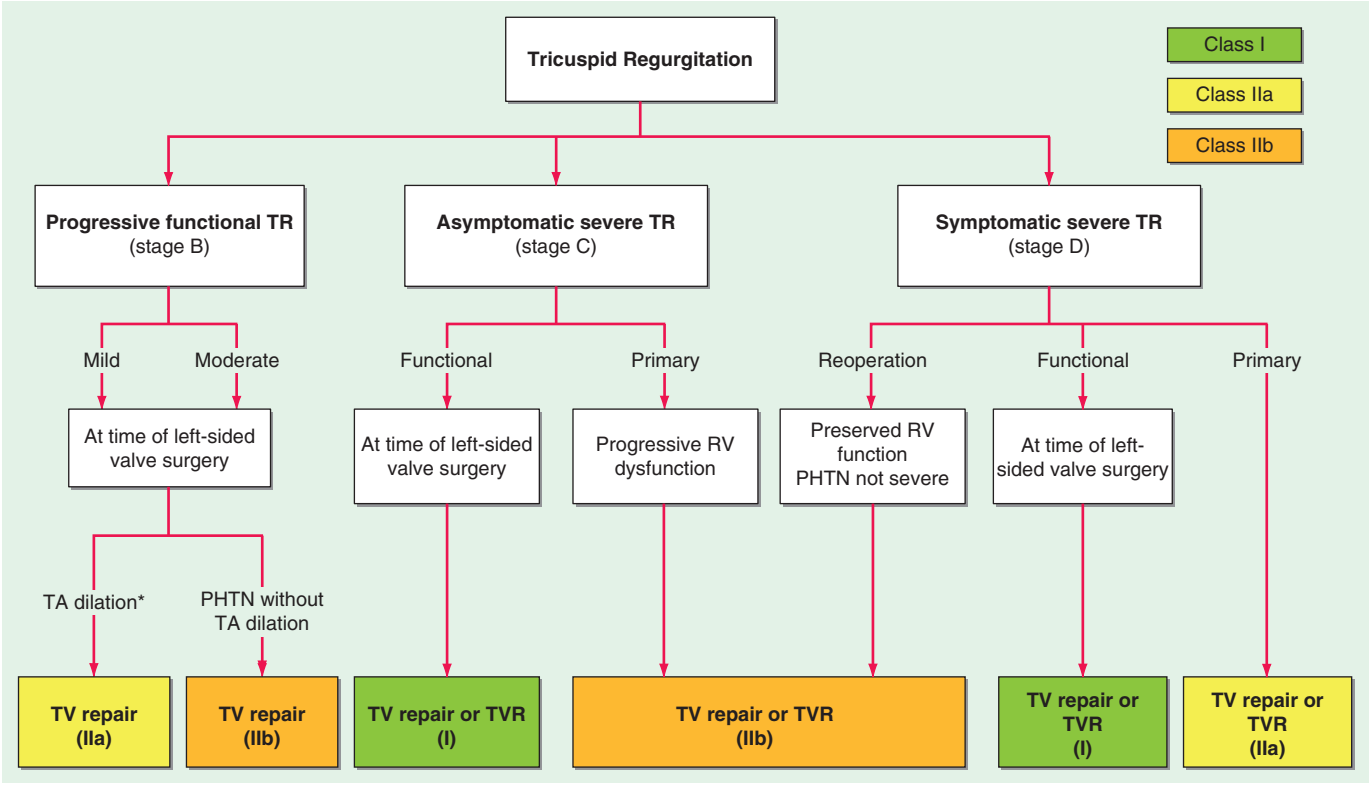

FIGURE 285-1 Management of tricuspid regurgitation. See legend for Fig. 283-2 for explanation of treatment recommendations (Class I, IIa, IIb) and disease stages (B, C, D). Preoperative coronary angiography should be performed routinely as determined by age, symptoms, and coronary risk factors. Cardiac catheterization and angiography may also be helpful when there is a discrepancy between clinical and noninvasive findings. PHTN, pulmonary hypertension; RV, right ventricular; TA, tricuspid annular; TTE, transthoracic echocardiogram; TR, tricuspid regurgitation; TV, tricuspid valve; TVR, tricuspid valve replacement. *TA dilation is defined by >40 mm on TTE (>21 mm/m2) or >70 mm on direct intraoperative measurement. *(Adapted from RA Nishimura et al: 2014 AHA/ACC Guideline for the Management of Patients with Valvular Heart Disease. J Am Coll Cardiol doi: 10.1016/j.jacc.2014.02.536, 2014, with permission.)*

left-sided valve surgery and is also undertaken frequently for treatment of even moderate TR in patients undergoing left-sided valve surgery who have tricuspid annular dilation (>40 mm), a history of right heart failure, or PA hypertension. Operation most often comprises repair rather than replacement in these settings and has become routine in most major surgical centers. Surgery may also infrequently be required for treatment of severe, primary TR with right heart failure not responsive to standard medical therapy or because of progressively declining RV systolic function. Reported perioperative mortality rates for isolated tricuspid valve surgery (repair and replacement) are high (~8-9%) and likely are influenced by the hazards encountered during reoperation on patients who have undergone previous left-sided valve surgery and have reduced RV function. Indwelling pacemaker or defibrillator leads can also pose technical challenges.

PULMONIC STENOSIS

Pulmonic valve stenosis (PS) is essentially a congenital disorder (Table 285-1). With isolated PS, the valve is typically domed. Dysplastic pulmonic valves are seen as part of the Noonan's syndrome (Chap. 302), which maps to chromosome 12. Much less common etiologies include carcinoid and obstructing tumors or bulky vegetations. The pulmonic valve is only very rarely affected by the rheumatic process.

PATHOPHYSIOLOGY

PS is defined hemodynamically by a systolic pressure gradient between the RV and main PA. RV hypertrophy develops as a consequence of sustained obstruction to RV outflow, and systolic ejection is prolonged. Compared with the ability of the LV to compensate for the pressure overload imposed by aortic stenosis (AS), RV dysfunction from afterload mismatch occurs earlier in the course of PS and at lower peak systolic pressures, because the RV adapts less well to this type of hemodynamic burden. With normal systolic function and CO, severe PS is defined by a peak systolic gradient across the pulmonic valve of >50 mmHg; moderate PS correlates with a peak gradient of 30–50 mmHg. PS rarely progresses in patients with peak gradients less than 30 mmHg, but may worsen in those with moderate disease due to valve thickening and calcification with age. The RA *a* wave elevates in relation to the higher pressures needed to fill a noncompliant, hypertrophied RV. A prominent RA *v* wave signifies functional TR from RV and annular dilation. The CO is maintained until late in the course of the disease.

SYMPTOMS

Patients with mild or even moderate PS are usually asymptomatic and first come to medical attention because of a heart murmur that leads to echocardiography. With severe PS, patients may report exertional dyspnea or early-onset fatigue. Anginal chest pain from RV oxygen supply-demand mismatch and syncope may occur with very severe forms of obstruction, particularly in the presence of a destabilizing trigger such as atrial fibrillation, fever, infection, or anemia.

PHYSICAL FINDINGS

The murmur of mild or moderate PS is mid-systolic in timing, crescendo-decrescendo in configuration, heard best in the left second interspace, and usually introduced by an ejection sound (click) in younger adults whose valves are still pliable. The ejection sound is the only right-sided acoustic event that decreases in intensity with inspiration. This phenomenon reflects premature opening of the pulmonic valve by the elevated RV end-diastolic (postatrial *a* wave) pressure. The systolic murmur increases in intensity during inspiration. With progressively severe PS, the ejection sound moves closer to the first heart sound and eventually becomes inaudible. A right-sided fourth heart sound may emerge. The systolic murmur peaks later and may persist through the aortic component of the second heart sound (A_2). Pulmonic valve