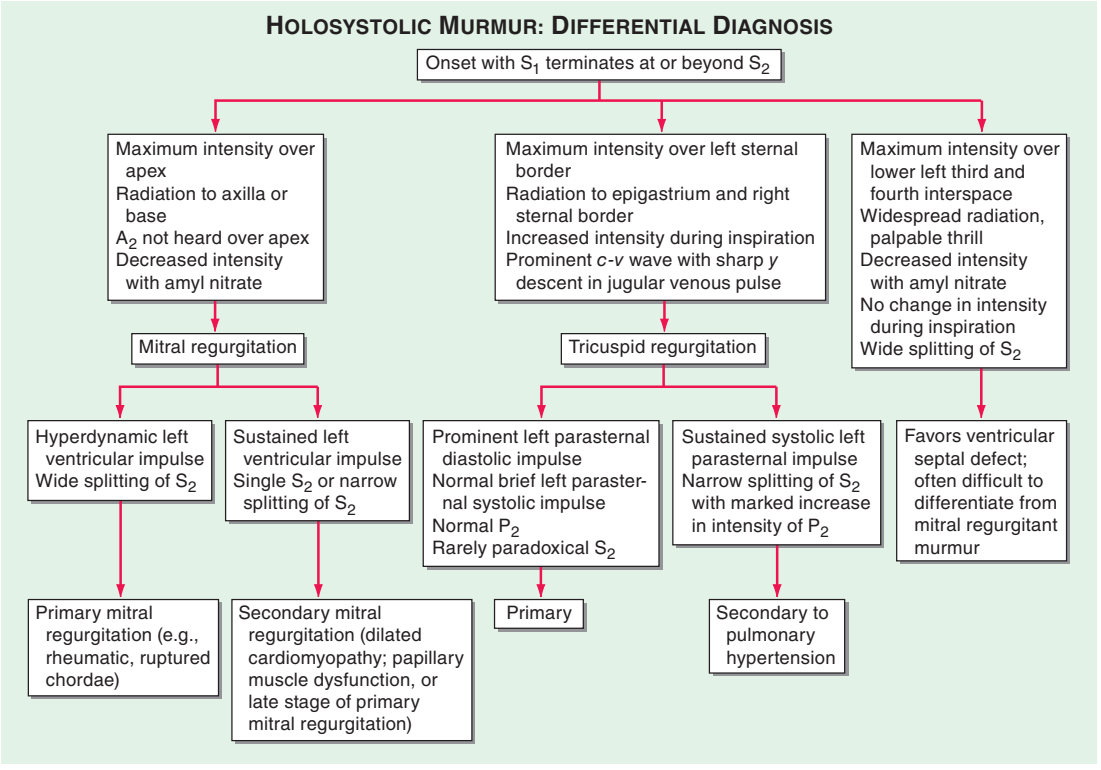

FIGURE 51e-5 **Differential diagnosis of a holosystolic murmur.**

absence of an audible murmur despite Doppler echocardiographic verification of TR. Causes of primary TR include myxomatous disease (prolapse), endocarditis, rheumatic disease, radiation, carcinoid, Ebstein's anomaly, and chordal detachment as a complication of right ventricular endomyocardial biopsy. TR is more commonly a passive process that results secondarily from annular enlargement due to right ventricular dilatation in the face of volume or pressure overload.

The holosystolic murmur of a VSD is loudest at the mid- to lower left sternal border (Fig. 51e-2) and radiates widely. A thrill is present at the site of maximal intensity in the majority of patients. There is no change in the intensity of the murmur with inspiration. The intensity of the murmur varies as a function of the anatomic size of the defect. Small, restrictive VSDs, as exemplified by the *maladie de Roger*, create a very loud murmur due to the significant and sustained systolic pressure gradient between the left and right ventricles. With large defects, the ventricular pressures tend to equalize, shunt flow is balanced, and a murmur is not appreciated. The distinction between post-MI ventricular septal rupture and MR has been reviewed previously.

DIASTOLIC HEART MURMURS

Early Diastolic Murmurs (Fig. 51e-1*E*) Chronic AR results in a high-pitched, blowing, decrescendo, early to mid-diastolic murmur that begins after the aortic component of S_2 (A_2) and is best heard at the second right interspace (Fig. 51e-6). The murmur may be soft and difficult to hear unless auscultation is performed with the patient leaning forward at end expiration. This maneuver brings the aortic root closer to the anterior chest wall. Radiation of the murmur may provide a clue to the cause of the AR. With primary valve disease, such as that due to congenital bicuspid disease, prolapse, or endocarditis, the diastolic murmur tends to radiate along the left sternal border, where it is often louder than appreciated in the second right interspace. When AR is caused by aortic root disease, the diastolic murmur may radiate along the right sternal border. Diseases of the aortic root cause dilation or distortion of the aortic annulus and failure of leaflet coaptation. Causes include Marfan syndrome with aneurysm formation, annuloaortic ectasia, ankylosing spondylitis, and aortic dissection.

Chronic, severe AR also may produce a lower-pitched mid to late, grade 1 or 2 diastolic murmur at the apex (Austin Flint murmur), which is thought to reflect turbulence at the mitral inflow area from the admixture of regurgitant (aortic) and forward (mitral) blood flow (Fig. 51e-1*G*). This lower-pitched, apical diastolic murmur can be distinguished from that due to MS by the absence of an opening snap and the response of the murmur to a vasodilator challenge. Lowering afterload with an agent such as amyl nitrite will decrease the duration and magnitude of the aortic–left ventricular diastolic pressure gradient, and thus, the Austin Flint murmur of severe AR will become shorter and softer. The intensity of the diastolic murmur of mitral stenosis (Fig. 51e-6) may either remain constant or increase with afterload reduction because of the reflex increase in cardiac output and mitral valve flow.

Although AS and AR may coexist, a grade 2 or 3 crescendo-decrescendo mid-systolic murmur frequently is heard at the base of the heart in patients with isolated, severe AR and is due to an increased volume and rate of systolic flow. Accurate bedside identification of coexistent AS can be difficult unless the carotid pulse examination is abnormal or the mid-systolic murmur is of grade 4 or greater intensity. In the absence of heart failure, chronic severe AR is accompanied by several peripheral signs of significant diastolic run-off, including a wide pulse pressure, a "water-hammer" carotid upstroke (Corrigan's pulse), and Quincke's pulsations of the nail beds. The diastolic murmur of *acute, severe AR* is notably shorter in duration and lower pitched than the murmur of chronic AR. It can be very difficult to appreciate in the presence of a rapid heart rate. These attributes reflect the abrupt rate of rise of diastolic pressure within the unprepared and noncompliant left ventricle and the correspondingly rapid decline in the aortic–left ventricular diastolic pressure gradient. Left ventricular diastolic pressure may increase sufficiently to result in premature closure of the mitral valve and a soft first heart sound. Peripheral signs of significant diastolic run-off are not present.

Pulmonic regurgitation (PR) results in a decrescendo, early to mid-diastolic murmur (*Graham Steell murmur*) that begins after the pulmonic component of S_2 (P_2), is best heard at the second left interspace, and radiates along the left sternal border. The intensity of the murmur may increase with inspiration. PR is most commonly due to dilation of the valve annulus from chronic elevation of the pulmonary artery pressure. Signs of pulmonary hypertension, including a right ventricular