can result in papillary muscle contusion and rupture, chordal detachment, or leaflet avulsion. TTE is indicated in all cases of suspected acute, severe MR to define its mechanism and severity, delineate left ventricular size and systolic function, and provide an assessment of suitability for primary valve repair.

A congenital, small muscular VSD (Chap. 282) may be associated with an early systolic murmur. The defect closes progressively during septal contraction, and thus, the murmur is confined to early systole. It is localized to the left sternal border (Fig. 51e-2) and is usually of grade 4 or 5 intensity. Signs of pulmonary hypertension or left ventricular volume overload are absent. Anatomically large and uncorrected VSDs, which usually involve the membranous portion of the septum, may lead to pulmonary hypertension. The murmur associated with the left-to-right shunt, which earlier may have been holosystolic, becomes limited to the first portion of systole as the elevated pulmonary vascular resistance leads to an abrupt rise in right ventricular pressure and an attenuation of the interventricular pressure gradient during the remainder of the cardiac cycle. In such instances, signs of pulmonary hypertension (right ventricular lift, loud and single or closely split S_2) may predominate. The murmur is best heard along the left sternal border but is softer. Suspicion of a VSD is an indication for TTE.

Tricuspid regurgitation (TR) with normal pulmonary artery pressures, as may occur with infective endocarditis, may produce an early systolic murmur. The murmur is soft (grade 1 or 2), is best heard at the lower left sternal border, and may increase in intensity with inspiration (Carvallo's sign). Regurgitant "*c-v*" waves may be visible in the jugular venous pulse. TR in this setting is not associated with signs of right heart failure.

Mid-Systolic Murmurs Mid-systolic murmurs begin at a short interval after S_1, end before S_2 (Fig. 51e-1*C*), and are usually crescendo-decrescendo in configuration. Aortic stenosis is the most common cause of a mid-systolic murmur in an adult. The murmur of AS is usually loudest to the right of the sternum in the second intercostal space (aortic area, Fig. 51e-2) and radiates into the carotids. Transmission of the mid-systolic murmur to the apex, where it becomes higher-pitched, is common (Gallavardin effect; see above).

Differentiation of this apical systolic murmur from MR can be difficult. The murmur of AS will increase in intensity, or become louder, in the beat after a premature beat, whereas the murmur of MR will have constant intensity from beat to beat. The intensity of the AS murmur also varies directly with the cardiac output. With a normal cardiac output, a systolic thrill and a grade 4 or higher murmur suggest severe AS. The murmur is softer in the setting of heart failure and low cardiac output. Other auscultatory findings of severe AS include a soft or absent A_2, paradoxical splitting of S_2, an apical S_4, and a late-peaking systolic murmur. In children, adolescents, and young adults with congenital valvular AS, an early ejection sound (click) is usually audible, more often along the left sternal border than at the base. Its presence signifies a flexible, noncalcified bicuspid valve (or one of its variants) and localizes the left ventricular outflow obstruction to the valvular (rather than sub- or supravalvular) level.

Assessment of the volume and rate of rise of the carotid pulse can provide additional information. A small and delayed upstroke (*parvus et tardus*) is consistent with severe AS. The carotid pulse examination is less discriminatory, however, in older patients with stiffened arteries. The electrocardiogram (ECG) shows signs of left ventricular hypertrophy (LVH) as the severity of the stenosis increases. TTE is indicated to assess the anatomic features of the aortic valve, the severity of the stenosis, left ventricular size, wall thickness and function, and the size and contour of the aortic root and proximal ascending aorta.

The obstructive form of hypertrophic cardiomyopathy (HOCM) is associated with a mid-systolic murmur that is usually loudest along the left sternal border or between the left lower sternal border and the apex (Chap. 287, Fig. 51e-2). The murmur is produced by both dynamic left ventricular outflow tract obstruction and MR, and thus, its configuration is a hybrid between ejection and regurgitant phenomena. The intensity of the murmur may vary from beat to beat and after provocative maneuvers but usually does not exceed grade 3. The murmur classically will increase in intensity with maneuvers that result in increasing degrees of outflow tract obstruction, such as a reduction in preload or afterload (Valsalva, standing, vasodilators), or with an augmentation of contractility (inotropic stimulation). Maneuvers that increase preload (squatting, passive leg raising, volume administration) or afterload (squatting, vasopressors) or that reduce contractility (β-adrenoreceptor blockers) decrease the intensity of the murmur. In rare patients, there may be reversed splitting of S_2. A sustained left ventricular apical impulse and an S_4 may be appreciated. In contrast to AS, the carotid upstroke is rapid and of normal volume. Rarely, it is bisferiens or bifid in contour (see Fig. 267-2*D*) due to mid-systolic closure of the aortic valve. LVH is present on the ECG, and the diagnosis is confirmed by TTE. Although the systolic murmur associated with MVP behaves similarly to that due to HOCM in response to the Valsalva maneuver and to standing/squatting (Fig. 51e-3), these two lesions can be distinguished on the basis of their associated findings, such as the presence of LVH in HOCM or a nonejection click in MVP.

The mid-systolic, crescendo-decrescendo murmur of congenital pulmonic stenosis (PS, Chap. 282) is best appreciated in the second and third left intercostal spaces (pulmonic area) (Figs. 51e-2 and 51e-4). The duration of the murmur lengthens and the intensity of P_2 diminishes with increasing degrees of valvular stenosis (Fig. 51e-1*D*). An early ejection sound, the intensity of which decreases with inspiration, is heard in younger patients. A parasternal lift and ECG evidence of right ventricular hypertrophy indicate severe pressure overload. If obtained, the chest x-ray may show poststenotic dilation of the main pulmonary artery. TTE is recommended for complete characterization.

Significant left-to-right intracardiac shunting due to an ASD (Chap. 282) leads to an increase in pulmonary blood flow and a grade 2–3 mid-systolic murmur at the middle to upper left sternal border

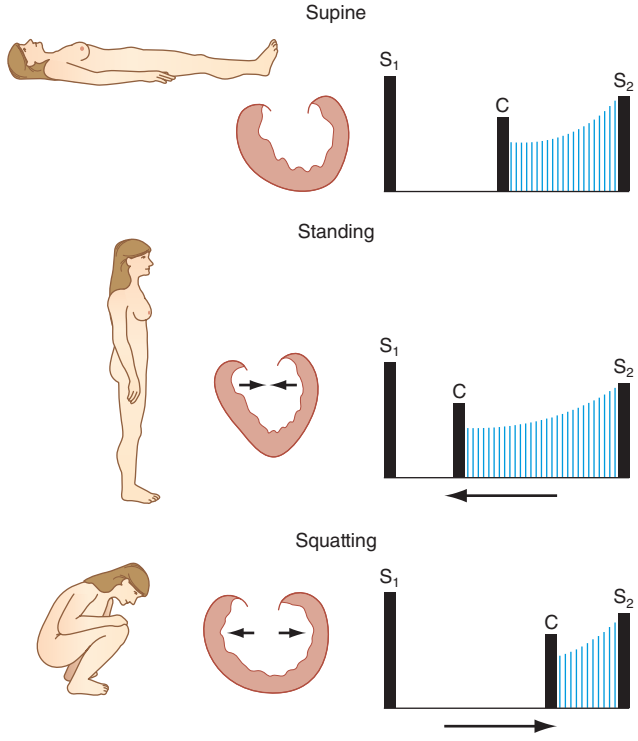

FIGURE 51e-3 A mid-systolic nonejection sound (C) occurs in mitral valve prolapse and is followed by a late systolic murmur that crescendos to the second heart sound (S_2). Standing decreases venous return; the heart becomes smaller; C moves closer to the first heart sound (S_1), and the mitral regurgitant murmur has an earlier onset. With prompt squatting, venous return and afterload increase; the heart becomes larger; C moves toward S_2; and the duration of the murmur shortens. *(From JA Shaver, JJ Leonard, DF Leon: Examination of the Heart, Part IV, Auscultation of the Heart. Dallas, American Heart Association, 1990, p 13. Copyright, American Heart Association.)*