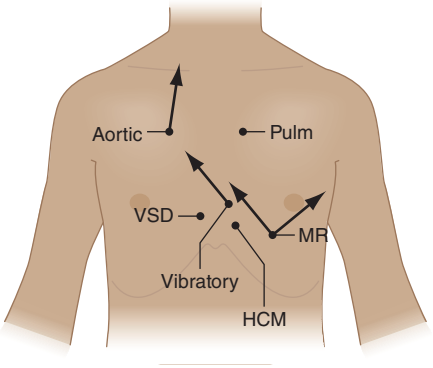

FIGURE 51e-2 Maximal intensity and radiation of six isolated systolic murmurs. HOCM, hypertrophic obstructive cardiomyopathy; MR, mitral regurgitation; Pulm, pulmonary stenosis; Aortic, aortic stenosis; VSD, ventricular septal defect. *(From JB Barlow: Perspectives on the Mitral Valve. Philadelphia, FA Davis, 1987, p 140.)*

during the respiratory cycle and the performance of simple bedside maneuvers complete the auscultatory examination. These features, along with recommendations for further testing, are discussed below in the context of specific systolic, diastolic, and continuous heart murmurs (Table 51e-1).

SYSTOLIC HEART MURMURS

Early Systolic Murmurs Early systolic murmurs begin with S_1 and extend for a variable period, ending well before S_2. Their causes are relatively few in number. *Acute, severe MR* into a normal-sized, relatively noncompliant left atrium results in an early, decrescendo systolic murmur best heard at or just medial to the apical impulse. These characteristics reflect the progressive attenuation of the pressure gradient between the left ventricle and the left atrium during systole owing to the rapid rise in left atrial pressure caused by the sudden volume load into an unprepared, noncompliant chamber and contrast sharply with the auscultatory features of chronic MR. Clinical settings in which acute, severe MR occur include (1) papillary muscle rupture complicating acute myocardial infarction (MI) (Chap. 295), (2) rupture of chordae tendineae in the setting of myxomatous mitral valve disease (MVP, Chap. 283), (3) infective endocarditis (Chap. 155), and (4) blunt chest wall trauma.

Acute, severe MR from papillary muscle rupture usually accompanies an inferior, posterior, or lateral MI and occurs 2–7 days after presentation. It often is signaled by chest pain, hypotension, and pulmonary edema, but a murmur may be absent in up to 50% of cases. The posteromedial papillary muscle is involved 6 to 10 times more frequently than the anterolateral papillary muscle. The murmur is to be distinguished from that associated with post-MI ventricular septal rupture, which is accompanied by a systolic thrill at the left sternal border in nearly all patients and is holosystolic in duration. A new heart murmur after an MI is an indication for transthoracic echocardiography (TTE) (Chap. 270e), which allows bedside delineation of its etiology and pathophysiologic significance. The distinction between acute MR and ventricular septal rupture also can be achieved with right heart catheterization, sequential determination of oxygen saturations, and analysis of the pressure waveforms (tall *v* wave in the pulmonary artery wedge pressure in MR). Post-MI mechanical complications of this nature mandate aggressive medical stabilization and prompt referral for surgical repair.

Spontaneous chordal rupture can complicate the course of myxomatous mitral valve disease (MVP) and result in new-onset or "acute on chronic" severe MR. MVP may occur as an isolated phenomenon, or the lesion may be part of a more generalized connective tissue disorder as seen, for example, in patients with Marfan syndrome. Acute, severe MR as a consequence of infective endocarditis results from destruction of leaflet tissue, chordal rupture, or both. Blunt chest wall trauma is usually self-evident but may be disarmingly trivial; it

TABLE 51e-1 PRINCIPAL CAUSES OF HEART MURMURS

Systolic Murmurs
Early systolic
Mitral
Acute MR
VSD
Muscular
Nonrestrictive with pulmonary hypertension
Tricuspid
TR with normal pulmonary artery pressure
Mid-systolic
Aortic
Obstructive
Supravalvular–supravalvular aortic stenosis, coarctation of the aorta
Valvular–AS and aortic sclerosis
Subvalvular–discrete, tunnel or HOCM
Increased flow, hyperkinetic states, AR, complete heart block
Dilation of ascending aorta, atheroma, aortitis
Pulmonary
Obstructive
Supravalvular–pulmonary artery stenosis
Valvular–pulmonic valve stenosis
Subvalvular–infundibular stenosis (dynamic)
Increased flow, hyperkinetic states, left-to-right shunt (e.g., ASD)
Dilation of pulmonary artery
Late systolic
Mitral
MVP, acute myocardial ischemia
Tricuspid
TVP
Holosystolic
Atrioventricular valve regurgitation (MR, TR)
Left-to-right shunt at ventricular level (VSD)
Early Diastolic Murmurs
Aortic regurgitation
Valvular: congenital (bicuspid valve), rheumatic deformity, endocarditis, prolapse, trauma, post-valvulotomy
Dilation of valve ring: aorta dissection, annuloaortic ectasia, cystic medial degeneration, hypertension, ankylosing spondylitis
Widening of commissures: syphilis
Pulmonic regurgitation
Valvular: post-valvulotomy, endocarditis, rheumatic fever, carcinoid
Dilation of valve ring: pulmonary hypertension; Marfan syndrome
Congenital: isolated or associated with tetralogy of Fallot, VSD, pulmonic stenosis
Mid-Diastolic Murmurs
Mitral
Mitral stenosis
Carey-Coombs murmur (mid-diastolic apical murmur in acute rheumatic fever)
Increased flow across nonstenotic mitral valve (e.g., MR, VSD, PDA, high-output states, and complete heart block)
Tricuspid
Tricuspid stenosis
Increased flow across nonstenotic tricuspid valve (e.g., TR, ASD, and anomalous pulmonary venous return)
Left and right atrial tumors (myxoma)
Severe AR (Austin Flint murmur)

Continuous Murmurs	
Patent ductus arteriosus	Proximal coronary artery stenosis
Coronary AV fistula	Mammary souffle of pregnancy
Ruptured sinus of Valsalva aneurysm	Pulmonary artery branch stenosis
Aortic septal defect	Bronchial collateral circulation
Cervical venous hum	Small (restrictive) ASD with MS
Anomalous left coronary artery	Intercostal AV fistula

Abbreviations: AR, aortic regurgitation; AS, aortic stenosis; ASD, atrial septal defect; AV, arteriovenous; HOCM, hypertrophic obstructive cardiomyopathy; MR, mitral regurgitation; MS, mitral stenosis; MVP, mitral valve prolapse; PDA, patent ductus arteriosus; TR, tricuspid regurgitation; TVP, tricuspid valve prolapse; VSD, ventricular septal defect.

Source: E Braunwald, JK Perloff, in D Zipes et al (eds): *Braunwald's Heart Disease,* 7th ed. Philadelphia, Elsevier, 2005; PJ Norton, RA O'Rourke, in E Braunwald, L Goldman (eds): *Primary Cardiology,* 2nd ed. Philadelphia, Elsevier, 2003.