

viding diagnostic insights into the pathophysiologic mechanisms underlying the acoustic findings heard in clinical practice.¹⁶²⁻¹⁶⁵ Contemporary physicians should take advantage of the valuable clinical information that can be obtained by such an inexpensive instrument and expedient and reliable tool as the stethoscope. The following section reviews the fundamental technique of cardiac auscultation, emphasizing the diagnostic value and practical clinical applications of this time-honored (but endangered) art in this time of need.¹⁶⁶

The Art and Technique of Cardiac Auscultation. Auscultation of the heart and vascular system is one of the most challenging and rewarding clinical diagnostic skills that can (and should) be learned and applied by every practicing physician. Proficiency in cardiac auscultation requires experience, repeated practice, and a great deal of patience (and patients). Most importantly, it requires a proper state of mind. (“we hear what we listen for”). Although the most vital component of the auscultatory apparatus lies between the earpieces, the proper use of a well-designed, efficient stethoscope cannot be overemphasized. To ensure optimal sound transmission, the well-crafted stethoscope should be airtight, with snug but comfortably-fitting earpieces, properly aligned metal binaurals, and flexible, double-barrel, thick-walled tubing, ⅜ inch in internal diameter and no more than 12 to 15 inches in length. A high-quality stethoscope should be equipped with both bell and diaphragm chest pieces. The bell, when applied gently to the skin, will “bring out” low frequency sounds and murmurs (eg, faint S₄ or S₃ gallop or diastolic rumble) and the diaphragm, when pressed firmly against the skin, will accentuate high-pitched acoustic events (eg, diastolic blowing murmur of AR). A unique triple-headed (Harvey) stethoscope has a third chest piece, the corrugated diaphragm, designed to amplify sound and provide an overview of the auscultatory findings heard. (Figure 14)

For maximum auscultatory yield, the examination should be conducted in a quiet, adequately lit room, with the patient comfortable and properly gowned to avoid embarrassment, yet with the precordium sufficiently exposed to enable adequate inspection, palpation, and auscultation. As a rule, most physicians conduct the examination from the right side of the patient and begin with the patient reclining in the supine position. A few moments taken by the physician to warm the stethoscope will be time well spent. The examiner then proceeds to listen over the second right intercostal space (aortic area) and slowly moves (“inches”) the stethoscope across to the second left intercostal space (pulmonic area), downward along the left sternal edge to the lower left sternal border (tricuspid area), and then laterally to the cardiac apex (mitral area), using both the diaphragm and bell chest pieces. Although some physicians prefer to

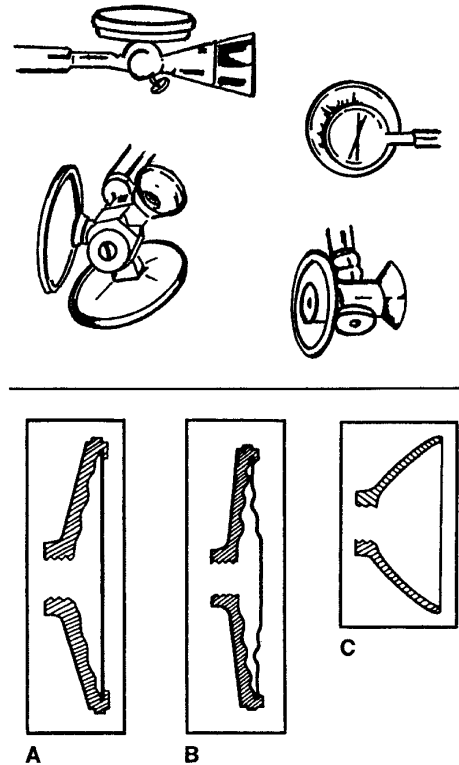


FIG 14. Composite of many types of double head and triple head (Harvey) stethoscopes (top) and stethoscope chest pieces (bottom). **A**, Flat diaphragm, excellent for all sounds and murmurs. Best for higher frequencies (eg, faint diastolic murmur of aortic regurgitation, splitting of heart sounds, systolic clicks, ejection sounds). **B**, Corrugated diaphragm, amplifying quality. Excellent for an “overview” of heart sounds, especially low frequency gallop sounds and murmurs. **C**, Bell, particularly useful in detecting faint, low frequency heart sounds and murmurs (eg, gallop sounds, diastolic rumble). (From Harvey WP. *Cardiac pearls*. Newton (NJ): Laennec; 1993. p. 217. With permission.)

reverse this sequence, it is important for the examiner to adopt a systematic method of auscultation that becomes automatic, beginning in one area and then carefully exploring all areas in an orderly and unhurried fashion, so that nothing is overlooked (Figure 15).^{167,168}

Next, the patient is turned to the left lateral decubitus position while the physician “tunes in” to low-frequency sounds and murmurs (eg, S_4 and S_3 gallops, diastolic rumble of mitral stenosis), using the bell of the stethoscope applied lightly to the chest wall at the cardiac apex barely making an air seal (Figure 16). Light pressure is essential because heavy pressure stretches the skin and converts the bell into a diaphragm, which thereby diminishes or eliminates (“filters out”) these low-frequency events. Then,

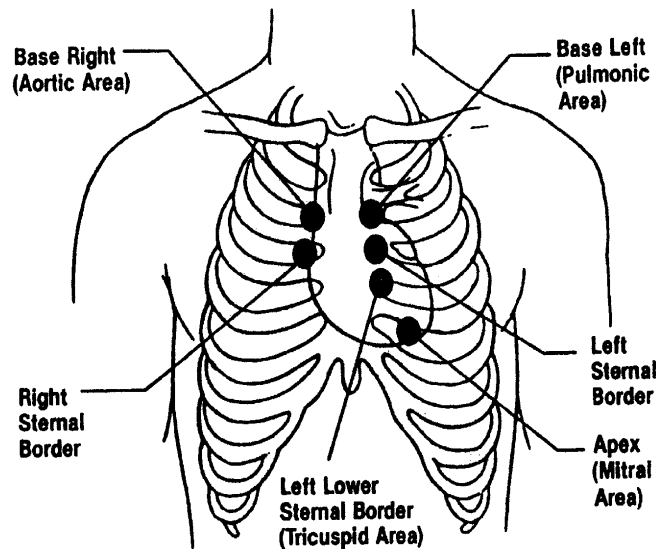


FIG 15. Sites of auscultation. These are the specific sites on the chest wall principally used for cardiac auscultation. See text for details. (Adapted from Harvey WP. *Cardiac Pearls*. Newton (NJ): Laennec; 1993. p. 20. With permission.)

with the patient in the sitting position, leaning forward with the breath held in deep expiration, the examiner listens over the base or left sternal border, to detect high-pitched sounds and murmurs (eg, diastolic murmur of AR, pulmonary hypertensive regurgitation [Graham Steell murmur], pericardial friction rub), using the diaphragm chest piece pressed firmly enough against the chest wall to leave a temporary imprint (after-ring) on the skin (Figure 17). In the presence of chronic obstructive pulmonary disease, the examiner should listen over the xiphoid or upper epigastric region, while the patient is in the upright position, to avoid interference by emphysematous lung tissue.

In clinical practice, it is customary to listen for specific heart sounds and murmurs over the traditional so-called valve areas, which are points over the precordium where events originating in each heart valve are best transmitted and heard. Sounds and murmurs of the aortic valve and aorta, for example, are well heard at the second right intercostal space (aortic area). Those from the pulmonic valve and pulmonary artery are usually best heard at the second left intercostal space (pulmonic area) or third (mid) left sternal border. The mid left sternal border is usually the best site to detect the diastolic murmur of AR. The lower left sternal border (tricuspid area) is the customary location for evaluation of the first heart sound, systolic clicks,

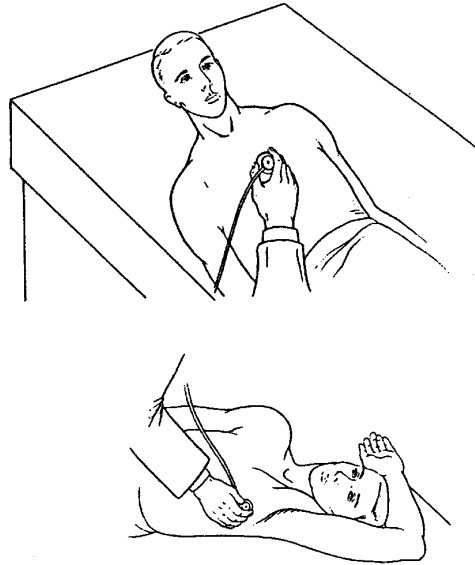


FIG 16. The patient should be examined in the semirecumbent position and gowned so that the precordium is easily and sufficiently exposed. During the course of the examination, a postural shift to the left lateral position may be required. Palpation of the left ventricle is then facilitated. The diastolic gallop and the rumble of mitral stenosis may become louder when the physician listens as the patient is turned. (From Segal BL. Clinical recognition of rheumatic heart disease. In Chizner MA, ed. Classic teachings in clinical cardiology: A tribute to W. Proctor Harvey, MD. Newton (NJ): Laennec; 1996. p. 994. With permission.)



FIG 17. The patient with aortic regurgitation should be examined in the sitting position. The high-frequency diastolic murmur is sometimes only heard in this position with the diaphragm of the stethoscope firmly applied to the chest wall after forced expiration. (From Segal BL. Clinical recognition of rheumatic heart disease. In Chizner MA, ed. Classic teachings in clinical cardiology: A tribute to W. Proctor Harvey, MD. Newton (NJ): Laennec; 1996. p. 1011. With permission.)

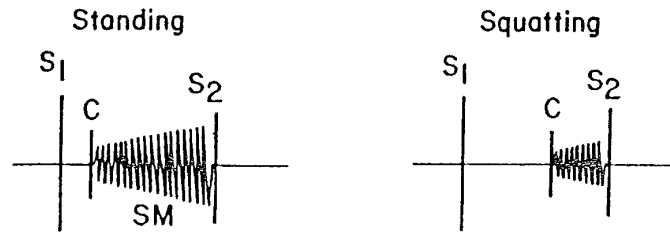


FIG 18. Schematic diagram showing the effects of standing and squatting on the mid-systolic click (C) and late-systolic murmur (SM) of MVP. Standing causes the click to move closer to the first heart sound (S₁) and the murmur to get longer and louder. Squatting causes the click to move closer to the second heart sound (S₂) and the murmur to get shorter and softer. (From de Leon AC Jr. A handbook on cardiac auscultation. Cedar Grove [NJ]: Laennec; 1989. With permission.)

right-sided S₄ and S₃ gallops, and tricuspid valve sounds and murmurs. The characteristic increase in intensity of the holosystolic murmur of TR with inspiration (Carvallo's sign) is best appreciated at this site. The holosystolic murmur of VSD, often accompanied by a palpable thrill, is also located over this area. The apex (mitral area) is usually best for identification of left-sided S₄ and S₃ gallops and murmurs of mitral valve origin. Aortic ejection sounds and murmurs, however, are often well heard in this location as well.¹⁶⁹

Important findings may also be present in other locations, such as the neck (transmitted systolic ejection murmur of valvular aortic stenosis, bruit of carotid arterial occlusive disease), clavicle (bone transmission of valvular aortic stenosis), supraclavicular fossa (continuous murmur of jugular venous hum heard in children, thyrotoxicosis, anemia, or pregnancy or in any hyperkinetic state), left axilla and posterior lung base ("band-like" radiation of holosystolic murmur of chronic MR), right sternal border (so-called right-sided diastolic murmur of AR [Harvey's sign] due to rightward displacement and aortic root pathology [eg, aortic dissection, aneurysm, Marfan's syndrome]), abdomen (bruit of renal artery stenosis), and over scars (continuous murmur of arteriovenous fistula, which can result in high-output heart failure). The site of maximal intensity and direction of transmission of certain heart sounds and murmurs therefore may prove to be of diagnostic value in the evaluation of their origin and clinical significance.

Changes in body position and bedside physiologic maneuvers (dynamic auscultation) may also be helpful adjuncts in enhancing the evaluation of specific acoustic events, such as the Valsalva maneuver, standing (increases systolic murmur of hypertrophic obstructive cardiomyopathy, earlier onset of systolic click, and longer and often louder murmur of MVP), and squatting (decreases systolic murmur of hypertrophic obstructive

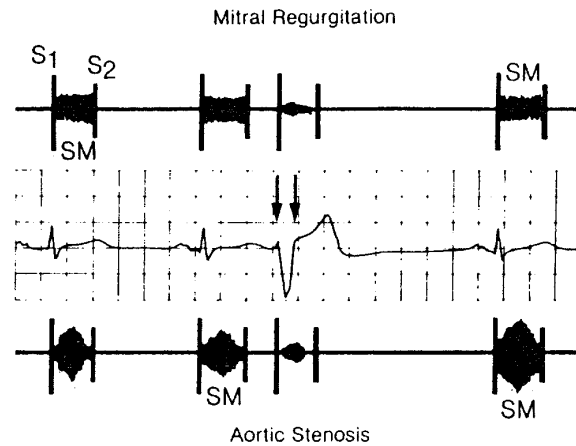


FIG 19. Systolic murmur (SM) of MR remains unchanged in intensity after pause. In contrast, systolic murmur of aortic stenosis is louder after pause following premature beat. (From Harvey WP. *Cardiac pearls*. Newton [NJ]: Laennec; 1993. With permission.)

tive cardiomyopathy, later onset of systolic click and murmur of MVP).¹⁷⁰⁻¹⁷³ As a practical clinical point, the squatting maneuver is ideally performed if the physician remains comfortably seated and listens for any subtle auscultatory changes while the patient squats and stands¹⁷⁴ (Figure 18). Paying careful attention to the effect of respiration on splitting and variation in intensity of heart sounds and murmurs, especially those generated within the right side of the heart, can be very useful as well and, therefore, should be listened for specifically. All right-sided heart sounds and murmurs (with the exception of the pulmonic ejection sound) should increase in intensity with inspiration. Isometric hand-grip exercise (or transient arterial occlusion of both arms with 2 blood pressure cuffs) increases the murmurs of MR, VSD, and AR and can also be of value. Alterations in cycle length as in atrial fibrillation or the long pause following a premature ventricular contraction are also helpful, when present, in the differentiation of murmurs at the bedside. The systolic ejection murmur of valvular aortic stenosis, for example, gets louder after the pause following shorter cycle lengths in atrial fibrillation or a premature ventricular beat. In contrast, the holosystolic murmur of MR remains essentially unchanged¹⁷⁵ (Figure 19). Although various pharmacologic interventions have also been used in the past to clarify the origin of heart murmurs (eg, amyl nitrite inhalation, which increases the systolic murmurs of valvular aortic stenosis/sclerosis and hypertrophic obstructive cardiomyopathy and decreases the murmurs of MR, VSD, and AR), their popularity has since waned.^{176,177}

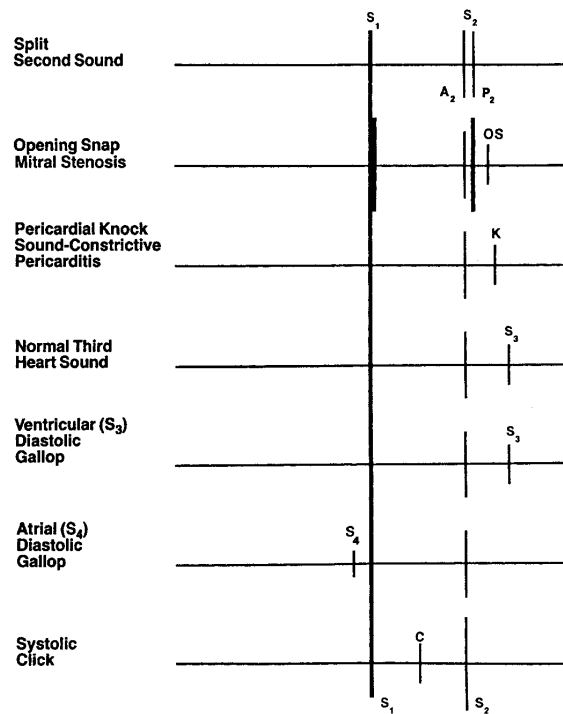


FIG 20. Comparison of timing of extra sounds and events. Note that S₁ and P₂ are accentuated with mitral stenosis. With constrictive pericarditis a pericardial knock sound (K) occurs later than the opening snap (OS) and S₁ is not accentuated. (From Harvey WP. *Cardiac pearls*. Newton (NJ): Laennec; 1993. p. 75. With permission.)

The key to successful auscultation is the accurate identification and timing of heart sounds and murmurs in the cardiac cycle (Figure 20). Proper timing can be achieved by using the classic “inching” technique. On the basis of the clinical fact that the second heart sound (S₂) is usually louder than the first heart sound (S₁) at the aortic area, by keeping S₂ in mind, as the stethoscope is moved (“inched”) down along the left sternal border to the apex, extra sounds or murmurs occurring before S₂ can be recognized as systolic, and after S₂ as diastolic. To detect and interpret these acoustic events correctly, one must develop the ability to listen selectively (“tune in”) to one specific aspect of the cardiac cycle at a time. While concentrating on S₁ first, the physician should pay special attention to intensity: faint S₁ (due to a long PR interval as in first degree AV block; weak ventricular contraction as in LV systolic dysfunction or heart failure, AMI, or dilated cardiomyopathy; and premature closure of the mitral valve as in acute severe AR), loud S₁ (due to a short PR interval; strong ventricular contraction as in hyperkinetic states; and delayed, high-velocity closure of a stiff but mobile mitral valve as in

rheumatic mitral stenosis), or varying S_1 (due to varying PR intervals as in complete heart block, VT, and changing cycle lengths in atrial fibrillation). After S_1 has been carefully appraised, S_2 is likewise concentrated on and analyzed with respect to intensity: loud S_2 (due to loud “tambour” A_2 in systemic hypertension or loud P_2 , heard even at the cardiac apex, in pulmonary hypertension), soft S_2 (due to decreased A_2 in severe calcific [immobile] aortic valve stenosis, faint and delayed P_2 in pulmonic stenosis), and effect of respiration on splitting—wide physiologic inspiratory splitting of S_2 (delay in P_2 in right bundle branch block, early A_2 in severe MR, VSD), wide so-called “fixed” splitting of S_2 (little or no respiratory variation in A_2 - P_2 as in atrial septal defect), or paradoxical splitting of S_2 (delay in A_2 with reversed split P_2 - A_2 on expiration, which narrows during inspiration as P_2 moves toward A_2 , as in left bundle branch block [LBBB], severe valvular aortic stenosis, and hypertrophic obstructive cardiomyopathy). Extra sounds in systole (aortic and pulmonic ejection sounds, as in congenital bicuspid aortic valve and pulmonic valve stenosis, and systolic clicks of MVP) and diastole (S_4 and S_3 gallops, opening snap of mitral stenosis, and pericardial knock sound of constrictive pericarditis) are then appraised.¹⁷⁸⁻¹⁸⁹ Then, the examiner concentrates and listens for murmurs in systole, murmurs in diastole, and continuous murmurs (eg, patent ductus arteriosus, jugular venous hum, arteriovenous fistula), if present.¹⁹⁰⁻¹⁹² Pericardial friction rubs may be mistaken for heart murmurs, but they can be differentiated by their rough, scratchy, superficial quality and by the fact that they have at least 2 or 3 components (systolic, diastolic, presystolic). They occur in patients with acute pericarditis, after AMI, heart surgery, or chest injury, and in association with uremia, malignancy, or connective tissue diseases.¹⁹³ An effective way to improve one’s auscultatory ability is to draw (“sketch”) the acoustic findings heard for every patient examined (Figure 21).

A heart murmur may be functional and have no pathologic significance (“innocent”), or it may be an important clue to the presence of underlying structural organic heart disease (“significant” or “guilty”).¹⁹⁴ Most heart murmurs are systolic and do not signify heart disease.¹⁹⁵ Many of these are related to physiologic increases in blood flow velocity. A large percentage of individuals with a systolic murmur therefore do not need referral to a specialist or further noninvasive cardiac evaluation. The most important task of the clinician is to determine if a given systolic murmur is significant or innocent. In the past few decades, the prevalence of diseases that cause “classic” heart murmurs has changed.^{196,197} In the United States, CAD and hypertensive heart disease and their complications, along with CHF (often secondary to dilated cardiomyopathy), now dominate the clinical practice of the majority of modern adult cardiologists.

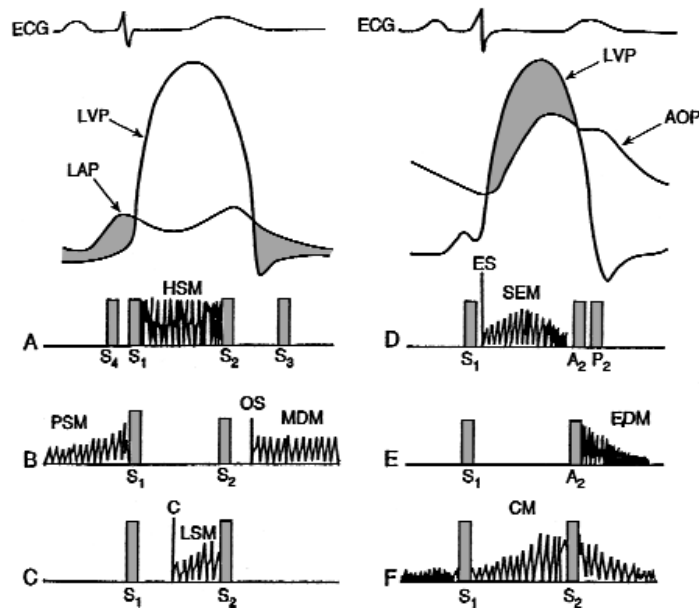


FIG 21. Intracardiac pressures and heart murmurs of the major cardiac valve abnormalities. See text for discussion of specific murmurs. *LVP*, LV pressure; *LAP*, left atrial pressure; *AOP*, aortic pressure; *HSM*, holosystolic murmur; *PSM*, presystolic murmur; *OS*, opening snap; *MDM*, mid-diastolic murmur; *C*, mid-systolic click; *LSM*, late-systolic murmur; *ES*, ejection sound; *SEM*, systolic ejection murmur; *EDM*, early-diastolic murmur; *CM*, continuous murmur. (From Crawford MH et al. *Curr Probl Cardiol* 1979;1:1. With permission.)

The major organic murmurs that must be considered include aortic sclerosis of elderly patients (which recently has been linked to atherosclerosis and an increased risk of AMI and stroke); valvular aortic stenosis, MR due to MVP, papillary muscle dysfunction (as seen in ischemia, AMI, cardiomyopathy, or LV failure from any cause), or calcified mitral annulus (especially in elderly women); and hypertrophic cardiomyopathy. Recent onset of a heart murmur may suggest infective endocarditis (eg, acute MR, AR) or a serious complication in a patient with AMI (eg, acute VSD, MR).

Careful analysis of a systolic murmur will enable the experienced listener to determine its duration (short to long), intensity or loudness (graded from 1 to 6 [Table 8]), quality (harsh, blowing, musical, etc), timing (early, mid, or late systolic, holosystolic), location and direction of radiation, and response to maneuvers (eg, squatting, Valsalva).¹⁹⁸⁻²⁰⁰ The same type of detailed analysis is conducted for diastolic and continuous murmurs. Early- and mid-systolic murmurs may be innocent or significant. Loud systolic murmurs (grade 3 or greater) are more likely to be hemodynamically significant and caused by underlying heart disease. Holosystolic, late-systolic,

TABLE 8. Grading of murmurs (Levine-Harvey)

Grade 1	Faintest murmur heard only with special effort (concentration or “tuning in” required)
Grade 2	Faint murmur but heard immediately
Grade 3	Moderately-loud murmur
Grade 4	Loud murmur associated with a palpable thrill
Grade 5	Very loud murmur heard with part of stethoscope touching chest wall
Grade 6	Loudest murmur heard with stethoscope removed from chest wall

and diastolic murmurs virtually always signify an abnormality of cardiac structure or function.^{201,202} The recognition that a systolic murmur is significant rather than innocent often rests on associated clinical findings (ie, the “company it keeps”), rather than on the characteristics of the murmur itself. The presence of symptoms suggestive of cardiovascular disease and/or associated abnormal cardiac physical (ECG and chest x-ray) findings may increase the likelihood that the murmur is significant (Table 9). Certain abnormal heart sounds that reflect cardiac pathology (eg, persistent [“fixed”] splitting of S_2 in atrial septal defect, ejection sound in congenital bicuspid aortic valve or pulmonic valvular stenosis, systolic click in MVP, loud “tambour” A_2 in systemic hypertension, loud, closely split, or single [fused] S_2 with an accentuation of P_2 in pulmonary hypertension) may all serve as immediate clues to the presence of a significant murmur. Unfortunately, it is easy to overlook the presence of these abnormal heart sounds unless one listens specifically for them. In contrast, the innocent systolic murmur, which can be found in children, young adults, athletes, and pregnant women and in those with a hyperkinetic circulatory state (as brought on by fever, anemia, exercise, excitement, hyperthyroidism), is a soft (grade 1-2/6), early- to mid-systolic, crescendo-decrescendo murmur, often with a musical, vibratory, or buzzing quality (Still’s murmur), heard best over the pulmonic area or mid left sternal border, but it may be heard at the apex and aortic areas as well. It is characteristically accompanied by normal respiratory splitting of S_2 , a physiologic S_3 at the cardiac apex waxing and waning with respiration, a jugular venous hum heard best with the bell placed over the right supraclavicular fossa with the head turned (“on a stretch”) to the opposite direction, and the absence of abnormal heart sounds (eg, ejection sounds, clicks, gallops) or other systolic or diastolic murmurs. When heard in the asymptomatic individual with normal ECG and chest x-ray findings (and no other clinical manifestations of heart disease), the innocent systolic murmur can be diagnosed with a high degree of certainty, without the use of more specialized (eg, echo) techniques (see below).²⁰³⁻²⁰⁷

A detailed discussion of the myriad heart sounds and murmurs that can be heard in daily practice is beyond the scope of this monograph. Some

TABLE 9. Clinical clues to significant (pathologic) systolic murmurs

Presence of clinical symptoms
Ischemic-type chest pain, CHF, syncope
Associated auscultatory findings
Loud in intensity (grade 3 or greater)
Long in duration (mid or late-peaking or holosystolic)
Prominent radiation to axilla or neck (carotids)
Changes in intensity during physiologic maneuvers (eg, Valsalva, squatting)
Associated diastolic murmur
Abnormal heart sounds
Loud S ₁ ; wide, "fixed" or paradoxical split S ₂ ; loud A ₂ or P ₂ , S ₄ or S ₃ gallops; mid-systolic click; aortic or pulmonic ejection sounds; opening snap; pericardial knock
Other physical examination findings
Abnormal jugular venous pulse
Elevated mean venous pressure
Large A or V waves
Abnormal arterial pulse/blood pressure
Wide pulse pressure
Pulsus alternans
Pulsus paradoxus
Brisk, rapid-rising pulse
Small, weak or slow-rising pulse
Cardiac arrhythmia (atrial fibrillation)
Abnormal precordial movements
Sustained LV apical or RV parasternal lift or heave (hypertrophy)
Diffuse, inferolaterally displaced impulse (LV enlargement)
Bifid LV apical impulse
"Ectopic" bulge (LV aneurysm)
ECG findings
LV or RV hypertrophy
Pathologic Q waves or ST-T-wave changes
Arrhythmias and/or conduction abnormalities
Chest x-ray findings
Cardiomegaly
Valve calcification
Abnormal pulmonary vasculature
Pulmonary venous congestion or edema

of the more common auscultatory findings encountered, which have stood the test of time and are proof of the continued value of cardiac auscultation, will be presented in the clinical vignettes that follow. The diagnostic clues on the cardiac clinical examination that enable today's physician to make the diagnosis of heart disease by clinical assessment *alone* will be highlighted (Tables 10 and 11).

Practical Clinical Applications of Cardiac Auscultation. In the context of a complete clinical cardiovascular assessment, the diagnostic information derived from the patient's history and other aspects of the cardiac clinical examination often directs the skilled examiner to search for spe-