Cardiac Auscultation: Rediscovering the Lost Art

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Abstract: Cardiac auscultation, long considered the centerpiece of the cardiac clinical examination, is rapidly becoming a lost art. Inadequate emphasis on the essentials of cardiac auscultation has resulted from the widespread availability of more elaborate and expensive “high-tech” diagnostic and therapeutic methods, particularly Doppler echocardiography. However, sophisticated high technology is not a substitute for a solid foundation in clinical cardiology including cardiac auscultation. When used properly, the stethoscope remains a valuable and cost-effective clinical tool that often enables many well-trained and experienced cardiac auscultators to make a rapid and accurate cardiac diagnosis with fewer, if any, additional studies. Not every patient needs every test.

Accordingly, this monograph reviews the fundamental principles of the art of cardiac auscultation. Emphasis is placed on the proper use of the stethoscope and the diagnostic and prognostic significance of the myriad heart sounds and murmurs present in patients with and without symptomatic heart disease. A practical clinical overview of the common auscultatory findings encountered in a variety of cardiac disease states and conditions will also be discussed. This monograph will inspire many practitioners to pick up their stethoscope, practice their cardiac examination, perfect their auscultatory skills, and reap the rewards of rediscovering this time-honored method of evaluating the cardiovascular system. (Curr Probl Cardiol 2008;33:326-408.)

Despite its long and rich tradition in clinical medicine, the time-honored art of cardiac auscultation is rapidly becoming a lost art. Most of today’s physicians have a difficult time identifying normal
and abnormal heart sounds and are often bewildered when confronted by a patient with a heart murmur. The reasons for this decline in cardiac auscultatory proficiency are a shortage of experienced clinician teachers skilled in the art of auscultation, a growing reliance on more elaborate and expensive technological advances, a volume-driven reimbursement system that provides minimal or no financial reward for time spent examining a patient, and a litigious environment that fosters the practice of “defensive medicine,” along with the ordering of many unnecessary, time-consuming, and potentially hazardous noninvasive and invasive diagnostic laboratory tests\(^1\) (Fig 1).

In the past, the stethoscope was considered the time-honored badge of the medical profession, but times and fashions change. In this age of increasing technologic sophistication, the stethoscope has become the forgotten instrument in cardiology.\(^2\) Although often seen worn around the practitioner’s neck or draped over the shoulder, in the so-called “cool position,” the stethoscope has become more of a decorative ornament than a diagnostic tool. In fact, many procedure-oriented cardiologists today do not even carry a stethoscope with them.

In this “high-tech” world of medicine, there is a growing misperception among medical trainees and younger practitioners that cardiac ausculta-
tion is antiquated, a “thing of the past.” These contemporary practitioners often question the value of the stethoscope and think it is a “waste of time” to listen to a patient when an echocardiography (echo) machine is available nearby. The echo transducer, however, is not a substitute for the stethoscope and may give a false diagnosis of cardiac disease (ie, “echocardiographic heart disease”) to a patient who has no murmur or an “innocent” murmur with only trivial or mild (“physiologic”) valvular regurgitation detected by color-flow Doppler techniques.

Cardiac auscultation should not become a lost art, or the stethoscope a medical relic. Despite the current emphasis on technology, the cardiac stethoscopic examination, when performed properly, remains a valuable and cost-effective clinical skill that often establishes the diagnosis, etiology, and severity of heart disease, forms the basis for further noninvasive and invasive testing when needed, and provides an added level of quality control by placing the interpretation of any additional laboratory tests in their proper perspective. At a time when the “fun” seems to have been taken away from the practice of medicine, auscultation of the heart restores the intellectual satisfaction of making a rapid and accurate cardiac diagnosis using one’s own wits and senses. Furthermore, by virtue of the “laying on of hands,” cardiac auscultation creates a personal bond with the patient that fosters the close rapport, trust, and confidence so important to the privileged doctor–patient relationship.3-18

The Art of Cardiac Auscultation

Auscultation of the heart is a fundamental yet difficult clinical skill to learn and master. Proficiency in the art of cardiac auscultation requires experience, repeated practice, and a great deal of patience. Most importantly, it requires a proper state of mind. The astute clinician should know what to listen for when examining a patient and to determine whether or not it is present.

Proper Use of the Stethoscope

Although the most crucial component of the auscultatory apparatus lies between the ear pieces, the proper use of a well-designed, efficient stethoscope cannot be overemphasized. To ensure optimal sound transmission, the stethoscope should be airtight, with snug but comfortably fitting earpieces, properly aligned metal binaurals, and flexible, double-barrel, thick-walled tubing that is as short as possible for efficiency (approximately 10 to 15 inches in length), yet compatible with a comfortable listening posture. A high-quality stethoscope should be equipped with both bell and diaphragm chest pieces. Proper use of these
Chest pieces will enhance the quality of heart sounds and murmurs heard and maximize the effectiveness of auscultation. The bell, when applied gently to the skin, will “bring out” low-frequency sounds and murmurs (e.g., fourth heart sound [S4] or third heart sound [S3] gallops, diastolic rumble of mitral stenosis [MS]). The diaphragm, when pressed firmly against the chest wall, will accentuate high-pitched auscultatory events (e.g., diastolic murmur of aortic regurgitation [AR], pericardial friction rub). A third chest piece (the corrugated diaphragm), present on the triple-headed (Harvey) stethoscope, amplifies sound and enables the examiner to “pick up” a wide range of frequencies by varying the pressure applied to the chest wall. Some modern stethoscopes incorporate both bell and diaphragm functions into a single chest piece (the tunable diaphragm). In these models, placing the piece lightly on the chest wall “brings out” the low-frequency sounds, while firm pressure accentuates the high frequencies. Electronic stethoscopes, equipped with amplification and ambient noise reduction technology, along with recording, storage, playback, and visual display capabilities are also now available.

The clinician should keep in mind, however, that to become proficient at cardiac auscultation, it takes more than just a good stethoscope—it is what is between the ears that counts! Auscultatory findings should always be judged in the context of the “company they keep,” namely, the other important components of the cardiac clinical examination. When possible, auscultation should be approached with the most likely diagnoses in mind. Prior to cardiac auscultation, the clinician should take advantage of the valuable information obtained from the patient’s history as well as the diagnostic clues derived from inspection and palpation. For example, the presence of chest pain, worsened by lying supine or during inspiration, and relieved by sitting up and leaning forward, should prompt a search for a pericardial friction rub. The finding of a large V-wave on inspection of the neck veins should alert the clinician to listen for the systolic murmur of tricuspid regurgitation (TR) along the lower left sternal border, increasing during inspiration. The presence of pulsus alternans (alternating strong and weak arterial pulse) on palpation of the radial arterial pulse should prompt a careful search for an S3 gallop along with a systolic murmur of mitral regurgitation (MR), often present in severe cardiac decompensation. A slow rise of the carotid arterial pulse warrants a search for the harsh systolic murmur of aortic stenosis (AS). Conversely, a rapid rise arterial pulse may be the clue to the finding of a faint blowing diastolic murmur of AR or the systolic murmur of hypertrophic obstructive cardiomyopathy (HOCM), which can be altered in intensity with certain maneuvers, e.g., squatting, standing, and Valsalva (see below).
These auscultatory findings are often discovered only when they are searched for specifically with intent listening and concentration.\textsuperscript{19-50}

\textbf{Auscultatory Tips}

To obtain the most information from cardiac auscultation, some general principles in applying the stethoscope should be followed.

1. The room should be as quiet as possible. This may be difficult to accomplish in an emergency department setting or in a hospital room with other patients and their visitors. By simply closing the door, turning off the television, and requesting that all conversation be terminated, the clinician can vastly improve the results of auscultation. As previously mentioned, modern electronic stethoscopes with ambient noise reduction capabilities are now available that have the ability to amplify heart sounds, while at the same time, filter out distracting background noise.

2. Both the examiner and the patient should be relaxed and comfortable. The patient should be properly gowned to avoid embarrassment; however, the chest should be sufficiently exposed to enable accurate auscultation. Proper auscultation should not be done through clothing. For maximal auscultatory yield, the stethoscope should be touching the patient’s bare chest.

3. To ensure patient comfort, the examiner should warm the diaphragm of the stethoscope with his or her hand before proceeding with auscultation. The room should also be kept warm to prevent shivering. Shivering may interfere with the examiner’s ability to accurately detect subtle characteristics of the heart sounds and murmurs.

4. The clinician should use an examining table or bed so that the patient can be examined in various positions, eg, supine, sitting upright, or turned to the left lateral decubitus position. The height of the examining table or bed should be adjusted to permit the patient (and examiner) to be comfortable.

5. When listening to a large-breasted woman, the left breast may need to be displaced away from the area of auscultation. The clinician may find it helpful to ask the patient to lift her breast upward and to the left or the examiner can use his or her nondominant hand to do so.

\textbf{Fundamentals of Auscultatory Technique}

The proper cardiac auscultatory technique in listening for normal and abnormal heart sounds and murmurs is summarized in \textit{Table 1}. When performing cardiac auscultation, the examiner should listen carefully. A superficial hurried stethoscopic examination can lead to inaccurate conclusions, even when performed by an experienced clinician.\textsuperscript{51-53}
**Systematic Approach to Cardiac Auscultation.** As a rule, most practitioners conduct the examination from the right side of the patient and begin with the patient reclining in the supine position. The examiner first listens over the patient’s 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 the bell chest pieces. Although some practitioners prefer to reverse this sequence, it is important for the examiner to adopt a systematic method of auscultation, beginning in one area and then carefully exploring all areas in an orderly and unhurried fashion, so that nothing is overlooked.

Next, the patient is turned to the left lateral decubitus position (which brings the heart closer to the chest wall) while the clinician “tunes in” to low-frequency sounds and murmurs (eg, S4 and S3 gallops, diastolic...
rumble of MS) using the bell of the stethoscope applied lightly at the cardiac apex, barely making an air seal. Light pressure is essential, since heavy pressure stretches the skin and converts the bell into a diaphragm, and thereby diminishes or eliminates (“filters out”) these low-frequency events (Fig 2). Then, with the patient in the sitting position, leaning forward with the breath held in deep expiration, the examiner listens over the base of the heart (right and left 2nd intercostal spaces) or left sternal border, to detect high-pitched sounds and murmurs, eg, diastolic murmur of AR, pulmonary hypertensive regurgitation (Graham Steell murmur), or pericardial friction rub. The diaphragm chest piece should be pressed firmly enough against the chest wall to leave a temporary imprint (after-ring) on the skin. The upright position also enables the clinician to further evaluate splitting of the second heart sound. Occasionally, normal individuals have expiratory splitting of the second heart sound in the supine position that becomes single in the upright position.

Everyone does not have the same capacity to hear sound. The ability to hear the faint high-frequency diastolic murmur of AR, for example, may be lost to those who are aging or even to the young who have listened to loud music for prolonged periods of time. It is possible to hear better by “selective listening.” That is, the examiner should close his or her eyes and “tune in” and concentrate on what he or she is listening for, blocking

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**FIG 2.** (Left) S4 and S3 gallop sounds are best heard with the patient turned to the left lateral decubitus position listening with the bell of the stethoscope applied lightly over the point of maximal impulse of the left ventricle barely making an air seal with the skin of the chest wall. (Reproduced with permission from Chizner MA. Clinical Cardiology Made Ridiculously Simple, 2nd edition. Miami, FL, MedMaster, Inc., 2007.) (Right) S4 and S3 gallops are usually best heard using light pressure. Firm pressure diminishes or eliminates these faint sounds. (Courtesy of W. Proctor Harvey, MD.)
out all other visual and auditory stimuli from consciousness. At times, accurate and efficient auscultation over the chest and neck is accomplished by having the patient temporarily stop breathing. In this way, breath sounds are not interfering with the clinician’s ability to hear.

**Timing of Heart Sounds and Murmurs: The “Inching” Technique.** The key to successful cardiac auscultation is the accurate timing of heart sounds and murmurs in the cardiac cycle. The first heart sound (S1) and second heart sound (S2) divide the cardiac cycle into systole and diastole. At normal heart rates, diastole is longer than systole. Therefore, the sound following the longer pause is S1, and the sound occurring immediately thereafter is S2. At fast heart rates, however, diastole shortens more than systole, and proper identification of S1 and S2 can be difficult. The listener may properly time S1 and S2 by palpation of the carotid artery or apical impulse. S1 occurs immediately before, and S2 after, the carotid pulse and apical impulse are felt. Although this method may be helpful, in clinical practice, accurate timing of heart sounds and murmurs can often best be achieved solely by listening, by employing a valuable auscultatory technique called “inching.” In most patients, S2 is louder than S1 over the aortic area, a point that can be helpful in distinguishing which sound is S1 and which is S2. By keeping S2 in mind as a reference as the stethoscope is moved or “inched” from the aortic area to the apex, any sound or murmur heard before S2 will be systolic in timing, and after S2, diastolic. This point can be helpful when there is a rapid cardiac rhythm and it is uncertain which sound is S1 and which is S2 (Fig 3).

**Traditional Areas of Auscultation.** 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 (Fig 4). For example,

- Sounds and murmurs of the aortic valve and aorta are well heard at the second right intercostal space (aortic area).
- Sounds and murmurs from the pulmonic valve and pulmonary artery are usually heard best at the second left intercostal space (pulmonic area) or (mid) left sternal border.
  The mid left sternal border is usually the best site to detect the diastolic murmur of AR (blood rushes back into the left ventricle [LV]).
- The lower left sternal border (tricuspid area) is the customary location for evaluation of S1, systolic clicks, 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. (In general, murmurs originating on the right side of the heart increase with inspiration.) The holosystolic murmur of ventricular septal defect (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.

**Other Areas of Auscultation.** Although the traditional areas of auscultation serve as a useful reference, cardiac auscultation should not be restricted to these sites alone. Important auscultatory findings may also be present in these other locations:

- Neck—transmitted systolic ejection murmur of valvular AS along the aortic branches, bruit of carotid arterial occlusive disease. Of note, transmitted murmurs of AS decrease in intensity as the stethoscope is
moved up the neck, whereas a carotid bruit is usually louder higher in the neck and becomes fainter as the stethoscope is inched down toward the chest.

- Clavicle—bone transmission of valvular AS. Bone is an excellent transmitter of sound, thus making the clavicles an ideal location to listen for the murmur of AS.

Supraclavicular fossa—continuous murmur of a jugular venous hum heard in children (normal), thyrotoxicosis, anemia, pregnancy, or any hyperkinetic state.

- Left axilla and posterior lung base—“band-like” radiation of the holosystolic murmur of chronic MR (when the anterior mitral leaflet is involved).
- Right sternal border—so-called “right-sided” diastolic murmur of the special type of AR due to aortic rightward displacement with aortic root pathology, eg, aortic dissection, aneurysm, Marfan’s syndrome. (The valvular type of AR is best heard along the left sternal border.)
- Abdomen—bruit of renal artery stenosis.

Over scars—continuous murmur of a peripheral arteriovenous fistula, which can result in high-output heart failure.

Thus, the site of maximal intensity and the direction of transmission of certain heart sounds and murmurs may prove to be of diagnostic value in the evaluation of their origin and clinical significance.

Certain heart sounds and/or murmurs may be faint and difficult to hear over the precordium, and, therefore, can be overlooked, especially in a patient with chronic obstructive pulmonary disease and an increase in anterior-posterior chest diameter. This is true if one listens over the usual areas of the chest. Listening over the inferior edge of the sternum (xiphoid area) or epigastrium (with the patient in the upright position), however, may help the clinician detect these sounds more easily. The examiner should keep in mind that, although MS can at times be “silent” (with no murmur present), in most of these cases, the bell of the stethoscope is not properly placed over the point of maximum impulse, a localized spot (which may be the size of a quarter) where the diagnostic diastolic rumble may be heard. The murmur can be missed unless the stethoscope is placed exactly over this small area.

**Dynamic Cardiac Auscultation.** Changes in body position and physiologic maneuvers (dynamic auscultation) alter the timing and/or intensity of auscultatory events and may help in the clinical evaluation of heart sounds and murmurs54-64 (Table 2).

**Changes in Position.** Prompt squatting (compresses veins in the legs and abdomen) causes the venous return to the heart (ie, preload) to increase. As a result, LV volume, chamber size, and stroke volume temporarily increase as does the intensity of many murmurs. Squatting also increases peripheral vascular resistance (ie, afterload). The latter may increase the murmurs of MR and AR. Although most murmurs become louder with squatting, two exceptions are the murmur of mitral valve prolapse (MVP) and that of HOCM, which become fainter. With squatting, the mitral valve in MVP becomes less redundant because of the increased ventricular volume and elevated systemic blood pressure. This results in a delay of the click and murmur of MVP to a later point in systole as well as a shorter murmur of decreased intensity (Fig 5). Squatting similarly decreases the intensity of the murmur of HOCM,
because the dynamic LV outflow obstruction created by the hypertrophied ventricular septum and the anterior leaflet of the mitral valve is relieved temporarily by the increased ventricular volume. While squatting decreases the murmur intensity of HOCM, it increases the intensity of most other murmurs due to the increased LV stroke volume (Fig 6). Thus, squatting can help distinguish HOCM from AS, since it decreases the murmur in HOCM (as it does in MVP) but increases the murmur in AS. Standing decreases venous return and stroke volume and therefore reverses these findings. The squatting maneuver is ideally performed if

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the practitioner remains comfortably seated and listens for any subtle auscultatory changes while the patient squats and stands. In those patients unable to perform the squatting maneuver, similar results can be induced by passive straight leg raising or by bending the patient’s knees toward his or her abdomen while he or she is in the supine position.

**The Valsalva Maneuver.** The Valsalva maneuver is a useful method for determining the cause of various heart murmurs. This maneuver is performed by having the patient forcefully attempt to exhale against a closed glottis after taking a normal breath. When performing the Valsalva maneuver, the clinician should ask the patient to “bear down” (ie, strain) as if they were having a baby or a bowel movement. This increases intrathoracic pressure, decreases venous return to the heart, and decreases LV volume. During the strain phase of the maneuver, the examiner should place his or her hand firmly against the patient’s abdomen to be sure that the muscles are tightening. An alternative method of performing the Valsalva maneuver is to instruct the patient to place their finger in their mouth, seal their lips around their finger, and “blow hard” against it. Since the initial effect of the Valsalva maneuver is to decrease venous return to the heart, most murmurs decrease in length and intensity. Two exceptions are the systolic murmur of MVP and that of HOCM. The systolic click and murmur of MVP moves earlier in systole (ie, closer to S1) during the strain phase of the Valsalva maneuver and the murmur becomes longer.
and often louder, since the valves are more redundant in the smaller ventricular cavity and flop and move back sooner into the left atrium (LA). The systolic murmur of HOCM increases in intensity (increased obstruction with reduced ventricular volume) during straining, while the systolic murmur of AS decreases in intensity (less forceful ventricular contraction with decreased ventricular volume). Following release of the Valsalva maneuver, there is a sudden increase in venous return. Right-sided murmurs that decreased tend to return to baseline intensity earlier (within one or two cardiac cycles) than left-sided murmurs (which return after several cardiac cycles). Of note, the Valsalva maneuver should not be held for more than 10 seconds. The decreased venous return to the heart that occurs causes a reduction in cardiac output and blood pressure, which may result in syncope. This maneuver should be avoided in patients with myocardial ischemia or a recent myocardial infarction (MI) since it will transiently decrease coronary blood flow.

**Effect of Respiration.** Noting the effect of respiration on heart sounds and murmurs can also provide important diagnostic clues to heart disease. Inspiration increases the venous return to the right side of the heart, causing a delayed pulmonic valve closure (P2) because the right ventricle (RV) requires a little longer to pump this normal increase in the amount of blood. Also, there is earlier closure of the aortic valve (A2) with
inspiration. Since less blood returns to the left side of the heart, there is less pressure in the LV, and the aortic valve can close more quickly against this reduced pressure. Normally, S2 is perceived as a single sound on expiration and splits on inspiration (A2 P2) due to the relative delay in closure of the pulmonic valve. Normal children and young adults, however, may have slight persistent expiratory splitting of S2 when supine, which disappears on sitting. Splitting of S2 that persists on expiration, particularly when the patient is examined in the sitting position, is usually abnormal.

In right bundle branch block (RBBB) and pulmonary stenosis (PS), splitting of S2 widens on inspiration but is also heard in expiration, since the pulmonary valve is even further delayed in its closure. In RBBB the delay is due to a slowly contracting RV; in PS the delay in pulmonic valve closure is due to delay of blood flow through the stenotic pulmonary valve. In atrial septal defect (ASD), where blood shunts from left to right atrium (RA), the splitting of S2 is “fixed” in inspiration and expiration, since there is increased flow into the RV (from both shunted blood and the periphery) during the entire respiratory cycle, thereby negating the usual influences of respiration on venous return to the right side of the heart. Of note, to make the diagnosis of ASD, a systolic murmur due to increased pulmonary flow (not due to flow through the defect) must be present; without the murmur, an ASD is not likely.

In paradoxical splitting of S2, as occurs in left bundle branch block (LBBB), AS, HOCM, and hypertension, the split becomes more noticeable during expiration (P2 A2), since the pulmonic valve in the resting state closes before the aortic valve in these conditions. (In LBBB, the LV does not contract fast enough; in AS, HOCM, and hypertension, it takes longer for blood to be ejected from the LV against resistance.) In pulmonary hypertension the splitting is narrow and the P2 component of S2 is particularly intense due to loud closure of the pulmonic valve resulting from the pulmonary hypertension.

Respiration is the best maneuver for differentiating between heart sounds and murmurs originating in the right and the left sides of the heart. All right-sided heart sounds and murmurs, with the exception of the pulmonic ejection sound (ES) of PS, increase in intensity with inspiration. The pulmonic ES does not increase in PS and may even decrease, because the higher pressure in the RV causes the valve leaflets to start to move upward even before RV systole; thus, there is less of a valve excursion and less sudden thrusting open of the valve leaflets when systole occurs.

Conversely, inspiration decreases the intensity of left-sided heart sounds and murmurs (decreased blood volume and flow on the left side during inspiration). When evaluating the effect of respiration, a helpful technique...
is to have the patient follow the examiner’s free hand and breathe in when his or her hand moves upward and breathe out when it moves downward. The patient should breathe smoothly and continuously.

**After a Premature Beat or Long Cycle Length in Atrial Fibrillation.** Sometimes a pause after a premature beat or following a long cycle length in atrial fibrillation may change the intensity of a murmur and provide further clues as to its cause. The systolic murmur of MR remains relatively unchanged in intensity in the beat after a premature ventricular contraction (PVC) or following a long cycle length in atrial fibrillation (since the LV to LA pressure gradient is minimally affected by alterations in cycle length). By contrast, the systolic (ejection) murmur of AS is louder in the beat after a PVC or following a pause in atrial fibrillation (greater filling of the LV, along with an increase in stroke volume, and greater flow of blood across the narrow aortic valve). Thus, careful auscultation can use a fortuitous premature beat or a variation in cycle lengths in atrial fibrillation to help establish the origin of a heart murmur (Fig 7).

**Isometric Handgrip Exercise and Transient Arterial Occlusion.** Isometric handgrip exercise can be performed by asking the patient to make a fist with both hands. This maneuver increases peripheral vascular
resistance and pressure (ie, afterload) and increases the murmurs of MR (more blood goes back into the LA), VSD (more blood goes from LV into RV), and AR (more blood goes back into the LV).

Transient arterial occlusion is performed by placing two blood pressure cuffs around the upper portion of both arms and inflating the cuffs simultaneously to pressures 20 to 40 mmHg above the patient’s peak systolic blood pressure for 20 seconds. This maneuver increases aortic impedance (ie, afterload) and also increases the murmurs of MR, AR, and VSD, but not murmurs due to other causes. Transient arterial occlusion has a sensitivity equal to that of sustained handgrip exercise. Since these maneuvers increase systemic vascular resistance, they should be avoided in patients with a recent MI, cerebrovascular disease, uncontrolled hypertension, or suspected aortic dissection.

**Pharmacologic Maneuvers.** Various pharmacologic interventions have been used in the past to clarify the origin of heart murmurs. Their popularity, however, has waned in recent years with the advent of other physiologic maneuvers and 2D Doppler echocardiography. An understanding of the physiologic mechanisms underlying the effects of pharmacologic agents on cardiac murmurs, however, contributes to the overall understanding of heart sounds and murmurs.

Inhalation of amyl nitrite is the most practical of these pharmacologic interventions because of its ease of administration and its very transient effect. As a potent vasodilator, inhalation of amyl nitrite causes a decrease in systemic blood pressure followed by a reflex increase in heart rate and myocardial contractility. During the initial hypotension following amyl nitrite inhalation, the murmurs of MR, VSD, and AR decrease while the murmurs of AS, PS, HOCM, and the innocent systolic flow murmur all increase because of the associated increase in stroke volume. During the later tachycardia phase, the murmur of MS also increases. As a result, amyl nitrite may distinguish the Austin Flint murmur of AR (which decreases) from that of the apical diastolic rumble of MS (which increases).

**Selective Listening.** When performing auscultation, the clinician should listen selectively (“tune in”) to one aspect of the cardiac cycle at a time rather than trying to hear everything at once and have a set sequence of listening. This detailed auscultation of heart sounds and murmurs may at first be difficult, but with continued practice and concentration, can be accomplished with a gratifyingly high degree of accuracy. For example, the examiner should first focus on S1, then on S2, then on systolic sounds and murmurs, then on diastolic sounds and murmurs, and then on continuous murmurs if present. 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 two or three components (systolic, diastolic, presystolic).

Paying close attention to the timing of heart sounds and murmurs within the cardiac cycle (systolic, diastolic, continuous), their intensity (loud, normal, or faint), frequency (high-, medium-, low-pitched), tonal quality (sharp, dull, muffled, snapping, tamour, harsh, blowing, rumbling), location, duration, and radiation may provide valuable clinical clues to underlying heart disease. An effective way to improve one’s auscultatory ability is to “sketch” the heart sounds and murmurs heard on each patient examined.

**Cardiophonetics.** Heart sounds and murmurs can also be simulated vocally by using words and letters to mimic their quality and timing. Learning to mimic heart sounds and murmurs may help the practitioner recognize auscultatory findings.65

**Heart Sounds: Normal and Abnormal**

**First Heart Sound**

The first heart sound (S1) is generated by vibrations resulting from closure of the mitral (M1) and tricuspid (T1) valves at the start of ventricular systole. S1 is usually perceived as a single sound (“lub”), although occasionally its two components (M1 and T1) can be readily appreciated. Since the LV contracts first, M1 occurs earlier, is the louder of the two components, and is best heard with the diaphragm of the stethoscope at the cardiac apex (mitral area). T1 is somewhat fainter and is heard at the lower left sternal border (tricuspid area). The clinician should pay close attention to the intensity of S1 (loud, normal, or faint). Most clinically significant abnormalities of S1 are of intensity rather than splitting (Table 3). Since S2 is normally louder than S1 at the aortic area, the clinician may identify a loud S1 if it is equal to or louder than S2 over this site. On the other hand, since S1 is louder than S2 at the cardiac apex, if S1 is fainter than S2 over this site, then the clinician may identify a reduced intensity S1.66-69

**Intensity of S1.** S1 intensity, which usually reflects mitral rather than tricuspid closure, can be affected by the PR interval, mitral valve structure, and ventricular contractility.

**Loud S1.** The intensity of S1 is augmented by any condition that increases the force of ventricular contraction and the rate of pressure development in the ventricle in systole or brings the heart closer to the chest wall. Thus, S1 is physiologically louder in children and young adults, in patients with a thin chest wall, and in hypercontractile states (eg,
exercise, tachycardia, anemia, hyperthyroidism, fever, pregnancy, excitement). The intensity of S1 is also increased if the mitral and tricuspid valve leaflets close from a wide open position at the onset of ventricular contraction since the excursion is large and the vibrations amplified. In the presence of a normal ventricular rate a loud S1 should provide a clue to the possibility of a cardiac problem:

- A loud S1 is heard in rheumatic MS. The loud S1 relates to the high LA pressure, which causes the mitral valve to remain open more widely until a very sharp high-velocity closing movement is produced by ventricular contraction. In fact, unless the mitral valve is immobilized by calcific deposits (at which time S1 becomes faint or absent), an increase in intensity of S1 implies that the valve is pliable, not heavily calcified, and that the patient may be a candidate for commissurotomy or balloon valvuloplasty rather than mitral valve replacement.

### TABLE 3. Clinical clues from the first heart sound

<table>
<thead>
<tr>
<th>Increased intensity of S₁</th>
<th>Mitral stenosis</th>
<th>Short PR interval</th>
</tr>
</thead>
<tbody>
<tr>
<td>Hyperkinetic states (eg, hyperthyroidism, fever, anemia, exercise, tachycardia, pregnancy)</td>
<td>Physiologically normal in children, young adults, patients with a thin chest wall and narrow A-P diameter</td>
<td>Holosystolic mitral valve prolapse</td>
</tr>
<tr>
<td>Physiologically normal in children, young adults, patients with a thin chest wall and narrow A-P diameter</td>
<td>Holosystolic mitral valve prolapse</td>
<td>Decreased intensity of S₁</td>
</tr>
<tr>
<td>Long PR interval (first-degree AV block)</td>
<td>Diminished LV contractility (eg, congestive heart failure, acute myocardial infarction, cardiomyopathy)</td>
<td>Premature mitral valve closure with high LV end-diastolic pressure (eg, acute severe aortic regurgitation)</td>
</tr>
<tr>
<td>Ineffective mitral valve closure due to loss of leaflet substance or thickening, fibrosis, calcification, or shortening of mitral valve apparatus</td>
<td>Increased tissue, air, or fluid between the heart and the stethoscope (eg, emphysema, obesity, large breasts, thick chest wall, increased A-P diameter, pericardial effusion)</td>
<td>Changing intensity of S₁</td>
</tr>
<tr>
<td>AV dissociation (varying PR intervals, eg., complete heart block, ventricular tachycardia). Atrial fibrillation</td>
<td>Wide splitting of S₁</td>
<td>RBBB</td>
</tr>
<tr>
<td>PVCs, ventricular tachycardia</td>
<td>Atrial septal defect</td>
<td></td>
</tr>
</tbody>
</table>

S₁, first heart sound; AV, atrioventricular; LV, left ventricular; RBBB, right bundle branch block; PVC, premature ventricular contraction; A-P, anterior-posterior.

• A loud S1 occurs with a short PR interval on the electrocardiogram (ECG), since the valve leaflets are in a wide open position at the onset of ventricular contraction and close more forcefully.

When a loud S1 is heard along with a holosystolic murmur of MR, the clinician should always consider the diagnosis of MVP. The loud S1 may be due to an increased amplitude of leaflet excursion beyond the line of closure and/or the merging of S1 with the systolic click of MVP (see below).

**Faint S1.** When the valve leaflets are in close apposition at the onset of ventricular contraction, the excursion is minimal, the vibrations are small, and the intensity of S1 is decreased.

A faint S1 may accompany a prolonged PR interval on the ECG (0.20-0.24 seconds), as occurs in first-degree heart block, since the mitral leaflets have had more time to “drift” to a partially closed position by the time ventricular contraction begins.

Decreased LV contractility due to LV systolic dysfunction will also tend to decrease S1 intensity.

In acute, severe AR (eg, from infective endocarditis), a sudden volume overload in the LV from the regurgitation results in a markedly elevated end-diastolic pressure, which may lead to premature closing of the mitral valve and therefore reduce the intensity of S1. The faint S1 is an important clue that the regurgitation is acute, where early surgical intervention can be lifesaving.

S1 may also be diminished in MR by thickening, fibrosis, calcification, and shortening of the valve apparatus, resulting in ineffective valve closure and loss of mobility.

Body build, chest configuration, and other extracardiac factors (eg, obesity, large breasts, increased chest wall thickness, and pericardial effusion) may also play a role in diminishing the intensity of S1 (as well as other auscultatory events) because of poor conduction of sound.

**Variable Intensity of S1.** A variation in the intensity of S1 may be detected during atrioventricular (AV) dissociation due to varying PR intervals (eg, complete heart block, ventricular tachycardia), and with changes in cycle lengths (eg, atrial fibrillation), since there is variation in position of the mitral leaflets at the time of ventricular contraction. The finding of a slow heart rate (~40 beats/min), accompanied by a varying intensity of S1 (due to AV dissociation), for example, is an auscultatory clue to the diagnosis of complete heart block. Likewise, a variable S1 during a wide complex regular tachycardia also suggests AV dissociation and is a clue to ventricular tachycardia. In atrial fibrillation, the intensity
of S1 is inversely related to the previous cycle length. A long cycle length produces a faint S1 and a short cycle length produces a loud S1.

**Wide Splitting of S1.** Wide splitting of S1 can be observed in conditions where there is a significant delay in RV contraction resulting in a late closure of the tricuspid valve in relation to the mitral valve. Widening of S1 is due most often to RBBB. In ASD, wide splitting of S1, in addition to a loud T1, is also present because the tricuspid valve is held open by the increased flow of blood from the RA to the RV. This results in valve closure from a more open position and at a greater velocity than normal. Wide splitting of S1 should be distinguished from other sounds that can simulate a split S1, eg, S4-S1, S1-ejection sound, S1-systolic click (see below).

**Second Heart Sound**

The second heart sound (S2) ("dub") is the sound produced by vibrations associated with closure of the aortic (A2) and pulmonic (P2) valves at the start of ventricular diastole. Since the pressure in the aorta is significantly higher than that in the pulmonary artery, the aortic valve normally closes before the pulmonic valve (A2 normally precedes P2) and A2 is louder than P2. S2 may thus be heard as a split sound (A2-P2) on inspiration, which increases venous return to the right side of the heart and prolongs flow through the pulmonary artery into the more compliant lungs (increased "hangout" time in the pulmonary circulation), delaying closure of the pulmonary valve still further. Inspiration also reduces blood return to the LA and LV so that there is less pressure in the LV, and the aortic valve closes sooner.

In contrast to S1, in which splitting is less important than changes in intensity, S2 reveals variations in both intensity and degree of splitting that provide significant clinical information. The clinician should determine whether S2 along with its A2 and P2 components are loud, normal, or faint and particularly note the effect of respiration on the splitting of S2 into its A2 and P2 components (Table 4). S2 is normally higher in pitch, shorter and sharper than S1. A2 is normally louder and earlier than P2 and can be heard well over the entire precordium.  

**Intensity and Splitting of S2. Loud S2.** A loud S2 will result from elevation of pressure in either of the great vessels, causing a forceful valve closure sound. Hence, the following:

- In systemic arterial hypertension there is a loud “tambour” (ringing, musical) A2
- In pulmonary hypertension (either idiopathic or secondary to other cardiac conditions, eg, left heart failure, MS) there is a loud (and often
palpable) P2 with narrow inspiratory splitting of S2. Of note, in normal individuals, only A2 is transmitted to the apex. P2 is not normally heard at the apex, except in young, thin individuals. If P2 is heard at the apex (along with A2), the clinician should consider pulmonary hypertension.

Faint S2. Impaired mobility of the aortic or pulmonic valve will result in a diminished intensity of their respective closure sounds, as follows:

- In severe calcific AS, there is a faint or inaudible A2.
- In PS, there is a faint P2.

**Abnormal Splitting of S2 (Wide, “Fixed,” Paradoxic)**

Splitting of S2 is best heard along the upper and mid left sternal border during normal breathing with the patient in the sitting position. As previously mentioned, wide “physiologic” splitting of S2 occurs when P2 is delayed (eg, RBBB, PS), or A2 occurs earlier (eg, MR, VSD). Audible splitting of S2 on expiration should be verified with the patient in the upright position, since healthy individuals occasionally have expiratory

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**TABLE 4. Clinical clues from splitting of the second heart sound**

<table>
<thead>
<tr>
<th>Wide “physiologic” splitting (increases with inspiration)</th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td>RBBB</td>
<td></td>
</tr>
<tr>
<td>LV ectopic beats</td>
<td>Due to delay of pulmonic valve closure</td>
</tr>
<tr>
<td>Pulmonic stenosis</td>
<td></td>
</tr>
<tr>
<td>Mitral regurgitation</td>
<td>Due to shortening of LV ejection time and early aortic valve closure</td>
</tr>
<tr>
<td>Large VSD</td>
<td></td>
</tr>
<tr>
<td>Idiopathic dilatation of pulmonary artery</td>
<td>Due to decreased impedance in the pulmonary bed</td>
</tr>
<tr>
<td>Wide “fixed” splitting (no change with inspiration)</td>
<td></td>
</tr>
<tr>
<td>Atrial septal defect</td>
<td>Due to increased pulmonary blood flow and decreased pulmonary vascular resistance</td>
</tr>
</tbody>
</table>

Paradoxical or reversed splitting (decreased with inspiration)

| LBBB |  |
| RV ectopic beats, RV pacing |  |
| Severe AS or hypertrophic cardiomyopathy | Due to delayed aortic valve closure |
| Severe LV dysfunction, eg, acute MI |  |
| Transient LV dysfunction during angina (rare) | Large PDA |

RBBB, right bundle branch block; LV, left ventricular; VSD, ventricular septal defect; LBBB, left bundle branch block; RV, right ventricular; AS, aortic stenosis; PDA, patent ductus arteriosus. (Reproduced with permission from Chizner MA. The Art of Cardiac Auscultation: Normal and Abnormal Heart Sounds. In: Chizner MA, editor. Classic Teachings in Clinical Cardiology: A Tribute to W. Proctor Harvey, MD. Cedar Grove, NJ: Laennec, 1996.)
splitting of S2 in the supine position. “Fixed” splitting of S2 is characteristic of ASD and paradoxical or reversed splitting of S2 occurs when A2 is delayed (eg, LBBB, RV paced rhythm, AS, HOCM). In the latter, A2 follows P2, and as P2 is delayed during inspiration, the sounds move together. Paradoxical splitting of S2 in AS and HOCM suggests that a significant LV outflow tract gradient is present. Transient paradoxical splitting of S2 can also occur during an episode of acute myocardial ischemia. As previously discussed, the clinician should keep in mind that P2 is normally heard over the pulmonic area and mid left sternal border and does not radiate to the apex, except in young, thin individuals or when pulmonary hypertension is present.

In many normal older patients (>50 years of age), splitting of S2 may be less discernible to the examiner, with a single audible S2 on inspiration and expiration. This results from a delayed A2 (eg, from LV systolic dysfunction or AS) and earlier P2, secondary to decreased pulmonary “hang-out” time (decreased compliance in the pulmonary vascular bed). An inaudible P2 may also exist in older adults with increased anteroposterior chest diameter. This may result in a single S2 heard during both expiration and inspiration (or single during inspiration but split on expiration), leading to the false impression of paradoxical splitting of S2; the A2 and P2 do not in this case fuse during inspiration, but rather the P2 just cannot be heard due to the increase in anteroposterior chest diameter on inspiration.

**Extra Heart Sounds in Systole**

**Ejection Sounds.** Normally, the opening of the aortic or pulmonic valves is acoustically silent. In certain cardiac conditions, brief, sharp, high-pitched sounds in early systole, occurring shortly after S1, may be heard and are referred to as aortic or pulmonic ejection sounds. Ejection sounds occur at the onset of ventricular ejection and systolic flow into the great vessels. They are heard best with the diaphragm chest piece and frequently are close enough to S1 to simulate splitting. The ES is usually the result of “doming” of a congenitally stenotic, but mobile and compliant, aortic or pulmonic valve and occurs when the valve is checked at its maximally distended position. The intensity of the ES correlates directly with the valve’s mobility and becomes faint or disappears with calcific fixation of the valve (Fig 8).

Aortic ejection sounds are widely transmitted over the precordium but are best heard at the aortic area and the cardiac apex (where they may even be loudest). In LV outflow obstruction, the ES localizes the obstruction to the aortic valve. The ES of PS is similar, but, unlike the
“DOMING” IN VALVULAR AORTIC STENOSIS

FIG 8. Schematic diagram of the mechanism of the aortic valvular ejection sound (ES) in congenital bicuspid valvular aortic stenosis. When the valve moves from its closed position in diastole to its open position in systole, a “doming effect” occurs because of restriction to complete opening. The ES occurs when the valve is checked at its maximally distended position. Another mechanism for the ES is poststenotic dilatation of the aorta. Ao, aortic valve; LA, left atrium; LV, left ventricle; SM, systolic murmur; S1, first heart sound; S2, second heart sound. (Courtesy of James A. Ronan Jr MD.)

aortic ES (which remains unchanged in inspiration and expiration), it decreases in intensity during inspiration (since the inflow of blood to the RV moves the stenotic pulmonic leaflets upward to a more open position, resulting in less systolic excursion). Also, the pulmonic ES is not heard at the apex (it is heard best at the base over the second left intercostal space [pulmonic area] or mid left sternal border).

In other cases, ejection sounds are due to abrupt “checking” of the rapid initial systolic distention of a dilated ascending aorta (eg, aneurysm, hypertension) or main pulmonary artery (vascular, rather than valvular, origin). They may also occur with forceful LV ejection (eg, thyrotoxicosis, anemia, pregnancy, exercise, high cardiac output states). Ejection sounds also occur with prosthetic (mechanical) aortic valve opening (see below).

It is important that the clinician becomes adept in the detection of ejection sounds, as they often serve as one of the first clues to the diagnosis of these conditions (Table 5). Worthy of mention, if S1 appears to be louder at the base than at the apex, the clinician should suspect that an ES may be masquerading as S1.72

Systolic Clicks. Systolic clicks are discrete high-frequency sounds caused by prolapse of the mitral valve leaflets into the LA during systole. Prolapse may also occur in the tricuspid valve. Isolated tricuspid valve
prolapse, however, occurs only rarely, and when it does, it usually accompanies MVP. Systolic clicks may be single or multiple. They are heard best at the cardiac apex or lower left sternal border and are usually mid to late systolic in timing, although occasionally they occur sufficiently early to simulate an ES. They are thought to be generated by the sudden tensing of the redundant mitral valve leaflets and elongated chordae tendineae when the mitral leaflet is checked at the farthest extent of its valve motion (Fig 9).

There can be a wide spectrum of auscultatory findings in MVP, even in the same patient. Sometimes, no click or clicks are present. At other times, an isolated systolic click (or clicks) with or without a mid to late (crescendo-decrescendo, or crescendo to S2) systolic murmur, holosystolic murmur, or musical “whoop” or “honk” of MR may be audible. One negative examination does not exclude the diagnosis. The anterior or posterior mitral valve leaflet or both leaflets can prolapse. The presence of a click or clicks without a murmur is often associated with prolapse of the anterior mitral leaflet.

Careful auscultation with a stethoscope remains a most valuable and cost-effective means of diagnosing MVP. In fact, the diagnostic acoustic hallmark, the systolic click, may even be heard in patients with no evidence of MVP on echocardiography (or angiography). These clicks, however, are often overlooked when the clinician does not properly time them or listen specifically for these high-pitched sounds in systole. As a rule, the findings are best detected using the diaphragm of the stethoscope. As previously mentioned, auscultation in multiple positions or after physiologic maneuvers (eg, Valsalva) may be required to “bring out” or enhance these auscultatory findings.73,74

<table>
<thead>
<tr>
<th>TABLE 5. Clinical clues from ejection sounds</th>
</tr>
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<tbody>
<tr>
<td>Aortic ejection sounds</td>
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<tr>
<td>Aortic valve abnormality (eg, congenital bicuspid aortic valve, AS, AI)—often heard best at apex</td>
</tr>
<tr>
<td>Dilatation of the aortic root (aneurysm, hypertension)</td>
</tr>
<tr>
<td>Forceful LV ejection (eg, thyrotoxicosis, anemia, pregnancy, exercise, high cardiac output states)</td>
</tr>
<tr>
<td>Prosthetic (mechanical) aortic valve opening</td>
</tr>
<tr>
<td>Pulmonic ejection sounds</td>
</tr>
<tr>
<td>Pulmonic valve abnormality (eg, PS)—fainter on inspiration</td>
</tr>
<tr>
<td>Dilatation of the pulmonary artery</td>
</tr>
</tbody>
</table>

AS, aortic stenosis; AI, aortic insufficiency; LV, left ventricular; PS, pulmonic stenosis. (Reproduced with permission from Chizner MA. The Art of Cardiac Auscultation: Normal and Abnormal Heart Sounds. In: Chizner MA, editor. Classic Teachings in Clinical Cardiology: A Tribute to W. Proctor Harvey, MD. Cedar Grove, NJ: Laennec, 1996.)
Extra Heart Sounds in Diastole

Third Heart Sound. The third heart sound (S3) is the sound produced by passive ventricular filling in early diastole. An S3 can be either left-sided or right-sided, reflecting a problem with either the left or the right side of the heart. An S3 commonly is normal (physiologic S3) in a healthy child or young adult, athlete, hyperkinetic states, or third trimester of pregnancy. It diminishes with age as the ventricles become less compliant and usually disappears by age 40 years. The S3 is an abnormal sound (pathologic S3 or S3 “gallop”) frequently heard in CHF due to
ventricular systolic dysfunction, and volume overload due to valvular regurgitation (eg, MR, AR) or left to right shunts (eg, ASD, VSD or patent ductus arteriosus) (Table 6).

S3 gallops are generally faint sounds and may be heard only intermittently (ie, waxes and wanes). The S3 is best heard with the bell of the stethoscope applied lightly to the cardiac apex with the patient turned to the left side (for left-sided gallops) or tricuspid area or xiphoid region (for right-sided gallops). Right-sided gallops increase with inspiration due to increased venous return and increased blood flow in the RV, while left-sided gallops are heard best on expiration due to the narrower diameter of the chest. Although often difficult to hear, the ability to detect these faint sounds improves with experience and practice.

In most adults with cardiac disease, an S3 gallop, particularly when accompanied by pulsus alternans and alternation in intensity of heart sounds (especially S2) and other accompanying murmurs, usually reflects impending or overt CHF. It can be heard in patients with LV systolic dysfunction and is associated with an abnormally elevated LV filling pressure, a reduced ejection fraction, and an elevated brain natriuretic peptide level. Its intensity often relates to the severity of LV failure, becoming louder as the condition worsens and growing fainter as compensation is restored. If loud and persistent, the S3 gallop reflects a significant decrease in LV function and carries a poor prognosis. An S3 gallop can also be heard in a variety of conditions not necessarily implying a failing ventricle. If blood rapidly accelerates into the ventricle because of an increased volume of flow, for example, an S3 may also occur as in patients with valvular regurgitation (eg, MR, AR, or TR) or left to right shunts.

The S3 can have great diagnostic value and prognostic implications. In an asymptomatic person younger than 40 years of age with an otherwise normal cardiac examination, an S3 (normal or physiologic) has a meaning

<table>
<thead>
<tr>
<th>TABLE 6. Clinical clues from the third heart sound</th>
</tr>
</thead>
<tbody>
<tr>
<td>● Physiologic S₃: normal early diastolic ventricular filling sound heard in healthy child or young adult, athlete, hyperkinetic states, or third trimester of pregnancy</td>
</tr>
<tr>
<td>● Pathologic S₃: abnormal sound heard in heart failure and/or cardiomyopathy (LV and RV systolic dysfunction), and mitral or aortic regurgitation, VSD or PDA (due to large volume of ventricular flow)</td>
</tr>
</tbody>
</table>

LV, left ventricular; RV, right ventricular; VSD, ventricular septal defect; PDA, patent ductus arteriosus.

entirely different from an S3 (pathologic) in a middle-aged or older adult with shortness of breath. In the latter, the S3 may now afford one of the earliest clues to the presence of cardiac decompensation and a high LV filling pressure due to severe LV systolic dysfunction or volume overload (eg, MR, AR).

Right-sided S3 gallops often are present in patients with dilated right ventricles and elevated right heart pressures. Although right-sided S3 gallops are generally best heard at the left sternal border or just beneath the xiphoid, these sounds may even be heard over the cardiac apex when the dilated RV occupies it. They are commonly heard in those patients with RV failure, TR, pulmonary hypertension, or cor pulmonale.

In rapid tachycardias the S3 and S4 can sometimes occur so close to one another as to fuse in a “summation” gallop, which may be louder than both the S1 and the S2.75-86

**Fourth Heart Sound.** The fourth heart sound (S4) is the sound produced by active ventricular filling as the atrium contracts in late diastole. An S4 gallop, although faint, is usually louder than the S3 gallop since it may be produced by healthier and more forceful hearts. An S4 gallop is more likely to be heard, therefore, with every beat. Some very low frequency S4 gallops, however, may be palpable at the apex as a presystolic movement, without being audible.

The S4 gallop is a hallmark of conditions where there is decreased ventricular compliance (diastolic dysfunction), which necessitates a more forceful atrial contraction for completion of ventricular filling. This change in compliance may be related to ventricular hypertrophy due to outflow obstruction (eg, AS, PS, HOCM), acute myocardial ischemia or infarction, fibrosis, or an increased afterload, eg, elevated aortic pressure (systemic hypertension) or pulmonary artery pressure (pulmonary hypertension) resulting in ventricular hypertrophy and stiffening of the ventricular wall (Table 7). An asymptomatic decrease in LV compliance may also accompany the aging process. Although an S4 can be heard in otherwise normal elderly patients, a palpable presystolic impulse (palpable S4) should not be present unless the LV is abnormal. Like an S3, an S4 can originate in either the LV or the RV. A right-sided S4 gallop is usually best heard at the lower left sternal border rather than at the apex and gets louder on inspiration.

An S4 gallop is a faint sound that may be detected, if carefully searched for using the bell of the stethoscope applied lightly to the cardiac apex with the patient in the left lateral position, in most patients with an acute MI who are in sinus rhythm due to the presence of a stiff, noncompliant, ischemic LV. It may become louder during the early phase of infarction.
or during an episode of angina pectoris. The S4 gallop may therefore provide a useful clue if it appears intermittently during attacks of chest pain suspected of being ischemic in etiology. The absence of an S4 in a patient with an acute MI should suggest inadequate auscultation or raise serious doubts as to the diagnosis. An S4 gallop may occur with or without clinical evidence of CHF in patients with sinus rhythm. Likewise, the detection of an S4 gallop, along with a loud “tambour” second heart sound (A2), can be the earliest auscultatory findings detected in hypertensive heart disease, often preceding ECG and other signs of LV hypertrophy or symptoms of cardiac decompensation. However, once systolic heart failure occurs, the S3 gallop may be noted for the first time.

An S4 gallop does not always indicate reduced LV compliance. S4 gallops are also commonly heard in patients with acute severe MR (eg, ruptured chordae tendineae or papillary muscle rupture), reflecting the persistence of vigorous LA contraction resulting in acceleration of blood flow into the LV. This contrasts sharply with the absence of an S4 gallop in chronic MR, since the LA is large in chronic MR, dilated, and unable to generate much contractile force.

The S4 gallop also occurs when there is prolongation of the PR interval with first-degree heart block (due to the delay in AV conduction that separates atrial contraction from ventricular contraction). Since an S4 gallop relies on effective atrial contraction, it is not present during atrial fibrillation. An S3 gallop, however, may still persist.\textsuperscript{87}

**Double First Sound Complex.** The proper identification of two heart sounds occurring around S1 (ie, double first sound complex) often provides a diagnostic challenge to the clinician. Sounds occurring around the time of S1 that must be differentiated from a split S1 include an S4, an ES, and an early systolic click. To distinguish an S4-S1 from a split S1

<table>
<thead>
<tr>
<th>Clinical clues from the fourth heart sound</th>
</tr>
</thead>
<tbody>
<tr>
<td>● Late diastolic (presystolic) ventricular “filling sound” due to forceful atrial contraction necessitated by decreased ventricular compliance (diastolic dysfunction), eg, AS, hypertension, coronary artery disease (acute MI, angina), cardiomyopathy (dilated, hypertrophic, restrictive), PS, pulmonary hypertension</td>
</tr>
<tr>
<td>● Acute severe mitral regurgitation (not chronic since the LA is dilated and unable to generate a forceful atrial contraction). Disappears in atrial fibrillation</td>
</tr>
<tr>
<td>● Long PR interval (first-degree AV block)</td>
</tr>
<tr>
<td>Normal in some apparently healthy older persons</td>
</tr>
</tbody>
</table>

AS, aortic stenosis; acute MI, acute myocardial infarction; PS, pulmonic stenosis; LA, left atrium.

(M1-T1) or S1-ES, the examiner should note that alternating the pressure with the bell causes the S4 gallop, which generally is of lower frequency, to fade in and out, but will not obliterate a split S1 or ES. Also, to distinguish an S4 gallop from an aortic ES, the clinician should note that an S4 usually is not heard over the aortic area.

Generally, the two components of S1 (M1 T1) are similar in frequency, are both heard with the diaphragm, and are most apparent at the left sternal border. Ejection sounds may be difficult to differentiate from a split S1. However, the separation of S1 from an ES or an early systolic click is generally wider than the separation in a split S1. (Aortic ejection sounds are loudest at the aortic area and apex; pulmonic ejection sounds are loudest at the pulmonic area, and split S1 is loudest at the tricuspid area and occasionally the apex, but not over the aortic or pulmonic areas.) The examiner should also note variation in intensity with inspiration (aortic ejection sounds do not vary with respiration).

If the clinician hears what appears to be a loud S1 in the aortic or pulmonic area, while remembering that S2 is normally louder than S1 in this location, the loud S1 in these areas should be an immediate clue that this represents an ES, unless the patient has an unusually loud S1 at the apex, as may occur with MS or a short PR interval. One should not confuse the tricuspid closure sound (T1) with the pulmonic ES, since the intensity of T1 tends to increase rather than decrease during inspiration (while the intensity of pulmonic ES decreases with inspiration).

The timing of a systolic click of MVP is usually later (mid to late systole), and if early, will vary in position with various maneuvers that alter ventricular volume (standing will cause the click to occur earlier in systole and closer to S1; squatting will cause the click to occur later in systole and closer to S2). These maneuvers, therefore, are helpful in distinguishing the systolic click of MVP from aortic and pulmonic ejection sounds. The ES is relatively fixed in timing in early systole, compared with S1, despite maneuvers that alter the ventricular volume.

Opening Snap. In the normal heart, the anterior and posterior leaflets of the mitral valve open widely and no sound is produced. In MS, the free edges of the two thickened mitral valve leaflets are bound together; however, the belly of the anterior leaflet is still flexible and domes forward at the onset of LV filling. When it reaches its limit, a high pitched sound, the opening snap (OS) occurs in early diastole (Fig 10). The OS is usually heard best with the diaphragm of the stethoscope at the lower left sternal border and apex but can transmit widely. The timing of the OS relative to aortic valve closure (A2-OS interval) is a valuable clue to the severity of MS. In general, the more severe the MS, the higher the LA
pressure, the earlier the OS, and the shorter the A2-OS interval. Of note, the intensity of an OS correlates with valve mobility. A loud OS (often accompanied by a loud S1) implies a pliable mitral valve that is not heavily calcified. Significant MS may be present in the absence of an OS, however, if the mitral valve leaflets are fixed and immobile. When an OS is suspected, the clinician should also listen for a loud S1 and P2 and search carefully for the characteristic diastolic rumble of MS (see below).

Pericardial Knock Sound. The pericardial knock sound of constrictive pericarditis is produced by the sudden arrest of blood flow by the rigid pericardial sac during diastolic filling of the ventricles. In general, the knock sound is higher in pitch and in intensity than the S3 gallop, is widely transmitted over the precordium, and usually becomes louder with inspiration.

The more severe the constrictive process, the earlier and louder the knock sound. Following successful surgery, the sound becomes later and

![Mechanism of opening snap of mitral stenosis](image)
fainter. Because of its characteristic higher frequency, the pericardial knock sound may be mistaken for an OS. The presence of this early diastolic heart sound, however, in association with an elevated JVP increasing during inspiration (Kussmaul’s sign) and in the absence of a loud S1 and diastolic rumble of MS, should provide the clinician with an immediate clue to the diagnosis of constrictive pericarditis. Nowadays, one should specifically search for these findings in the patient who presents with unexplained heart failure (especially right-sided) following cardiac surgery.89

Tumor Plop. An early diastolic heart sound called a “tumor plop” may also be created by an LA myxoma. The sound is heard as the mobile pedunculated tumor attached by a long stalk to the interatrial septum abruptly stops short as it descends through the mitral valve into the LV during early diastole. Although the sound is usually later in diastole than an OS and lower in pitch, it may vary in intensity as the tumor is altered in position with various postural changes. At times, however, the tumor plop has the same early diastolic timing as an OS and can be confused with it.

Heart Murmurs: Systolic, Diastolic, and Continuous

A heart murmur is defined as a prolonged series of audible vibrations that arises when rapid blood flow occurs across a normal structure (eg, an innocent systolic murmur), turbulent flow occurs across an abnormal valve (eg, congenital bicuspid aortic valve) or a constricted valve (eg, AS, PS), regurgitant flow occurs through an incompetent valve (eg, MR, AR), flow occurs into a dilated vessel (eg, the aortic root or pulmonary artery in systemic or pulmonary hypertension, respectively), or blood is shunted from a high to a low pressure area through an abnormal opening (eg, VSD).90-92

Classification and Grading of Heart Murmurs

Heart murmurs are described according to their timing in the cardiac cycle (systolic, diastolic, or continuous), intensity (faint, loud), frequency (low, high), duration (short, prolonged), configuration (eg, crescendo [increases progressively in intensity], decrescendo [decreases progressively in intensity], crescendo-decrescendo [diamond shaped], plateau [relatively constant]), location, radiation, quality (eg, harsh, blowing, rumbling), and the effect of respiration and/or other physiologic maneuvers (eg, position change, Valsalva) on their intensity. The intensity of a murmur is graded on a scale of 1 to 6. A grade 1 murmur is a faint murmur heard only with special effort; a grade 4 murmur is a loud
murmur accompanied by a palpable thrill, and a grade 6 murmur is audible with the stethoscope removed from the chest wall (Table 8).

Heart murmurs are present in many individuals, both with and without cardiac disease. In the last few decades, the prevalence of cardiac diseases that cause the most common heart murmurs heard in clinical practice has changed. With the declining incidence of acute rheumatic fever, the most common conditions include aortic sclerosis of the elderly (which recently has been linked to atherosclerosis and an increased risk of acute MI and stroke), valvular AS, MR due to MVP, papillary muscle dysfunction (as seen in acute myocardial ischemia or infarction, dilated cardiomyopathy, LV systolic dysfunction from any cause), or calcified mitral annulus (especially in elderly females), and HOCM. The recent onset of a heart murmur (eg, acute MR, AR) may provide a clue to infective endocarditis, especially if accompanied by a fever, or a serious mechanical complication (eg, acute VSD, MR) in a patient with acute MI.

The major systolic, diastolic, and continuous murmurs are summarized in Table 9.

### Systolic Murmurs

The most common auscultatory finding on cardiac auscultation is a systolic murmur. These murmurs can be “innocent” (normal) or “significant” (pathologic).

**Innocent versus Significant Systolic Murmur.** It is particularly important to be able to distinguish between an innocent and a significant (or “guilty”) murmur. Early and mid systolic murmurs less than grade 3 may be “innocent” or “significant” when accompanied by other signs or symptoms of cardiac pathology. Loud systolic murmurs (grade 3 or greater) are more likely to be hemodynamically significant and due to underlying heart disease. The loudness of a murmur, however, while commonly reflecting the severity of the underlying abnormality, does not always correlate. For example, flow across a small VSD is frequently

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

(Reproduced with permission from Chizner MA. Clinical Cardiology Made Ridiculously Simple, 2nd edition. Miami, FL: MedMaster, Inc., 2007.)
associated with a loud murmur because of the turbulence, whereas the systolic murmur of severe AS may lessen in intensity or even be inaudible with CHF and a low cardiac output. The length of a murmur is often more indicative of the severity of a lesion than its intensity. Holosystolic, late systolic, diastolic, and continuous murmurs (with the exception of jugular venous hum and mammary souffle of pregnancy) virtually always signify an abnormality of cardiac structure or function.

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 physical findings may increase the likelihood that the murmur is significant. Certain abnormal heart sounds that reflect cardiac pathology may all serve as immediate clues to the presence of a “significant” murmur. These abnormal sounds include “fixed” splitting of S2 in ASD, an ES in congenital bicuspid aortic valve or PS, a systolic click in MVP, a loud “tambour” A2 in systemic hypertension, and a loud, closely split or single (fused) S2 with an accentuation of P2 in pulmonary hypertension. 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 that can be found in children, young adults, athletes, pregnant women, and those with a hyperkinetic circulatory state (as brought on by fever, anemia, exercise, excitement, hyperthyroidism) is a faint (grade 1-2/6), early to mid-systolic, crescendo-

<table>
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<th>TABLE 9. Clinical clues to heart disease from cardiac auscultation: Heart murmurs</th>
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<tr>
<td>Clue</td>
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<tr>
<td>● Systolic murmurs</td>
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<tr>
<td>Early-mid</td>
</tr>
<tr>
<td>Holosystolic</td>
</tr>
<tr>
<td>Late systolic</td>
</tr>
<tr>
<td>● Diastolic murmurs</td>
</tr>
<tr>
<td>Early (high-frequency)</td>
</tr>
<tr>
<td>Mid-late (low frequency)</td>
</tr>
<tr>
<td>● Continuous murmurs</td>
</tr>
</tbody>
</table>

(Reproduced with permission from Chizner MA. The diagnosis of heart disease by clinical assessment alone. Curr Probl Cardiol 2001;26:344.)
decrescendo murmur, often with a musical, vibratory, or buzzing quality (Still’s murmur). It occurs during early RV or LV ejection and is heard best over the pulmonic area or mid left sternal border but may be heard at the apex and aortic areas as well. It is characteristically accompanied by normal respiratory splitting of S2, a physiologic S3 at the cardiac apex, waxing and waning with respiration, and a jugular venous hum, in the absence of abnormal heart sounds (eg, ejection sounds, clicks, gallops) or other systolic or diastolic murmurs. When heard in the asymptomatic individual without any clinical manifestations of heart disease (along with a normal ECG and chest X-ray), the “innocent” systolic murmur can be diagnosed with a high degree of certainty, without the need for more specialized imaging techniques (eg, echocardiography) (Fig 11).  

**Early to Mid Systolic (Ejection) Murmurs.** Systolic murmurs can be divided into ejection murmurs and regurgitant murmurs (Fig 12). Ejection murmurs begin after S1, are crescendo-decrescendo in configuration (ie, they have a rise and fall in their intensity), and stop before S2. They reflect turbulent blood flow across the aortic or pulmonic valve or LV or RV outflow tract.

As previously mentioned, a short, early to mid systolic ejection murmur heard best at the right or left second intercostal space or left sternal border...
is common in children and young adults and is considered an “innocent” (normal or physiologic) systolic flow murmur. Significant systolic murmurs, however, may present as a systolic ejection murmur of similar character, length, and configuration. The judgment that cardiac pathology is present, therefore, is often based not only on the presence of the murmur but also on other abnormal physical findings, eg, an ES, a systolic click, abnormal S2 splitting, or an abnormal gallop.

A frequent clinical problem is the differentiation of AS from aortic valve sclerosis, ie, aortic leaflet thickening without stenosis. Aortic sclerosis is common in elderly patients; 25% of those >65 years of age are affected. The condition is often diagnosed when a short early to mid systolic murmur, generally of grade 1 or 2 intensity, with no ES, a normal

FIG 12. Systolic murmurs can be divided into ejection murmurs and regurgitant murmurs. (Left) The classic holosystolic (pansystolic) murmur of MR begins with S1 and continues up to and through the aortic component of S2, since LV pressure continues to exceed LA pressure. (Right) The classic mid systolic ejection murmur occurs during the period of LV ejection. It begins after S1, is crescendo-decrescendo in nature, and stops before S2. (Reproduced with permission from Vandenbelt RJ, Ronan JA Jr, Bedynek JL Jr. Cardiology: A Clinical Approach, 2nd edition. Chicago, IL: Yearbook Medical Publishers, 1987.)
carotid pulse, and a normal S2 is detected in an otherwise asymptomatic elderly patient during a routine physical examination. When a systolic murmur becomes prolonged (ie, occupies more and more of systole), the likelihood of organic heart disease increases.

In general, all forms of obstruction to left or right ventricular outflow (eg, valvular AS, PS) result in the presence of a systolic ejection murmur. As a rule, the more severe the degree of obstruction, the longer the corresponding murmur, peaking later in systole. The location of maximal intensity and the direction of radiation of the murmur (aortic area to right shoulder for valvular AS, pulmonic area to left shoulder for PS) provide important clues to the site of obstruction (Fig 13).

The murmur of AS can also radiate to the apex, where it may assume a more musical quality (Gallavardin phenomenon). The clinician should remember to also listen over both clavicles; since bone is such a good transmitter of sound, the murmur of AS may even be louder here than in the neck. If a high-frequency musical systolic murmur is heard at the apex, the clinician should always rule out AS. The patient is usually an elderly man, who has an increase in the anterior-posterior diameter of the chest.

In HOCM the systolic flow murmur across the aortic outflow tract originates in the LV cavity and is usually maximal at the lower left sternal border and apex, with relatively little radiation to the carotids. The murmur is usually midsystolic and harsh in quality. As previously mentioned, it increases on standing and decreases on squatting, because the dynamic outflow obstruction created by the proximity of the anterior mitral valve leaflet and the hypertrophied ventricular septum is relieved temporarily by the increased ventricular volume on squatting. Additional auscultatory findings may include a paradoxical splitting of S2 (delayed closure of the aortic valve) when the LV tract obstruction is severe, and a systolic murmur of MR at the cardiac apex (HOCM may cause MR by distorting the mitral valve apparatus). The murmur of HOCM can be increased by a Valsalva maneuver or by having the patient move from the squatting to the standing position; this reduces venous return and decreases the size of the LV, thereby increasing the obstruction to blood flow.

It should be noted that early to mid systolic murmurs are not necessarily related to “ejection.” For example, the murmur of MR may be early systolic (acute MR) or midsystolic (papillary muscle dysfunction), in addition to late systolic (MVP or papillary muscle dysfunction) or holosystolic (chronic MR) (see below).
Auscultatory findings in mild, moderate, and severe aortic stenosis. An aortic ejection sound (ES) follows the first heart sound (1) in the mild type. A midsystolic ejection murmur (SM) ends before the normal aortic component ($A_2$) of the second heart sound (2). With progression of the disease (moderate stenosis), the systolic ejection murmur peaks later in systole and splitting is minimal since the faint aortic component is synchronous with the pulmonic component ($P_2$). The ejection sound becomes faint or absent as the valve becomes less mobile. A fourth heart sound or atrial gallop (a) (reflecting decreased LV compliance), along with a late peaking systolic ejection murmur, and a paradoxically split second heart sound (2) are heard in the severe type. The faint aortic component is delayed, occurring after the pulmonic component and splitting as the pulmonic area is wider on expiration than inspiration. (Reproduced with permission from Segal BL. Clinical recognition of rheumatic heart disease. In: Chizner MA, editor. Classic Teachings in Clinical Cardiology: A Tribute to W. Proctor Harvey, MD. Newton, NJ: Laennec, 1996. p. 1007.)
Holosystolic Murmurs. If a murmur extends throughout all of systole (ie, holosystolic or pansystolic murmur), the clinician should think of three possibilities: MR, TR, and VSD (Fig 14). MR is the most common of the three. The location of maximal murmur intensity, radiation, and, in the case of TR, inspiratory increase in murmur intensity all help to determine which of the three entities is present. The classic holosystolic murmur of MR or TR begins with S1 and continues up to or even through S2, since ventricular pressure continues to exceed atrial pressure. Similarly, a holosystolic murmur occurs in VSD, since LV pressure continues to exceed RV pressure throughout all of systole.

Although often plateau in shape, murmurs of MR may have a mid systolic accentuation or be distinctly louder in early or late systole. In general, the severity of chronic MR is not reflected in the intensity of the systolic murmur, but by the accompanying diastolic events. Mild MR is characterized only by a systolic murmur. Moderate MR is accompanied by an S3 gallop. The high-frequency blowing murmur occupies all of systole (and obliterates S1 and S2), and the low-frequency S3 gallop occurs in early diastole. Severe MR has an associated diastolic flow rumble, which results from increased mitral flow in diastole. S2 in severe MR is widely split over the pulmonic area and widens further on inspiration (due to early closure of the aortic valve). The holosystolic murmur of chronic MR is usually high in frequency and is best heard at the apex but may radiate to the left axilla and back (when the anterior leaflet is involved) or to the aorta and base of the heart (when the posterior leaflet is involved).

The holosystolic murmur of TR (associated with pulmonary hypertension) is heard best at the lower left sternal border and increases in intensity during inspiration (Carvallo’s sign). This murmur may be quite elusive. It is often sharply localized and of low intensity. Significant TR may even be present without a murmur, in which case a large jugular V-wave or pulsatile liver may be the clue.

The holosystolic murmur of VSD (congenital or acquired, eg, secondary to rupture of the interventricular septum in acute MI) is a harsh murmur heard best along the left sternal border, does not radiate significantly, and is often accompanied by a palpable thrill (ie, grade 4 or higher).

At times, it may be difficult to differentiate between the murmur of valvular AS and MR, especially when the murmur of MR at the apex is early or midsystolic, as may occur with ischemic MR (due to papillary muscle dysfunction) or functional MR (secondary to LV dilatation) and radiates to the base (aortic area). As previously mentioned, a valuable clue is to listen specifically to the murmur after a pause (with a PVC or
When one hears a holosystolic murmur, three conditions should come to mind: mitral regurgitation, tricuspid regurgitation, and ventricular septal defect. (A) Note holosystolic murmur (SM) of chronic mitral regurgitation that radiates in a “band-like” fashion from the apex to the left axilla and posterior lung base. When hemodynamically significant, an S3 gallop and a short diastolic murmur (dm) or “flow rumble” is also present. (B) Note holosystolic murmur (sm) of tricuspid regurgitation heard best at the lower left sternal border, which increases in intensity (SM) during inspiration (Carvallo’s sign). Exp, expiration; insp, inspiration. (C) Note holosystolic murmur of acute ventricular septal defect secondary to rupture of the interventricular septum louder along the left sternal border than the apex in a patient with an acute myocardial infarction. A systolic thrill can frequently be palpated. (Courtesy of W. Proctor Harvey, MD.)
atrial fibrillation). The murmur of AS increases in intensity, whereas the murmur of MR shows little change. The clinician should keep in mind that most patients with isolated aortic valve disease are in normal sinus rhythm. The presence of atrial fibrillation in and of itself may provide a clue to mitral valve disease. Aortic valve disease is also more common in men, whereas mitral valve disease is more common in women.

Although the systolic murmur of chronic MR, TR, or VSD is usually holosystolic, patients with acute severe MR into a noncompliant LA often have a loud early to mid systolic murmur that diminishes as the pressure gradient between the LV and the LA decreases in late systole. An early systolic murmur is also a feature of TR occurring in the absence of pulmonary hypertension, and a very small VSD in which the shunt is confined to early systole. When a large VSD occurs in the setting of severe pulmonary hypertension, the systolic murmur may decrease or disappear in late systole.

Often, mild valvular regurgitation, detected by color-flow Doppler echocardiography, is not associated with an audible murmur despite optimal cardiac auscultation. This degree of mild (“physiologic”) regurgitation usually does not indicate clinical heart disease. Trivial MR can be detected by Doppler echocardiography in up to 45% of normal individuals, and TR in up to 70%. An over-interpretation of the significance of mild valvular regurgitation by echocardiographers frequently results in a misdiagnosis of “echocardiographic heart disease,” resulting in unnecessary patient anxiety and further testing in the future.

**Late Systolic Murmurs.** Although the systolic murmur of MR is usually holosystolic, the murmur can also be late systolic. Late systolic murmurs may occur from MR that is secondary to ischemia (papillary muscle dysfunction) or LV dilatation (functional MR). As previously mentioned, MVP can present with just a single systolic click, multiple clicks, or a click(s) along with a late systolic (or sometimes holosystolic) murmur. Occasionally, a loud systolic “whoop” or “honk” of MR will be heard. When the murmur of MR is confined to late systole, the regurgitation is usually mild.

**Diastolic Murmurs**

Diastolic murmur begin with or after S2 and end at or before S1. All diastolic murmurs are pathologic. They include the following:

† Early diastolic murmurs (AR and PR)
† Mid/late rumbling murmurs (MS and tricuspid stenosis [TS])
In general, the loudness of a diastolic murmur correlates with the severity of the underlying abnormality.

**Early Diastolic Murmurs.** Aortic or pulmonic regurgitation results in an early diastolic murmur due to back flow through the aortic or pulmonic valves during diastole. In the case of AR, the murmur is of high frequency, decrescendo in configuration, and “blowing” in character. To simulate the murmur, the clinician should purse his or her lips tightly and blow. Most commonly, the diastolic murmur is loudest along the left sternal border and aortic area (Fig 15). When heard best along the right sternal border in the third and fourth intercostal spaces (so-called “right-sided” diastolic murmurs), the clinician should consider unusual causes of AR due to aortic root pathology (eg, dissection, aneurysm) (Fig 16). The practitioner may previously have suspected its presence because of an aortic ES, an abnormal right second interspace lift (suggesting aneurysmal dilatation of the aorta), or a history of previous hypertension, current chest or interscapular back pain, and unequal upper extremity pulses, suggesting aortic dissection. When the diastolic murmur is musical (“cooing dove” murmur), it usually signifies eversion or perforation of an aortic cusp, as may occur with infective endocarditis.

**FIG 15.** A patient with a congenital bicuspid aortic valve and aortic regurgitation. Note the high-frequency diastolic murmur (DM) heard along the third left sternal border (3L) and the aortic ejection sound (ES), the hallmark of such a valve, which can be heard at the apex as well as over the aortic area. This faint aortic diastolic murmur might be overlooked unless one listens with the patient sitting upright, leaning forward, breath held in deep expiration, applying firm enough pressure on the diaphragm of the stethoscope to leave an imprint on the chest wall. (Courtesy of W. Proctor Harvey, MD.)
Although often readily apparent, a faint, grade 1-2/6 murmur of AR can easily be missed because its frequency closely approximates ambient room noise (eg, air conditioning). A careful search is needed to properly elicit the murmur, with the patient sitting upright and leaning forward, with the breath held in full expiration, and the examiner applying firm pressure on the diaphragm of the stethoscope, enough to leave an imprint on the skin. The clinician should keep in mind that certain factors, eg, the systemic arterial vasodilation of pregnancy, can decrease the intensity and audibility of regurgitant murmurs, eg, AR and MR. Patients with AR often have a systolic murmur as well due to the large stroke volume even in the absence of any obstruction. When loud, the systolic flow murmur may distract the inexperienced clinician from the subtle but diagnostic diastolic murmur. The peripheral findings of a rapid rising arterial pulse or wide pulse pressure often are the first clues that an AR murmur will be heard.
A poorly appreciated and seldom used maneuver for auscultation of the diastolic murmur of AR is to have the patient lying on his or her stomach, propped up on the elbows. This position moves the heart closer to the chest wall and is also especially useful to detect a pericardial friction rub and enhance the intensity of heart sounds and murmurs in some patients having a pericardial effusion. The clinician should keep in mind that patients having a slight leak of the aortic valve can be completely asymptomatic. It is only when the leak becomes significant that the murmur, being louder, is heard, and other symptoms and signs occur. The typical “to and fro” systolic and diastolic murmurs of advanced degrees of AR are best heard along the mid left sternal border. If, when taking the blood pressure, the patient has a very wide pulse pressure (eg, 160-170/40 down to 0), the presence of a loud aortic systolic murmur (even with a palpable thrill) represents flow, not AS.

The clinical presentation of a regurgitant pulmonary valve varies depending on whether pulmonary artery pressure is normal or elevated. Pulmonary valve insufficiency with normal pulmonary artery pressure is usually the result of congenital valve insufficiency, in association with idiopathic dilatation of the pulmonary artery. The murmur is usually of low or medium frequency, best heard at the second left interspace or left sternal border. It starts at some interval immediately after P2, most often has a crescendo-decrescendo configuration, and increases with inspiration. In the congenital variety, all clinical parameters other than the murmur may be normal.

When PR occurs along with pulmonary hypertension, the diastolic murmur may assume the same high-frequency, decrescendo character as that noted in AR. However, there are often stigmata of pulmonary hypertension, such as a loud P2, a pulmonic ES, and an abnormal RV lift (RV hypertrophy) to help distinguish it from AR.

Of note, mild “physiologic” PR can be detected by Doppler echocardiography in a high percentage (up to 88%) of normal individuals and usually does not indicate clinical heart disease. AR is encountered much less frequently in normal persons and its incidence increases with advancing age.

**Middiastolic and Presystolic Murmurs.** Murmurs resulting from turbulent flow across the mitral valve tend to be of low frequency and thus are best heard with the bell of the stethoscope lightly applied at the cardiac apex with the patient in the left lateral position. MS results in a diastolic rumbling murmur, usually preceded by an OS. In normal sinus rhythm, a presystolic crescendo murmur up to a loud S1 is common. Similar murmurs may be heard with obstructing atrial myxomas, but the
latter commonly fluctuate with the patient’s position, as the tumor moves toward or away from the mitral valve opening.

In MS the typical rumbling diastolic murmur with presystolic accentuation is best heard over the cardiac apex using light pressure on the bell of the stethoscope with the patient turned to the left lateral decubitus position. The early to mid diastolic (Fig 17) part occurs during the phase of early ventricular filling; the presystolic accentuation of the murmur occurs in patients with sinus rhythm during the phase of atrial contraction (but has been reported even with atrial fibrillation). With mild MS only a presystolic murmur may be present. The duration of the murmur is more reliable than its intensity as an index of the severity of valve obstruction. When the stenosis is severe, the diastolic murmur is prolonged.

The murmur of MS is often missed. Improper positioning and/or use of the bell of the stethoscope are important reasons. Frequently, the murmur is confined to a very small area (the size of a quarter) over the cardiac apex, and the bell of the stethoscope must be applied over this small area with the patient turned to the left. The left lateral decubitus position brings the apex closer to the chest wall and overlying stethoscope, which increases the audibility of the low intensity murmur. Appreciating a loud S1, a high-pitched OS and a loud P2 are helpful clues. The clinician should always keep in mind, if a loud S1 is heard, that a search for the diastolic rumble of MS should be conducted. If the murmur is not audible, exercise (eg, sit-ups) may increase mitral flow and “bring out” the murmur.
Tricuspid valve events, in contrast to the mitral valve, are usually best appreciated along the lower left sternal border rather than the cardiac apex. Tricuspid valve obstruction, although rare, results in a rumbling diastolic murmur similar to that noted in MS. As a right-sided event, inspiratory augmentation in intensity of the murmur helps to distinguish its tricuspid valve origin.

In severe AR, the regurgitant jet from the aorta into the LV may strike the ventricular surface of the anterior mitral leaflet, moving it toward a more closed position. At the same time, blood flow from the LA to the LV tends to move the anterior leaflet to a more open position. The relative narrowing of the effective mitral valve orifice may result in a mid or late diastolic low-pitched rumbling murmur due to mitral inflow at the cardiac apex ("Austin-Flint" murmur), which can mimic the murmur of MS (although there is no OS). Ventricular vibrations caused by the AR jet itself is another proposed mechanism for the Austin-Flint murmur. The Austin-Flint murmur is present only when the degree of AR is moderate or severe.

**Continuous Murmurs.** Continuous murmurs last throughout all of systole and continue uninterrupted into at least early diastole. They are generated by continuous flow from a vessel or chamber with high pressure into a vessel or chamber with low pressure. Continuous murmurs can result from an abnormal communication between the aorta and pulmonary artery (eg, PDA), abnormal connections between arteries and veins (eg, arteriovenous fistulas), abnormal flow in veins (eg, venous hums), or abnormal flow in arteries (eg, renal artery stenosis, carotid artery occlusive disease). Continuous jugular venous hums are frequently heard in children and young adults, especially during pregnancy (along with the mammary arterial souffle—a systolic/diastolic murmur due to blood flow through the superficial mammary arteries heard over the breast in late pregnancy and lactation), and in thyrotoxicosis, in anemia, or in persons with a hyperdynamic circulatory state. These innocent venous hums are best heard over the right internal jugular vein at the base of the neck with the patient’s head turned to the opposite direction ("on a stretch") but occasionally may be loud enough to be transmitted to the upper chest where they may be confused with a serious cardiac murmur (Fig 18). Gentle pressure over the jugular vein generally eliminates the hum, as does turning the patient’s head to the forward position. The mammary souffle can usually be obliterated with firm pressure applied to the diaphragm of the stethoscope over the engorged breast.

Many continuous murmurs do not actually occupy the total cardiac cycle. Characteristically they begin in systole and spill over into diastole.
The recognition of the exact cause of the continuous murmur is aided by the location of its maximal intensity (murmur peaking at S2, murmur peaking in both systole and diastole, or murmur accentuated in diastole alone or systole alone). The typical continuous “machinery” murmur of PDA, for example, is maximal at the first and second left intercostal spaces and peaks and “envelops” S2 (Fig 19). Peripheral AV fistulas produce a continuous murmur with systolic accentuation. When present, they should be considered a potential cause for CHF. Severe systemic arterial stenosis may also result in a continuous murmur that typically peaks in systole. Sinus of Valsalva aneurysms may cause a continuous murmur when they rupture into the right heart (usually following trauma or exertion). They are heard maximally at the lower sternal border or xiphoid area and peak in diastole. The continuous murmur in coronary AV fistula tends to crescendo-decrescendo both in systole and in diastole, the diastolic component being louder. The murmur of a venous hum peaks in diastole, not over S2.

Miscellaneous

Pericardial Friction Rub. Normally, movement of the heart within the smooth, lubricating pericardial sac is silent. With roughening or inflammation of the pericardium, a “friction rub” may be produced. The pericardial friction rub is a coarse, leathery, or scratchy series of sounds best heard along the left sternal border, exerting firm pressure on the
PATENT DUCTUS ARTERIOSUS

FIG 19. The auscultatory findings in a patient with patent ductus arteriosus. Note continuous murmur heard over the pulmonic area that envelops the second heart sound ($S_2$). If the murmur does not envelop $S_2$, although sounding continuous, it may be a clue to a cause other than patent ductus. (Courtesy of W. Proctor Harvey, MD.)

diaphragm of the stethoscope with the patient sitting upright, breath held in deep expiration. These to and fro noises represent friction during the different parts of the cardiac cycle. They may be mistaken for both systolic and diastolic heart murmurs but can be differentiated by their rough, superficial quality (sounds closer to the listening ear) and by the fact that they have at least two or three components that are synchronous with ventricular contraction (systolic), ventricular relaxation (early diastolic), and/or atrial contraction (late diastolic or presystolic) (Fig 20). In approximately one-third of patients, only two components are heard (usually the systolic component and the presystolic component). The pericardial friction rub is often quite changeable in character from day to day or even minute to minute. The transient nature of the sounds also provides a clue to their pericardial origin. They occur in patients with acute pericarditis, after acute MI, cardiac surgery, chest injury, and in association with uremia, malignancy, or connective tissue diseases. Rubs have been compared to the creaking leather of a new saddle, the sound of sandpaper, or the crunching sound made by stepping on dry snow. Pericardial friction rubs generally become louder with inspiration and may occur even with a large pericardial effusion.

Prosthetic Valve Sounds. Ejection sounds may accompany the opening movement of a mechanical prosthetic aortic valve, eg, the Starr–Edwards
ball-in-cage valve, Bjork–Shiley tilting-disk, and St. Jude bileaflet prosthesis. The relative intensity of these sounds varies according to the type and design of the prosthetic valve. The ball-in-cage valve produces the loudest sounds, simulating the rolling of dice as the ball “jiggles” at the top of the cage during systole. They are normally absent, however, with a porcine heterograft (tissue) valve, eg, Hancock–Carpentier–Edwards bioprosthesis. Alteration in intensity, presence, and timing of these distinctive sounds and the characteristic features of associated murmurs provide clues to the state of prosthetic valve function. The disappearance or absence of previously heard opening or closing sounds with a mechanical aortic prosthesis and the development of a new murmur usually indicates significant prosthetic valve dysfunction, eg, due to thrombus, tissue ingrowth, or ball variance.

Early diastolic sounds may also be caused by the opening movement of a mechanical mitral valve prosthesis, eg, Starr–Edwards ball-in-cage, Bjork–Shiley tilting-disk, and St. Jude bileaflet valve. The sounds vary in intensity and quality, depending upon the design of the prosthesis. A change in the intensity or timing of the prosthetic mitral valve opening

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**PERICARDIAL FRICTION RUB**

FIG 20. A typical three-component pericardial friction rub (as, vs, vd), which generally gets louder with inspiration is present. To detect a pericardial friction rub, listen along the left sternal border exerting firm pressure on the diaphragm of the stethoscope with the patient sitting upright, breath held in deep expiration. The ventricular systolic component (VS) in this patient is loud and has a musical component. (Courtesy of W. Proctor Harvey, MD.)
sound on serial auscultation may serve as the first clue to the presence of a malfunction of the artificial valve.

**Approach to the Patient with a Heart Murmur.** Careful and accurate clinical evaluation of the patient with a heart murmur is one of the most common and important tasks that a clinician may be called on to perform in the daily practice of medicine. Proper interpretation will enable the experienced examiner to make appropriate and cost-effective management decisions regarding the need for further diagnostic laboratory studies, medical and/or surgical intervention, risk of noncardiac surgery, pregnancy, competitive and/or recreational sports, job-related activities, and eligibility for military service or life insurance.

The approach to the patient with a heart murmur may vary greatly depending on its intensity, timing, location, radiation, and response to various maneuvers, and the presence or absence of other cardiac or noncardiac symptoms or signs that suggest that the murmur is clinically “innocent,” ie, not indicative of cardiac pathology, or “significant,” ie, secondary to structural heart disease. One schematic approach to the patient with a cardiac murmur is depicted in the algorithm in Fig 21. Although color-flow Doppler echocardiography can provide important information about patients with heart murmurs, it is not a necessary test for many patients with cardiac murmurs and usually adds little but cost to...

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**FIG 21.** Schematic approach to the patient with a heart murmur. *If an electrocardiogram or chest X-ray has been obtained and is abnormal, echocardiography is indicated. (Courtesy of Robert A. O’Rourke, MD.)
the evaluation of asymptomatic patients with a short, grade 1-2/6 early to mid systolic murmur, and otherwise normal clinical findings (ie, “innocent” systolic murmur). As valuable as echocardiography may be, optimal cardiac auscultation by a well-trained clinician who can recognize an innocent systolic murmur with confidence results in less frequent use of expensive additional testing.

In general, most patients with diastolic or continuous murmurs (except jugular venous hum or mammary souffle pregnancy) or systolic murmurs that are loud (ie, grade 3 or greater), holosystolic, late systolic, or become louder or longer with physiologic maneuvers, eg, Valsalva or squatting to standing (suggesting the diagnosis of HOCM or MVP), or are accompanied by abnormal cardiac symptoms and/or signs, or by an abnormal ECG and/or chest X-ray should ordinarily undergo further diagnostic testing (ie, echo) and subsequent workup.

Worthy of emphasis, many recent studies indicate that, with improved sensitivity of Doppler ultrasound devices, valvular regurgitation may be detected through the tricuspid and pulmonic valves in a very high percentage of young healthy individuals and through left-sided valves in a variable but lower percentage. Thus, echocardiographic interpretation of mild or trivial (“physiologic”) valvular regurgitation may lead to the echocardiographic diagnosis of cardiac disease in patients with no clinical heart disease.107-117

Auscultatory Findings in Various Cardiac Disease States and Conditions

Cardiac disease states and conditions may cause a multitude of abnormal auscultatory findings. These auscultatory findings often enable the experienced clinician to arrive at a rapid and accurate cardiac diagnosis. Although heart disease may not always be detectable or quantifiable on cardiac auscultation, much of the information which is now being obtained by echocardiography and other imaging techniques can also be obtained safely, more conveniently, and less expensively by careful attentive auscultation of the heart and vascular system.

Coronary Artery Disease

Coronary artery disease (CAD) represents, by far, the most common cardiac problem encountered in daily clinical practice. Annually, there are more than 5 million emergency department visits for evaluation of chest discomfort suggestive of acute cardiac ischemia. Over 1 million Americans experience a new or recurrent MI each year. Many more are evaluated and treated for stable or unstable coronary syndromes.
Although the cardiac examination may be entirely normal in patients with stable CAD, careful auscultation during an acute ischemic attack may reveal supportive evidence. Clues may include a transient S4 gallop (due to decreased LV compliance) and S3 gallop (secondary to decreased LV systolic function), along with an apical systolic murmur of MR (due to papillary muscle dysfunction), or rarely, a paradoxically split S2 (due to delay of aortic valve closure secondary to poor LV function) that disappears when the pain subsides.

In patients who present with unstable angina or non-ST elevation MI, evidence of significant LV systolic dysfunction, ie, an S3 gallop and a new or worsening systolic murmur of MR (along with hypotension, tachycardia, and/or new or worsening rales), portends a higher likelihood of severe underlying CAD and a much higher risk for an adverse clinical outcome.

The spectrum of auscultatory findings in patients with an acute ST elevation MI varies greatly depending on the location and extent of myocardial damage. The blood pressure and/or pulse rate may be increased in anterior MI (due to sympathetic nervous system overactivity related to fever, pain, fear, or anxiety) and decreased in inferior MI (due to excess vagal tone and/or ischemia of the sinus and AV nodes). A normal heart rate usually indicates that the patient is not under significant hemodynamic compromise. Persistent sinus tachycardia, however, may signify significant LV systolic dysfunction and is therefore a poor prognostic sign. S1 may be faint if LV contractility is diminished, or if the PR interval is prolonged, as may occur in first-degree AV block in the setting of inferior wall MI. Variation in the intensity of S1 can be appreciated when the PR interval varies, as in Mobitz Type I (Wenckebach) second-degree AV block, complete heart block, and ventricular tachycardia.

In the setting of an anteroseptal MI, abnormally wide splitting of S2 or paradoxical splitting of S2 may be present due to the development of a new RBBB or LBBB, respectively, and is generally associated with a poor prognosis (since this usually reflects an extensive amount of damage to the LV). In addition to LBBB, paradoxical splitting of S2 may also be heard when there is severe LV systolic dysfunction. An S4 gallop, indicative of forceful atrial contraction into a noncompliant LV (diastolic dysfunction), can be heard (or felt) in almost all patients (who are in normal sinus rhythm) during or shortly after the acute ischemic event. Although the presence of an S4 gallop may not be specific enough to be diagnostic, when listened for properly by an experienced examiner, its absence argues strongly against an acute MI. An S3 gallop, along with
pulsus alternans, may also be present in as many as 25% of post-MI patients, but only if significant LV systolic dysfunction, ie, damage to 25% or more of the myocardium, has developed, and, as such, portends an adverse prognosis.

A midsystolic murmur of MR from papillary muscle dysfunction may occur at least transiently in 30 to 50% or more of patients within the first 24 hours and reflects ischemia of the papillary muscle rather than irreversible injury. In patients with acute ischemic MR secondary to papillary muscle dysfunction, prompt resolution of ischemia or restoration of coronary blood flow to an infarct-related vessel (by means of thrombolysis or percutaneous coronary intervention) may restore mitral valve competence with complete disappearance of the murmur. Approximately 10% of patients with ST segment elevation (transmural) MI have a pericardial friction rub, generally appearing 48 to 72 hours after symptom onset. In patients who receive reperfusion therapy, however, the incidence of pericardial friction rubs has diminished to 5%, presumably as a result of limiting transmural extension of the infarction. Although pericarditis itself is usually benign and self-limiting, it may be a clue to a relatively large transmural MI. Overall, patients with pericarditis have lower ejection fractions and a higher incidence of CHF.

If the patient with an acute MI develops the sudden onset of respiratory distress and/or shock, together with a new holosystolic murmur heard best along the left sternal border, or the cardiac apex, the clinician should consider the diagnosis of an acute VSD due to perforation of the ventricular septum or acute MR due to papillary muscle rupture, respectfully. The murmur of a VSD is harsh, holosystolic, and crescendo-decrescendo, is heard best at the left sternal border, and is frequently accompanied by a palpable systolic thrill. The murmur does not get louder with inspiration (as is characteristic of TR) nor does it radiate well to the axilla (as does MR). The murmur of papillary muscle rupture is typically loudest at the cardiac apex and may be accompanied by a palpable thrill. It is holosystolic but decrescendo (tapers off) in the latter part of systole.

In a patient with acute inferior wall MI, distention of the neck veins, along with Kussmaul’s sign (an inspiratory increase in jugular venous pressure), hypotension, and clear lungs, provides valuable clues to the presence of concomitant RV infarction and should direct the clinician to listen specifically along the lower left sternal border for right-sided S4 and S3 gallops and a systolic murmur of acute TR, often deceptively short, faint, or absent during expiration, increasing or “brought out” during inspiration.118-140
Congestive Heart Failure (CHF)

Overt or decompensated CHF is a very common clinical condition. In fact, CHF is the most frequently used cardiovascular hospital diagnosis-related group discharge diagnosis in the United States today. It is estimated that nearly 5 million Americans have CHF, and approximately 500,000 new cases are diagnosed each year. In general, CHF can be divided into two main categories: systolic dysfunction (due to impaired LV contractility), which is most common, and diastolic dysfunction (due to reduced LV filling).

LV Systolic Dysfunction. CHF due to LV systolic dysfunction is a common complication of many types of heart disease, eg, CAD with ischemic LV damage (ie, acute MI, ischemic cardiomyopathy), systemic arterial hypertension, dilated cardiomyopathy, and valvular heart disease with its pressure load (eg, AS) and/or volume overloading (eg, MR, AR) of the heart. The auscultatory findings can confirm the diagnosis or CHF when the patient’s history is suggestive. If CHF is due to an underlying valve lesion, specific features of that structural abnormality may be heard.

An immediate clue to the presence of LV systolic dysfunction is pulsus alternans (alternating strong and weak arterial pulse) along with alternation of heart sounds (particularly S2) and heart murmurs, if present. An S3 gallop will often be heard at the cardiac apex using light pressure on the bell of the stethoscope with the patient in the left lateral position if searched for specifically. In many cases, a faint systolic murmur of MR due to papillary muscle dysfunction and a diminished S1 due to a weak LV contraction, along with an accentuated P2 due to pulmonary hypertension, can also be heard. These findings represent some of the most commonly overlooked clues to the presence of LV systolic dysfunction in patients being examined today.

Although an S3 gallop is only a fairly sensitive (40-50%) marker of LV systolic dysfunction, when heard, it is a highly specific (90%) indicator of an elevated LV filling pressure, a reduced LV ejection fraction, and an elevated brain natriuretic peptide level. In fact, the absence of an S3 gallop, when listened for by an experienced examiner, argues strongly against an ejection fraction less than 30%. As a rule, the S3 gallop is a reliable and inexpensive tool for evaluating the patient’s clinical course and response to therapy. Its continued loud presence usually implies a poor prognosis. With effective therapy, the S3 gallop can be expected to decrease or disappear completely. The presence of an S3 gallop also has prognostic significance. An S3 gallop is associated with a higher risk of
progression to symptomatic CHF in those with asymptomatic LV dysfunction and a higher risk of hospitalization for CHF or death from pump failure in patients with overt CHF.

In some patients, the auscultatory features of right heart failure may predominate. Along with neck vein distention, ascites, and edema, these include right-sided S4 and S3 gallop sounds, and the systolic murmur of TR, louder along the lower left sternal border, increasing during inspiration.\textsuperscript{141-144}

**LV Diastolic Dysfunction.** In patients presenting with CHF and a loud S4 gallop (but with no S3 gallop), the clinician should suspect the possibility that the CHF results from diastolic dysfunction. LV diastolic dysfunction is usually associated with LV hypertrophy (CAD, small vessel disease eg, diabetes mellitus), restrictive cardiomyopathy), or the aging process. Although LV contractility is normal (or supranormal), there is impaired LV filling to such an extent that CHF and even acute (“flash”) pulmonary edema may result.

Diastolic dysfunction is responsible for \(~30 \text{ to } 50\%\) of all CHF episodes, particularly older patients with associated CAD and/or hypertension. An atrial (S4) gallop is the major auscultatory finding in these patients. Unlike the S3 gallop, the S4 gallop does not by itself denote ventricular decompensation. Instead, it is a clinically useful sign of decreased LV compliance (“stiff LV”). It can be detected, if carefully searched for, over the cardiac apex using light pressure on the bell of the stethoscope with the patient in the left lateral position. Diastolic dysfunction should also be suspected in a patient with CHF who has a normal-sized heart.

**Systemic Arterial Hypertension**

Systemic hypertension is the most common disease-specific reason for practitioner visits in the United States today. Most patients with systemic hypertension have no specific complaints or clinical manifestations other than an elevated systolic and/or diastolic blood pressure (ie, “silent killer”).

On cardiac auscultation, an apical S4 gallop due to LV hypertrophy and decreased LV compliance (diastolic dysfunction) is frequently heard in the presence of a loud “tambour” A2 (over the aortic area). There may also be an aortic systolic ejection murmur reflecting turbulence across the aortic valve, and on occasion, a “functional” diastolic murmur of AR caused by dilatation of the aortic ring (which may lessen in intensity or even disappear coincident with reduction in blood pressure from antihypertensive drug therapy). The S4 gallop can be the earliest clinical finding.
detected in patients with hypertensive heart disease and often precedes ECG and other signs of LV hypertrophy. When the LV can adapt no further to an increase in afterload, an S3 gallop along with the murmur of MR due to papillary muscle dysfunction appears, denoting the presence of LV systolic dysfunction. Although 95% of hypertensive patients have essential hypertension, the initial physical examination should be “directed” toward searching for clues to an identifiable secondary cause, eg, listening for an abdominal or flank bruit, which may be a sign of renal artery stenosis.145

Valvular Heart Disease

Valvular heart disease is one of the major types of cardiac disease encountered in clinical practice. In its earliest stages, valvular dysfunction may be detected by the presence of a specific heart murmur (produced by stenosis or regurgitation). When performed by an experienced examiner, cardiac auscultation has a sensitivity of 70% and a specificity of 98% for detection of valvular dysfunction.146-148

Aortic Stenosis. AS is the most common fatal valvular heart lesion in adults. In the older population (ie, those over 65 years of age), “degenerative” calcification of a normal trileaflet aortic valve (aortic sclerosis), now considered to be an inflammatory process related to atherosclerosis, has emerged as the most common cause.

AS may be recognized in a variety of ways. The disease is often discovered during a routine clinical examination when a heart murmur is heard. In other instances, the patient may present with angina pectoris, exertional dyspnea, episodes of effort-related lightheadedness, or a true syncopal spell. Most patients with mild to moderate valvular AS are asymptomatic and develop symptoms only when the valve obstruction becomes severe.

The hallmark of valvular AS is a harsh, grunting, crescendo-decrescendo midsystolic ejection murmur, best heard over the aortic area, radiating into the neck (carotids), over the clavicles (bone conduction), and down to the apex, where it may have a high-frequency musical quality (so-called “Gallavardin phenomenon”). In many patients, particularly the elderly, the midsystolic ejection murmur is atypical and may be heard only at the cardiac apex. The murmur of AS may be distinguished from that of MR because it is not holosystolic, and unlike the murmur of MR, it becomes louder in the beat after a PVC or following a pause in atrial fibrillation.

The length of the murmur is key. The murmur of aortic sclerosis and/or mild stenosis is not very long and does not peak late. As the severity of
the stenosis increases with time, the murmur becomes loud and prolonged, peaking later in systole; it is then accompanied by an S4 gallop (due to enhanced atrial contraction against a stiff, noncompliant hypertrophied LV); a single (absent A2) or paradoxically split S2 (secondary to prolongation of LV ejection due to severe outflow obstruction); and a palpable systolic thrill directed towards the right clavicle; along with a small, weak, slow-rising, late-peaking (pulsus parvus et tardus) arterial pulse (which may be absent in the elderly, due to thickening and lack of distensibility of the arterial wall), and a forceful, slow and sustained LV apical heave. The duration of the murmur is more important than intensity as an indicator of the severity of the obstruction (Fig 22). If advanced LV failure is present, however, the systolic murmur may become short or even absent, consequent to the marked decrease in cardiac output and reduced forward flow across the aortic valve.

Younger adults (particularly males) with isolated AS most often have a congenitally bicuspid valve. Consequently, the disease may be manifested as an aortic ES heard best at the aortic area and cardiac apex, not varying with respiration, along with a midsystolic ejection murmur of AS and, in some cases, an early high-pitched blowing diastolic murmur of AR (Fig 23). An S4 gallop in these young individuals (without other reasons for LV hypertrophy) usually indicates a hemodynamically significant degree of stenosis.149-152
Aortic Regurgitation. Chronic Aortic Regurgitation. In an age of declining incidence of rheumatic fever and syphilis, AR may be due to primary valve disease, eg, congenital bicuspid aortic valve, calcific degeneration, previous infective endocarditis, or more commonly, aortic root disease, eg, ascending aortic aneurysm, cystic medial necrosis, aortic dissection, and/or severe longstanding hypertension.

Early in the course of the disease, the patient may have symptoms attributable to an augmented stroke volume, ie, a forceful heartbeat and prominent arterial pulsations in the neck. When clinical deterioration occurs, patients may present with symptoms of left heart failure (fatigue, weakness, exertional dyspnea, orthopnea, paroxysmal nocturnal dyspnea), night sweats, or angina (due to decreased coronary artery flow secondary to the low diastolic pressure in the aorta). Patients with chronic AR usually remain asymptomatic for decades, often until the fourth or fifth decade of life, before signs and symptoms of CHF occur.

Cardiac auscultation characteristically reveals a high-frequency blowing decrescendo diastolic murmur best heard with the diaphragm applied firmly along the left sternal border with the patient in the sitting position.
leaving forward with breath held in deep inspiration (Fig 24). A wide arterial pulse pressure, ie, high systolic and very low diastolic BP (<40 mmHg and which can sometimes be heard down to 0 mmHg) may be present when AR is severe. A low-frequency apical diastolic rumble (Austin-Flint murmur) may also be noted in up to 60% of patients with moderate or severe AR. A systolic ejection murmur along the left sternal border (due to increased forward flow across the aortic valve) often is present and does not necessarily imply concomitant AS.

In the patient with chronic AR, the aortic systolic murmur (due to the large LV stroke volume) followed by the diastolic murmur can create the characteristic “to and fro” systolic and diastolic murmurs of AR. Moving the stethoscope from the left sternal border (to-FRO) to the aortic area (TO-fro) will increase the systolic murmur’s intensity. The systolic murmur in some patients can be loud (grade 4) and accompanied by a palpable systolic thrill and resemble valvular AS. It is important to realize that, in such a patient, if the systolic murmur peaks in early to mid systole, and peripheral signs of AR exist, eg, a rapid rising arterial pulse along
with a wide pulse pressure on the blood pressure readings (160-170/40-30 down to 0), no matter how loud the systolic murmur or how faint the diastolic murmur, there is no AS present, only significant regurgitation.

In general, the louder the diastolic murmur, the more severe the AR. The severity of AR, however, correlates better with the duration of the diastolic murmur rather than with its intensity. In trivial AR the diastolic murmur is very brief. In severe AR, the murmur may be holodiastolic. A low-frequency mid-diastolic and presystolic murmur (Austin-Flint murmur) superimposed on the high-frequency diastolic murmur of AR (to-fro-FRO) may also be heard. When the murmur of AR is faint, its intensity can be increased by isometric exercise, eg, a handgrip, or squatting, which increases peripheral vascular resistance. Conversely, the decreased systemic vascular resistance of pregnancy may decrease the intensity of AR. At times, the diastolic murmur of AR is louder along the right sternal border than the left and is a clue to aortic root disease, eg, aortic dissection, ascending aortic aneurysm (see below).

**Acute Aortic Regurgitation.** Acute AR can result from aortic dissection, infective endocarditis, prosthetic valve dysfunction, or trauma. The patient with acute AR usually has marked symptoms of left heart failure, which often begins abruptly and progresses rapidly to full-blown pulmonary edema (since the sudden severe leak of the aortic valve does not allow time for the LV to adapt). The peripheral arterial pulse shows a rapid rate of rise. In contrast to chronic AR, however, in acute AR the pulse pressure may not be widened, the diastolic blood pressure may be low normal, and the “to and fro” systolic and diastolic murmurs, usually heard along the left sternal border, are shorter in duration; S1 is faint or absent (an important clue to premature mitral valve closure), and there is marked sinus tachycardia, often in the setting of acute pulmonary edema (with a normal sized heart) (Fig 25). As S1 intensity diminishes or disappears, S2 may be mistaken for the absent S1, the aortic diastolic murmur misinterpreted as a systolic murmur, and AR missed completely. Prompt recognition and urgent surgical intervention in these patients can be lifesaving.153

**Mitral Regurgitation. Chronic Mitral Regurgitation.** With the decline of rheumatic fever, MVP has become the most common cause of valvular MR. Myxomatous degeneration of the mitral valve is a very common cause of mild MR in asymptomatic adults, especially in women. The severity of MR may increase with age, particularly in men.

Patients with mild to moderate chronic MR are relatively symptom-free for decades or life. LV systolic performance is satisfactory until late in the course, at which time LV function begins to deteriorate. The patient with
chronic MR may then present with a slow, insidious onset of CHF as the valvular leak becomes progressively more severe (“MR begets MR”).

The murmur of chronic MR is typically high frequency and blowing in quality, heard best at the apex, commonly radiating laterally and posteriorly in a “band-like” fashion into the axilla, left infrascapular area and back, and at times, up the spine to the top of the head (when the anterior leaflet is involved). Occasionally, the murmur of MR may radiate toward the left sternal border and base (when the posterior leaflet of the mitral valve is involved). As previously discussed, the severity of MR is not reflected in the intensity of the systolic murmur, but rather by the accompanying diastolic events. A late systolic murmur or holosystolic murmur with a late systolic accentuation usually is a clue to mild MR, especially when an S3 gallop is absent. When hemodynamically significant, the holosystolic murmur is accompanied by an S3 gallop and a short diastolic “flow rumble” at the cardiac apex. It should be noted, however, that in patients with significant MR due to acute MI with severe LV systolic dysfunction, hypotension, and a low cardiac output, the systolic murmur may be barely audible or even absent (“silent MR”). The absence of an apical systolic murmur, therefore, although decreasing the likelihood of significant MR, does not exclude it, especially in the presence of clinical shock states. Likewise, systemic arterial vasodilation of pregnancy decreases peripheral vascular resistance and can diminish the intensity of the systolic murmur of MR.154,155
Acute Mitral Regurgitation. Acute MR (caused by spontaneous chordae tendineae rupture, infective endocarditis, or papillary muscle rupture in the setting of acute MI) is a potentially lethal condition characterized by the abrupt onset of pulmonary edema and severe perfusion failure. In contrast to chronic MR, the murmur of acute MR is louder in early to mid systole, tapers off well before S2 (as LA pressure rises rapidly and decreases regurgitant flow), tends to radiate anteriorly and upward to the base and, at times, even into the neck (when the posterior leaflet is affected) where it can be mistaken for the murmur of valvular AS (Fig 26). In the acute form (predominantly in middle-aged or older males), normal sinus rhythm (rather than atrial fibrillation) and a prominent S4 gallop (normal-sized, vigorously contracting LA) are the rule. In contrast, atrial fibrillation and an S3 gallop, but no S4 gallop (due to a large, at times even giant, poorly contractile LA), are typical of the chronic type.156

**FIG 26.** Schematic representation of acute mitral regurgitation. Note the holosystolic murmur decreases in the latter part of systole because left atrial (LA) pressure significantly increases in a normal-sized (or slightly enlarged) left atrium to about mid systole and then decreases the pressure gradient and thus the regurgitant flow and murmur in late systole. [Courtesy of W. Proctor Harvey, MD.]
Mitral Valve Prolapse. The most common mitral valve abnormality is MVP, affecting ~2% of the population. There are two distinct clinical subsets of patients with this disorder. The first group is characterized by a mid systolic click with or without a late systolic murmur of mild MR. Young women (20 to 50 years of age), especially those with a thin, lean body habitus, constitute the majority of individuals affected. Most of these patients are asymptomatic.

The second group of patients with MVP are mostly men (40 to 70 years of age) who have a high likelihood of having progressive MR that necessitates mitral valve surgery. Another problem to keep in mind is that chordal rupture can cause a sudden increase in the severity of MR and an abrupt onset of severe symptoms.

In a patient with MVP the classic auscultatory findings are the systolic click along with a mid to late or holosystolic murmur or musical “whoop” or “honk” of MR. When the degree of MVP is mild, the murmur begins in late systole and tends to have a crescendo configuration. This is because the valve is competent in early systole and prolapses into the LA in late systole. In severe MVP there is a loud S1 followed by a holosystolic murmur. The loud S1 is caused by fusion of the systolic click with S1. These findings vary considerably in intensity and timing in systole with dynamic auscultation. As previously discussed, maneuvers that reduce ventricular volume (eg, standing) make the click and murmur earlier, closer to S1, whereas those that increase ventricular volume (eg, squatting) delay the click and murmur, closer to S2 (Fig 5).

In the hands of an experienced clinician, the stethoscope is the best instrument to diagnose MVP. Although echocardiography is very useful in identifying MVP, it is not needed in the majority of patients to make the diagnosis. In fact, the systolic click may be heard even when MVP cannot be demonstrated on echo.

The auscultatory findings of MVP are quite variable between patients and even in the same patient when examined at different times. The clinician can listen to a patient with MVP on one occasion and hear click(s) and on a second occasion hear no clicks or murmurs at all. The absence of the classic auscultatory findings during a single visit, therefore, does not definitively determine the presence of the disorder. It is important to carefully examine the patient in not only the supine position, but during standing, squatting, and re-standing as well. It is often only on re-standing after squatting that the murmur of MVP becomes apparent.

Worthy of mention, the click and murmur of MVP may be masked or disappear altogether with the increased blood volume and ventricular dimensions that occur during pregnancy, returning after delivery in the
postpartum state. Likewise, beta-blocking agents (frequently used to treat these patients) decrease heart rate and contractility and increase ventricular volume (allowing more time for ventricular filling) and may attenuate or abolish the classic auscultatory findings of MVP. These auscultatory phenomena, therefore, may help explain why patients with MVP may have no acoustic findings on one occasion yet prominent findings on another.157-162

**Rheumatic Mitral Stenosis.** Rheumatic fever is the major cause of MS. Although we have witnessed a dramatic decline in acute rheumatic fever in the United States (due to control of group A streptococcal infection), strep infections and acute rheumatic fever have recently reemerged as clinical problems. MS may be present for a lifetime with few or no symptoms. In most cases, there is a long asymptomatic phase, followed by subtle limitation of activity. Pregnancy (and its associated increase in blood volume) and the onset of rapid atrial fibrillation often precipitate symptoms, eg, dyspnea, fatigue, and palpitations (which may be associated with systemic embolism).

During the presymptomatic phase, a loud S1 and a faint presystolic murmur or short diastolic murmur may be all that is heard. In patients with symptomatic MS (which typically develops years to decades after the initial attack of acute rheumatic fever), the auscultatory findings are more obvious and include a loud S1, a loud closely split or single (fused) S2 due to a loud P2, a high-pitched, sharp early diastolic OS along with the characteristic low-frequency diastolic rumble of MS (Fig 27).

As previously mentioned, to best detect the diastolic rumble, the clinician should turn the patient to the left lateral position, feel the point of maximal impulse of the LV, and place the bell of the stethoscope lightly over this area. Failure to examine the patient in the left lateral decubitus position accounts for most of the missed diagnoses of MS. If a diastolic rumble of MS is present, it is almost always heard over this localized spot. In some patients, brief exercise, eg, sit-ups, increases flow and “brings out” the murmur.

Presystolic accentuation of the murmur occurs in sinus rhythm but has been reported even in atrial fibrillation when severe MS is present (in which case the mitral gradient persists throughout all of diastole). In general, the closer the OS is to S2 (the higher the LA pressure), and the longer the diastolic rumble, the greater the severity of the stenosis. Although the OS is present in most patients with MS, it may be absent in patients with stiff, fibrotic, or calcified leaflets. Absence of the OS in severe MS suggests that mitral valve replacement rather than commissurotomy or balloon valvuloplasty may be necessary.
As pulmonary hypertension develops, signs of RV failure, eg, a loud palpable P2, a right-sided S4 and S3 gallop, and a holosystolic murmur of TR at the lower left sternal border or even the apex when the RV is greatly enlarged, increasing with inspiration, along with a high-frequency early diastolic murmur of PR over the pulmonic area or mid left sternal border become evident. In most patients with MS, however, the presence of a high-pitched early diastolic murmur usually indicates coexistent AR.163-165

**Tricuspid Regurgitation.** The auscultatory findings of TR depend on whether the patient’s pulmonary pressure is high or normal. “High-
pressure” TR is commonly due to left-sided heart disease; low-pressure TR commonly results from infective endocarditis of the tricuspid valve.

The murmur of high-pressure TR is holosystolic because the elevated RV pressure exceeds RA pressure throughout systole. The holosystolic murmur is best heard at the left lower sternal border, or apex (if the enlarged RV has replaced the normal position of the LV), becoming louder during inspiration (Carvallo’s sign), and is accompanied by a right-sided S3 and/or S4 gallop and “flow rumble,” which also increase in intensity with inspiration, along with prominent “V”-waves in the neck veins, a pulsatile liver, ascites, and edema. When TR is due to pulmonary hypertension, there is a loud P2 and a high-pitched decrescendo diastolic murmur of PR that often increases in intensity during inspiration.

In a febrile IV drug abuser with tricuspid valve endocarditis, the systolic murmur may be unimpressive, or heard only on inspiration, not on expiration. If pulmonary and RV pressures are normal, the murmur of TR is confined to early systole since by mid systole RA and RV pressure are the same, thus eliminating the gradient causing the murmur (Fig 28). Of note, most (up to 70%) normal individuals have mild (“physiologic”) TR on Doppler-echo, which is not significant and goes undetected by auscultation. Although experienced examiners are generally accurate in diagnosing the systolic murmur of severe TR, mild and even moderate TR may cause minimal or even no auscultatory findings.166-168

**Hypertrophic Obstructive Cardiomyopathy**

HOCM, previously called idiopathic hypertrophic subaortic stenosis, is a genetic heart muscle disease that occurs in about 1 of 500 births. It is
one of the most common causes of sudden death in athletes below 30 years of age. A patient with HOCM may be asymptomatic or present with shortness of breath and chest discomfort, as well as palpitations, light-headedness, or syncope with exertion (due to dynamic LV outflow tract obstruction). Symptoms may begin at any age and often do not appear until mid-life (age 30s or 40s). Symptoms can develop at different rates, with long periods of stability, and often vary from day to day. Unfortunately, HOCM may cause no symptoms until the tragic episode of sudden cardiac death occurs.

The classic auscultatory finding is a midsystolic murmur. The systolic murmur of HOCM can confuse even the most skilled examiner. It peaks around mid systole, is best heard along the left sternal border and apex, and does not radiate well into the carotids or axilla. It can be faint and unimpressive, mimicking an innocent systolic murmur or systolic ejection murmur across a sclerotic aortic valve, or loud and long, and be mistaken for the murmur of MR or VSD. As previously mentioned, certain physiologic maneuvers, eg, standing and Valsalva, will increase the obstruction and make the murmur louder. The murmur may become fainter or disappear completely following prompt squatting (and passive leg raising), only to return even louder still, once the patient resumes the standing position (Fig 6). Additional clinical clues help to confirm the diagnosis, eg, a quick rising arterial pulse (which distinguishes HOCM from AS where the arterial pulse is slow to rise), a prominent S4 gallop, a palpable presystolic and double systolic apical impulse (“triple ripple”), a paradoxically split S2 (when LV outflow tract obstruction is severe), and the absence of an aortic ES or diastolic murmur of AR.169-170

**Aortic Dissection**

Aortic dissection is most common in middle-aged to older males with a history of hypertension. Patients with Marfan’s syndrome and women in their last trimester of pregnancy are also at increased risk. The abrupt onset of excruciating chest pain, “ripping” or “tearing” in quality, radiating to the back (interscapular area), arms, neck, jaw, abdomen, and even down the legs, in a middle-aged or older hypertensive male, a young woman in the last trimester of her pregnancy, or in a patient with Marfan’s syndrome, should lead the astute practitioner to consider aortic dissection. Type A dissections originate in the ascending aorta, usually within a few centimeters of the aortic valve, and either extend around the aortic arch into the descending aorta (type 1) or are limited to the ascending aorta (type 2). An auscultatory clue to an acute proximal aortic dissection is a new high-frequency diastolic blowing murmur of AR,
heard best along the right sternal border (third and fourth intercostal spaces) as compared to the left (which is usually the case for murmurs of valvular AR) (Fig 16).

The clinician should keep in mind the following formula: Chest pain (radiating through to the interscapular region) + hypertension + right-sided aortic regurgitation murmur = dissection of the first portion of ascending aorta (Harvey’s sign). This can provide a valuable clue to the diagnosis and thus enable prompt lifesaving surgical intervention.171-174

**Pericardial Disease**

In this modern era of aggressive intervention for acute coronary syndromes, investigation into such conditions as pericardial disease that are at especially high risk for developing hemorrhagic complications if inadvertently treated with thrombolytic agents and/or anticoagulant drugs is of the utmost importance.175-178

**Acute Pericarditis.** In a patient with the sudden onset of sharp, centrally located chest pain radiating to the neck, shoulder, and trapezius ridge, aggravated by deep breathing and the recumbent posture, relieved when sitting up and leaning forward, the presence of a superficial, scratchy, grating, leathery-quality three-component pericardial friction rub, heard best along the left sternal border, generally increasing in intensity with inspiration, suggests acute pericarditis.

The pericardial friction rub, although pathognomonic of pericarditis, may be missed on auscultation since it can be remarkably evanescent. Its absence, therefore, does not exclude the diagnosis. When a rub is not heard initially in a suspected case of pericarditis, frequent, repeated auscultation using firm pressure on the diaphragm of the stethoscope with the patient sitting upright and leaning forward can be rewarding in its detection. As a rule, it is wise never to diagnose a “one-component pericardial friction rub.” Unless two or three components, corresponding to atrial systole, ventricular systole (most common), and early diastolic filling of the ventricle (least common) are heard, the diagnosis of a pericardial friction rub should be withheld since most one-component sounds are usually scratchy systolic murmurs. A pericardial effusion does not always cause the heart sounds to be reduced in intensity and may not eliminate the presence of a pericardial friction rub. Rubs can be heard even in the presence of large pericardial effusions. It should be noted, however, that the diagnosis of acute pericarditis is made by documenting the clinical syndrome of chest pain, and a pericardial friction rub (along with characteristic ECG abnormalities), rather than by the presence of a pericardial effusion. In fact, acute pericarditis may be present without
significant (or any) pericardial effusion on echocardiography. Echocardiography, therefore, is useful for confirming the diagnosis when it shows even a small pericardial effusion, but the absence of effusion does not exclude the diagnosis.

In a patient with an underlying malignancy (especially lung, breast, lymphoma), nonpenetrating (eg, steering wheel) chest injury, or end-stage renal disease on dialysis, a new pericardial friction rub suggests pericardial metastases or traumatic or uremic pericarditis, respectively. Alternatively, pericarditis may be the cause of recurrent chest pain in the patient several days after an acute MI. As previously mentioned, pericardial friction rubs may be present when an infarction has extended to the pericardium, ie, transmural MI, and as such denote an infarct that is larger in size (lower ejection fraction), with a higher incidence of CHF.

**Constrictive Pericarditis.** Constrictive pericarditis is characterized by a thick, rigid, scarred, pericardium that restricts filling of all four chambers of the heart. It is usually a chronic consequence of acute or viral pericarditis but may occur with carcinoma (especially breast and bronchogenic), prior radiation therapy to the chest for malignancy, and particularly following previous cardiac surgery.

The presence of a high-pitched, early diastolic sound (pericardial knock), often becoming louder with inspiration (in the absence of a loud S1 or diastolic rumble) in the setting of unexplained, especially right-sided heart failure with markedly distended neck veins, ascites, and edema should prompt the clinician to suspect the diagnosis of constrictive pericarditis. Although usually a chronic consequence of acute or viral pericarditis, nowadays, one should search specifically for these findings in the patient who has had prior radiation therapy to the chest for malignancy, and particularly after previous heart surgery.179,180

**The Athlete’s Heart**

Clinical evaluation of the well-trained athlete may present a challenge to the clinician. Auscultatory findings that would be considered “abnormal” in less well-conditioned individuals are not uncommon in healthy young athletes. The practitioner should be aware that the physiologic effects of training may produce changes in the cardiovascular system that can mimic pathologic heart disease (so-called “athletic heart syndrome”). Common auscultatory findings in highly trained athletes include a slow heart rate, a grade 1-2/6 (“innocent”) systolic murmur, wide splitting of S1 and S2, an S3 and S4 gallop, and a jugular venous hum.

The distinction between the athlete’s heart and cardiac disease has important implications. The erroneous diagnosis of heart disease in a
normal athlete may have unfortunate consequences, including limitation of physical activity or disqualification from participation in competition. On the other hand, with certain cardiac conditions, participation in competitive athletics carries the risk for sudden death. Dynamic cardiac auscultation can often help clarify the situation. For example, athletes with a systolic murmur that becomes fainter with squatting or louder and/or longer with standing or during a Valsalva maneuver should be evaluated for HOCM, the most common cause of sudden death in athletes below the age of 30. Determining which murmurs are pathologic and which are benign is perhaps the most challenging aspect of preparticipation evaluation of the athlete.\textsuperscript{181-183}

Conclusion

This monograph is a timely reminder of the continued importance of cardiac auscultation in the contemporary practice of medicine. Despite the growing reliance on technological advances, cardiac auscultation remains a valuable and cost-effective clinical skill, often enabling the well-trained clinician to arrive at a rapid and accurate cardiac diagnosis, in many cases, without recourse to more elaborate and expensive “high-tech” investigative methods. Auscultation of the heart, however, is not an easy skill to learn and master. It requires both continued interest on the part of the student and expert mentoring by experienced clinician teachers proficient in the art of auscultation. Once learned, cardiac auscultation must be practiced repeatedly to be performed skillfully.

There are many challenges to teaching and learning cardiac auscultation. Traditional teaching methods, eg, textbooks and didactic classroom lectures, followed by a brief demonstration of heart sounds and murmurs, have yielded disappointing results. The best way to gain proficiency and greater accuracy and confidence in cardiac auscultation is to listen carefully to large numbers of actual patients under the expert guidance of an experienced clinician auscultator, obtaining “real-time” confirmation and immediate direct feedback. Regrettably, such master clinician teachers are becoming a vanishing breed. After decades of neglect and apathy, today’s medical trainees are now suffering the consequences, as there are few, if any, expert cardiac auscultators around to teach the finer points of this time-honored, but virtually lost, clinical art.

Self-directed learning with computer-based technology, eg, multimedia CD-ROMs, computer-aided auscultation devices, audio tapes or recordings heard online or downloaded onto an iPOD or other portable MP3 player (so-called “heart songs”), and heart sound simulators or manne-

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quins (eg, Harvey, the cardiology patient simulator) have all been shown to be of value at improving and reinforcing cardiac auscultatory skills. These “high-tech” adjuncts can supplement deficiencies in clinical training and enable clinicians at any level to learn, practice, and perfect their cardiac auscultatory skills at their own time, convenience, and pace.

**FIG 29.** Remembrances of master cardiac clinician and medical educator, W. Proctor Harvey, MD, who elevated the teaching and practice of clinical cardiology, particularly cardiac auscultation, to an art form. Restoration of the lost art of cardiac auscultation may be a difficult task but is well worth the effort. (Reproduced with permission from Chizner MA. Clinical Cardiology Made Ridiculously Simple, 2nd edition. Miami, FL, MedMaster, Inc., 2007.)
Despite the recognized value of these sophisticated adjunctive methods, however, they are no substitute for a “hands-on” patient-centered mentor approach to teaching and learning cardiac auscultation. The challenge for modern-day clinical educators, therefore, is to decide on what specific aspects of cardiac auscultation are most important and practical to teach and learn in today’s fast-paced health care environment.

Perhaps we should concentrate on the basics. Is there a heart murmur? Is it systolic or diastolic? Does it have the characteristics of an “innocent” murmur or a “significant” murmur? Does it change with respiration, position (eg, standing or squatting), or certain physiologic maneuvers (eg, Valsalva)? Is S2 split, and if so, how? Is there an S4 or S3 gallop? Are there any other extra heart sounds present (eg, systolic click, ejection sound, opening snap)? Does the patient have a pericardial friction rub?

Familiarity with these essentials of cardiac auscultation can dispel the panic and sense of intimidation often felt by today’s medical trainees when asked to listen to a patient’s heart. Only then will contemporary clinicians be willing to incorporate cardiac auscultation into routine clinical practice and teaching and pass along this time-honored art enthusiastically to the next generation of practicing physicians and other health care professionals.

We must not allow cardiac auscultation to become a lost art or the stethoscope a medical relic. Auscultation of the heart remains the cornerstone of the cardiac clinical examination, and when skillfully performed, can lead to fewer misdiagnoses, foster improved patient trust and confidence, and provide more effective and economically sound cardiovascular care. Rediscovering the lost art of cardiac auscultation and restoring it to its rightful place and “time-honored” status may be a tedious task, but it is well worth the effort (Fig 29).184-227

Dedication

I wish to dedicate, in loving memory, this monograph on the lost art of cardiac auscultation to Dr. W. Proctor Harvey, who instilled in those of us fortunate enough to have been trained by him a love for clinical cardiology that has greatly enriched our professional lives. From the passion for cardiology that he instilled in us springs the inspiration to pass along his rich legacy and to carry on the teaching tradition that is his.

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