06 - 246 Physical Examination of the Cardiovascular System

246 Physical Examination of the Cardiovascular System

who stand to benefit the most from specific, low-cost prevention interventions, including screening for and treatment of hypertension and elevated cholesterol. Simple, low-cost interventions, such as the “polypill”—a regimen of aspirin, a statin, and an antihypertensive agent—now show trial data with reductions in events for both primary and secondary prevention and have been added to the WHO Essential Medicines list. Third, resources should be allocated to acute, as well as secondary, prevention interventions. For countries with limited resources, a critical first step in developing a comprehensive plan is better assessment of cause-specific mortality and morbidity, as well as the prevalence, of the major preventable risk factors. In the meantime, the HICs must continue to bear the burden of research and development aimed at prevention and treatment, being mindful of the economic limitations of many countries. The concept of the epidemiologic transition provides insight into how to alter the course of the CVD epidemic. The efficient transfer of low-cost preven tive and therapeutic strategies could alter the natural course of this epidemic and thereby reduce the excess global burden of preventable CVD. ■ ■FURTHER READING Boutari C et al: A 2022 update on the epidemiology of obesity and a call to action: As its twin COVID-19 pandemic appears to be reced ing, the obesity and dysmetabolism pandemic continues to rage on. Metabolism 133:155217, 2022. Clerkin KJ et al: COVID-19 and cardiovascular disease. Circulation 141:1648, 2020. Gaziano T, Gaziano JM: Global burden of cardiovascular disease, in Heart Disease: A Textbook of Cardiovascular Medicine, 12th ed, Braunwald E (ed). Philadelphia, Elsevier, 2022. Mensah G et al: Global burden of cardiovascular diseases and risks, 1990-2022. J Am Coll Cardiol 82:2350, 2023. Tsao CW et al: Heart disease and stroke statistics–2022 update: A report from the American Heart Association. Circulation 145:e153, 2022. Section 2 Diagnosis of Cardiovascular Disorders

Physical Examination

of the Cardiovascular

System Patrick T. O’Gara, Joseph Loscalzo The approach to a patient with known or suspected cardiovascular disease begins with the time-honored traditions of a directed history and a targeted physical examination. The scope of these activities depends on the clinical context, ranging from an elective ambulatory follow-up visit to a more urgent bedside encounter. There has been a gradual decline in physical examination skills over the past few decades at every level, from student to faculty specialist, a development of great concern to both clinicians and medical educators. Classic cardiac find ings are recognized by only a minority of internal medicine and family practice residents. Despite popular perceptions, clinical performance does not improve predictably with experience; instead, the acquisi tion of new examination skills may become more difficult for a busy individual practitioner. Less time is now devoted to mentored cardio vascular examinations during the training of students and residents. One widely recognized outcome of these trends is the progressive utilization of noninvasive imaging studies to establish the presence and

severity of cardiovascular disease even when the examination findings imply a low pretest probability of significant pathology. Proponents of the use of hand-held ultrasound devices to identify and character ize structural cardiac disease have called for its incorporation into educational curricula. Techniques to improve competency in bedside examination skills include repetition, patient-centered teaching con ferences, visual display feedback of auscultatory events using Doppler echocardiographic imaging, and simulation-based training. The use of digital stethoscopes may enhance learning and is foundational to the application of computer- or artificial intelligence–assisted evaluation of auscultatory events.

CHAPTER 246 Physical Examination of the Cardiovascular System
The findings from the history and physical examination can help establish the presence, severity, and prognosis of several cardiovascular diseases. For example, observations regarding heart rate and blood pressure, signs of pulmonary congestion, and the presence of mitral regurgitation (MR) contribute importantly to bedside risk assessment in patients with acute coronary syndromes and can inform clinical decision-making before the results of cardiac biomarker testing are known. The prognosis of patients with heart failure can be predicted on the basis of the jugular venous pressure (JVP) and the presence or absence of a third heart sound (S3). Accurate characterization of car diac murmurs provides important insight into the natural history of many valvular and congenital heart lesions. Finally, the important role played by the physical examination in enhancing the clinician-patient relationship cannot be overstated. ■ ■THE GENERAL PHYSICAL EXAMINATION The examination begins with an assessment of the general appear ance of the patient, with notation of age, posture, demeanor, and overall health status. Is the patient in pain or resting quietly, dyspneic or diaphoretic? Does the patient choose to avoid certain body posi tions to reduce or eliminate pain, as might be the case with suspected acute pericarditis? Are there clues indicating that dyspnea may have a pulmonary cause, such as a barrel chest deformity with an increased anterior-posterior diameter, tachypnea, and pursed-lip breathing? Skin pallor, cyanosis, and jaundice can be appreciated readily and provide additional clues. The appearance of a chronically ill-appearing emaci ated patient may suggest the presence of long-standing heart failure or another systemic disorder, such as a malignancy. Various genetic syndromes, often with cardiovascular involvement, can also be rec ognized easily, such as trisomy 21, Marfan syndrome, and Holt-Oram syndrome. Height and weight should be measured routinely, and both body mass index and body surface area should be calculated. Knowl edge of the waist circumference and the

waist-to-hip ratio can be used to predict long-term cardiovascular risk. Mental status, level of alert ness, and mood should be assessed continuously during the interview and examination. Skin Central cyanosis occurs with significant right-to-left shunt ing at the level of the heart or lungs, allowing deoxygenated blood to reach the systemic circulation. Peripheral cyanosis or acrocyanosis, in contrast, is usually related to reduced extremity blood flow due to small vessel constriction, as seen in patients with severe heart failure, shock, or peripheral vascular disease; it can be aggravated by the use of β-adrenergic blockers with unopposed α-mediated vasoconstriction. Differential cyanosis refers to isolated cyanosis affecting the lower but not the upper extremities in a patient with a large patent ductus arterio sus (PDA) and secondary pulmonary hypertension with right-to-left to shunting at the great vessel level. Telangiectasias on the lips, tongue, and mucous membranes, as part of the Osler-Weber-Rendu syndrome (hereditary hemorrhagic telangiectasia), resemble spider nevi and can be a source of right-to-left shunting when also present in the lung. Malar telangiectasias also are seen in patients with advanced mitral ste nosis (MS) or scleroderma. An unusually tan or bronze discoloration of the skin may suggest hemochromatosis as the cause of the associated systolic heart failure. Jaundice, which may be visible first in the sclerae, has a broad differential diagnosis but, in the appropriate setting, can be consistent with advanced right heart failure and congestive hepatomeg aly. Various hereditary lipid disorders sometimes are associated with

subcutaneous xanthomas, particularly along the tendon sheaths or over the extensor surfaces of the extremities. Severe hypertriglyceridemia can be associated with eruptive xanthomatosis and lipemia retinalis. Palmar crease xanthomas are specific for type III hyperlipoprotein emia. Pseudoxanthoma elasticum, a disease associated with premature atherosclerosis, is manifested by a leathery, cobblestoned appearance of the skin in the axilla and neck creases and by angioid streaks on funduscopic examination. Extensive lentigenes have been described in a variety of development delay–cardiovascular syndromes, including Carney’s syndrome, which includes multiple atrial myxomas. Cutane ous manifestations of sarcoidosis such as lupus pernio and erythema nodosum may suggest this disease as a cause of an associated dilated cardiomyopathy, especially with heart block, intraventricular conduc tion delay, or ventricular tachycardia. PART 6 Disorders of the Cardiovascular System Head and Neck Dentition and oral hygiene should be assessed in every patient both as a source of potential infection and as an index of general health. A high-arched palate is a feature of Marfan syndrome and other connective tissue disease syndromes. Bifid uvula has been described in patients with Loeys-Dietz syndrome, and orange tonsils are characteristic of Tangier disease. The ocular manifestations of hyperthyroidism have been well described. Many patients with con genital heart disease have associated hypertelorism, low-set ears, or micrognathia. Blue sclerae are a feature of osteogenesis imperfecta. An arcus senilis pattern lacks specificity as an index of coronary heart dis ease risk. The funduscopic examination is an often-underused method by which to assess the microvasculature, especially among patients with established atherosclerosis, hypertension, or diabetes mellitus. A mydriatic agent may be necessary for optimal visualization. A fundu scopic examination should be performed routinely in the assessment of patients with suspected endocarditis and those with a history of acute visual change. Branch retinal artery occlusion or visualization of a Hollenhorst plaque can narrow the differential diagnosis rapidly in the appropriate setting. Relapsing polychondritis may manifest as an inflamed pinna or, in its later stages, as a saddle-nose deformity because of destruction of nasal cartilage; granulomatosis with polyan giitis can also lead to a saddle-nose deformity. Chest Midline sternotomy, right anterior thoracotomy, left postero lateral thoracotomy,

or infraclavicular scars should not be overlooked and may provide the first clue regarding an underlying cardiovascular disorder in patients unable to provide a relevant history. A prominent venous collateral pattern may suggest subclavian or vena caval obstruc tion. If the head and neck appear dusky and slightly cyanotic and the venous pressure is grossly elevated without visible pulsations, a diag nosis of superior vena cava syndrome should be entertained. Thoracic cage abnormalities have been well described among patients with connective tissue disease syndromes. They include pectus carinatum (“pigeon chest”) and pectus excavatum (“funnel chest”). Obstructive lung disease is suggested by a barrel chest deformity, especially with tachypnea, pursed-lip breathing, and use of accessory muscles. The characteristically severe kyphosis and compensatory lumbar, pelvic, and knee flexion of ankylosing spondylitis should prompt careful auscultation for a murmur of aortic regurgitation (AR). Straight back syndrome refers to the loss of the normal kyphosis of the thoracic spine and has been described in patients with mitral valve prolapse (MVP) and its variants. In some patients with cyanotic congenital heart dis ease, the chest wall appears to be asymmetric, with anterior displace ment of the left hemithorax. The respiratory rate and pattern should be noted during spontaneous breathing and sleep, with additional attention to depth, audible wheezing, and stridor. Lung examination can reveal adventitious sounds indicative of pulmonary edema, pneu monia, or pleuritis. Abdomen In some patients with advanced obstructive lung dis ease, the point of maximal cardiac impulse may be in the epigastrium. The liver is frequently enlarged and tender in patients with chronic heart failure. Systolic pulsations over the liver signify severe tricus pid regurgitation (TR). Splenomegaly may be a feature of infective endocarditis, particularly when symptoms have persisted for weeks

or months. Ascites is a nonspecific finding but may be present with advanced chronic right heart failure, constrictive pericarditis, hepatic cirrhosis, or an intraperitoneal malignancy. The finding of an elevated JVP implies a cardiovascular etiology. In nonobese patients, the aorta typically is palpated between the epigastrium and the umbilicus. The sensitivity of palpation for the detection of an abdominal aortic aneu rysm (pulsatile and expansile mass) decreases as a function of body size. Because palpation alone is not sufficiently accurate to establish this diagnosis, a screening ultrasound examination is advised when appropriate. The presence of an arterial bruit over the abdomen sug gests high-grade atherosclerotic disease, although precise localization is difficult. Extremities The temperature and color of the extremities, the presence of clubbing, arachnodactyly, and pertinent nail findings can be surmised quickly during the examination. Clubbing implies the presence of central right-to-left shunting, although it has also been described in patients with endocarditis. Its appearance can range from cyanosis and softening of the root of the nail bed, to the classic loss of the normal angle between the base of the nail and the skin, to the skeletal and periosteal bony changes of hypertrophic osteoarthropathy, which is seen rarely in patients with advanced lung or liver disease. Patients with the Holt-Oram syndrome have an unopposable, “fin gerized” thumb, whereas patients with Marfan syndrome may have arachnodactyly and a positive “wrist” (overlapping of the thumb and fifth finger around the wrist) or “thumb” (protrusion of the thumb beyond the ulnar aspect of the hand when the fingers are clenched over the thumb in a fist) sign. The Janeway lesions of endocarditis are nontender, slightly raised hemorrhagic lesions on the palms and soles, whereas Osler’s nodes are tender, raised nodules on the pads of the fingers or toes. Splinter hemorrhages are classically identified as linear petechiae in the midposition of the nail bed and should be distinguished from the more common traumatic petechiae, which are seen closer to the distal edge. Lower extremity or presacral edema in the setting of an elevated JVP defines volume overload and may be a feature of chronic heart

failure or constrictive pericarditis. Lower extremity edema in the absence of jugular venous hypertension may be due to profound hypoalbu minemia as seen in nephrotic syndrome or liver failure. Other causes include lymphatic or venous obstruction or, more commonly, venous insufficiency, as would be further suggested by the appearance of varicosities, venous ulcers (typically medial in location), and brownish cutaneous discoloration from hemosiderin deposition (eburnation). Pitting edema can also be seen in patients who use dihydropyridine calcium channel blockers. A Homans' sign (posterior calf pain on active dorsiflexion of the foot against resistance) is neither specific nor sensitive for deep venous thrombosis. Muscular atrophy or the absence of hair along an extremity is consistent with severe arterial insuffi ciency or a primary neuromuscular disorder. ■ ■CARDIOVASCULAR EXAMINATION Jugular Venous Pressure and Waveform The JVP is the single most important bedside measurement from which to estimate the vol ume status. The internal jugular vein is preferred because the external jugular vein is valved and not directly in line with the superior vena cava and right atrium. Nevertheless, the external jugular vein has been used to discriminate between high and low central venous pressure (CVP). Precise estimation of the central venous or right atrial pressure from bedside assessment of the jugular venous waveform can be dif ficult. Venous pressure traditionally has been measured as the vertical distance between the top of the jugular venous pulsation and the ster nal inflection point (angle of Louis). A distance >4.5 cm at 30° eleva tion is considered abnormal. However, the actual distance between the mid-right atrium and the angle of Louis varies considerably as a func tion of both body size and the patient angle at which the assessment is made (30°, 45°, or 60°). The use of the sternal angle as a reference point leads to systematic underestimation of CVP, and this method should be used less for semiquantification than to distinguish a normal from an elevated CVP. The use of the clavicle may provide an easier reference

for standardization. Venous pulsations above this level in the sitting position are clearly abnormal, as the distance between the clavicle and the right atrium is at least 10 cm. The patient should always be placed in the sitting position, with the legs dangling below the bedside, when an elevated pressure is suspected in the semisupine position. It should also be noted that bedside estimates of CVP are made in centimeters of water, and must be converted to millimeters of mercury to provide correlation with accepted hemodynamic norms (1.36 cmH2O = 1.0 mmHg). The venous waveform sometimes can be difficult to distinguish from the carotid pulse, especially during casual inspection. Nev ertheless, the venous waveform has several characteristic features, and its individual components can be appreciated in most patients (Fig. 246-1). The arterial pulsation is not easily obliterated with palpation; the venous waveform in patients with sinus rhythm is usually biphasic, while the carotid pulse is monophasic; and the jugular venous pulsation should change with changes in posture or inspiration (unless the venous pressure is quite elevated). The venous waveform is divided into several distinct peaks. The a wave reflects right atrial presystolic contraction and occurs just after the electrocardiographic P wave, preceding the first heart sound (S1). A prominent a wave is seen in patients with reduced right ventricu lar compliance; a cannon a wave occurs with atrioventricular (AV) dissociation and right atrial contraction against a closed tricuspid valve. In a patient with a wide complex tachycardia, the apprecia tion of cannon a waves in the jugular venous waveform identifies the rhythm as ventricular in origin. The a wave is not present with atrial fibrillation. The x descent defines the fall in right atrial pres sure after tricuspid valve opening, and occurs after inscription of the a wave. The c wave, which occurs as the closed tricuspid valve is pushed into the right atrium during early ventricular systole, interrupts this x descent and is followed by a further descent. The v wave represents atrial filling (atrial diastole)

and occurs during ventricular systole. The height of the v wave is determined by right atrial compliance as well as the volume of blood returning to the right atrium either antegrade from the cavae or retrograde through an incompetent tricuspid valve. In patients with TR, the v wave is accentuated and the subsequent fall in pressure (y descent) is rapid. With progressive degrees of TR, the v wave merges with the c wave, and the right atrial and jugular vein waveforms become “ventricu larized.” The y descent, which follows the peak of the v wave, can become prolonged or blunted with obstruction to right ventricular inflow, as may occur with tricuspid stenosis or pericardial tampon ade. Normally, the venous pressure should fall by at least 3 mmHg with inspiration. Kussmaul’s sign is defined by either a rise or a lack of fall of the JVP with inspiration and is classically associated with constrictive pericarditis, although it has been reported in patients with restrictive cardiomyopathy, massive pulmonary embolism, right ventricular infarction, and advanced left ventricular (LV) sys tolic heart failure. It is also a common, isolated finding in patients after cardiac surgery without other hemodynamic abnormalities. Venous hypertension sometimes can be elicited by passive leg elevation or performance of the abdominojugular reflux maneuver. When these signs are positive, a volume-overloaded state with lim ited compliance of an overly distended or constricted venous system is present. Abdominojugular reflux is produced with firm and consistent pressure over the upper portion of the abdomen, prefer ably over the right upper quadrant, for >15 s. A positive response is defined by a sustained rise of >3 cm in the JVP during the applica tion of firm abdominal pressure. The response should be assessed after 10 s of continuous pressure to allow for respiratory artifacts and tensing of the abdominal muscles to subside. Patients must be coached to refrain from breath holding or a Valsalva-like maneuver during the procedure. Performance of the abdominojugular reflux maneuver is useful in predicting a pulmonary artery wedge pressure

> “15 mmHg in patients with heart failure. Although the JVP estimates right ventricular filling pressure, it has a predictable relationship with the pulmonary artery wedge pressure. In a large study of patients with advanced heart failure, the

A V C CHAPTER 246 X Y IV II I A Physical Examination of the Cardiovascular System
V A Severe V Y A C X Mild A V Y C Normal Y X II III I B P ECG A V X V JVP Y II K I C FIGURE 246-1 A. Jugular venous pulse wave tracing (top) with heart sounds (bottom) in a patient with reduced right ventricular compliance. The A wave represents right atrial presystolic contraction and occurs just after the electrocardiographic P wave and just before the first heart sound (I). In this example, the A wave is accentuated and larger than normal due to decreased right ventricular compliance, as also suggested by the right-sided S4 (IV). The C wave may reflect the carotid pulsation in the neck and/or an early systolic increase in right atrial pressure as the right ventricle pushes the closed tricuspid valve into the right atrium. The x descent follows the A wave just as atrial pressure continues to fall. The V wave represents atrial filling during ventricular systole and peaks at the second heart sound (II). The y descent corresponds to the fall in right atrial pressure after tricuspid valve opening.

B. Jugular venous wave forms in mild (middle) and severe (top) tricuspid regurgitation, compared with normal (bottom), with phonocardiographic representation of the corresponding heart sounds

below. With increasing degrees of tricuspid regurgitation, the waveform becomes “ventricularized.” C. Electrocardiogram (ECG) (top), jugular venous waveform (JVP) (middle), and heart sounds (bottom) in pericardial constriction. Note the prominent and rapid y descent, corresponding in timing to the pericardial knock (K). (Reproduced with permission from J Abrams: Synopsis of Cardiac Physical Diagnosis, 2nd ed. Boston, Butterworth Heinemann, 2001.) presence of a right atrial pressure >10 mmHg (as predicted on bedside examination) had a positive value of 88% for the prediction of a pulmo nary artery wedge pressure of >22 mmHg. In addition, an elevated JVP has prognostic significance in patients with both symptomatic heart failure and asymptomatic LV systolic dysfunction. The presence of an elevated JVP is associated with a higher risk of subsequent hospitaliza tion for heart failure, death from heart failure, or both.

Assessment of Blood Pressure Measurement of blood pressure usually is delegated to a medical assistant but should be repeated by the examining clinician. Accurate measurement depends on body position, arm size, time of measurement, place of measurement, type of device, device size, technique, and examiner. In general, physician-recorded blood pressures are higher than both nurse-recorded pressures and self-recorded pressures at home. Blood pressure is best measured in the seated position with the arm at the level of the heart and the feet on the floor with the back supported, using an appropriately sized cuff, after 5–10 min of relaxation. When it is measured in the supine position, the arm should be raised to bring it to the level of the mid-right atrium. The length and width of the blood pressure cuff bladder should be 80 and 40% of the arm’s circumference, respectively. A common source of error in practice is to use an inappropriately small cuff, resulting in marked overestimation of true blood pressure, or an inappropriately large cuff, resulting in underestimation of true blood pressure. The cuff should be inflated to 30 mmHg above the expected systolic pres sure and the pressure released at a rate of 2–3 mmHg/s. Systolic and diastolic pressures are defined by the first and fifth Korotkoff sounds, respectively. Very low (even 0 mmHg) diastolic blood pressures may be recorded in patients with chronic, severe AR or a large arteriove nous fistula because of enhanced diastolic “run-off.” In these instances, both the phase IV and phase V Korotkoff sounds should be recorded. Blood pressure is best assessed at the brachial artery level, though it can be measured at the radial, popliteal, or pedal artery level. In gen eral, systolic pressure increases and diastolic pressure decreases when measured in more distal arteries. Blood pressure should be measured in both arms, and the difference should be <10 mmHg. A blood pres sure differential that exceeds this threshold may be associated with atherosclerotic or inflammatory subclavian artery disease, supravalvu lar aortic stenosis, aortic coarctation, or aortic dissection. Systolic leg pressures are usually as much as 20 mmHg higher than systolic arm pressures. Greater leg–arm pressure differences are seen in patients with chronic severe AR as well as patients with extensive and calcified lower extremity peripheral arterial disease. The ankle-brachial index (systolic pressure in the dorsalis pedis and/or posterior tibial artery divided by the higher of the two brachial artery pressures) is a powerful predictor of long-term cardiovascular mortality.

PART 6 Disorders of the Cardiovascular System The blood pressure measured in an office or hospital setting may not accurately reflect the pressure in other venues. “White coat hyperten sion” (elevated clinic blood pressure and normal out of clinic blood pressure) is defined by at least three separate clinic-based measure ments >130/80 mmHg and at least two non-clinic-based measure ments <130/80 mmHg in the absence of any evidence of target organ damage. Individuals with white coat hypertension may not benefit from drug therapy, although they may be

more likely to develop sus tained hypertension over time. Masked hypertension (normal or low clinic blood pressure but elevated out of clinic blood pressure) should be suspected when normal or even low blood pressures are recorded in the office in patients with advanced atherosclerotic disease, especially when evidence of target organ damage is present or bruits are audible. Higher systolic blood pressures measured with a 24-h ambulatory blood pressure device are associated with a higher risk of cardiovas cular disease and all-cause death independent of blood pressures mea sured in the outpatient setting. Orthostatic hypotension is defined by a fall in systolic pressure >20 mmHg or in diastolic pressure >10 mmHg within 3 minutes of assump tion of the upright posture from a supine position. There may also be a lack of a compensatory tachycardia, an abnormal response that sug gests autonomic insufficiency, as may be seen in patients with diabetes mellitus or Parkinson's disease. Orthostatic hypotension is a common cause of postural lightheadedness/syncope and should be assessed routinely in patients for whom this diagnosis might pertain. It can be exacerbated by advanced age, dehydration, certain medications, food, deconditioning, and ambient temperature/humidity. Arterial Pulse The carotid artery pulse occurs just after the ascending aortic pulse. The aortic pulse is best appreciated in the epigastrium, just above the level of the umbilicus. Peripheral arterial

pulses that should be assessed routinely include the subclavian, bra chial, radial, ulnar, femoral, popliteal, dorsalis pedis, and posterior tibial. In patients in whom the diagnosis of either temporal arteritis or polymyalgia rheumatica is suspected, the temporal arteries also should be examined. Although one of the two pedal pulses may not be palpable in up to 10% of normal subjects, the pair should be sym metric. The integrity of the arcuate system of the hand is assessed by Allen's test, which is performed routinely before instrumentation of the radial artery. The pulses should be examined for their symmetry, volume, timing, contour, amplitude, and duration. If necessary, simul taneous auscultation of the heart can help identify a delay in the arrival of an arterial pulse. Simultaneous palpation of the radial and femoral pulses may reveal a femoral delay in a patient with upper extremity hypertension and suspected aortic coarctation. The carotid upstrokes should never be examined simultaneously or before listening for a bruit. Light pressure should always be used to avoid precipitation of carotid hypersensitivity syndrome and syncope in a susceptible elderly individual. The arterial pulse usually becomes more rapid and spiking as a function of its distance from the heart, a phenomenon that reflects the muscular status of the more peripheral arteries and the summation of the incident and reflected waves. In general, the character and con tour of the arterial pulse depend on the stroke volume, ejection veloc ity, vascular compliance, and systemic vascular resistance. The pulse examination can be misleading in patients with reduced cardiac output and in those with stiffened arteries from aging, chronic hypertension, or peripheral arterial disease. The character of the pulse is best appreciated at the carotid level (Fig. 246-2). A weak and delayed pulse (pulsus parvus et tardus) defines severe aortic stenosis (AS). Some patients with AS may also have a slow, notched, or interrupted upstroke (anacrotic pulse) with a thrill S4 S4 S1 A2 P2 P2 S1 A2 Dicrotic notch A Dicrotic notch B S4 S4 P2 P2 A2 A2 S1 S1 Dicrotic notch C Dicrotic notch D S4 S1 A2 P2 Dicrotic notch E FIGURE 246-2 Schematic diagrams of the configurational changes in carotid pulse and their differential diagnoses. Heart sounds are also illustrated. A. Normal. S4, fourth heart sound; S1, first heart sound; A2, aortic component of second heart sound; P2, pulmonic component of second heart sound. B. Aortic stenosis. Anacrotic pulse with slow upstroke (tardus) to a reduced peak (parvus). C. Bisferiens pulse with two peaks in systole. This pulse is rarely appreciated in patients with severe aortic regurgitation. D. Bisferiens pulse in hypertrophic obstructive cardiomyopathy. There is a rapid upstroke to the first peak

(percussion wave) and a slower rise to the second peak (tidal wave). E. Dicrotic pulse with peaks in systole and diastole. This waveform may be seen in patients with sepsis or during intraaortic balloon counterpulsation with inflation just after the dicrotic notch. (Reproduced with permission from K Chatterjee, W Parmley [eds]: Cardiology: An Illustrated Text/ Reference. Philadelphia, Gower Medical Publishers, 1991.)

or shudder. With chronic severe AR, by contrast, the carotid upstroke has a sharp rise and rapid fall-off (Corrigan's or water-hammer pulse). Some patients with advanced AR may have a bifid or bisferiens pulse, in which two systolic peaks can be appreciated. A bifid pulse is also described in patients with hypertrophic obstructive cardiomyopathy (HOCM), with inscription of percussion and tidal waves. A bifid pulse is easily appreciated in patients on intraaortic balloon counterpulsation (IABP), in whom the second pulse is diastolic in timing. Pulsus paradoxus refers to a fall in systolic pressure >10 mmHg with inspiration that is seen in patients with pericardial tamponade but also is described in those with massive pulmonary embolism, hemorrhagic shock, severe obstructive lung disease, and tension pneumothorax. Pulsus paradoxus is measured by noting the difference between the systolic pressure at which the Korotkoff sounds are first heard (during expiration) and the systolic pressure at which the Korotkoff sounds are heard with each heartbeat, independent of the respiratory phase. Between these two pressures, the Korotkoff sounds are heard only intermittently and during expiration. The cuff pressure must be decreased slowly to appreciate the finding. It can be difficult to mea sure pulsus paradoxus in patients with tachycardia, atrial fibrillation, or tachypnea. A pulsus paradoxus may be palpable at the brachial artery or femoral artery level when the pressure difference exceeds 15 mmHg. This inspiratory fall in systolic pressure is an exaggerated consequence of interventricular dependence. Pulsus alternans, in contrast, is defined by beat-to-beat variability of pulse amplitude. It is present only when every other phase I Korotkoff sound is audible as the cuff pressure is lowered slowly, typically in a patient with a regular heart rhythm and independent of the respira tory cycle. Pulsus alternans is seen in patients with severe LV systolic Major arteries Ankle-brachial index Ankle systolic pressures Right common carotid a. Axillary a. Deep brachial a. Left arm, systolic pressure in the brachial a. Brachial a. Common iliac a. Radial a. Ulnar a. Deep femoral a. Femoral a. Popliteal a. Posterior tibial artery a. Left ankle, systolic pressure in the posterior tibial a. and the dorsalis pedis a. Anterior tibial artery a. Dorsalis pedis a. A B FIGURE 246-3 A. Anatomy of the major arteries of the leg. B. Measurement of the ankle systolic pressure. The ankle-brachial index (ABI) is calculated by dividing the lower of the two ankle pressures (i.e., the dorsalis pedis or posterior tibia) by the higher of the two arm pressures (i.e., left or right arm). Left and right ABIs should be recorded. (Adapted from NA Khan et al: Does the clinical examination predict lower extremity peripheral arterial disease? JAMA 295:536, 2006.)

dysfunction and is thought to be due to cyclic changes in intracel lular calcium and action potential duration. When pulsus alternans is associated with electrocardiographic T-wave alternans, the risk for an arrhythmic event appears to be increased.

Ascending aortic aneurysms can rarely be appreciated as a pulsatile mass in the right parasternal area. Appreciation of a prominent abdom inal aortic pulse should prompt noninvasive imaging with ultrasound or computed tomography for better characterization. Femoral and/or popliteal artery aneurysms should be sought in patients with abdomi nal aortic aneurysm disease. CHAPTER 246 Physical Examination of the Cardiovascular System

The level of a claudication-producing arterial obstruction can often be identified on physical examination (Fig. 246-3). For example, in a patient with calf claudication, a decrease in pulse amplitude between the common femoral and popliteal arteries will localize the obstruction to the level of the superficial femoral artery, although inflow obstruc tion above the level of the common femoral artery may coexist. Aus cultation for carotid, subclavian, abdominal aortic, and femoral artery bruits should be routine. However, the correlation between the pres ence of a bruit and the degree of vascular obstruction is poor. A cervi cal bruit is a weak indicator of the degree of carotid artery stenosis; the absence of a bruit does not exclude the presence of significant luminal obstruction. If a bruit extends into diastole or if a thrill is present, the obstruction is usually severe. Another cause of an arterial bruit is an arteriovenous fistula with enhanced flow. The likelihood of significant lower extremity peripheral arterial disease increases with typical symptoms of claudication, cool skin, abnormalities on pulse examination, or the presence of a vascular bruit. Abnormal pulse oximetry (a >2% difference between finger and toe oxygen saturation) can be used to detect lower extremity peripheral Dorsalis pedis a. Posterior tibial artery a.

arterial disease and is comparable in its performance characteristics to the ankle-brachial index.

Inspection and Palpation of the Heart The LV apex beat may be visible in the midclavicular line at the fifth intercostal space in thin-chested adults. Visible pulsations anywhere other than this expected location are abnormal. The left anterior chest wall may heave in patients with an enlarged or hyperdynamic left or right ventricle. As noted previously, a visible right upper parasternal pulsation may be suggestive of ascending aortic aneurysm disease. In thin, tall patients and patients with advanced obstructive lung disease and flattened diaphragms, the cardiac impulse may be visible in the epigastrium and should be distinguished from a pulsatile liver edge. PART 6 Disorders of the Cardiovascular System Palpation of the heart begins with the patient in the supine position at 30° and can be enhanced by placing the patient in the left lateral decubitus position. The normal LV impulse is <2 cm in diameter and moves quickly away from the fingers; it is better appreciated at end expiration, with the heart closer to the anterior chest wall. Character istics such as size, amplitude, and rate of force development should be noted. Enlargement of the LV cavity is manifested by a leftward and down ward displacement of an enlarged apex beat. A sustained apex beat is a sign of pressure overload, such as that which may be present in patients with AS or chronic hypertension. A palpable presystolic impulse corresponds to the fourth heart sound (S4) and is indicative of reduced LV compliance and the forceful contribution of atrial contraction to ventricular filling. A palpable third sound (S3), which is indicative of a rapid early filling wave in patients with heart failure, may be pres ent even when the gallop itself is not audible. A large LV aneurysm may sometimes be palpable as an ectopic impulse, discrete from the apex beat and often dyskinetic. HOCM may very rarely cause a triple cadence beat at the apex with contributions from a palpable S4 and the two components of the bisferiens systolic pulse. Right ventricular pressure or volume overload may create a sternal lift. Signs of either TR (cv waves in the jugular venous pulse) and/or pulmonary arterial hypertension (a loud single or palpable P2) would be confirmatory. The right ventricle can enlarge to the extent that left-sided events are obscured. A zone of retraction between the right ventricular and LV impulses sometimes can be appreciated in patients with right ventricle pressure or volume overload when they are placed in the left lateral decubitus position. Systolic and diastolic thrills signify turbulent and high-velocity blood flow. Their locations help identify the origin of heart murmurs. ■ ■CARDIAC AUSCULTATION Heart Sounds Ventricular systole is defined by the interval between the first (S1)

and second (S2) heart sounds (Fig. 246-4). The first heart sound (S1) includes mitral and tricuspid valve closure. Normal split ting can be appreciated in young patients and those with right bundle branch block, in whom tricuspid valve closure is relatively delayed. The intensity of S1 is determined by the distance over which the anterior leaflet of the mitral valve must travel to return to its annular plane, leaf let mobility, LV contractility, and the PR interval. S1 is classically loud in the early phases of rheumatic MS and in patients with hyperkinetic circulatory states or short PR intervals. S1 becomes softer in the later stages of MS when the leaflets are rigid and calcified, after exposure to β-adrenergic receptor blockers, with long PR intervals, and with LV contractile dysfunction. The intensity of heart sounds, however, can be reduced by any process that increases the distance between the stethoscope and the responsible cardiac event, including mechanical ventilation, obstructive lung disease, obesity, pneumothorax, and a pericardial effusion. Aortic and pulmonic valve closure constitutes the second heart sound (S2). With normal or physiologic splitting, the A2–P2 interval increases with inspiration and narrows during expiration. This physi ologic interval will widen with right bundle branch block because of the further delay in pulmonic valve closure and in patients with severe MR because of the premature closure of the aortic valve. An unusually

EXPIRATION INSPIRATION A2 P2 A2 P2 A Normal S1 S2 S1 S2 A2 P2 A2 P2 B Atrial septal

defect S1 S2 S1 S2 A2 P2 A2 P2 C Expiratory splitting

with inspiratory

increase (RBBB,

idiopathic dilatation PA) S1 S2 S1 S2 A2 P2 A2 P2 D Reversed splitting

(LBBB, aortic

stenosis) S1 S2 S1 S2 A2 P2 A2 P2 E Close fixed

splitting

(pulmonary

hypertension) S1 S2 S1 S2 FIGURE 246-4 Heart sounds. A. Normal. S1, first heart sound; S2, second heart sound; A2, aortic component of the second heart sound; P2, pulmonic component of the second heart sound. B. Atrial septal defect with fixed splitting of S2. C. Physiologic but wide splitting of S2 with right bundle branch block (RBBB). PA, pulmonary artery. D. Reversed or paradoxical splitting of S2 with left bundle branch block (LBBB). E. Narrow splitting of S2 with pulmonary hypertension. (Reproduced with permission from NO Fowler: Diagnosis of Heart Disease, Springer Nature; 1991.) narrowly split or even a singular S2 is a feature of pulmonary arterial hypertension. Fixed splitting of S2, in which the A2–P2 interval is wide and does not change during the respiratory cycle, occurs in patients with a secundum atrial septal defect (ASD). Reversed or paradoxical splitting refers to a pathologic delay in aortic valve closure, such as that which occurs in patients with left bundle branch block, right ventricu lar pacing, severe AS, HOCM, and acute myocardial ischemia. With reversed or paradoxical splitting, the individual components of S2 are

audible at end expiration, and their interval narrows with inspiration, the opposite of what would be expected under normal physiologic conditions. P2 is considered loud when its intensity exceeds that of A2 at the base, when it can be palpated in the area of the proximal main pulmonary artery (second left interspace), or when both components of S2 can be appreciated at the lower left sternal border or apex. The intensity of A2 and P2 decreases with AS and pulmonic stenosis (PS), respectively. In these conditions, a single S2 may result. Systolic Sounds An ejection sound is a high-pitched early systolic sound that corresponds in timing to the upstroke of the carotid pulse. It usually is associated with congenital bicuspid aortic or pulmonic valve disease; however, ejection sounds are also sometimes audible in patients with isolated aortic or pulmonary root dilation and normal semilunar valves. The ejection sound that accompanies bicuspid aortic valve disease becomes softer and then inaudible as the valve calcifies and becomes more rigid. The ejection sound that accompanies PS moves closer to the first heart sound as the severity of the stenosis increases. In addition, the pulmonic ejection sound is the only rightsided acoustic event that decreases in intensity with inspiration. Ejec tion sounds are often heard more easily at the lower left sternal border than they are at the base. Nonejection sounds (clicks), which occur after the onset of the carotid upstroke, are related to MVP and may be single or multiple. The nonejection click may introduce a murmur. This click-murmur complex will move away from the first heart sound with maneuvers that increase ventricular preload, such as squatting. On standing, the click and murmur move closer to S1.

Diastolic Sounds The high-pitched opening snap (OS) of MS occurs after a very short interval after the second heart sound. The A2–OS interval is inversely proportional to the height of the left atrial–left ventricular diastolic pressure gradient. The intensity of both S1 and the OS of MS decreases with progressive calcification and rigid ity of the anterior mitral leaflets. The pericardial knock (PK) is also high-pitched and occurs slightly later than the OS, corresponding in timing to the abrupt cessation of ventricular expansion after tricuspid valve opening and to an exaggerated y descent seen in the jugular venous waveform in patients with constrictive pericarditis. A tumor plop is a lower-pitched sound that rarely can be heard in patients with atrial myxoma. It may be appreciated only in certain positions and arises from the diastolic prolapse of the tumor across the mitral valve. The third heart sound (S3) occurs during the rapid filling phase of ventricular diastole. It can be a normal finding in children, adolescents, and young adults; however, in older patients, it signifies heart failure. A left-sided S3 is a low-pitched sound best heard over the LV apex. A right-sided S3 is usually better heard over the lower left sternal border and becomes louder with inspiration. A left-sided S3 in patients with chronic heart failure is predictive of cardiovascular morbidity and mortality. Interestingly, an S3 is equally prevalent among heart failure patients with preserved and reduced LV ejection fraction. The fourth heart sound (S4) occurs during the atrial filling phase of ventricular diastole and indicates LV presystolic expansion. An S4 is more common among patients who derive significant benefit from the atrial contribution to ventricular filling, such as those with chronic LV hypertrophy or active myocardial ischemia. An S4 is not present with atrial fibrillation. Cardiac Murmurs Heart murmurs result from audible vibrations that are caused by increased turbulence and are defined by their timing within the cardiac cycle. Not all murmurs are indicative of structural heart disease, and the accurate identification of a benign or functional systolic murmur often can obviate the need for additional testing in healthy subjects, especially children and adolescents. The duration, frequency, configuration, and intensity of a heart murmur are dictated by the magnitude, variability, and duration of the responsible pressure difference between two cardiac chambers, the two ventricles, or the ventricles and their respective great

arteries. The intensity of a heart murmur is graded on a scale of 1 to 6; a thrill is present with murmurs of grade 4 or greater intensity. Other attributes of the murmur that aid in its accurate identification include its location, radiation, and response to bedside maneuvers. Although clinicians can detect and correctly identify heart murmurs with only fair reliability, a careful and complete bedside examination usually can identify individuals with valvular heart disease for whom transthoracic echocardiography and clinical follow-up are indicated and exclude subjects for whom no further evaluation is necessary. Systolic murmurs can be early, mid, late, or holosystolic in timing (Fig. 246-5). Acute severe MR results in a decrescendo early systolic murmur, the characteristics of which are related to the progressive attenuation of the LV to left atrial pressure gradient during systole because of the steep and rapid rise in left atrial pressure in this context. Severe MR associated with posterior leaflet prolapse or flail radiates anteriorly and to the base, where it can be confused with the murmur of AS. MR that is due to anterior leaflet involvement radiates posteri orly and to the axilla. With acute TR in patients with normal pulmo nary artery pressures, an early systolic murmur that may increase in intensity with inspiration may be heard at the left lower sternal border, with regurgitant cv waves visible in the jugular venous pulse. A midsystolic murmur begins after S1 and ends before S2; it is typi cally crescendo-decrescendo in configuration. AS is the most com mon cause of a midsystolic murmur in an adult. It is often difficult to estimate the severity of the valve lesion on the basis of the physi cal examination findings, especially in older hypertensive patients with stiffened carotid arteries or patients with low cardiac output in whom the intensity of the systolic heart murmur is misleadingly soft. Examination findings consistent with severe AS would include parvus et tardus carotid upstrokes, a late-peaking grade 3 or greater

ECG ECG LVP AOP LVP CHAPTER 246 LAP HSM EDM Physical Examination of the Cardiovascular System
S1 S2 S1 A2 ECG ECG LVP LVP AOP LAP MSM PSM MDM S1 A2 S1 S2 A B FIGURE 246-5 A. Top. Graphic representation of the systolic pressure difference (green shaded area) between left ventricle and left atrium with phonocardiographic recording of a holosystolic murmur (HSM) indicative of mitral regurgitation. ECG, electrocardiogram; LAP, left atrial pressure; LVP, left ventricular pressure; S1, first heart sound; S2, second heart sound. Bottom. Graphic representation of the systolic pressure gradient (green shaded area) between left ventricle and aorta in patient with aortic stenosis. A midsystolic murmur (MSM) with a crescendo-decrescendo configuration is recorded. AOP, aortic pressure. B. Top. Graphic representation of the diastolic pressure difference between the aorta and left ventricle (blue shaded area) in a patient with aortic regurgitation, resulting in a decrescendo, early diastolic murmur (EDM) beginning with A2. Bottom. Graphic representation of the diastolic left atrial–left ventricular gradient (blue areas) in a patient with mitral stenosis with a mid-diastolic murmur (MDM) and late presystolic murmurs (PSM). midsystolic murmur, a soft A2, a sustained LV apical impulse, and an S4. It is sometimes difficult to distinguish aortic sclerosis from more advanced degrees of valve stenosis. The former is defined by focal thickening and calcification of the aortic valve leaflets that is not severe enough to result in obstruction. These valve changes are associ ated with a Doppler jet velocity across the aortic valve of 2.5 m/s or less. Patients with aortic sclerosis can have grade 2 or 3 midsystolic murmurs identical in their acoustic characteristics to the murmurs heard in patients with more advanced degrees of AS. Other causes of a midsystolic heart murmur include pulmonic valve stenosis (with or without an ejection sound), HOCM, increased pulmonary blood flow in patients with a large ASD and left-to-right shunting, and several states associated with accelerated blood flow in the absence

of struc tural heart disease, such as fever, thyrotoxicosis, pregnancy, anemia, and normal childhood/adolescence. The murmur of HOCM has features of both obstruction to LV out flow and MR, as would be expected from knowledge of the pathophysi ology of this condition. The systolic murmur of HOCM usually can be distinguished from other causes on the basis of its response to bedside maneuvers, including Valsalva, passive leg raising, and standing/ squatting. In general, maneuvers that decrease LV preload (or increase LV contractility) will cause the murmur to intensify, whereas maneu vers that increase LV preload or afterload will cause a decrease in the intensity of the murmur. Accordingly, the systolic murmur of HOCM becomes louder during the strain phase of the Valsalva maneuver and after standing quickly from a squatting position. The murmur becomes softer with passive leg raising and when squatting. The murmur of AS is typically loudest in the second right interspace with radiation into the carotids, whereas the murmur of HOCM is best heard between the lower left sternal border and the apex. The murmur of PS is best heard in the second left interspace. The midsystolic murmur associated with enhanced pulmonic blood flow in the setting of a large ASD is usually loudest at the mid-left sternal border.

A late systolic murmur, heard best at the apex, indicates MVP. As previously noted, the murmur may or may not be introduced by one or more nonejection clicks. Differential radia tion of the murmur, as previously described, may help identify the specific leaflet involved by the myxomatous process. The click-murmur complex behaves in a manner directionally similar to that demonstrated by the murmur of HOCM during the Valsalva and stand/squat maneuvers (Fig. 246-6). The murmur of MVP can be identified by the accompanying nonejec tion click.

PART 6 Disorders of the Cardiovascular System Holosystolic murmurs are plateau in con figuration and reflect a continuous and wide pressure gradient between the left ventricle and left atrium with chronic MR, the left ventricle and right ventricle with a ventricular septal defect (VSD), and the right ventricle and right atrium with TR. In contrast to acute MR, in chronic MR, the left atrium is enlarged and its compliance is normal or increased to the extent that there is little if any further increase in left atrial pressure from any increase in regurgitant volume. The murmur of MR is best heard over the cardiac apex. The intensity of the murmur increases with maneuvers that increase LV afterload, such as sustained hand grip. The murmur of a VSD (without significant pulmo nary hypertension) is holosystolic and loudest at the mid-left sternal border, where a thrill is usually present. The murmur of TR is loud est at the lower left sternal border, increases in intensity with inspiration (Carvallo’s sign), and is accompanied by visible cv waves in the jugular venous wave form and, on occasion, by pulsatile hepatomegaly. S1 S1 C C M M S2 S2 FIGURE 246-6 Behavior of the click (C) and murmur (M) of mitral valve prolapse with changes in loading (volume, impedance) and contractility. S1, first heart sound; S2, second heart sound. With standing (left side of figure), volume and impedance decrease, as a result of which the click and murmur move closer to S1. With squatting (right), the click and murmur move away from S1 due to the increases in left ventricular volume and impedance (afterload). Ao, aorta; LV, left ventricle. (Adapted from RA O’Rourke, MH Crawford: Curr Prob Cardiol 1:9, 1976.) Diastolic Murmurs In contrast to some systolic murmurs, dia stolic heart murmurs always signify structural heart disease (Fig. 246-5). The murmur associated with acute, severe AR is relatively soft and of short duration because of the rapid rise in LV diastolic pressure and the progressive diminution of the aortic-LV diastolic pressure gradient. In contrast, the murmur of chronic severe AR is classically heard as a decrescendo, blowing diastolic murmur along the left sternal border in patients with primary valve pathology and sometimes along the right sternal border in patients with primary aortic root pathology. When the

jet of AR is directed posteriorly along the anterior leaflet of the mitral valve, the intensity of the murmur may be attenuated, and sever ity of the valve lesion underestimated. Detection of subtle AR murmurs can be enhanced by auscultation and end-expiration with the patient leaning forward. With chronic, severe AR, the pulse pressure is wide and the arterial pulses are bounding in character. Signs of significant diastolic run-off are often absent in the acute phase. The murmur of pulmonic regurgitation is also heard along the left sternal border. It is most commonly due to pulmonary hypertension and enlargement of the annulus of the pulmonic valve. S2 is single and loud and may be palpable. There is a right ventricular/parasternal lift that is indicative of chronic right ventricular pressure overload. A less impressive murmur of PR is present after repair of tetralogy of Fallot or pulmonic valve atresia. In this postoperative setting, the murmur is softer and lowerpitched, and the severity of the accompanying pulmonic regurgitation can be underestimated significantly. MS is the classic cause of a mid- to late-diastolic murmur, which is best heard over the apex in the left lateral decubitus position, is lowpitched or rumbling, and is introduced by an OS in the early stages of the rheumatic disease process. Presystolic accentuation refers to an increase in the intensity of the murmur just before the first heart sound and occurs in patients with sinus rhythm. It is absent in patients with atrial fibrillation. The auscultatory findings in patients with rheumatic

Impedance Ao LV Contractility Volume tricuspid stenosis typically are obscured by left-sided events, although they are similar in nature to those described in patients with MS. “Functional” mitral or tricuspid stenosis refers to the generation of mid-diastolic murmurs that are created by increased and accelerated transvalvular diastolic flow, even in the absence of valvular obstruc tion, in the setting of severe MR, severe TR, or a large ASD with leftto-right shunting. The Austin Flint murmur of chronic severe AR is a low-pitched mid- to late apical diastolic murmur that sometimes can be confused with MS. The Austin Flint murmur typically decreases in intensity after exposure to vasodilators, whereas the murmur of MS may be accompanied by an OS and also may increase in intensity after vasodilators because of the associated increase in cardiac output. Unusual causes of a mid-diastolic murmur include atrial myxoma, complete heart block, and acute rheumatic mitral valvulitis. Continuous Murmur A continuous murmur is predicated on a pressure gradient that persists between two cardiac chambers or blood vessels across systole and diastole. The murmurs typically begin in systole, envelop the second heart sound (S2), and continue through some portion of diastole. They can often be difficult to dis tinguish from individual systolic and diastolic murmurs in patients with mixed valvular heart disease. The classic example of a continu ous murmur is that associated with a PDA, which usually is heard in the second or third interspace at a slight distance from the sternal border. Other causes of a continuous murmur include a ruptured sinus of Valsalva aneurysm with creation of an aortic–right atrial or right ventricular fistula, a coronary or great vessel arteriovenous fistula, and an arteriovenous fistula constructed to provide dialysis access. There are two types of benign continuous murmurs. The cervical venous hum is heard in children or adolescents in the supra clavicular fossa. It can be obliterated with firm pressure applied to the diaphragm of the stethoscope, especially when the subject turns their head toward the examiner. The mammary soufflé of pregnancy relates to enhanced arterial blood flow through engorged breasts. The diastolic component of the murmur can be obliterated with firm pres sure over the stethoscope.

TABLE 246-1 Effects of Physiologic Interventions on the Intensity of Heart Murmurs and Sounds Respiration Right-sided murmurs and sounds generally increase with inspiration, except for the

PES. Left-sided murmurs and sounds are usually louder during expiration. Valsalva Maneuver Most murmurs decrease in length and intensity. Two exceptions are the systolic murmur of HOCM, which usually becomes much louder, and that of MVP, which becomes longer and often louder. After release of the Valsalva maneuver, right-sided murmurs tend to return to control intensity earlier than do left-sided murmurs. After VPB or AF Murmurs originating at normal or stenotic semilunar valves increase in the cardiac cycle after a VPB or in the cycle after a long cycle length in AF. By contrast, systolic murmurs due to AV valve regurgitation do not change or become shorter (MVP). Positional Changes With standing, most murmurs diminish, with two exceptions being the murmur of HOCM, which becomes louder, and that of MVP, which lengthens and often is intensified. With squatting, most murmurs become louder, but those of HOCM and MVP usually soften and may disappear. Passive leg raising usually produces the same results. Exercise Murmurs due to blood flow across normal or obstructed valves (e.g., PS, MS) become louder with both isotonic and submaximal isometric (hand grip) exercise. Murmurs of MR, VSD, and AR also increase with hand grip exercise. However, the murmur of HOCM often decreases with nearly maximum hand grip exercise. Left-sided S4 and S3 sounds are often accentuated by exercise, particularly when due to ischemic heart disease. Abbreviations: AF, atrial fibrillation; AR, aortic regurgitation; HOCM, hypertrophic obstructive cardiomyopathy; MR, mitral regurgitation; MS, mitral stenosis; MVP, mitral valve prolapse; PES, pulmonic ejection sound; PR, pulmonic regurgitation; PS, pulmonic stenosis; TR, tricuspid regurgitation; TS, tricuspid stenosis; VPB, ventricular premature beat; VSD, ventricular septal defect. Dynamic Auscultation Diagnostic accuracy can be enhanced by the performance of simple bedside maneuvers to identify heart murmurs and characterize their significance (Table 246-1). Except for the pulmonic ejection sound, right-sided events increase in intensity with inspiration and decrease with expiration; left-sided events behave oppositely (100% sensitivity, 88% specificity). As previously noted, the intensity of the murmurs associated with MR, VSD, and AR will increase in response to maneuvers that increase LV afterload, such as hand grip and vasopressors. The intensity of these murmurs will decrease after exposure to vasodilating agents. Squatting is associated with an abrupt increase in LV preload and afterload, whereas rapid standing results in a sudden decrease in preload. In patients with MVP, the click and murmur move away from the first heart sound with squatting because of the delay in onset of leaflet prolapse at higher ventricular volumes. With rapid standing, however, the click and murmur move closer to the first heart sound as prolapse occurs earlier in systole at a smaller chamber dimension. The murmur of HOCM behaves similarly, becoming softer and shorter with squatting (95% sensitivity, 85% specificity) and longer and louder on rapid standing (95% sensitivity, 84% specificity). A change in the intensity of a systolic murmur in the first beat after a premature beat or in the beat after a long cycle length in patients with atrial fibrillation suggests valvular AS rather than MR, particularly in an older patient in whom the murmur of the AS may be well transmitted to the apex (Gallavardin effect). Of note, however, the systolic murmur of HOCM also increases in intensity in the beat after a premature beat. This increase in intensity of any LV outflow murmur in the beat after a premature beat relates to the combined effects of enhanced LV filling (from the longer diastolic period) and postextrasystolic potentiation of LV contractile function. In either instance, forward flow will accelerate, causing an increase in the gradient across the LV outflow tract (dynamic or fixed) and a louder systolic murmur. In contrast, the intensity of the murmur of MR does not change in a postpremature beat, because there is relatively little change in the nearly constant LV to left atrial pressure gradient or

further alteration in mitral valve flow. Bedside exercise can sometimes be performed to increase cardiac output and, secondarily, the intensity of both systolic and diastolic heart murmurs. Most left-sided heart murmurs decrease in intensity and duration during the strain phase of the Valsalva maneuver. The murmurs associated with MVP and HOCM are the two notable exceptions. The Valsalva maneuver also can be used to assess the integrity of the heart and vasculature in the setting of advanced heart failure.

CHAPTER 246 Prosthetic Heart Valves The first clue that prosthetic valve dysfunction may contribute to recurrent symptoms is frequently a change in the quality of the heart sounds or the appearance of a new murmur. The heart sounds with a bioprosthetic valve resemble those generated by native valves. A mitral bioprosthesis usually may be associated with a grade 1 to 2 midsystolic murmur along the left sternal border (created by turbulence across the valve struts as they project into the LV outflow tract) as well as by a soft mid-diastolic murmur that occurs with normal LV filling. This diastolic murmur often can be heard only in the left lateral decubitus position and after exercise. A high-pitched or holosystolic apical murmur is indicative of pathologic MR due to a paravalvular leak and/or intra-annular bioprosthetic regurgitation from leaflet degeneration, for which diagnostic noninvasive imaging is indicated. Clinical deterioration can occur rapidly after the first expression of mitral bioprosthetic valve failure. A tissue valve in the aortic position is always associated with a grade 1 to 3 midsystolic murmur at the base or just below the suprasternal notch. A diastolic murmur of AR is abnormal in any circumstance. Mechanical valve dysfunction may first be suggested by a decrease in the intensity of either the opening or the closing sound. A high-pitched apical systolic murmur in patients with a mechanical mitral prosthesis and a diastolic decrescendo murmur in patients with a mechanical aortic prosthesis indicate paravalvular regurgita tion. Patients with prosthetic valve thrombosis may present clinically with signs of shock, muffled heart sounds, and soft murmurs in both systole and diastole. Pericardial Disease A pericardial friction rub is nearly 100% specific for the diagnosis of acute pericarditis, although the sensitivity of this finding is not nearly as high, because the rub may come and go over the course of an acute illness or be very difficult to elicit. The rub is heard as a leathery or scratchy three-component or two-component sound, although it may be monophasic. Classically, the three com ponents are ventricular systole, rapid early diastolic filling, and late presystolic filling after atrial contraction in patients in sinus rhythm. It is necessary to listen to the heart in several positions. Additional clues to the presence of acute pericarditis may be present from the history and 12-lead electrocardiogram. The rub typically disappears as the volume of any pericardial effusion increases. Pericardial tamponade can be diagnosed with a sensitivity of 98%, a specificity of 83%, and a positive likelihood ratio of 5.9 (95% confidence interval 2.4–14) by a pulsus paradoxus that exceeds 12 mmHg in a patient with a large pericardial effusion. Physical Examination of the Cardiovascular System

The findings on physical examination are integrated with the symptoms previously elicited with a careful history to construct an appropriate differential diagnosis and proceed with indicated imaging and laboratory assessment. The physical examination is an irreplace able component of clinician-patient interaction. Educational efforts to enhance competence may result in improved diagnostic efficiency. The adjunctive use of point-of-care ultrasound and artificial intelligence tools is expected to increase. ■ ■FURTHER READING Chamsi-Pasha MA et al: Handheld echocardiography: Current state and future perspectives. Circulation 136:2178, 2017. Drazner MH et al: Value of clinician assessment of hemodynamics in advanced heart failure: The ESCAPE trial. Circ Heart Fail 1:170, 2008. Fanaroff AC et al: Does this patient with chest pain have acute coronary syndrome? The Rational Clinical Examination Systematic Review. JAMA 314:1955, 2015.

Revision #1
Created 2026-01-06 16:33:43 UTC by Omar Ayman
Updated 2026-01-06 16:33:43 UTC by Omar Ayman