13 History and Physical Examination: An Evidence-Based Approach

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Evaluation of the patient with known or suspected cardiovascular disease begins with a directed history and targeted physical examination, the scope and duration of which depend on the clinical context of the patient encounter. Elective, ambulatory investigations allow comparatively more time for the development of a comprehensive assessment, whereas emergency department visits and urgent bedside consultations necessitate a more focused strategy. The elicitation of the history, with its emphasis on major cardiovascular symptoms and their change over time, demands a direct interaction between the clinician and patient, and should not be delegated to another nor inferred from information gleaned from a cursory chart review. The history also affords a unique opportunity to assess the patient's personal attitudes, intelligence, comprehension, acceptance or denial, motivation, fear, and prejudices. Such insights allow a more informed understanding of the patient's preferences and values regarding shared decision making. The interview also can reveal genetic or familial influences and the impact of other medical conditions on the manifesting illness. Although time constraints have limited the emphasis on careful history taking, the information gathered from the patient interview remains essential to inform the design of an efficient diagnostic and treatment plan.

Physical examination skills have declined. Only a minority of internal medicine and family practice residents recognizes classic cardiac findings in relevant diseases. Performance does not predictably improve with experience.[1] Residency work hours and health care system efficiency standards have severely restricted the time devoted to the mentored cardiovascular examination. In 2020, the SARS-CoV-2 virus pandemic drastically limited in-person interactions, catalyzed a movement to virtual visits (VV) and challenged clinicians to develop alternative means for patient assessment through real-time video observations. It is anticipated that VV will become an established feature of ambulatory patient follow-up. Less attention to bedside skills and declining confidence in the powers of observation have led to increasing use of noninvasive imaging, including the use of handheld ultrasound. Educational efforts, which utilize repetition, patient-centered teaching conferences, simulation, and visual display feedback of auscultatory and Doppler echocardiographic findings, can improve physical examination performance.[2–6]

The evidence base that links the findings from the history and physical examination to cardiovascular disease severity and prognosis is more robust for heart failure, valvular heart disease, and coronary artery disease than for other conditions. For example, vital signs and the presence of pulmonary congestion and mitral regurgitation (MR) contribute importantly to bedside risk assessment in patients with acute coronary syndromes (ACSs). The diagnosis of heart failure is fundamentally made at the bedside from symptoms and signs that reflect congestion and/or inadequate end-organ perfusion; these findings have been correlated with invasive hemodynamic measurements as well as with outcomes[7]: irregularly irregular pulse, a heart murmur suggestive of MR, a heart rate greater than 60 beats/min, and an elevated jugular venous pressure (JVP).[7] Accurate auscultation provides important insight into many valvular and congenital heart lesions. This chapter reviews the fundamentals of the cardiovascular history and physical examination and the evidence to support their utility. A diagnostic test is considered reasonably reliable if the kappa statistic is at least 0.4. A positive likelihood ratio (LR) (sensitivity/[1 – specificity]) increases the likelihood of the condition; a negative LR (1 – sensitivity)/specificity) decreases the likelihood of the condition.[8]

THE HISTORY

The major signs and symptoms associated with cardiac disease include chest discomfort (see Chapter 35), dyspnea, fatigue, edema, palpitations (see Chapter 61), and syncope (see Chapter 71). In most cases, careful attention to the specific characteristics of chest discomfort—quality, location, radiation, triggers, mode of onset, and duration—along with alleviating factors and associated symptoms can narrow the differential diagnosis (see Chapter 35). Angina pectoris can usually be differentiated from the pain associated with pulmonary embolism, pericarditis, aortic dissection, esophageal reflux, or costochondritis. Cough, hemoptysis, and cyanosis may provide additional clues as to the cause of chest pain. Claudication, limb pain, edema, and skin discoloration usually indicate a vascular disorder. The cardiovascular clinician also should be familiar with common manifestations of acute

stroke and transient ischemic attack, such as sudden weakness, sensory loss, incoordination, and visual disturbance. The sudden onset of symptoms and associated diaphoresis should always elicit concern that a cardiovascular cause underlies the patient's complaints.

Typical angina should satisfy three characteristics: (1) substernal discomfort, (2) initiated by exertion or stress, and (3) relieved with rest or sublingual nitroglycerin. Chest discomfort with two of these three criteria is considered atypical angina; pain with one or none of these features is considered nonanginal. When age and sex are considered, the diagnostic accuracy for CAD using these criteria is reasonable (receiver operator curve [ROC] area under the curve [AUC] 0.713). Incorporating a history of diabetes, hypertension, smoking, and dyslipidemia improves the diagnostic accuracy (ROC AUC 0.791).[9]

Several aspects of the presenting symptom of chest pain increase or decrease the likelihood of ACS. For example, pain that is sharp (LR, 0.3; 95% CI, 0.2 to 0.5), pleuritic (LR, 0.2; 95% CI, 0.1 to 0.3), positional (LR, 0.3; 95% CI, 0.2 to 0.5), or reproducible with palpation (LR, 0.3; 95% CI, 0.2 to 0.4) usually is noncardiac, whereas discomfort that radiates to both arms or shoulders (LR, 4.1; 95% CI, 2.5 to 6.5) or is precipitated by exertion (LR, 2.4; 95% CI, 1.5 to 3.8) has a much higher likelihood of reflecting myocardial ischemia. Less classic symptoms (i.e., anginal equivalents) such as indigestion, belching, and dyspnea, also should command the clinician's attention when other features of the presentation suggest ACS, even in the absence of chest discomfort.

Women, elderly persons, and patients with diabetes more commonly present with a less typical clinical picture. A history of a prior abnormal stress test (LR, 3.1; 95% CI, 2.0 to 4.7), known CAD (LR, 2.0; 95% CI, 1.4 to 2.6) or the presence of peripheral arterial disease (PAD) (LR, 2.7; 95% CI, 1.5 to 4.8) increases the likelihood that the pain indicate an ACS.[10] However, the accuracy of traditional risk factors and symptoms for the diagnosis of ACS is weak. Clinical prediction models that incorporate aspects of the history and examination with serum biomarkers of cardiac injury (troponins) and ECG findings provide better diagnostic accuracy, especially when they have been externally validated (Table 13.1).

Dyspnea may occur with exertion or in recumbency (orthopnea) or even on standing (platypnea). Paroxysmal nocturnal dyspnea of cardiac origin usually occurs 2 to 4 hours after onset of sleep; the dyspnea is sufficiently severe to compel the patient to sit upright or stand and then subsides gradually over several minutes. The patient's partner should be questioned about any signs of sleep-disordered breathing, such as loud snoring or periods of apnea. Pulmonary embolism often associates with dyspnea of sudden onset.

Patients may use a variety of terms to describe their awareness of the heartbeat (palpitations), such as "flutters," "skips," or "pounding." The likelihood of a cardiac arrhythmia modestly increases with a known history of cardiac disease (LR, 2.03; 95% CI, 1.33 to 3.11) and decreases when symptoms resolve within 5 minutes (LR, 0.38; 95% CI, 0.22 to 0.63) or when associated with panic disorder (LR, 0.26; 95% CI, 0.07 to 1.01). A report of a regular, rapid-pounding sensation in the neck (LR, 177; 95% CI, 25 to 1251) or visible neck pulsations associated with palpitations (LR, 2.68; 95% CI, 1.25 to 5.78) increases the likelihood that atrioventricular nodal reentrant tachycardia (AVNRT) is the responsible arrhythmia. The absence of a regular, rapid-pounding sensation in the neck makes detecting AVNRT much less likely (LR, 0.07; 95% CI, 0.03 to 0.19).[11]

Cardiac syncope occurs suddenly, with rapid restoration of full consciousness thereafter. Patients with neurocardiogenic syncope may experience early warning signs (nausea, yawning), appear ashen and diaphoretic, and revive more slowly, albeit without signs of seizure or a prolonged postictal state. The complete history consists of information pertaining to traditional cardiovascular risk factors, a general medical history, occupation, social habits, activities, medications, drug allergies or intolerance, family history, and systems review. In most instances, the history, examination, and limited testing can establish the cause of syncope (Table 13.2).[12]

It is important to obtain a semiquantitative assessment of symptom severity and to document any change over time. The New York Heart Association (NYHA) and the Canadian Cardiovascular Society (CCS) functional classification systems are useful for both patient care and clinical research, despite their inherent limitations. Current technology now allows patients to self-report symptoms directly into the patient record using iterative responsive survey instruments, which can be quantified and may better reflect the patient's experience with their cardiovascular condition in contrast to the provider's interpretation of the patient's symptoms.[13]

THE GENERAL PHYSICAL EXAMINATION

The physical examination can help determine the cause of a given symptom, assess disease severity and progression, and evaluate the impact of specific therapies. It also can identify the presence of early-stage disease in patients without signs or symptoms. In general, the physical examination should be undertaken in a hypothesis-driven

TABLE 13.2 History and Exam Findings Suggestive of Cardiac Syncope

1. Known heart disease
2. Abnormal cardiovascular physical exam
3. Family history of sudden death or drowning
4. Male sex
5. Age >35 years at time of syncope
6. Two or fewer previous episodes
7. Palpitations
8. Chest pain or dyspnea

TABLE 13.1 Value of Selected History and Exam Findings For Diagnosis of Acute Coronary Syndrome

SYMPTOM	POSITIVE LR (95% CI)	PPV (%)	NEGATIVE LR (95% CI)	NPV (%)
Radiation to both arms	2.6 (1.8–3.7)	28	0.93 (0.89–0.96)	12
Radiation to left arm	1.3 (1.2–1.4)	16	0.88 (0.81–0.96)	12
Typical chest pain	1.9 (0.94–2.9)	22	0.52 (0.35–0.69)	7
Increase with exertion	1.5–1.8 (NA)	18–21	0.66–0.83 (NA)	9–11
Radiation to neck or jaw	1.5 (1.3–1.8)	18	0.91 (0.87–0.95)	12
Associated diaphoresis	1.3–1.4 (NA)	15	0.91–0.93 (NA)	12
Exam Findings				
Systolic BP <100	3.9 (0.98–15)	37	0.98 (0.95–1.0)	13
Tachypnea	1.9 (0.99–3.5)	22	0.95 (0.89–1.0)	12
Pain reproduced with palpation	0.28 (0.14–0.54)	4.0	1.2 (1.0–1.2)	15

BP, Blood pressure; *LR,* likelihood ratio; *NPV,* negative predictive value; *PPV,* positive predictive value.

manner[14] in which the pretest probability of a specific diagnosis is altered by a specific finding. Depending upon the characteristics of this finding, a post-test probability can be established to guide further testing as appropriate.

General Appearance

The examination begins with an appreciation of the general appearance of the patient, including age, posture, demeanor, and general health status. Is the patient in pain, resting quietly, or visibly diaphoretic? Does the patient choose to avoid certain positions to reduce or eliminate pain? The pain of acute pericarditis, for example, often diminishes with sitting up, leaning forward, or breathing shallowly. Pursing of the lips, a breathy quality to the voice, and an increased anteroposterior chest diameter would favor a pulmonary rather than a cardiovascular cause of dyspnea, although disorders in both etiologic categories may contribute in an individual patient. Pallor suggests anemia as a possible underlying disorder in patients with exercise intolerance or dyspnea, independent of cardiovascular disease. Cyanosis and jaundice also bear noting. Specific genetic cardiovascular disorders may be discernible from the patient's appearance. Emaciation suggests chronic heart failure or another systemic disorder (e.g., malignancy, infection).

The vital signs, including height, weight, temperature, pulse rate, blood pressure (in both arms), respiratory rate, and peripheral oxygen saturation, are used to determine the urgency of the evaluation and provide initial clues as to the presence of a cardiovascular disorder. The height and weight permit calculation of body mass index (BMI) and body surface area (BSA). Waist circumference (measured at the iliac crest) and waist-to-hip ratio (using the widest circumference around the buttocks) powerfully predict long-term cardiovascular risk. In patients with palpitations, a resting heart rate less than 60 beats/min may increase the likelihood of a clinically significant arrhythmia (LR, 3.00; 95% CI, 1.27 to 7.08).[11] Observation of the respiratory pattern may reveal signs of disordered breathing (e.g., Cheyne-Stokes respirations, obstructive sleep apnea), a finding associated with reduced survival in patients with severe systolic heart failure.[15] Mental status should be assessed and is an important gauge of adequate cerebral and systemic perfusion.

Frailty is defined as a state of decreased physiologic reserve and vulnerability to stressors. Several scales are available that incorporate quantifiable criteria such as unintentional weight loss, grip strength, gait speed, serum albumin, and hemoglobin (Table 13.3).[16] Frailty assessment, a common tool in the evaluation of patients with heart failure, is a routine feature of the preprocedural appraisal of elderly patients referred for heart valve intervention.

Skin

Central cyanosis is present with significant right-to-left shunting at the level of the heart or lungs. It also is a feature of hereditary methemoglobinemia. Peripheral cyanosis or acrocyanosis of the fingers, toes, nose, and ears is characteristic of the reduced blood flow that accompanies small-vessel constriction seen in severe heart failure, shock, or peripheral vascular disease. Differential cyanosis affecting the lower but not the upper extremities occurs with a patent ductus arteriosus (PDA) and pulmonary artery hypertension with right-to-left shunting at the great vessel level. Hereditary telangiectases on the lips, tongue, and mucous membranes (a finding in Osler-Weber-Rendu syndrome) resemble spider nevi; when present in the lungs, they can cause right-to-left shunting and central cyanosis. Telangiectasias also are seen in patients with scleroderma with or without pulmonary hypertension. Livedo reticularis, a lace-like purplish dislocation of the skin that imparts a mottled or reticulated appearance (Fig. 13.1), can occur on exposure to cold in normal individuals, but is also observed in a variety of conditions resulting in sluggish cutaneous blood flow, such as cardiogenic shock or certain autoimmune diseases. Tanned or bronze discoloration of the skin in unexposed areas can suggest iron overload and hemochromatosis. With jaundice, often first appreciated in the sclerae, the differential diagnosis is broad in scope. Ecchymoses often occur with either anticoagulant and/or antiplatelet use, whereas petechiae characterize thrombocytopenia, and purpuric skin lesions can be seen with infective endocarditis and other causes of leukocytoclastic vasculitis. Various lipid disorders can manifest with xanthomas, located subcutaneously, along tendon sheaths, or over the extensor surfaces of the extremities. Xanthomas within the palmar creases are specific for type III hyperlipoproteinemia. The leathery, cobblestone, "plucked chicken" appearance of the skin in the axillae and skinfolds of a young person is characteristic of pseudoxanthoma elasticum, a disease with multiple cardiovascular manifestations, including premature atherosclerosis. Extensive lentiginoses (freckle-like brown macules and café-au-lait spots over the trunk and neck) may be part of developmental delay–associated cardiovascular syndromes (LEOPARD, LAMB, and Carney) with multiple atrial myxomas, atrial septal defect (ASD), hypertrophic cardiomyopathy, and valvular stenoses. In a patient with heart failure or syncope, cardiovascular sarcoid should be suspected in the presence of lupus pernio, erythema nodosum, or granuloma annulare. Certain vascular disorders such as erythromelalgia, chilblains, frostbite, or lymphangitis also may be readily apparent from examination of the skin in the appropriate context.

Head and Neck

All patients should undergo assessment of the state of dentition, both as a source of infection and as an index of general health and hygiene. A high-arched palate is a feature of Marfan and other connective tissue disease syndromes. A large protruding tongue with parotid enlargement may suggest amyloidosis. Patients with Loeys-Dietz syndrome characteristically have a bifid uvula. Orange tonsils are typical of Tangier disease. Ptosis and ophthalmoplegia suggest muscular dystrophies, and congenital heart disease often is accompanied by hypertelorism,

TABLE 13.3 The Fried Criteria for Frailty

CHARACTERISTIC	METRICS
Shrinking (Unintentional weight loss)	>10 pound or >5% of total body weight in past year.
Weakness (Reduced hand grip strength)	Maximum isometric contraction in dominant hand over three attempts using hand dynamometer.
Exhaustion (Self-reported exhaustion)	Questions from the Center for Epidemiologic Studies—Depression Scale.
Slowness (Slow gait speed)	Slowest quintile according to gender/height based on time to walk 15 feet.
Inactivity (Low self-reported physical activity)	Lowest quintile of expended kcal/week using activity questionnaire.

Frail: Greater than or equal to 3 criteria present.
Intermediate/Prefrail: 1 or 2 criteria present.
From Joyce E. Frailty in advanced heart failure. *Heart Fail Clin*. 2016;12(3):363–374.

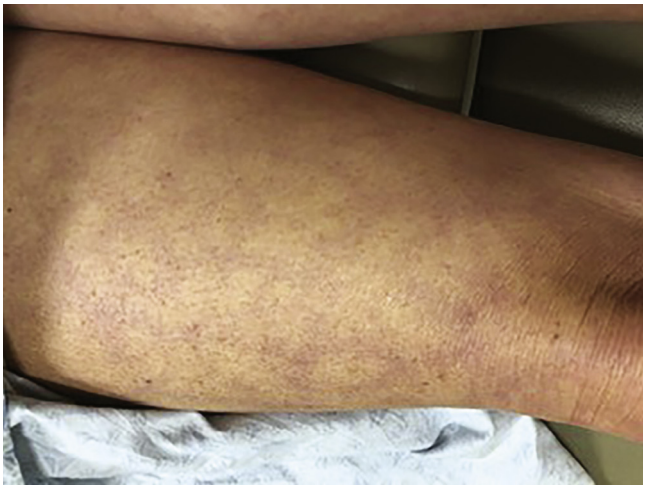


FIGURE 13.1 Appearance of livedo reticularis suggesting decreased skin perfusion.

low-set ears, micrognathia, and a webbed neck, as with Noonan, Turner, and Down syndromes. Proptosis, lid lag, and stare point to Graves hyperthyroidism. Patients with osteogenesis imperfecta may have blue sclerae, mitral or aortic regurgitation (AR), and a history of recurrent nontraumatic skeletal fractures.

Attention to the extraocular movements and the size and symmetry of the pupils may reveal a neurologic disorder. The oft-omitted funduscopic examination can aid in the evaluation of patients with hypertension, atherosclerosis, diabetes, endocarditis, neurologic signs or symptoms, or known carotid or aortic arch disease. Lacrimal gland hyperplasia is sometimes a feature of sarcoidosis. The "mitral facies" of rheumatic mitral stenosis (pink-purplish patches with telangiectasias over the malar eminences) also can accompany other disorders associated with pulmonary hypertension and reduced cardiac output. Relapsing polychondritis is suggested by inflammation of the pinnae and nasal cartilage in association with a saddle-nose deformity.

Extremities

Inspection and palpation can quickly ascertain the temperature of the extremities and the presence of clubbing, arachnodactyly, and nail changes. Clubbing implies the presence of central shunting. An unopposable "fingerized" thumb and shortened forearm bones occur in Holt-Oram syndrome. Arachnodactyly characterizes the Marfan syndrome. Janeway lesions (nontender, slightly raised areas of hemorrhage on the palms and soles), Osler's nodes (tender, raised nodules on the pads of the fingers or toes), and splinter hemorrhages (linear petechiae in the mid-nailbed) may be signs of infective endocarditis. Ulcerations and tissue loss of the fingertips may suggest thromboangiitis obliterans in the appropriate context.

Lower extremity or presacral edema with elevated JVP occurs in many volume-overloaded states, including heart failure. With a normal JVP, additional signs of venous disease, such as extensive varicosities, medial ulcers, or brownish pigmentation from hemosiderin deposition, suggest chronic venous insufficiency. A history of lower extremity vein ligation and "stripping" should be recognized. Edema also can occur with dihydropyridine calcium channel blocker therapy. Anasarca seldom occurs in heart failure, unless the condition is long standing, untreated, and accompanied by severe hypoalbuminemia. Asymmetric swelling can reflect local or unilateral venous thrombosis, the sequelae of previous vein graft harvesting, or lymphatic obstruction (lymphedema). Homan sign (calf pain elicited by forceful dorsiflexion of the foot) is neither specific nor sensitive for deep vein thrombosis. Muscular atrophy and the absence of hair in an extremity should suggest chronic arterial insufficiency or a neuromuscular disorder. Redistribution of fat from the extremities to central/abdominal stores (lipodystrophy) in some patients with HIV infection may relate to antiretroviral treatment and is associated with insulin resistance and several features of the metabolic syndrome.

Chest and Abdomen

Cutaneous venous collaterals over the anterior chest suggest chronic obstruction of the superior vena cava (SVC) or subclavian vein, especially in the presence of indwelling catheters or leads from cardiac implantable electrical devices (CIEDs). Asymmetric breast enlargement or arm swelling ipsilateral to an implanted device also may be present. Thoracic cage abnormalities, such as pectus carinatum (pigeon chest) or pectus excavatum (funnel chest), may accompany connective tissue disorders; the barrel chest of emphysema or advanced kyphoscoliosis may be associated with cor pulmonale. The severe kyphosis of ankylosing spondylitis should prompt careful auscultation for AR and scrutiny of the electrocardiogram (ECG) for first-degree atrioventricular (AV) block. The "straight back syndrome" (loss of normal kyphosis of the thoracic spine) can accompany mitral valve prolapse (MVP). A thrill may be present over well-developed intercostal artery collaterals in patients with aortic coarctation.

Patients with emphysema may exhibit prominence of the cardiac impulse in the epigastrium. The liver often is enlarged and tender in heart failure; systolic hepatic pulsations signify severe tricuspid regurgitation (TR). Patients with infective endocarditis of long duration may have splenomegaly. Ascites can develop with advanced and chronic right heart failure or constrictive pericarditis. The abdominal aorta normally may be palpated between the epigastrium and the umbilicus in thin patients and in children. The sensitivity of palpation for the detection of abdominal aortic aneurysm (AAA) disease increases as a function of aneurysm diameter and varies inversely with body size. Arterial bruits in the abdomen should be sought.

Careful chest auscultation is an essential component of the cardiovascular exam and is of prime importance when the presenting complaint is dyspnea. Technologic advances have provided important insights into often underappreciated pulmonary auscultatory phenomena (Fig. 13.2) that are commonly encountered in the evaluation of patients with cardiovascular disease.[17] Point of care ultrasound in emergency rooms and intensive care units have assumed increasing importance in the bedside evaluation of dyspnea.[18,19]

THE CARDIOVASCULAR EXAMINATION

Jugular Venous Pressure and Waveform

The JVP aids in the estimation of volume status. The external (EJV) or internal (IJV) jugular vein may be used, although the IJV is preferred because the EJV is valved and not directly in line with the SVC and right atrium. The EJV is easier to visualize when distended, and its appearance can help to discriminate between low and high central venous pressure (CVP). An elevated left EJV pressure may also signify a persistent left-sided SVC or compression of the innominate vein from an intrathoracic structure. If an elevated CVP is suspected but venous pulsations cannot be appreciated, the patient should be asked to sit upright with the feet dangling. With subsequent pooling of blood in the lower extremities, venous pulsations may be evident. SVC syndrome should be suspected if the venous pressure is elevated, pulsations are still not discernible, the face is swollen, and the skin of the head and neck appears dusky or cyanotic. When hypovolemia is suspected as a cause of hypotension, the patient may need to be lowered to a supine position to assess the waveform in the right supraclavicular fossa.

The venous waveform can sometimes be difficult to distinguish from the carotid artery pulse. The venous waveform has several characteristic features (Fig. 13.3; Table 13.4) and its individual components can usually be identified. The *a* and *v* waves, and *x* and *y* descents, are defined by their temporal relation to electrocardiographic events and heart sounds. The estimated height of the venous pressure indicates the CVP or right atrial pressure. Although observers vary widely in their estimates of the CVP, knowledge that the pressure is elevated, and not its specific value, can inform diagnosis and management.

The bedside venous pressure is usually estimated by the vertical distance between the top of the venous pulsation and the sternal inflection point, where the manubrium meets the sternum (angle of Louis). A distance of greater than 3 cm is considered abnormal. However, the distance between the angle of Louis and the mid–right atrium varies considerably as a function of body size and position. In general, use of the sternal angle as a reference leads to systematic underestimation of venous pressure. In practice, however, it is difficult to use even relatively simple thoracic landmarks. Measurements obtained by critical care nurses often will vary by several centimeters. Venous pulsations above the clavicle with the patient in the sitting position are clearly abnormal, because the distance from the right atrium is at least 10 cm. Estimated CVP correlates only modestly with direct measurement. Measurements made at the bedside, in units of centimeters of blood or water, require conversion to millimeters of mercury (1.36 cm H_2O = 1.0 mm Hg), for comparison with values measured with catheterization. Remote assessment of the JVP using video "chat" in patients with heart failure and reduced ejection fraction was demonstrated to be feasible and of comparable accuracy to bedside estimation in a pilot study using invasively measured right atrial pressure as the reference standard.[20] These findings have implications for both remote in-hospital and virtual ambulatory patient assessment (see later).

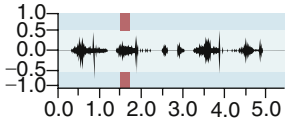
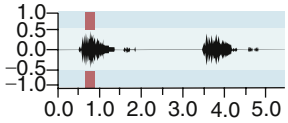
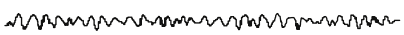
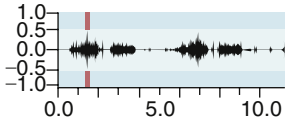
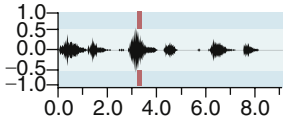
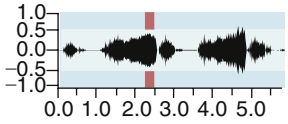
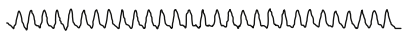
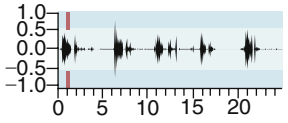
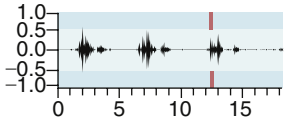
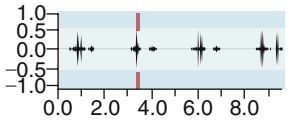
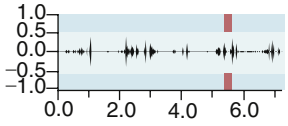
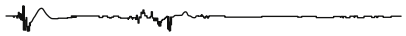
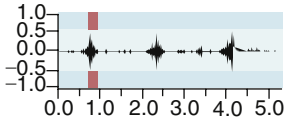
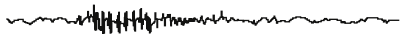
Respiratory sound	Amplitude–Time plot	
Acoustics	Unexpanded time	Expanded time
A Tracheal sound White noise Typical frequency, 100–5000 Hz Drop of energy at 800 Hz		
B Normal (vesicular) lung sound Low-pass-filtered noise Typical frequency, 100–1000 Hz Drop of energy at 200 Hz		
C Bronchial breathing Strong expiratory component An intermediate sound between tracheal and normal breathing		
D Stridor Sinusoid Fundamental frequency, >500 Hz		
E Wheeze Sinusoid Typical frequency, >100–5000 Hz Typical duration, >80 msec		
F Rhonchus Sinusoid Typical frequency, about 150 Hz Typical duration, >80 msec		
G Fine crackle Rapidly dampened wave deflection Typical frequency, about 650 Hz Typical duration, about 5 msec		
H Coarse crackles Rapidly dampened wave deflection Typical frequency, about 350 Hz Typical duration, about 15 msec		
I Pleural friction rub Rhythmic succession of short sounds Typical frequency, <350 Hz Typical duration, >15 msec		
J Squawk Sinusoid Typical frequency, 200–300 Hz Typical duration, about 200 msec Followed or preceded by crackles		

FIGURE 13.2 Respiratory sounds and the acoustic waveforms. (From Bohadana A, Izbicki G, Kraman SS. Fundamentals of lung auscultation. *N Engl J Med*. 2014;370:2053.)

The venous waveforms include several distinct peaks: *a, c,* and *v* (see Fig. 13.3, Table 13.4). The *a* wave reflects right atrial presystolic contraction, occurs just after the electrocardiographic P wave, and precedes the first heart sound (S_1). Patients with reduced right ventricular (RV) compliance from any cause can have a prominent *a* wave. A cannon *a* wave occurs with AV dissociation and right atrial contraction against a closed tricuspid valve. *The presence of cannon a waves in a patient with wide complex tachycardia identifies the rhythm as ventricular in origin.* The *a* wave is absent with atrial fibrillation (AF). The *x* descent reflects the fall in right atrial pressure after the *a* wave peak. The *c* wave interrupts this descent as ventricular systole pushes the closed valve into the right atrium. In the neck, the carotid pulse also may contribute to the *c* wave. As depicted in Figure 13.3, the *x* descent follows because of atrial diastolic suction created by ventricular systole pulling the tricuspid valve downward. In normal persons, the *x* descent is the predominant waveform in the jugular venous pulse. The *v* wave represents atrial filling, occurs at the end of ventricular systole, and follows just after S_2. Its height is determined

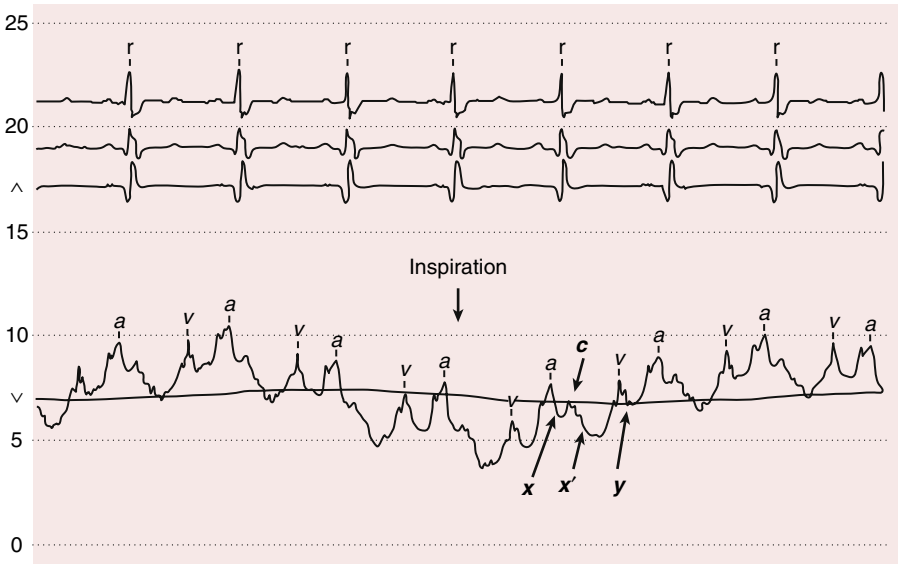


FIGURE 13.3 The normal jugular venous waveform recorded at cardiac catheterization. Note the inspiratory fall in pressure and the dominant *x*/*x*′ descent.

TABLE 13.4 Distinguishing Jugular Venous Pulse from Carotid Pulse

FEATURE	INTERNAL JUGULAR VEIN PULSE	CAROTID ARTERY PULSE
Appearance of pulse	Undulating two troughs and two peaks for every cardiac cycle (biphasic)	Single brisk upstroke (monophasic)
Response to inspiration	Height of column falls and troughs become more prominent	No respiratory change to contour
Palpability	Generally not palpable (except in severe TR)	Palpable
Effect of pressure	Can be obliterated with gentle pressure at base of vein/clavicle	Cannot be obliterated

TR, Tricuspid regurgitation.

by right atrial compliance and by the volume of blood returning to the right atrium from all sources. The *v* wave is smaller than the *a* wave because of the normally compliant right atrium. In patients with ASD, the *a* and *v* waves may be of equal height; in TR, the *v* wave is accentuated (Video 13.1). With TR, the *v* wave will merge with the *c* wave because retrograde valve flow and antegrade right atrial filling occur simultaneously. The *y* descent follows the *v* wave peak and reflects the fall in right atrial pressure after tricuspid valve opening. Resistance to ventricular filling in early diastole blunts the *y* descent, as is the case with pericardial tamponade or tricuspid stenosis. The *y* descent will be steep when ventricular diastolic filling occurs early and rapidly, as with pericardial constriction, restrictive cardiomyopathy, or isolated, severe TR.

The normal venous pressure should fall by at least 3 mm Hg with inspiration. A rise in venous pressure (or its failure to decrease) with inspiration (Kussmaul sign) is associated with constrictive pericarditis, and with restrictive cardiomyopathy, pulmonary embolism, RV infarction, and advanced systolic heart failure. A Kussmaul sign (Video 13.2) is seen with right-sided volume overload and reduced RV compliance. Normally, the inspiratory increase in right-sided venous return is accommodated by increased RV ejection, facilitated by an increase in the capacitance of the pulmonary vascular bed. In states of RV diastolic dysfunction and volume overload, the right ventricle cannot accommodate the enhanced volume, and the pressure rises. Increased pulmonary vascular resistance may also limit RV ejection and contribute to the Kussmaul phenomenon.

The abdominojugular reflux maneuver or passive leg elevation can elicit venous hypertension. The abdominojugular reflux maneuver requires firm and consistent pressure over the upper abdomen, preferably the right upper quadrant, for at least 10 seconds. Classically, a positive abdominojugular reflux sign has been defined as a rise of more than 3 cm in the venous pressure sustained for at least 15 seconds, although in practice a shorter time duration is usually accepted. The patient should be coached to refrain from holding their breath or performing a Valsalva-like maneuver, which can falsely elevate the venous pressure. A positive abdominojugular reflux sign can predict heart failure in patients with dyspnea, as well as a pulmonary artery wedge pressure higher than 15 mm Hg.

Measuring the Blood Pressure (see also Chapter 26)

Auscultatory measurement of blood pressure yields lower systolic and higher diastolic values than direct intra-arterial recording. Nurse or pharmacist-recorded blood pressure usually is closer to the patient's average daytime blood pressure than physician measured blood pressure. Blood pressure should be measured with the patient in the seated position, back supported, feet on the floor, with the arm at the level of the heart, using an appropriate-size cuff (Table 13.5), after 5 minutes of rest, repeated 5 minutes later, and the readings averaged. The use of an inappropriately small cuff can result in overestimation of the true blood pressure, an issue of particular relevance in obese patients.

On occasion, the Korotkoff sounds may disappear soon after the first sound, only to recur later before finally disappearing as phase 5. This auscultatory gap is more likely to occur in older, hypertensive patients with target organ damage. The systolic pressure should be recorded at the first Korotkoff sound and not when the sound reappears. This finding should be distinguished from *pulsus paradoxus* (see later). Korotkoff sounds may be heard all the way down to 0 mm Hg with the cuff completely deflated in children, in pregnant patients, in patients with chronic severe AR, or in the presence of a large arteriovenous fistula. In these cases, both the phases 4 and 5 pressures should be noted.

Blood pressure should be measured in both arms either in rapid succession or simultaneously; normally the measurements should differ by less than 10 mm Hg, independent of handedness. As many as 20% of normal subjects, however, exhibit a left-right arm blood pressure differential of more than 10 mm Hg in the absence of symptoms or other examination findings. A blood pressure differential of more than 10 mm Hg can be associated with subclavian artery disease, supravalvular aortic stenosis (SVAS), aortic coarctation, or aortic dissection. Systolic leg pressures may exceed arm pressures by as much as 20 mm Hg; greater leg-arm systolic blood pressure differences are seen in patients with severe AR (Hill sign) and patients with extensive and calcified (noncompressible) lower extremity PAD. Leg blood pressure should be measured using large thigh cuffs with auscultation at the popliteal artery or using a standard large arm cuff on the calf with simultaneous auscultation or palpation at the posterior tibial artery. Measurement of lower extremity blood pressures constitutes the basis of the ankle-brachial index (ABI) (see Chapter 43).

Consideration should be given to ambulatory blood pressure monitoring when uncertainty exists about the significance of recordings obtained in the clinic. This approach is especially useful for the patient

TABLE 13.5 Important Aspects of Blood Pressure Measurement

- Patient should be seated comfortably, with back supported and legs uncrossed and the upper arm bared.
- Upper arm should be at heart level.
- Cuff length and width should be 80% and 40% of arm circumference, respectively.
- Cuff should be deflated at <3 mm Hg/sec.
- Column or dial should be read to nearest 2 mm Hg.
- First audible Korotkoff sound is systolic pressure; last sound, diastolic pressure.
- There should be no talking between subject and observer (or other person).

From Daskalopolou SS, Rabi DM, Zarnke KB, et al. The 2015 Canadian Hypertension Education Program recommendations for blood pressure measurement, diagnosis, assessment of risk, prevention, and treatment of hypertension. *Can J Cardiol*. 2015;31:549–568; and Ringrose JS, McLean D, Ao P, et al. Effect of cuff design on auscultatory and oscillometric blood pressure measurements. *Am J Hypertens*. 2016;29(9):1063–1069.

with suspected "white coat hypertension" (see Chapter 26).[21] Measurement of normal or even low blood pressures in the clinic with evidence of hypertensive end organ damage should suggest masked hypertension,[22] which occurs more often than clinicians appreciate and may be present in the absence of obstructive PAD that can lower extremity blood pressure.

Orthostatic hypotension (a fall in blood pressure of more than 20 mm Hg systolic and/or more than 10 mm Hg diastolic in response to moving from the supine to the standing position within 3 minutes) may be accompanied by a lack of compensatory tachycardia, a response suggestive of autonomic insufficiency, as can occur in patients with diabetes or Parkinson disease. The heart rate–blood pressure response to standing also depends on age, hydration, medications, food, conditioning, and ambient temperature and humidity. In patients with postural orthostatic tachycardia syndrome (POTS), blood pressure does not usually fall on standing.

An increase in pulse pressure can represent increased vascular stiffness, usually secondary to aging or atherosclerosis. Aortic stiffness is increased in patients with Marfan syndrome and other connective tissue disorders and may contribute to risk for dissection. Peripheral indices may not correlate well with central aortic stiffness, which is a primary determinant of ventricular-vascular coupling.

Assessing the Pulses

The carotid artery pulse wave occurs within 40 milliseconds of the ascending aortic pulse and reflects aortic valve and ascending aortic function. The temporal arteries can be easily palpated to aid in the diagnosis of temporal arteritis. One of the two pedal pulses may not be palpable in a normal subject because of unusual anatomy (posterior tibial, less than 5%; dorsal pedis, less than 10%), but each pair should be symmetric. True congenital absence of a pulse is rare, and in most cases, pulses can be detected with a handheld Doppler device when not palpable. Simultaneous palpation of the brachial or radial pulse with the femoral pulse should be performed in young patients with hypertension to screen for aortic coarctation.

The contour of the pulses depends on the stroke volume, ejection velocity, vascular capacity and compliance, and systemic resistance. The palpable pulse reflects the merging of the antegrade pulsatile flow of blood and reflection of the propagated pulse returning from the periphery. The amplitude of the arterial pulse increases with distance from the heart. Normally, the incident (percussion) wave begins with systolic ejection (just after S_1) and is the predominant monophasic pulse appreciated at the bedside (Fig. 13.4). The incisura or dicrotic notch identifies aortic valve closure. A *bounding* pulse may occur in hyperkinetic states such as fever, anemia, and thyrotoxicosis,

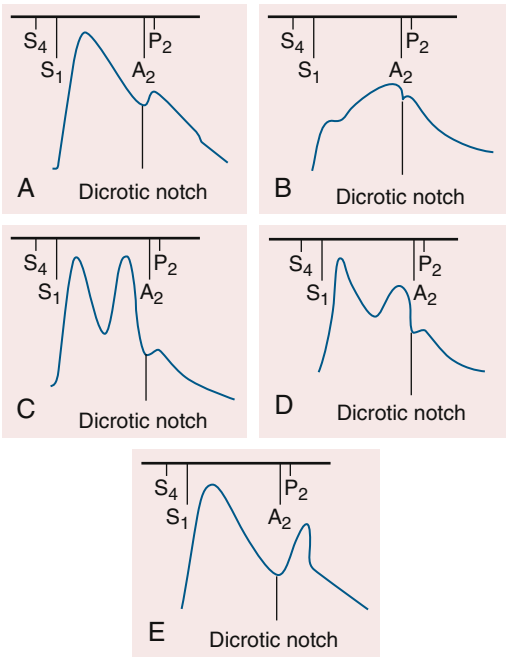


FIGURE 13.4 Carotid pulse waveforms and heart sounds. **A,** Normal. **B,** Aortic stenosis. Anacrotic pulse with slow upstroke and peak near S_2. **C,** Severe aortic regurgitation (AR): bifid pulse with two systolic peaks. **D,** Hypertrophic obstructive cardiomyopathy (HCM): bifid pulse with two systolic peaks. The second peak (tidal or reflected wave) is of lower amplitude than the initial percussion wave. **E,** Bifid pulse with systolic and diastolic peaks as may occur with sepsis or intra-aortic balloon counterpulsation. A_2, Aortic component of S_2; P_2, pulmonic component of S_2. (From Chatterjee K. Bedside evaluation of the heart: the physical examination. In: Chatterjee K, Parmley W, eds. *Cardiology: An Illustrated Text/Reference*. Philadelphia: JB Lippincott; 1991; and Braunwald E. The clinical examination. In: Braunwald E, Goldman L, eds. *Primary Cardiology*. 2nd ed. Philadelphia: WB Saunders; 2003:36.)

or in pathologic states such as severe bradycardia, AR, or arteriovenous fistula. A *bifid* pulse is created by two distinct pressure peaks. This phenomenon may occur with fever or after exercise in a normal person and is consistent with increased vascular compliance. With chronic severe AR, a large stroke volume ejected rapidly into a noncompliant arterial tree produces a reflected wave of sufficient amplitude to be palpated during systole, rendering the pulse bifid. Hypertrophic cardiomyopathy (HCM) can rarely produce a bifid systolic pulse with percussion and tidal waves (see Fig. 13.4). Diastolic augmentation of pressure with an intra-aortic balloon pump also results in a bifid pulse, though with the two components separated by aortic valve closure.

A fall in systolic pressure of more than 10 mm Hg with inspiration (*pulsus paradoxus*) is considered pathologic and a sign of pericardial tamponade or severe pulmonary disease; this phenomenon also can occur in obesity and pregnancy without clinical disease. Pulsus paradoxus is detected 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 beat, independent of respiratory phase. Between these two pressures, the sounds will be heard only intermittently (during expiration). Appreciation of this finding requires a slow decrease of the cuff pressure. Conditions such as tachycardia, AF, and tachypnea make its assessment difficult. Pulsus paradoxus may be palpable at the brachial artery when the pressure difference exceeds 15 mm Hg (see Chapter 86). Pulsus paradoxus is not specific for pericardial tamponade and can accompany massive pulmonary embolus, hemorrhagic shock, severe obstructive lung disease, or tension pneumothorax.

Pulsus alternans is defined by the beat-to-beat variability of the pulse amplitude (Fig. 13.5). It is present when only every other phase 1 Korotkoff sound is audible as the cuff pressure is slowly lowered, in

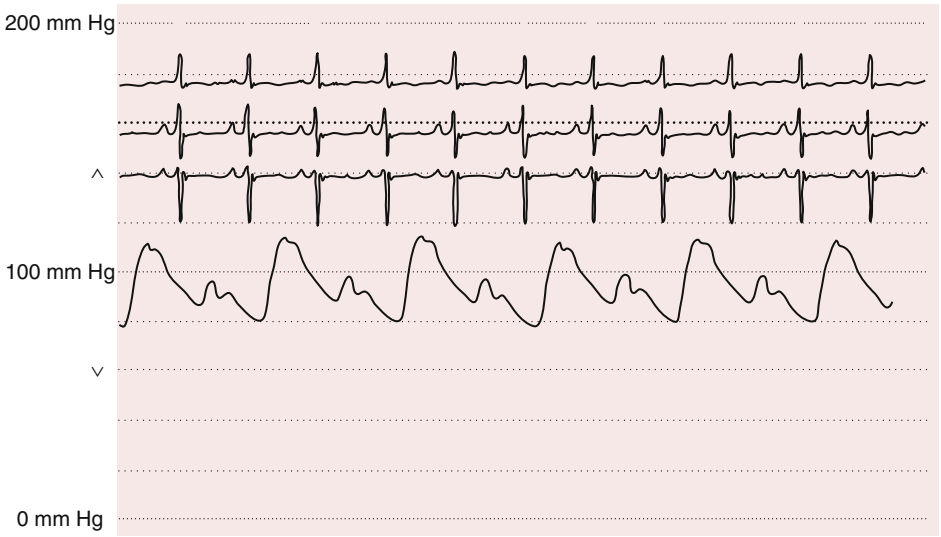


FIGURE 13.5 Pulsus alternans in a patient with severe left ventricular systolic dysfunction. The systolic pressure varies from beat to beat, independent of the respiratory cycle. The rhythm is sinus throughout.

a patient with a regular heart rhythm, independent of the respiratory cycle. Pulsus alternans generally occurs in severe heart failure, severe AR, hypertension, and hypovolemic states. It is attributed to cyclic changes in intracellular calcium and action potential duration. Association with electrocardiographic T wave alternans appears to increase arrhythmic risk.

Severe aortic stenosis may be suggested by a weak and delayed pulse (*pulsus parvus et tardus*) and is best appreciated by careful palpation of the carotid arteries (see Fig. 13.4; see Chapter 72). The delay is assessed during simultaneous auscultation of the heart sounds; the carotid upstroke should coincide with S_1. This finding is less specific in older, hypertensive patients with reduced vascular compliance and stiffer carotid arteries. An abrupt carotid upstroke with rapid fall-off characterizes the pulse of chronic AR (Corrigan or water-hammer pulse). The carotid upstroke also is rapid in older patients with isolated systolic hypertension and wide pulse pressures.

Pulsation of the abdominal aorta can be appreciated in the epigastric area. Femoral and popliteal artery aneurysms should be sought in patients with AAA disease or underlying connective tissue disease.

The history and physical examination findings can help assess the level of arterial obstruction in patients with lower extremity claudication (see Chapter 43). Auscultation for carotid, subclavian, aortic, and femoral artery bruits should be routine. The correlation between the presence of a bruit and the degree of vascular obstruction is weak. Extension of a bruit into diastole or a thrill generally indicates severe obstruction. Other causes of a bruit include arteriovenous fistulas and enhanced flow through normal arteries as, for example, in a young patient with fever.

Integrating the clinical history and presence of atherosclerotic risk factors improves the accuracy of the examination for the identification of lower extremity PAD. In an asymptomatic patient, the presence of a femoral bruit (LR, 4.8; 95% CI, 2.4 to 9.5) or any abnormality of the pulse (LR, 3.1; 95% CI, 3.1 to 6.6) increases the likelihood of PAD. The likelihood of significant PAD increases when there are lower extremity symptoms and cool skin (LR, 5.9; 95% CI, 4.1 to 8.6), pulse abnormalities (LR, 4.7; 95% CI, 2.2 to 9.9), or any bruit (LR, 5.6; 95% CI, 4.7 to 6.7). Abnormal pulse oximetry, defined by a more than 2% difference between finger and toe oxygen saturation, can also indicate lower extremity PAD and is comparable to the ABI (LR, 30.0; 95% CI, 7.6 to 121 versus LR, 24.8; 95% CI, 6.2 to 99.8).[23]

Inspection and Palpation of the Heart

The apical heartbeat may be visible in thin-chested adults. The left anterior chest wall may heave in patients with enlarged and hyperdynamic left ventricles. Right upper parasternal and sternoclavicular pulsations suggest ascending aortic aneurysm disease. A left parasternal lift indicates RV pressure or volume overload. A pulsation in the third intercostal space to the left of the sternum can indicate pulmonary artery hypertension. In very thin, tall patients, or in patients with emphysema and flattened diaphragms, the RV impulse may be visible in the epigastrium and should be distinguished from a pulsatile liver edge.

Palpation of the heart should begin with the patient in the supine position inclined at 30 degrees. If the heart is not palpable in this position, the patient should be examined either in the left lateral decubitus position with the left arm above the head or in the seated position, leaning forward. The point of maximal impulse normally is over the left ventricular (LV) apex beat and should be located in the midclavicular line at the fifth intercostal space. It is smaller than 2 cm in diameter and moves quickly away from the fingers. It is best appreciated at end-expiration, when the heart is closest to the chest wall. The normal impulse may not be palpable in obese or muscular patients or in those with thoracic cage deformities. LV cavity enlargement displaces the apex beat leftward and downward. A sustained apex beat is a sign of LV pressure overload (as in aortic stenosis or hypertension). A palpable, presystolic impulse corresponds to a fourth heart sound (S_4) and reflects the atrial contribution to ventricular diastolic filling of a noncompliant left ventricle. A prominent, rapid early filling wave in patients with advanced systolic heart failure may result in a palpable third sound (S_3), which may be present when the gallop itself is not audible (Video 13.3). A large ventricular aneurysm may yield a palpable and visible ectopic impulse discrete from the apex beat. HOCM rarely may cause a triple cadence apex beat, with contributions from a palpable S_4 and the two components of the systolic pulse.

A parasternal lift occurs with RV pressure or volume overload. Signs of TR (jugular venous *cv* waves) and/or pulmonary artery hypertension (loud, single, or palpable P_2) should be sought. An enlarged RV can give rise to a precordial lift that can extend across the precordium and obscure left-sided findings. Rarely, patients with severe MR will have a prominent left parasternal impulse because of systolic expansion of the left atrium and forward displacement of the heart. Lateral retraction of the chest wall may be present with isolated RV enlargement secondary to posterior displacement of the systolic LV impulse. Systolic and diastolic thrills signify turbulent, high-velocity blood flow. Their locations help to identify the origins of heart murmurs.

Auscultation of the Heart

Heart Sounds

First Heart Sound (S_1)

The normal first heart sound (S_1) comprises mitral (M_1) and tricuspid (T_1) valve closure. The two components usually are best heard at the lower left sternal border in younger subjects. Normal splitting of S_1 is accentuated with complete right bundle branch block. S_1 intensity increases in the early stages of rheumatic mitral stenosis when the valve leaflets are still pliable, in hyperkinetic states, and with short P-R intervals (less than 160 milliseconds). S_1 becomes softer in the late stages

of stenosis, when the leaflets are rigid and calcified, with contractile dysfunction, beta-adrenergic receptor blockers, and long P-R intervals (greater than 200 milliseconds). Other factors that can decrease the intensity of the heart sounds and murmurs include mechanical ventilation, obstructive lung disease, obesity, pendulous breasts, pneumothorax, and pericardial effusion.

Second Heart Sound (S_2)

The second heart sound (S_2) comprises aortic (A_2) and pulmonic (P_2) valve closure. With normal, or physiologic, splitting, the A_2–P_2 interval increases during inspiration and narrows with expiration. The individual components are best heard at the second left interspace with the patient in the supine position. The A_2–P_2 interval widens with complete right bundle branch block because of delayed pulmonic valve closure, and with severe MR because of premature aortic valve closure. Unusually narrow but physiologic splitting of S_2, with an increase in the intensity of P_2 relative to A_2, indicates pulmonary artery hypertension. With fixed splitting, the A_2–P_2 interval is wide and remains unchanged during the respiratory cycle, indicating ostium secundum ASD. Reverse, or paradoxical, splitting occurs as a consequence of a pathologic delay in aortic valve closure, as may occur with complete left bundle branch block, RV apical pacing, severe aortic stenosis, HCM, and myocardial ischemia. A_2 normally is louder than P_2 and can be heard at most sites across the precordium. When both components can be heard at the lower left sternal border or apex, or when P_2 can be palpated at the second left interspace, pulmonary hypertension is present. The intensity of A_2 and P_2 decreases with aortic and pulmonic stenosis, respectively. A single S_2 may result.

Systolic Sounds

An ejection sound is a high-pitched, early systolic sound that coincides in timing with the upstroke of the carotid pulse and usually is associated with congenital bicuspid aortic or pulmonic valve disease, or sometimes with aortic or pulmonic root dilation and normal semilunar valves. The ejection sound accompanying pulmonic valve disease decreases in intensity with inspiration, the only right-sided cardiac event to behave in this manner. Ejection sounds disappear as the culprit valve loses its pliability over time. They often are better heard at the lower left sternal border than at the base of the heart. Nonejection clicks, which occur after the upstroke of the carotid pulse, are related to MVP. A systolic murmur may or may not follow. With standing, ventricular preload and afterload decrease and the click and murmur move closer to S_1. With squatting, ventricular preload and afterload increase, the prolapsing mitral valve tenses later in systole, and the click and murmur move away from S_1 (Fig. 13.6).

Diastolic Sounds

The high-pitched opening snap (OS) of mitral stenosis occurs a short distance after S_2; the A_2–OS interval is inversely proportional to the height of the left atrial (LA)-LV diastolic pressure gradient. The intensity of both S_1 and OS decreases with progressive calcification and rigidity of the anterior mitral leaflet. A pericardial knock (PK) is a high-pitched early diastolic sound, which corresponds in timing to the abrupt cessation of ventricular expansion after AV valve opening and to the prominent *y* descent seen in the jugular venous waveform in patients with constrictive pericarditis. A tumor "plop" rarely is heard with atrial myxoma; it is a low-pitched sound sometimes only heard in certain positions that arises from the diastolic prolapse of the tumor across the mitral valve. A diastolic murmur may be present, although most myxomas cause no sound. A third heart sound (S_3) occurs during the rapid filling phase of ventricular diastole. An S_3 may be normally present in children, adolescents, and young adults, but indicates systolic heart failure in older adults and carries important prognostic weight. A left-sided S_3 is a low-pitched sound best heard over the LV apex with the patient in the left lateral decubitus position, whereas a right-sided S_3 is usually heard at the lower left sternal border or in the subxiphoid position with the patient supine and may become louder with inspiration. A fourth heart sound (S_4) occurs during the atrial filling phase of ventricular diastole and is thought to indicate presystolic ventricular expansion. An S_4 is especially common in patients with accentuated atrial contribution to ventricular filling (e.g., LV hypertrophy).

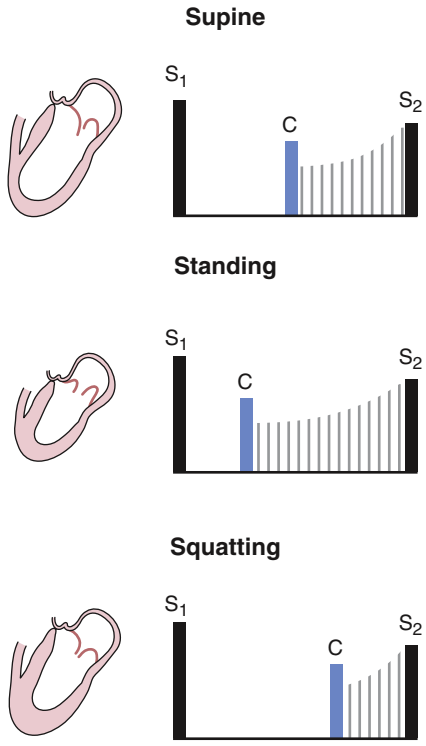


FIGURE 13.6 Behavior of the nonejection click (*C*) and systolic murmur of mitral valve prolapse. With standing, venous return decreases, the heart becomes smaller, and prolapse occurs earlier in systole. The click and murmur move closer to S_1. With squatting, venous return increases, causing an increase in left ventricular chamber size. The click and murmur occur later in systole and move away from S_1.

Cardiac Murmurs

Heart murmurs result from audible vibrations caused by increased turbulence and are defined by their timing within the cardiac cycle (Tables 13.6 and 13.7; Figs. 13.7 and 13.8). Not all murmurs indicate valvular or structural heart disease. The accurate identification of a functional (benign) systolic murmur can obviate the need for echocardiography in many healthy subjects. The magnitude, dynamic change, and duration of the pressure difference between two cardiac chambers, or between the ventricles and their respective great arteries, dictate the duration, frequency, configuration, and intensity of murmurs. Intensity is graded on a scale of 1 to 6; a palpable thrill characterizes murmurs of grade 4 or higher intensity. Other important attributes that aid in identification include location, radiation, and response to bedside maneuvers, including quiet respiration.

Systolic Murmurs

Systolic murmurs are early, midsystolic, late, or holosystolic in timing. Acute severe MR results in a decrescendo, early systolic murmur because of the steep rise in pressure within the noncompliant left atrium (see Fig. 13.8). Severe MR associated with posterior mitral leaflet prolapse or flail radiates anteriorly and to the base; MR caused by anterior leaflet involvement radiates posteriorly and to the axilla. With acute TR in patients with normal pulmonary artery pressures, an early systolic murmur, which increases in intensity with inspiration, may be audible at the lower left sternal border, and regurgitant *cv* waves may be

TABLE 13.6 Principal Causes of Heart Murmurs

Systolic Murmurs

Early Systolic

Mitral—acute MR

VSD
- Muscular
- Nonrestrictive with pulmonary hypertension

Tricuspid—TR with normal pulmonary artery pressure

Midsystolic

Aortic
- Obstructive
  - Supravalvular—supravalvular aortic stenosis, coarctation of the aorta
  - Valvular—aortic stenosis and sclerosis
  - Subvalvular—discrete, tunnel, or HCM
- Increased flow, hyperkinetic states, AR, complete heart block
- Dilation of ascending aorta, atheroma, aortitis

Pulmonary
- Obstructive
  - Supravalvular—pulmonary artery stenosis
  - Valvular—pulmonic valve stenosis
  - Subvalvular—infundibular stenosis (dynamic)
- Increased flow, hyperkinetic states, left-to-right shunt (e.g., ASD)
- Dilation of pulmonary artery

Late Systolic

Mitral—MVP, acute myocardial ischemia

Tricuspid—tricuspid valve prolapse

Holosystolic

Atrioventricular valve regurgitation (MR, TR)

Left-to-right shunt at ventricular level (VSD)

Diastolic Murmurs

Early Diastolic

Aortic regurgitation
- Valvular—congenital (bicuspid valve), rheumatic deformity, endocarditis, prolapse, trauma, postvalvulotomy
- Dilation of valve annulus—aortic dissection, annuloaortic ectasia, cystic medial degeneration, hypertension, ankylosing spondylitis
- Widening of commissures—syphilis

Pulmonic regurgitation
- Valvular—postvalvulotomy, endocarditis, rheumatic fever, carcinoid
- Dilation of valve annulus—pulmonary hypertension; Marfan syndrome
- Congenital—isolated or associated with tetralogy of Fallot, VSD, pulmonic stenosis

Mid-diastolic

Mitral
- Mitral stenosis
- Carey Coombs murmur (mid-diastolic apical murmur in acute rheumatic fever)
- Increased flow across nonstenotic mitral valve (e.g., MR, VSD, PDA, high-output states, complete heart block)

Tricuspid
- Tricuspid stenosis
- Increased flow across nonstenotic tricuspid valve (e.g., TR, ASD, anomalous pulmonary venous return)

Left and right atrial tumors (myxoma)

Severe or eccentric AR (Austin Flint murmur)

Late Diastolic

Presystolic accentuation of mitral stenosis murmur

Austin Flint murmur of severe or eccentric AR

Continuous Murmurs

PDA

Coronary arteriovenous fistula

Ruptured sinus of Valsalva aneurysm

Aortic septal defect

Cervical venous hum

Anomalous left coronary artery

Proximal coronary artery stenosis

Mammary soufflé of pregnancy

Pulmonary artery branch stenosis

Bronchial collateral circulation

Small (restrictive) ASD with mitral stenosis

Intercostal arteriovenous fistula

AR, Aortic regurgitation; *ASD*, atrial septal defect; *HCM*, hypertrophic cardiomyopathy; *MR*, mitral regurgitation; *MVP*, mitral valve prolapse; *PDA*, patent ductus arteriosus; *TR*, tricuspid regurgitation; *VSD*, ventricular septal defect.

From Braunwald E, Perloff JK. Physical examination of the heart and circulation. In Zipes DP, et al., eds. *Braunwald's Heart Disease: A Textbook of Cardiovascular Medicine*. 7th ed. Philadelphia: Saunders; 2005:77–106; and Norton PJ, O'Rourke RA. Approach to the patient with a heart murmur. In: Braunwald E, Goldman L, eds. *Primary Cardiology*. 2nd ed. Philadelphia: Elsevier; 2003:151–168.

visible in the jugular venous pulse. Midsystolic murmurs begin after S_1 and end before S_2; they usually are crescendo-decrescendo in configuration. Aortic stenosis or sclerosis causes most midsystolic murmurs in adults. Accurate characterization of the severity of aortic stenosis at the bedside depends on cardiac output, stiffness of the carotid arteries, and associated findings. Other causes of a midsystolic heart murmur include HCM, pulmonic stenosis, and increased pulmonary blood flow in patients with a large ASD and a left-to-right shunt. An isolated grade 1 or 2 midsystolic murmur in the absence of symptoms or other signs of heart disease is a benign finding that does not warrant further evaluation, including echocardiography. A mid-to-late, apical systolic murmur usually indicates MVP; one or more nonejection clicks may be present. A similar murmur may be heard transiently during an episode of acute myocardial ischemia. The intensity of the murmur will vary with LV afterload, as is also the case for the murmur of chronic, secondary MR associated with ischemic or dilated cardiomyopathy. Holosystolic murmurs, which are plateau in configuration, derive from the continuous and wide pressure gradient between two cardiac chambers—the left ventricle and left atrium with chronic MR, the right ventricle and right atrium with chronic TR, and the left ventricle and right ventricle with membranous ventricular septal defect (VSD) without pulmonary hypertension. MR is best heard over the cardiac apex, TR at the lower left sternal border, and a VSD murmur at the mid-left sternal border, where a thrill is palpable in most patients. TR most commonly is secondary to annular dilation from RV enlargement with papillary muscle displacement and failure of tricuspid leaflet coaptation. Pulmonary artery hypertension also may be present.

Diastolic Murmurs

Diastolic murmurs invariably signify cardiac disease. Chronic AR causes a high-pitched decrescendo early to mid-diastolic murmur. With primary aortic valve disease, the murmur is best heard along the left sternal border, whereas with root enlargement and secondary AR, the murmur may radiate along the right sternal border. A midsystolic murmur caused by augmented and accelerated blood flow is also present with moderate to severe AR and need not signify valve or

TABLE 13.7 Interventions for Altering Intensity of Cardiac Murmurs

Respiration: Right-sided murmurs generally increase with inspiration. Left-sided murmurs usually are louder during expiration.

Valsalva maneuver: Most murmurs decrease in length and intensity. Two exceptions are the systolic murmur of HCM, 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 baseline intensity earlier than left-sided murmurs.

Exercise: Murmurs caused by blood flow across normal or obstructed valves (as in pulmonic and mitral stenosis) become louder with both isotonic and isometric (handgrip) exercise. Murmurs of MR, VSD, and AR also increase with handgrip exercise.

Positional changes: With standing, most murmurs diminish; two exceptions are the murmur of HCM, which becomes louder, and that of MVP, which lengthens and often is intensified. With squatting, most murmurs become louder, but those of HCM and MVP usually soften and may disappear. Passive leg raising usually produces the same results as squatting.

Post–ventricular premature beat or AF: Murmurs originating at normal or stenotic semilunar valves increase in intensity during the cardiac cycle after a ventricular premature beat or in the beat after a long cycle length in AF. By contrast, systolic murmurs caused by AV valve regurgitation do not change, diminish (papillary muscle dysfunction), or become shorter after a premature beat (MVP).

Pharmacologic interventions: During the initial relative hypotension after amyl nitrite inhalation, murmurs of MR, VSD, and AR decrease in intensity, whereas the murmur of AS increases in intensity because of increased stroke volume. During the later tachycardia phase, murmurs of mitral stenosis and right-sided lesions also become louder. This intervention may help distinguish the murmur of the Austin Flint phenomenon from that of mitral stenosis. The response in MVP often is biphasic (softer and then louder than control).

Transient arterial occlusion: Transient external compression of both brachial arteries by bilateral cuff inflation to 20 mm Hg greater than peak systolic pressure augments the murmurs of MR, VSD, and AR, but not murmurs from other causes.

AR, Aortic regurgitation; *HCM*, hypertrophic cardiomyopathy; *MR*, mitral regurgitation; *MVP*, mitral valve prolapse; *VSD*, ventricular septal defect.

From Bonow RO, Carabello BA, Chatterjee K, et al. ACC/AHA 2006 guidelines for the management of patients with valvular heart disease: a report of the American College of Cardiology/American Heart Association Task Force on Practice Guidelines (Writing Committee to Revise the 1998 Guidelines for the Management of Patients with Valvular Heart Disease) developed in collaboration with the Society of Cardiovascular Anesthesiologists endorsed by the Society for Cardiovascular Angiography and Interventions and the Society of Thoracic Surgeons. *J Am Coll Cardiol*. 2006;48:e18.

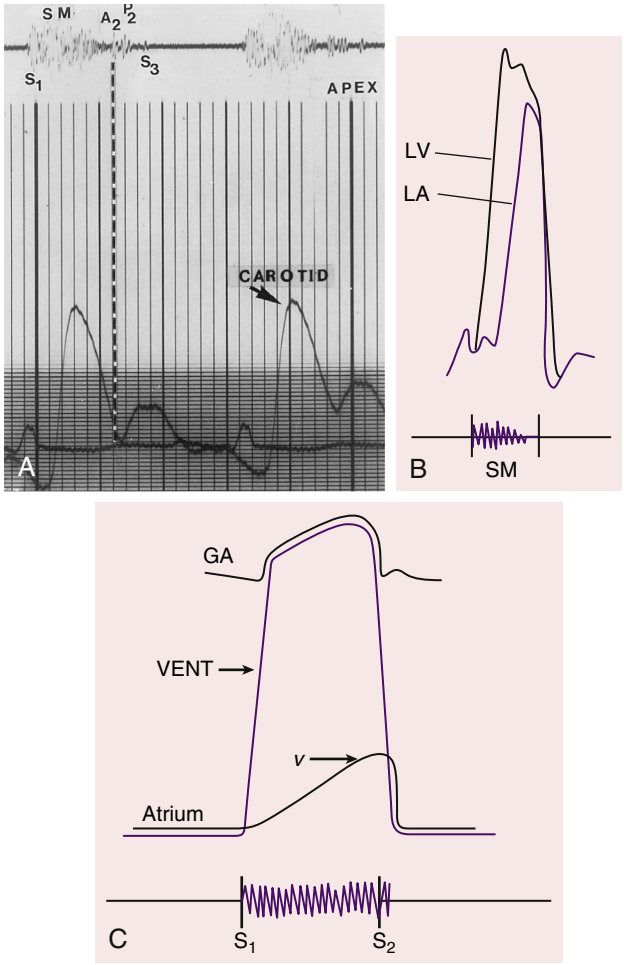


FIGURE 13.8 **A,** Phonocardiogram (*top*) obtained in a patient with acute severe mitral regurgitation (MR) showing a decrescendo early systolic murmur and diastolic filling sound (S_3). **B,** Left ventricular (LV) and left atrial (LA) pressure waveforms demonstrating the abrupt rise in LA pressure and attenuation of the LV-LA pressure gradient, resulting in the duration and configuration of the murmur. **C,** Illustration of great artery (GA) and ventricular (VENT) and atrial pressures with corresponding phonocardiogram in chronic MR or TR. Note the holosystolic timing and plateau configuration of the murmur, both of which derive from the large ventricular-atrial pressure gradient throughout systole. *SM,* Systolic murmur; *v*, v wave. (From Braunwald E, Perloff JK. Physical examination of the heart and circulation. In: Zipes D, et al. eds. *Braunwald's Heart Disease: A Textbook of Cardiovascular Medicine*. 7th ed. Philadelphia: Saunders; 2005:97.)

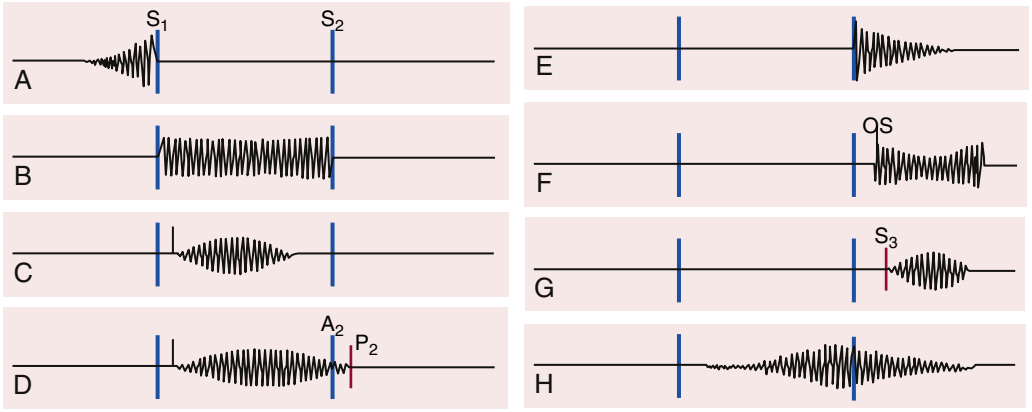


FIGURE 13.7 Diagram of principal heart murmurs. **A,** Presystolic accentuation of the murmur of mitral stenosis with sinus rhythm. **B,** Holosystolic murmur of chronic, severe mitral regurgitation or tricuspid regurgitation, or ventricular septal defect without severe pulmonary hypertension. **C,** Ejection sound and crescendo-decrescendo murmur of bicuspid aortic stenosis. **D,** Ejection sound and crescendo-decrescendo murmur that extends to P_2 in bicuspid pulmonic stenosis. **E,** Early decrescendo diastolic murmur of aortic regurgitation or pulmonic regurgitation. **F,** Opening snap (OS) and mid-diastolic rumble of mitral stenosis. **G,** Diastolic filling sound (S_3) and mid-diastolic murmur associated with severe MR, TR, or atrial septal defect with significant left-to-right shunt. **H,** Continuous murmur of patent ductus arteriosus that envelops S_2. (Modified from Wood P. *Diseases of the Heart and Circulation*. Philadelphia: Lippincott; 1968; and O'Rourke RA, Braunwald E. Physical examination of the cardiovascular system. In: Kasper D, Braunwald E, Fauci A, et al. eds. *Harrison's Principles of Internal Medicine*. 16th ed. New York: McGraw-Hill; 2005:1309.)

outflow tract obstruction. The diastolic murmur is both softer and of shorter duration in acute AR, as a result of the rapid rise in LV diastolic pressure and the diminution of the aortic-LV diastolic pressure gradient. Additional features of acute AR include tachycardia, a soft S_1, and the absence of peripheral findings of significant diastolic run-off. The murmur of pulmonic regurgitation (PR) is heard along the left sternal border and most often is due to annular enlargement from chronic pulmonary artery hypertension (Graham-Steele murmur). Signs of RV pressure overload are present. PR also can occur with a congenitally deformed valve and is invariably present after repair of tetralogy of Fallot. In these settings, the murmur is relatively softer and lower-pitched. The severity of PR after surgical repair can be underappreciated. Mitral stenosis is the classic cause of a mid- to late diastolic murmur (see Fig. 13.7). The stenosis also may be "silent"—for example, in patients with low cardiac output or large body habitus. The murmur is best heard over the apex with the patient in the left lateral decubitus position, is low-pitched (rumbling), and is introduced by an OS in the early stages of the disease. Presystolic accentuation (an increase in the intensity of the murmur in late diastole following atrial contraction) occurs in patients in sinus rhythm. Left-sided events usually obscure findings in patients with rheumatic tricuspid stenosis. Functional mitral stenosis or tricuspid stenosis refers to mid-diastolic murmurs created by increased, accelerated transvalvular flow, without valvular obstruction, in the setting of severe MR or TR, respectively, or ASD with a large left-to-right shunt. The low-pitched mid- to late apical diastolic murmur sometimes associated with AR (Austin Flint murmur) can be distinguished from mitral stenosis on the basis of its response to vasodilators and the presence of associated findings. Less common causes of a mid-diastolic murmur include atrial myxoma, complete heart block, and acute rheumatic mitral valvulitis (Carey Coombs murmur).

Continuous Murmurs

The presence of a continuous murmur implies a pressure gradient between two chambers or vessels during both systole and diastole. These murmurs begin in systole, peak near S_2, and continue into diastole. They can be difficult to distinguish from systolic and diastolic murmurs in patients with mixed aortic or pulmonic valve disease. Examples are the murmurs associated with PDA, ruptured sinus of Valsalva aneurysm, and coronary, great vessel, or hemodialysis-related arteriovenous fistulas. The cervical venous hum and mammary soufflé of pregnancy are two benign variants.

Dynamic Auscultation

Simple bedside maneuvers can help identify heart murmurs and characterize their significance (see Table 13.7). Right-sided events, except for the pulmonic ejection sound, increase with inspiration and decrease with expiration; left-sided events behave oppositely (100% sensitivity, 88% specificity). The intensity of the murmurs associated with MR, VSD, and AR will increase in response to maneuvers that increase LV afterload (e.g., handgrip, vasopressor administration) and decrease after exposure to vasodilating agents (e.g., amyl nitrite). The response of the murmur associated with MVP to standing and squatting has previously been described. The murmur of HOCM behaves in a directionally similar manner, becoming softer and shorter with squatting (95% sensitivity, 85% specificity) and longer and louder on rapid standing (95% sensitivity, 84% specificity). The intensity of the murmur of HCM also increases with the Valsalva maneuver (65% sensitivity, 95% 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 AF, suggests aortic stenosis rather than MR, particularly in an older patient, in whom the murmur of aortic stenosis is well transmitted to the apex (Gallavardin effect). Systolic murmurs that are due to LV outflow obstruction, including those caused by aortic stenosis, will increase in intensity in the beat following a premature beat because of the combined effects of enhanced LV filling and post-extrasystolic potentiation of contractile function. Forward flow accelerates, causing an increase in the gradient and a louder murmur. The intensity of the murmur of MR does not change in the post-premature beat, because relatively little further increase occurs in mitral valve flow or change in the LV-LA gradient.

INTEGRATED, EVIDENCE-BASED APPROACH TO SPECIFIC CARDIAC DISORDERS

Heart Failure (see Part VI)

History

Both exertional and resting symptoms should be investigated. Common signs and symptoms include dyspnea, fatigue, exercise limitation, orthopnea, and edema. In a review of 22 studies of adult patients presenting to an emergency department with dyspnea, the probability of heart failure was best predicted by a past history of heart failure (LR, 5.8; 95% CI, 4.1 to 8.0), paroxysmal nocturnal dyspnea (LR, 2.6; 95% CI, 1.5 to 4.5), a third heart sound (LR, 11; 95% CI, 4.9 to 25), or AF (LR, 3.8; 95% CI, 1.7 to 8.8).[24] An initial clinical impression of heart failure as noted by a physician was one of the stronger clinical predictors of this diagnosis (LR, 4.4; 95% CI, 1.8 to 10.0). With the exception of paroxysmal nocturnal dyspnea, these same features also predicted heart failure when there was concomitant pulmonary disease. The addition of testing for N-terminal pro–B-type natriuretic peptide (NT-pro-BNP) increases diagnostic accuracy only modestly (C-statistic, 0.83 versus 0.86).[7]

Severe and sudden onset dyspnea indicates acute pulmonary edema, typically precipitated by ischemia, arrhythmia, sudden left-sided valvular regurgitation, and/or accelerated hypertension. It is important to exclude other causes such as pulmonary embolism and pneumothorax. The extent of limitation also should be defined because functional capacity, as assessed by NYHA classification, strongly and independently predicts the risk of death for patients with heart failure. Self-reported functional capacity and objectively measured cardiovascular performance can differ substantially. Symptoms that occur at rest may have greater predictive value for the diagnosis of heart failure than exertional symptoms. Orthopnea is not specific for heart failure and can occur in patients with asthma, ascites, or gastroesophageal reflux. Trepopnea, which is dyspnea or discomfort experienced in the lateral decubitus position, also may be present. Patients with heart failure prefer sleeping on their right side, and trepopnea probably accounts for the predominance of right-sided pleural effusions in this population. Shortness of breath may be particularly noticeable when bending forward, termed bendopnea. It is associated with higher supine right atrial and pulmonary capillary wedge pressures and is mediated by further elevations in these pressures with bending over. It is even more likely present when the resting cardiac index is low.[25] Paroxysmal nocturnal dyspnea also is common in patients with heart failure. Cheyne-Stokes respirations may be apparent when the patient is awake.[26] The prevalence of central sleep apnea or Cheyne-Stokes respirations ranges from 20% to 62% in various heart failure studies,[15] and either of these disorders is associated with an increased mortality risk.

Clinically evident edema or weight gain over days indicates volume excess but lags the clinical redistribution of intravascular volume from the splanchnic beds to the central veins. In patients with advanced right-sided heart failure, uncomfortable hepatomegaly and ascites may predominate. Patients with chronic heart failure often lack pulmonary rales or lower extremity edema. Gastrointestinal symptoms such as early satiety, nausea, vomiting, and belching are also common and are related to decreases in gastrointestinal blood flow and bowel edema, particularly in those with cardiac cachexia.[27]

Few studies have explored the predictive values of various signs and symptoms of heart failure. In a systematic review,[28] orthopnea only modestly predicted increased filling pressures. Dyspnea and edema were similarly useful but were most predictive when combined with physical examination findings (S_3, tachycardia, elevated JVP, low pulse pressure, rales, abdominojugular reflux sign). When combined with other findings, a total of three or more symptoms or signs predicted a greater than 90% likelihood of increased filling pressures if severe LV dysfunction was not known. By contrast, if one or no findings or symptoms were present, the likelihood of increased filling pressures was less

than 10%. The commonly used Framingham criteria for heart failure diagnosis in patients with reduced ejection fraction have only modest specificity (63%) and sensitivity (63%).

The distinction between heart failure with reduced ejection fraction, and that with preserved ejection fraction, can be made at the bedside with modest accuracy. Systolic function is more likely to be preserved when patients are female or older and have an increased BMI, but such findings lack adequate specificity and sensitivity to guide therapy. Furthermore, diastolic dysfunction does not exclude systolic dysfunction.

Physical Examination

In most patients with heart failure who require hospitalization, the reason for admission is volume overload; failure to relieve it has negative prognostic impact. Four signs are commonly used to predict elevated filling pressures: jugular venous distention/abdominojugular reflux sign, presence of an S_3 and/or S_4, rales, and pedal edema. In general, the use of a combination of findings, rather than reliance on isolated findings, improves diagnostic accuracy. Some clinicians advocate assessment of the heart failure patient along two basic axes—volume status ("dry" or "wet") and perfusion status ("warm" or "cold")—as a useful guide to therapy. This approach has prognostic usefulness, particularly in assessing patients at discharge after admission for heart failure. For example, patients discharged with a "wet" or "cold" profile experience worse outcomes (HR, 1.5; 95% CI, 1.1 to 12.1; $P = 0.017$) compared with those discharged "warm and dry" (HR 0.9; 95% CI, 0.7 to 2.1; $P = 0.5$).[28] Advanced training may be required to achieve this level of diagnostic precision with the physical examination.

Jugular Venous Pressure

The JVP provides the readiest bedside estimate of LV filling pressure. In the Evaluation Study of Congestive Heart Failure and Pulmonary Artery Catheterization Effectiveness (ESCAPE) trial, 82% of patients whose estimated right atrial pressure was higher than 8 mm Hg (10.5 cm H_2O) had a measured right atrial pressure higher than 8 mm Hg. The same investigators also identified 9 of the 11 patients with pressures lower than 8 mm Hg.[28] Although the JVP estimates RV filling pressure, it has a predictable relationship with pulmonary artery wedge pressure. Drazner and colleagues[28] found that the right atrial pressure reliably predicted the pulmonary artery wedge pressure; the positive predictive value of a right atrial pressure higher than 10 mm Hg for a pulmonary artery wedge pressure higher than 22 mm Hg was 88%. In addition, the pulmonary artery systolic pressure could be estimated as twice the wedge pressure. In the ESCAPE trial, an estimated right atrial pressure higher than 12 mm Hg and two-pillow orthopnea were the only bedside parameters that provided incremental value to the prediction of a pulmonary artery wedge pressure higher than 22 mm Hg, and compared favorably with BNP levels.[28] Echocardiography and BNP determinations may not always provide incremental value to the clinical assessment of heart failure by experienced observers.[29]

An elevated JVP has prognostic significance. Drazner and associates[28] demonstrated that the presence of jugular vein distention signifying elevated pressures at the time of enrollment in a large clinical heart failure trial (11% of the Studies of Left Ventricular Dysfunction [SOLVD] treatment study participants), after adjustment for other markers of disease severity, predicted heart failure hospitalizations (relative risk [RR], 1.32; 95% CI, 1.08 to 1.62), death from pump failure (RR, 1.37; 95% CI, 1.07 to 1.75), and death plus heart failure hospitalization (RR, 1.30; 95% CI, 1.11 to 1.53) (**eFig. 13.1**). The investigators extended these observations to asymptomatic subjects enrolled in the SOLVD prevention study, among whom jugular vein distention was less common (1.7% of the study population).[28] In a contemporary clinical trial, physical examination evidence of congestion remained predictive of outcome and independent of symptoms, risk scores, and natriuretic peptide levels.[30] Signs of congestion have also been shown to be independently associated with cardiovascular death, heart failure hospitalization, and all-cause mortality in heart failure with preserved ejection fraction.[31]

In patients presenting with dyspnea, the abdominojugular reflux sign is useful in predicting heart failure (LR, 6.0; 95% CI, 0.8 to 51) and suggests a pulmonary artery wedge pressure higher than 15 mm Hg (LR, 6.7; 95% CI, 3.3 to 13.4). The presence of jugular vein distention, either at rest or induced, had the best combination of sensitivity (81%), specificity (80%), and predictive accuracy (81%) for elevation of the pulmonary artery wedge pressure. Ultrasound assessment of IJV dimension, both at rest and during the Valsalva maneuver, associates independently with prognosis in ambulatory heart failure patients with reduced ejection fraction.[32]

Third and Fourth Heart Sounds

The third heart sound (S_3) predicts ejection fraction poorly because it reflects primarily diastolic rather than systolic performance. In patients with heart failure, an S_3 is equally prevalent in those with and without LV systolic dysfunction. A rigorous assessment of the S_3 was conducted by Marcus and colleagues in 100 patients with various cardiovascular conditions undergoing elective cardiac catheterization. Cardiology fellows ($n = 18$; K statistic, 0.37; $P < 0.001$) and faculty ($n = 26$; K statistic, 0.29; $P = 0.003$) performed better than residents ($n = 102$; no significant agreement) in the identification of a phonocardiographically confirmed S_3.[33] Furthermore, an S_3 predicted an increase in both LV end-diastolic pressure (LVEDP) (greater than 15 mm Hg) and BNP (greater than 100 pg/mL) and depressed ventricular systolic function (ejection fraction less than 0.50), although sensitivities were low (32% to 52%).[34] An S_4 had comparable sensitivity (40% to 46%) but inferior specificity (72% to 80% for an S_4 versus 87% to 92% for an S_3). A third heart sound frequently may be heard in patients referred for cardiac transplant evaluation but is a poor predictor of elevated filling pressures. Alternatively, the lack of an S_3 cannot exclude a diagnosis of heart failure, but its presence reliably indicates ventricular dysfunction.

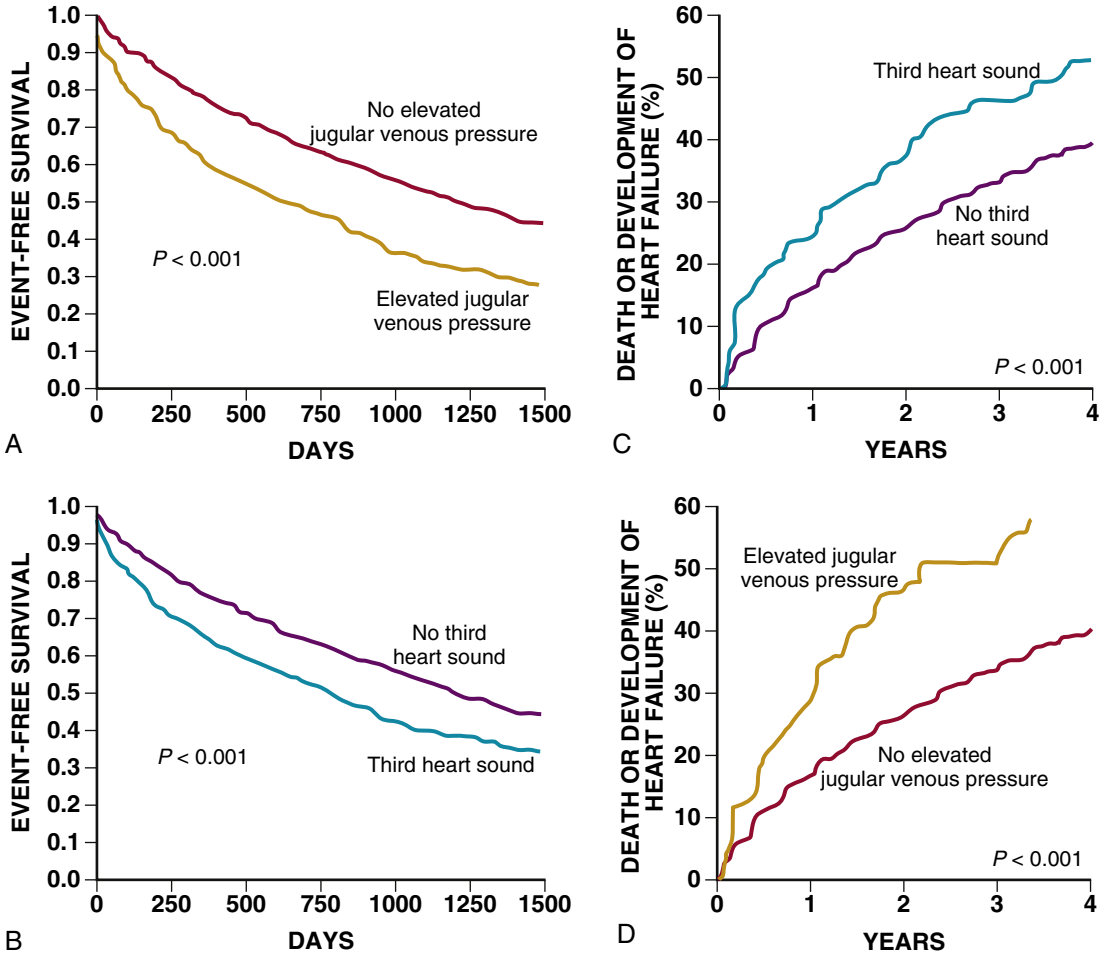
The prognostic value of an S_3 in chronic heart failure was established in the SOLVD treatment and prevention studies.[28] The investigators found that an S_3 predicted cardiovascular morbidity and mortality (see eFig. 13.1). The RR for heart failure hospitalization and death in patients with an S_3 was of comparable magnitude in the prevention and treatment cohorts. These observations remained significant after adjustment for markers of disease severity and were even more powerful when combined with the presence of an elevated JVP. An S_3 also predicts a higher risk of adverse outcomes in other settings, such as that of MI or noncardiac surgery.

Rales and Edema

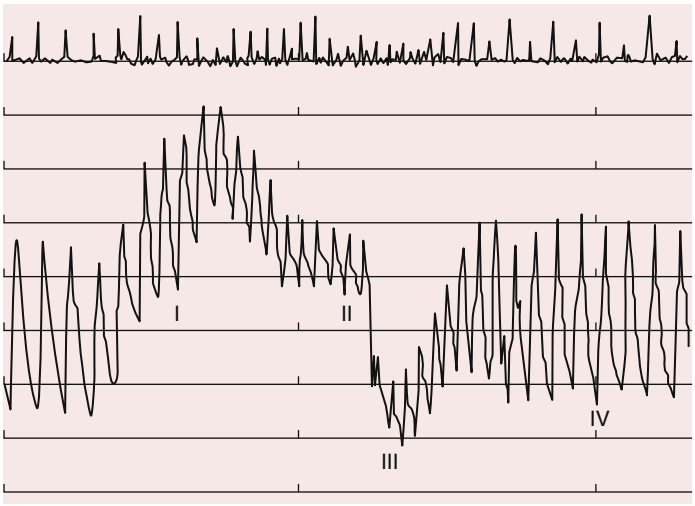
In patients with chronic heart failure, approximately 75% to 80% of participants lacked rales despite elevated pulmonary artery wedge pressures, presumably because of enhanced lymphatic drainage. Recent studies have incorporated the findings from lung ultrasound (LR 7.4; 95% CI 4.2 to 12.8) in the assessment of emergency room patients with acute heart failure[35] and in ambulatory heart failure patients.[36] In the latter setting, lung ultrasound findings can identify patients with worse prognosis. When pulmonary adventitious sounds are present, specific characteristics may help elucidate a pulmonary rather than cardiac disorder (see Fig. 13.2). The chest radiograph similarly lacked sensitivity for increased filling pressures in these studies. Pedal edema is neither sensitive nor specific for the diagnosis of heart failure and has low predictive value as an isolated variable.

Valsalva Maneuver

The blood pressure response to the Valsalva maneuver can be measured noninvasively using a blood pressure cuff or commercially available devices. The Valsalva maneuver consists of four phases (Figs. 13.9 and 13.10). In a normal response, Korotkoff sounds are audible only during phases I and IV, because the systolic pressure normally rises at the onset and release of the strain phase. Two abnormal responses to the Valsalva maneuver in heart failure are recognized: (1) absence of the phase IV overshoot and (2) the square-wave response (see Fig. 13.10). The absent overshoot pattern indicates decreased systolic function; the square-wave response indicates elevated filling pressures and appears to be independent of ejection fraction. The responses can be quantified using the pulse amplitude ratio if the pulse pressure is measured during the maneuver. This ratio compares the minimum pulse pressure at the end of the strain phase against the maximum pulse pressure at the onset of the strain phase; a higher ratio is consistent with a square-wave response.



EFIGURE 13.1 Kaplan-Meier plots demonstrating prognostic value of an elevated jugular venous pressure and third heart sound (S_3) in symptomatic (**A** and **B**) and asymptomatic (**C** and **D**) patients with heart failure who also had systolic dysfunction. (**A** and **B** from Drazner MH, Rame JE, Stevenson LW, Dries DL. Prognostic importance of elevated jugular venous pressure and a third heart sound in patients with heart failure. *N Engl J Med*. 2001;345:574; **C** and **D** from Drazner MH, Rame JE, Dries DL. Third heart sound and elevated jugular venous pressure as markers of the subsequent development of heart failure in patients with asymptomatic left ventricular dysfunction. *Am J Med*. 2003;114:431.)



Phase I: Increase in systolic pressure with initial strain due to increase in intrathoracic pressure

Phase II: Decrease in stroke volume and pulse pressure and reflex tachycardia with continued strain due to decrease in venous return and increase in vascular resistance

Phase III: Brief, sudden decrease in systolic pressure due to sudden decrease in intrathoracic pressure

Phase IV: Overshoot of systolic pressure and reflex bradycardia due to increased venous return and decreased systemic vascular resistance

FIGURE 13.9 The normal Valsalva response. (From Nishimura RA, Tajik AJ. The Valsalva maneuver—3 centuries later. *Mayo Clin Proc.* 2004;79:577.)

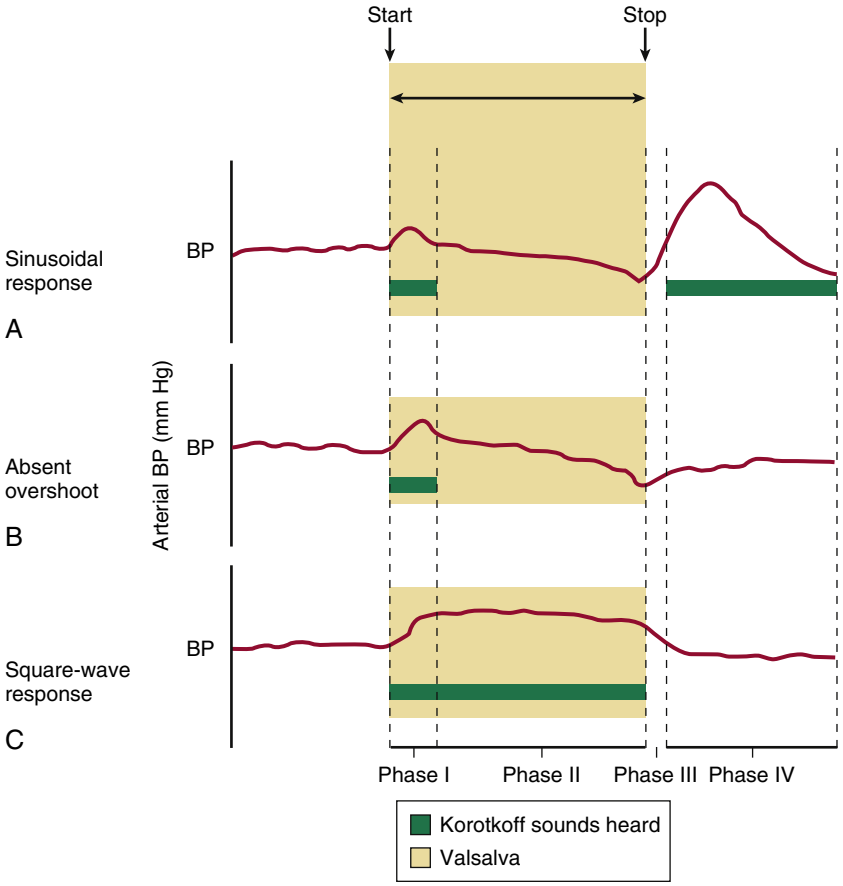


FIGURE 13.10 Abnormal Valsalva responses assessed using the pattern of Korotkoff sounds. **A,** Normal, sinusoidal response with sounds intermittent during strain and release. **B,** Briefly audible sounds during initial strain phase suggests only impaired systolic function in absence of fluid overload. **C,** Persistence of Korotkoff sounds throughout strain phase suggests elevated left ventricular filling pressures. *BP,* Blood pressure. (From Shamsham F, Mitchell J. Essentials of the diagnosis of heart failure. *Am Fam Physician.* 2000;61:1319.)

Other Findings

In the absence of hypertension, the pulse pressure is determined by the stroke volume and vascular stiffness and can be used to assess cardiac output. In a cohort of patients with chronic systolic heart failure, the proportional pulse pressure ([systolic – diastolic]/systolic) correlated well with cardiac index (correlation coefficient [r] = 0.82; $P < 0.001$), stroke volume index ($r = 0.78$; $P < 0.001$), and the inverse of systemic vascular resistance ($r = 0.65$; $P < 0.001$). Using a proportional pulse pressure of 25%, the cardiac index could be predicted: if the value was lower than 25%, the cardiac index was less than 2.2 L/min/m^2 in 91% of patients; if the value was higher than 25%, the cardiac index was higher than 2.2 L/min/m^2 in 83% of patients.[37] Circulation time has also been used to assess the cardiac output. Using oxygen as the indicator, a time from a breath hold to the nadir of finger oximetry of greater than 34 seconds has been associated with a cardiac output of less than 4 L/min.[38]

Heart rate is also a powerful indicator of prognosis in heart failure. A resting heart rate (in sinus rhythm) of greater than 70 to 75 beats/min is an independent predictor of mortality. When the heart rate is decreased by ivabradine on a background of beta-blocker therapy in patients with sinus rhythm, heart failure hospitalizations are decreased.[39] An attenuated heart rate increase with immediate standing (e.g., ≤3 beats/min) may also reflect the dysautonomia of heart failure and has been associated with death or heart failure hospitalization. A greater increase of heart rate with standing over time is associated with greater heart failure hospitalization-free survival.[40] In patients admitted with acute decompensated heart failure, a heart rate greater than the systolic blood pressure (e.g., a shock index ≥0.9) has been associated with in-hospital and 3-month mortality.[41]

Clinical experience may be the most helpful in assessing hemodynamic status. A good assessment for systemic perfusion and cardiac index appears to be overall clinical impression, the so-called "cold" profile. The gestalt of specialized heart failure clinicians performed better than proportional pulse pressure, systolic blood pressure, cool extremities, or fatigue in predicting an invasively measured cardiac index lower than 2.3 L/min/m^2.[28] This prediction rule has not been reported in other patient groups, in larger cohorts, or in more contemporary studies. Pleural effusions also are common in patients with heart failure, in whom they typically are right-sided, as noted previously. Dullness to percussion is the simplest finding to elicit in identifying a pleural effusion and is superior (LR, 8.7; 95% CI, 2.2 to 33.8)

to auscultatory percussion, decreased breath sounds, asymmetric chest expansion, increased vocal resonance, crackles, or pleural friction rubs. By contrast, absence of reduced tactile vocal fremitus makes a pleural effusion less likely (negative LR, 0.21; 95% CI, 0.12 to 0.37).[42]

Patient with a Left Ventricular Assist Device

The physical examination assessment of the left ventricular assist device (LVAD) patient should be organized around the hemodynamics of durable LVAD support (e.g., volume status, presence of native aortic valve opening, mean blood pressure, end-organ perfusion) as well as the complications unique to the mechanically supported circulation (e.g., driveline infection, blood loss from either gastrointestinal bleeding or hemolysis, and neurologic complications). Jugular venous distension, lower extremity edema, and abdominal distension will indicate right heart failure, often without concomitant left heart failure. Blood pressure may be obtained with a traditional cuff but may require Doppler assessment. In general, the first Korotkoff sound obtained by Doppler will be reported interchangeably as either the systolic or mean blood pressure; a mean blood pressure of less than 90 mm Hg is optimal (eFig. 13.2**).** The pulse should be palpated by hand at either the radial, brachial, or carotid locations; a palpable pulse should be intermittent and reflective of intermittent opening of the native aortic valve. Auscultation of the LVAD itself will elicit sounds unique to the specific LVAD device; regular changes in the LVAD hum can reflect intrinsic speed changes (a Lavare cycle) in order to provide an artificial "pulse" to the LVAD itself. Other changes to the cadence maybe indicative of LVAD dysfunction (e.g., thrombosis.)

Valvular Heart Disease (see also Part VIII)

A careful history and physical examination can reveal much regarding lesion severity, natural history, indications for intervention, and outcomes in patients with valvular heart disease. The history in patients with known or suspected valvular heart disease should rely on the use of a functional classification scheme and assessment of patient frailty when appropriate. Onset of even mild functional limitation is generally an indication for mechanical correction of the responsible valve lesion. Valvular heart disease most often is first suspected because of a heart murmur but many patients go undetected until presentation with symptoms or the discovery of a valve lesion on an echocardiogram performed for another indication.[43,44] Cardiologists can detect systolic heart murmurs with fair reliability (interobserver kappa coefficient, 0.30 to 0.48), and usually can confirm or rule out aortic stenosis, HOCM, MR, MVP, TR, and functional murmurs. The use of handheld ultrasound devices may improve detection and accuracy rates.[45]

Mitral Stenosis

In patients with mitral stenosis, survival declines following symptom onset and worsens with increasing degrees of functional limitation (NYHA class) and as pulmonary hypertension increases. Findings on physical examination vary with the chronicity of the disease, heart rate, rhythm, and cardiac output. It can be difficult to estimate the severity of the valve lesion in older patients with less pliable valves, rapid AF, or low cardiac output. Severe mitral stenosis is suggested by (1) a long or holodiastolic murmur, indicating a persistent LA-LV gradient; (2) a short A_2-OS interval, consistent with higher LA pressure; (3) a loud P_2 (or single S_2) and/or an RV lift, suggestive of pulmonary hypertension; and (4) elevated JVP with *cv* waves, hepatomegaly, and lower extremity edema—all signs of right heart failure with TR. Neither the intensity of the diastolic murmur nor the presence of presystolic accentuation in patients with sinus rhythm accurately reflects lesion severity.

Mitral Regurgitation

The symptoms associated with MR depend on its severity and time course of development. Acute severe MR that occurs with papillary muscle rupture or infective endocarditis usually results in sudden and profound dyspnea from pulmonary edema. Examination findings may be misleading because the LV impulse usually is neither enlarged nor displaced, and the systolic murmur is early in timing and decrescendo in configuration (see Fig. 13.10). The murmur also may be louder at the lower left sternal border or in the axilla than at the apex. A new systolic murmur developing early after an MI may not be audible in a ventilated or obese patient.

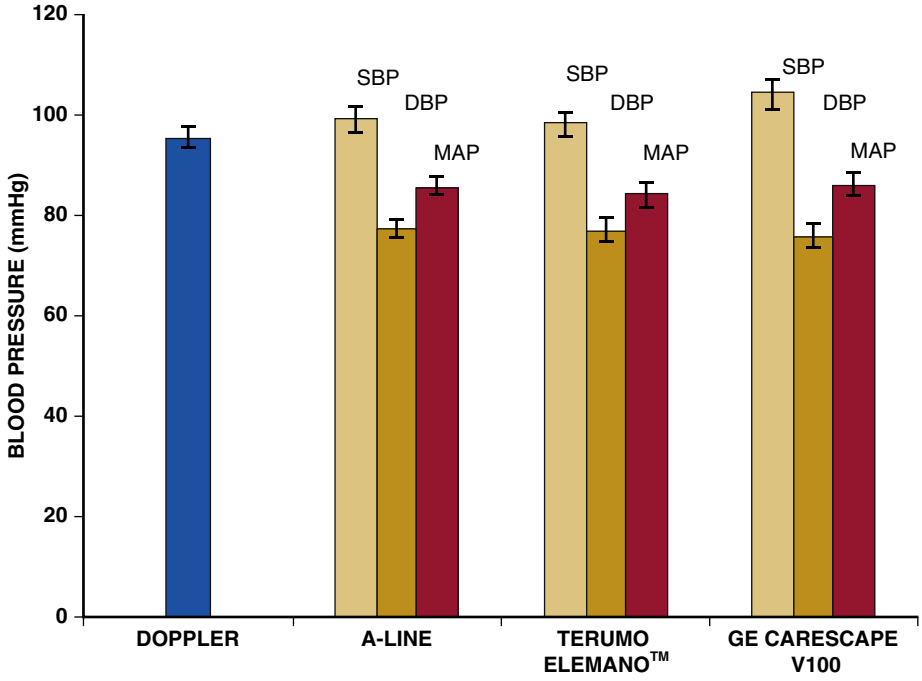
Several findings suggest chronic severe MR: (1) an enlarged, displaced, but dynamic LV apex beat; (2) an apical systolic thrill (murmur intensity of grade 4 or greater); (3) a mid-diastolic filling complex comprising an S_3 and a short, low-pitched murmur, indicative of accelerated and enhanced diastolic mitral inflow; (4) wide but physiologic splitting of S_2 caused by early aortic valve closure; and (5) a loud P_2 or RV lift. The findings in patients with MVP can vary, depending on LV loading conditions. The combination of a nonejection click and mid- to late systolic murmur predicts MVP best, as confirmed by transthoracic echocardiography criteria (LR, 2.43). Dynamic auscultation should be performed. The murmur associated with secondary MR in patients with reduced LV systolic function is often of low intensity and can be difficult to hear unless specifically sought.

Aortic Stenosis

A slowly rising carotid upstroke (*pulsus tardus*), reduced carotid pulse amplitude (*pulsus parvus*), reduced intensity of A_2, and mid- to late peaking of the systolic murmur help gauge the severity of aortic stenosis. The intensity of the murmur depends on cardiac output and body size (peak momentum transfer) and does not reliably reflect stenosis severity. In a 35-year follow-up study of 2014 apparently healthy middle-aged Norwegian men, the presence of even a low-grade systolic murmur was associated with an almost fivefold increased age-adjusted risk for aortic valve replacement.[46] No single physical examination finding has both high sensitivity and high specificity for the diagnosis of severe aortic stenosis, and only a reduced carotid upstroke amplitude may independently predict outcome. Clinical experience has established the difficulty of assessing carotid upstroke characteristics in older patients, in patients with hypertension, and in low-output states. Distinguishing the murmur of hemodynamically significant aortic stenosis from that caused by lesser degrees of stenosis is also challenging. Even with aortic sclerosis, the murmur can be of grade 2 or 3 intensity, although it peaks in midsystole. The carotid upstroke should be normal, A_2 should be preserved, and the electrocardiogram should lack evidence of LV hypertrophy. Nevertheless, TTE often is necessary to clarify this distinction, especially in older patients with hypertension. Signal analysis of digitally captured cardiovascular sounds using spectral display can distinguish the murmur of aortic sclerosis from a murmur resulting from hemodynamically significant aortic stenosis. The differential diagnosis of a systolic murmur related to LV outflow obstruction includes valvular aortic stenosis, HOCM, discrete membranous subaortic stenosis (DMSS), and SVAS. The presence of an ejection sound indicates a valvular cause. HOCM can be distinguished on the basis of the response of the murmur to the Valsalva maneuver and standing or squatting. Patients with DMSS will commonly have a diastolic murmur indicative of AR but not an ejection sound, whereas in patients with SVAS, the right arm blood pressure is more than 10 mm Hg greater than the left arm blood pressure. Assessment of fragility status in elderly patients with aortic stenosis is a routine feature of the multidisciplinary team evaluation (see Table 13.3).

Aortic Regurgitation

Patients with acute severe AR present with pulmonary edema and symptoms and signs of low forward cardiac output. Tachycardia is invariably present; systolic blood pressure is not elevated, and the pulse pressure may not be significantly widened. S_1 is soft because of premature closure of the mitral valve. The intensity and duration of the diastolic murmur are attenuated by the rapid rise in LV diastolic pressure and diminution of the aortic-LV diastolic pressure gradient. In patients with acute type A aortic dissection, the presence of a diastolic murmur (present in almost 30% of cases)



EFIGURE 13.2 Comparison of blood pressure assessment by Doppler, arterial-line, and noninvasive autonomic cuffs in patients with a continuous-flow left ventricular assist device. Note that the blood pressure assessed by mean Doppler is approximately 4 mm Hg below the arterial line systolic blood pressure and approximately 10 mm Hg greater the arterial line mean blood pressure. (From Lanier GM, Orlanes K, Hayashi Y. Validity and reliability of a novel slow cuff-deflation system for noninvasive blood pressure monitoring in patients with continuous-flow left ventricular assist device. *Circ Heart Fail*. 2013;6:1005–1012.)

does little to change the pretest probability of dissection. Acute severe AR is poorly tolerated and mandates emergency surgery. Typical symptoms associated with chronic, severe AR include dyspnea, fatigue, chest discomfort, and palpitations. A decrescendo diastolic blowing murmur suggests chronic AR. A midsystolic murmur indicative of augmented LV outflow is invariably heard at the base. Aortic stenosis may coexist. The absence of a diastolic murmur significantly reduces the likelihood of moderate or greater AR (LR, 0.1), whereas the presence of a typical diastolic murmur increases the likelihood of moderate or greater AR (LR, 4.0 to 8.3). In addition, in patients with chronic AR, the intensity of the murmur correlates with the severity of the lesion. A grade 3 diastolic murmur has an LR of 4.5 (95% CI, 1.6 to 14.0) for distinguishing severe AR from mild or moderate AR.[47] Data conflict regarding the significance of an Austin Flint murmur. Little evidence supports the historical claims of the importance of almost all the eponymous peripheral signs of chronic AR, which number at least 12. The Hill sign (brachial-popliteal systolic blood pressure gradient higher than 20 mm Hg) may be the single exception (sensitivity of 89% for moderate to severe AR), although its supporting evidence base also is weak.

Tricuspid Valve Disease

Left-sided valve lesions often obscure the symptoms and signs of tricuspid stenosis. An elevated JVP together with a delayed *y* descent, abdominal ascites, and edema suggests severe tricuspid stenosis. Auscultatory findings are difficult to appreciate but mimic those in mitral stenosis and may be accentuated with inspiration. The symptoms of TR resemble those of tricuspid stenosis. Severe TR causes elevated JVP with prominent *cv* waves, a parasternal lift, pulsatile liver, ascites, and edema. The intensity of the holosystolic murmur of TR increases with inspiration (Carvallo sign). Murmur intensity does not accurately reflect the severity of the valve lesion. Primary and secondary causes of TR should be distinguished.

Pulmonic Valve Disease

Pulmonic stenosis may cause exertional fatigue, dyspnea, lightheadedness, and chest discomfort ("right ventricular angina"). Syncope denotes severe obstruction. The midsystolic murmur of pulmonic stenosis is best heard at the second left interspace. With severe pulmonic stenosis, the interval between S_1 and the pulmonic ejection sound narrows, and the murmur peaks in late systole and may extend beyond A_2. P_2 becomes inaudible. Signs of significant RV pressure overload include a prominent jugular venous *a* wave and a parasternal lift. PR occurs most commonly as a secondary manifestation of significant pulmonary artery hypertension and annular dilation, but it may also reflect a primary valve disorder (e.g., congenital bicuspid valve) or develop as a complication of RV outflow tract surgery, in which case characteristics of the murmur and Doppler echocardiographic signs differ. Symptoms vary as a function of the severity of PA hypertension and the level of RV compensation. The diastolic murmur of secondary PR (Graham Steell) can be distinguished from that caused by AR on the basis of its increase in intensity with inspiration, its later onset (after A_2 and with P_2), and its slightly lower pitch. When a typical murmur is audible, the likelihood of PR increases (LR, 17), but the absence of a murmur does not exclude PR (LR, 0.9). With severe pulmonary artery hypertension and PR, P_2 is usually palpable and there are signs of RV pressure and volume overload on examination.

Prosthetic Heart Valves

The differential diagnosis of functional limitation after valve replacement surgery includes prosthetic valve dysfunction, prosthesis-patient mismatch, arrhythmia, and impaired ventricular function. Prosthetic valve dysfunction can occur as a result of thrombosis, pannus ingrowth, infection, or structural deterioration. Symptoms and signs mimic those of native valve disease and may arise acutely or develop gradually. The first clue that prosthetic valve dysfunction may be present often is 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 bioprosthesis in the mitral position usually may be associated with a grade 1-2 midsystolic murmur (from turbulence created by systolic flow across the valve struts that project into the LV outflow tract) and a soft, mid-diastolic murmur that occurs with normal LV filling. The diastolic murmur usually is heard only in the left lateral decubitus position at the apex. A high-pitched or holosystolic apical murmur signifies para- or transvalvular regurgitation that requires echocardiographic verification and careful follow-up evaluation. Depending on the magnitude of the regurgitant volume, a diastolic murmur may be audible. Clinical deterioration can occur rapidly after initial manifestation of bioprosthetic failure.

A bioprosthesis in the aortic position is invariably associated with a midsystolic murmur at the base usually of grade 1-2 intensity. A diastolic murmur of AR is abnormal under any circumstance and merits additional investigation. A decrease in the intensity of either the opening or closing sounds of a mechanical prosthesis, depending on its type, is a worrisome finding. A high-pitched apical systolic murmur in patients with a mechanical mitral prosthesis, or a decrescendo diastolic murmur in patients with a mechanical aortic prosthesis, indicates paravalvular regurgitation or prosthetic dysfunction. Signs of hemolysis should be sought. Patients with prosthetic valve thrombosis may present with shock, muffled heart sounds, and soft murmurs. Pannus ingrowth is usually associated with an increase in the intensity of a systolic murmur and other signs indicative of prosthetic valve stenosis.

Acute Coronary Syndromes (see Chapters 35–39)

Risk stratification of patients with ACS informs decision making regarding the intensity and pace of management and is recommended by international guidelines.[48–50] Clinical findings indicative of high risk of short-term death or MI in patients with non-ST-elevation ACS include age greater than 75 years, tachycardia, hypotension, signs of pulmonary congestion, and/or a new or worsening murmur of MR.

Pericardial Disease (see also Chapter 86)

Pericarditis

The typical pain of acute pericarditis starts abruptly, is sharp, and varies with position. It can radiate to the trapezius ridge. Associated fever or history of a recent viral illness may provide additional clues. A pericardial friction rub is almost 100% specific for the diagnosis, although its sensitivity is not as high, because the rub may wax and wane over the course of an acute illness or may be difficult to elicit. This leathery or scratchy, typically two- or three-component sound also may be monophasic. It usually is necessary to auscultate the heart with the patient in several positions. The ECG may display concave upward ST segment elevation and P-R segment deviation (elevation in lead aVR, depression in lead II). A transthoracic ECG is routinely obtained to assess the volume and appearance of any effusion and to look for early signs of hemodynamic compromise.

Pericardial Tamponade

Pericardial tamponade occurs when intrapericardial pressure equals or exceeds right atrial pressure. The time course of its development depends on the volume of the effusion, the rate at which it accumulates, and pericardial compliance. The most common associated symptom is dyspnea (sensitivity, 87% to 88%). Hypotension (sensitivity, 26%) and muffled heart sounds (sensitivity, 28%) are relatively insensitive indicators of tamponade. A pulsus paradoxus greater than 12 mm Hg in a patient with a large pericardial effusion predicts tamponade with a sensitivity of 98%, a specificity of 83%, and a positive LR of 5.9 (95% CI, 2.4 to 14).[51] Echocardiography is indicated in all patients with suspected pericardial tamponade.

TABLE 13.8 Cardiovascular Virtual Visits: Benefits and Challenges

Benefits
• Improved access for patients
• Review in the home environment
• Medication reconciliation
• Caregiver/co-resident engagement
• Provider flexibility and engagement
• Data aspects of exam reviewable (BMI, weight, BP, HR, rhythm, urine output/quality)
• Qualitative exam possible (general appearance, neuro/mental status, skin/pallor, jugular venous distension, edema)
Challenges
• Lack of tactile and auscultatory capacity
• Reimbursement
• Change in expectations/culture
• Technology/Internet availability
• Patient and provider unfamiliarity with the technical aspects of the virtual visit
• Depersonalization of the patient-provider relationship

Constrictive Pericarditis

Constrictive pericarditis is an uncommon clinical entity that occurs with previous chest irradiation, cardiac or mediastinal surgery, chronic tuberculosis, malignancy, or a prior episode of acute pericarditis. Dyspnea, fatigue, weight gain, abdominal bloating, and leg swelling dominate the clinical presentation. The diagnosis most often is first suspected after inspection of the JVP and waveforms, with elevation and inscription of the classic M or W contour caused by prominent *x* and *y* descents and a Kussmaul sign. Evidence of pleural effusions and ascites often can be found. On rare occasion, a PK is audible. Distinction from restrictive cardiomyopathy often is not possible on the basis of the history and physical examination alone.

VIRTUAL VISITS

The COVID-19 global pandemic has accelerated the adoption of video-assisted patient encounters (VVs) into clinical practice and challenged clinicians to improve their ability to detect certain physical examination findings remotely. Despite their obvious differences from the typical in-person encounter, VVs may offer several benefits, though limitations clearly exist (Table 13.8). Evidence suggests that patients can become comfortable with VVs; in a survey of U.S. veterans, most were supportive of such encounters.

Blood pressure, HR, pulse regularity, weight, and even urine output are examples of data that can be obtained from most patients. Remote history taking may benefit from the ability to observe the patient in their home, interview other caretakers and/or home residents, and review prescription bottles. Such information is often more easily obtained during a virtual as opposed to in-person visit. These issues are particularly relevant in rural areas and urban environments when getting to in-person encounters are limited by transportation, frailty, and scheduling.

HF management may be particularly suited to the VV. Congestion can be reasonably assessed during the VV. Observations regarding JVP, peripheral edema, and weight are all possible. JVP by VV tracks closely with bedside JVP and invasively measured right atrial pressure. In a prospective study of 31 patients with 63 remote video assisted evaluations, agreement between video assisted and bedside JVP assessment was greater than 90%.[20]

FUTURE DIRECTIONS

Concerns regarding the escalating costs of medical care may reinforce the value of the traditional history and examination to guide appropriate use of imaging and invasive diagnostic modalities. Patients' perceptions of the quality of their care is often associated with the performance of the history and examination. These considerations should spur additional efforts to establish the accuracy and predictive value of bedside findings with contemporary measurement science across a spectrum of cardiovascular disorders.

The mentored patient evaluation should be revisited as a dedicated component of training programs, along with mechanisms to allow practice, repetition, and feedback. Improved teaching methods using simulation-based training aids are effective.[5] Electronic and digital stethoscopes may allow for computer automation and spectral display as means not only to enhance learning but also to improve the accuracy of diagnosis, while maintaining the physical link between the patient and provider.[6,52,53] The addition of handheld ultrasound may also improve learner performance, but whether it should replace the stethoscope is a point of contention.[54–57] Continued improvements in the technical performance characteristics and declining costs of these devices are attractive features, as is the possibility of initiating treatment at the point of care without the need for additional testing in many cases.[58,59] Insonation (e.g., ultrasound) may in fact become a pillar of the bedside exam in a way that Laennec introduced device facilitated auscultation 200 years ago.[60] Dissemination of such technology has been accelerated by the SARS-CoV-2 pandemic.[61]

Increasingly, patients will be acquiring their own digital data to review with their health care providers; how such information will be used for the patient's benefit remains to be seen.[62] Finally, the VV will become a routine part of clinical practice. Further refinements in the technology, comfort with these encounters by both patient and provider, and the decisions by payors to recognize these important care paradigms will ultimately change the delivery of cardiovascular care.

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Future Directions

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