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GOLDMAN'S
CECIL
MEDICINE

西氏内科学

第24版

心血管疾病分册

LEE GOLDMAN
ANDREW I. SCHAFER

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LEE GOLDMAN, MD
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LEE GOLDMAN, MD
Dean of the Faculties of Health Sciences and Medicine
Executive Vice President for Health and Biomedical Sciences
Harold and Margaret Hatch Professor of the University
Professor of Medicine and of Epidemiology
Columbia University
New York, New York

ANDREW I. SCHAFER, MD
Chairman, Department of Medicine
The E. Hugh Luckey Distinguished Professor of Medicine
Weill Cornell Medical College
Physician-in-Chief
New York-Presbyterian Hospital/Weill Cornell Medical Center
New York, New York

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PREFACE

The 24TH Edition of *Goldman's Cecil Medicine* symbolizes a time of extraordinary advances in medicine and in technological innovations for the dissemination of information. This textbook and its associated electronic products incorporate the latest medical knowledge in formats that are designed to appeal to learners who prefer to access information in a variety of ways.

The contents of *Cecil* have remained true to the tradition of a comprehensive textbook of medicine that carefully explains the *why* (the underlying normal physiology and pathophysiology of disease, now at the cellular and molecular as well as the organ level) and the *how* (now frequently based on Grade A evidence from randomized controlled trials). Descriptions of physiology and pathophysiology include the latest genetic advances in a practical format that strives to be useful to the nonexpert. Medicine has entered an era when the acuity of illness and the limited time available to evaluate a patient have diminished the ability of physicians to satisfy their intellectual curiosity. As a result, the acquisition of information, quite easily achieved in this era, is often confused with knowledge. We have attempted to counteract this tendency with a textbook that not only informs but also stimulates new questions and gives a glimpse of the future path to new knowledge. Grade A evidence is specifically highlighted in the text and referenced at the end of each chapter. In addition to the information provided in the textbook, the Cecil website supplies expanded content and functionality. In many cases, the full articles referenced in each chapter can be accessed from the Cecil website. The website is also continuously updated to incorporate subsequent Grade A information, other evidence, and new discoveries.

The sections for each organ system begin with a chapter that summarizes an approach to patients with key symptoms, signs, or laboratory abnormalities associated with dysfunction of that organ system. As summarized in Table 1-1, the text specifically provides clear, concise information regarding how a physician should approach more than 100 common symptoms, signs, and laboratory abnormalities, usually with a flow diagram, a table, or both for easy reference. In this way, *Cecil* remains a comprehensive text to guide diagnosis and therapy, not only for patients with suspected or known diseases but also for patients who may have undiagnosed abnormalities that require an initial evaluation.

Just as each edition brings new authors, it also reminds us of our gratitude to past editors and authors. Previous editors of *Cecil Medicine* include a short but remarkably distinguished group of leaders of American medicine: Russell Cecil, Paul Beeson, Walsh McDermott, James Wyngaarden, Lloyd H. Smith, Jr., Fred Plum, J. Claude Bennett, and Dennis Ausiello. As we welcome new associate editors—Wendy Levinson, Donald W. Landry, Anil Rustgi, and W. Michael Scheld—we also express our appreciation to Nicholas LaRusso and other associate editors from the previous editions on whose foundation we have built. Our returning associate editors—William P. Arend, James O. Armitage, David Clemmons, Jeffrey M. Drazen, and Robert C. Griggs—continue to make critical contributions to the selection of authors and the review and approval of all manuscripts. The editors, however, are fully responsible for the book as well as the integration among chapters.

The tradition of *Cecil Medicine* is that all chapters are written by distinguished experts in each field. We are also most grateful for the editorial assistance in New York of Theresa Considine and Silva Sergenian. These individuals and others in our offices have shown extraordinary dedication and equanimity in working with authors and editors to manage the unending flow of manuscripts, figures, and permissions. We also thank Faten Aberra, Reza Akari, Robert C. Brunham, Ivan Ciric, Seema Daulat, Gregory F. Erikson, Kevin Ghassemi, Jason H. Huang, Caron Jacobson, Lisa Kachnic, Bryan T. Kelly, Karen Krok, Heather Lehman, Keiron Leslie, Luis Marcos, Michael Overman, Eric Padron, Bianca Maria Piraccini, Don W. Powell, Katy Ralston, James M. Swain, Tania Thomas, Kirsten Tillisch, Ali Turabi, Mark Whiteford, and Y. Joseph Woo, who contributed to various chapters. At Elsevier, we are most indebted to Dolores Meloni and Linda McKinley, and also thank Cathy Carroll, Taylor Ball, Virginia Wilson, Linda Van Pelt, Suzanne Fannin, and Steve Stave, who have been critical to the planning and production process under the direction of Mary Gatsch. Many of the clinical photographs were supplied by Charles D. Forbes and William F. Jackson, authors of *Color Atlas and Text of Clinical Medicine*, Third Edition, published in 2003 by Elsevier Science Ltd. We thank them for graciously permitting us to include their pictures in our book. We have been exposed to remarkable physicians in our lifetimes and would like to acknowledge the mentorship and support of several of those who exemplify this paradigm—Robert H. Gifford, Lloyd H. Smith, Jr., Frank Gardner, and William Castle. Finally, we would like to thank the Goldman family—Jill, Jeff, Abigail, Mira, Daniel, and Robyn Goldman—and the Schafer family—Pauline, Eric, Pam, John, Evan, and Kate—for their understanding of the time and focus required to edit a book that attempts to sustain the tradition of our predecessors and to meet the needs of today's physician.

LEE GOLDMAN, MD
ANDREW I. SCHAFER, MD

CONTENTS

SECTION VIII: CARDIOVASCULAR DISEASE

VIII

CARDIOVASCULAR DISEASE

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APPROACH TO THE PATIENT WITH POSSIBLE CARDIOVASCULAR DISEASE

LEE GOLDMAN

Patients with cardiovascular disease may present with a wide range of symptoms and signs, each of which may be caused by noncardiovascular conditions. Conversely, patients with substantial cardiovascular disease may be asymptomatic. Because cardiovascular disease is a leading cause of death in the United States and other developed countries, it is crucial that patients be evaluated carefully to detect early cardiovascular disease, that symptoms or signs of cardiovascular disease be evaluated in detail, and that appropriate therapy be instituted. Improvements in diagnosis, therapy, and prevention have contributed to a 70% or so decline in age-adjusted cardiovascular death rates in the United States since the 1960s. However, the absolute number of deaths from cardiovascular disease in the United States has not declined proportionately because of the increase in the population older than 40 years as well as the aging of the population in general.

In evaluating a patient with known or suspected heart disease, the physician must determine quickly whether a potentially life-threatening condition exists. In these situations, the evaluation must focus on the specific issue at hand and be accompanied by the rapid performance of appropriately directed additional tests. Examples of potentially life-threatening conditions include acute myocardial infarction (Chapter 73), unstable angina (Chapter 72), suspected aortic dissection (Chapter 78), pulmonary edema (Chapter 59), and pulmonary embolism (Chapter 98).

USING THE HISTORY TO DETECT CARDIOVASCULAR SYMPTOMS

Patients may complain spontaneously of a variety of cardiovascular symptoms (Table 50-1), but sometimes these symptoms are elicited only by obtaining a careful, complete medical history. In patients with known or suspected cardiovascular disease, questions about cardiovascular symptoms are key components of the history of present illness; in other patients, these issues are a fundamental part of the review of systems.

Chest Pain

Chest discomfort or pain is the cardinal manifestation of myocardial ischemia resulting from coronary artery disease or any condition that causes myocardial ischemia by an imbalance of myocardial oxygen demand compared with myocardial oxygen supply (Chapter 71). New, acute, often ongoing pain may indicate an acute myocardial infarction, unstable angina, or aortic dissection; a pulmonary cause, such as acute pulmonary embolism or pleural irritation; a musculoskeletal condition of the chest wall, thorax, or shoulder; or a gastrointestinal abnormality, such as esophageal reflux or spasm, peptic ulcer disease, or cholecystitis (Table 50-2). The chest discomfort of myocardial infarction commonly occurs without an immediate or obvious precipitating clinical cause and builds in intensity for at least several minutes; the sensation can range from annoying discomfort to severe pain (Chapter 73). Although a variety of adjectives may be used by patients to describe the sensation, physicians must be suspicious of any discomfort, especially if it radiates to the neck, shoulder, or arms. The probability of an acute myocardial infarction can be estimated by integrating information from the history, physical examination, and electrocardiogram (Fig. 50-1).

The chest discomfort of unstable angina is clinically indistinguishable from that of myocardial infarction except that the former may be precipitated more clearly by activity and may be more rapidly responsive to antianginal therapy (Chapter 72). Aortic dissection (Chapter 78) classically presents with the sudden onset of severe pain in the chest and radiating to the back; the location of the pain often provides clues to the location of the dissection. Ascending aortic dissections commonly present with chest discomfort radiating to the back, whereas dissections of the descending aorta commonly present with back pain radiating to the abdomen. The presence of back pain or a history of hypertension or other predisposing factors, such as Marfan syndrome, should prompt a careful assessment of peripheral pulses to determine whether the great vessels are affected by the dissection and of the chest radiograph to evaluate the size of the aorta. If this initial evaluation is suggestive, further testing with transesophageal echocardiography, computed tomography (CT), or magnetic resonance imaging (MRI) is indicated. The pain of pericarditis (Chapter 77) may simulate that of an acute myocardial infarction, may be primarily pleuritic, or may be continuous; a key physical finding is a pericardial rub. The pain of pulmonary embolism (Chapter 98) is commonly pleuritic in nature and is associated with dyspnea; hemoptysis also may be present. Pulmonary hypertension (Chapter 68) of any cause may be associated with chest discomfort with exertion; it commonly is associated with severe dyspnea and often is associated with cyanosis.

Recurrent, episodic chest discomfort may be noted with angina pectoris and with many cardiac and noncardiac causes (Chapter 71). A variety of stress tests (Table 50-3) can be used to provoke reversible myocardial ischemia in susceptible individuals and to help determine whether ischemia is the pathophysiologic explanation for the chest discomfort (Chapter 71).

Dyspnea

Dyspnea, which is an uncomfortable awareness of breathing, is commonly due to cardiovascular or pulmonary disease. A systematic approach (see Fig. 83-3 in Chapter 83) with selected tests nearly always reveals the cause. Acute dyspnea can be caused by myocardial ischemia, heart failure, severe hypertension, pericardial tamponade, pulmonary embolism, pneumothorax, upper airway obstruction, acute bronchitis or pneumonia, or some drug overdoses (e.g., salicylates). Subacute or chronic dyspnea is also a common presenting or accompanying symptom in patients with pulmonary disease (Chapter 83). Dyspnea also can be caused by severe anemia (Chapter 161) and can be confused with the fatigue that often is noted in patients with systemic and neurologic diseases (Chapters 264 and 403).

In heart failure, dyspnea typically is noted as a hunger for air and a need or an urge to breathe. The feeling that breathing requires increased work or effort is more typical of airway obstruction or neuromuscular disease. A feeling of chest tightness or constriction during breathing is typical of bronchoconstriction, which is commonly caused by obstructive airway disease (Chapters 87 and 88) but also may be seen in pulmonary edema. A feeling of heavy breathing, a feeling of rapid breathing, or a need to breathe more is classically associated with deconditioning.

In cardiovascular conditions, chronic dyspnea usually is caused by increases in pulmonary venous pressure as a result of left ventricular failure (Chapters 58 and 59) or valvular heart disease (Chapter 75). Orthopnea, which is an exacerbation of dyspnea when the patient is recumbent, is due to increased work of breathing because of either increased venous return to the pulmonary vasculature or loss of gravitational assistance in diaphragmatic effort. Paroxysmal nocturnal dyspnea is severe dyspnea that awakens a patient at night and forces the assumption of a sitting or standing position to achieve gravitational redistribution of fluid.

Palpitations

Palpitations (Chapter 62) describe a subjective sensation of an irregular or abnormal heartbeat. Palpitations may be caused by any arrhythmia (Chapters 64 and 65) with or without important underlying structural heart disease. Palpitations should be defined in terms of the duration and frequency of the episodes; the precipitating and related factors; and any associated symptoms of chest pain, dyspnea, lightheadedness, or syncope. It is crucial to use the history to determine whether the palpitations are caused by an irregular or a regular heartbeat. The feeling associated with a premature atrial or ventricular contraction, often described as a "skipped beat" or a "flip-flopping of the heart," must be distinguished from the irregularly irregular rhythm of atrial fibrillation and the rapid but regular rhythm of supraventricular tachycardia. Associated symptoms of chest pain, dyspnea, lightheadedness, dizziness, or diaphoresis suggest an important effect on cardiac output and mandate further evaluation. In general, evaluation begins with ambulatory electrocardiography (ECG) (Table 50-4), which is indicated in patients who have palpitations in the presence of structural heart disease or substantial accompanying symptoms. Depending on the series, 9 to 43% of patients have important underlying heart disease. In such patients, more detailed evaluation is warranted (see Fig. 62-1).

Lightheadedness or *syncope* (Chapter 62) can be caused by any condition that decreases cardiac output (e.g., bradyarrhythmia, tachyarrhythmia, obstruction of the left ventricular or right ventricular inflow or outflow, cardiac tamponade, aortic dissection, or severe pump failure), by

TABLE 50-1 CARDINAL SYMPTOMS OF CARDIOVASCULAR DISEASE

Chest pain or discomfort
Dyspnea, orthopnea, paroxysmal nocturnal dyspnea, wheezing
Palpitations, dizziness, syncope
Cough, hemoptysis
Fatigue, weakness
Pain in extremities with exertion (claudication)

reflex-mediated vasomotor instability (e.g., vasovagal, situational, or carotid sinus syncope), or by orthostatic hypotension (see Table 62-1 in Chapter 62). Neurologic diseases (e.g., migraine headaches, transient ischemic attacks, or seizures) also can cause transient loss of consciousness. The history, physical examination, and ECG are often diagnostic of the cause of syncope (see Table 62-2 in Chapter 62). Syncope caused by a cardiac arrhythmia usually occurs with little warning. Syncope with exertion or just after conclusion of exertion is typical of aortic stenosis and hypertrophic obstructive cardiomyopathy. In many patients, additional testing is required to document central nervous system disease, the cause of reduced cardiac output, or carotid sinus

TABLE 50-2 CAUSES OF CHEST PAIN

CONDITION	LOCATION	QUALITY	DURATION	AGGRAVATING OR RELIEVING FACTORS	ASSOCIATED SYMPTOMS OR SIGNS
CARDIOVASCULAR CAUSES					
Angina	Retrosternal region; radiates to or occasionally isolated to neck, jaw, epigastrium, shoulder, or arms (left common)	Pressure, burning, squeezing, heaviness, indigestion	<2-10 min	Precipitated by exercise, cold weather, or emotional stress; relieved by rest or nitroglycerin; atypical (Prinzmetal's) angina may be unrelated to activity, often early morning	S_4 or murmur of papillary muscle dysfunction during pain
Rest or unstable angina	Same as angina	Same as angina but may be more severe	Usually <20 min	Same as angina, with decreasing tolerance for exertion or at rest	Similar to stable angina but may be pronounced; transient heart failure can occur
Myocardial infarction	Substernal and may radiate like angina	Heaviness, pressure, burning, constriction	≥30 min but variable	Unrelieved by rest or nitroglycerin	Shortness of breath, sweating, weakness, nausea, vomiting
Pericarditis	Usually begins over sternum or toward cardiac apex and may radiate to neck or left shoulder; often more localized than the pain of myocardial ischemia	Sharp, stabbing, knifelike	Lasts many hours to days; may wax and wane	Aggravated by deep breathing, rotating chest, or supine position; relieved by sitting up and leaning forward	Pericardial friction rub
Aortic dissection	Anterior chest; may radiate to back	Excruciating, tearing, knifelike	Sudden onset, unrelenting	Usually occurs in setting of hypertension or predisposition, such as Marfan syndrome	Murmur of aortic insufficiency, pulse or blood pressure asymmetry; neurologic deficit
Pulmonary embolism (chest pain often not present)	Substernal or over region of pulmonary infarction	Pleuritic (with pulmonary infarction) or angina-like	Sudden onset; minutes to <1 hr	May be aggravated by breathing	Dyspnea, tachypnea, tachycardia; hypotension, signs of acute right ventricular failure, and pulmonary hypertension with large emboli; rales, pleural rub, hemoptysis with pulmonary infarction
Pulmonary hypertension	Substernal	Pressure; oppressive	Similar to angina	Aggravated by effort	Pain usually associated with dyspnea; signs of pulmonary hypertension
NONCARDIAC CAUSES					
Pneumonia with pleurisy	Localized over involved area	Pleuritic, localized	Brief or prolonged	Painful breathing	Dyspnea, cough, fever, dull to percussion, bronchial breath sounds, rales, occasional pleural rub
Spontaneous pneumothorax	Unilateral	Sharp, well localized	Sudden onset, lasts many hours	Painful breathing	Dyspnea; hyperresonance and decreased breath and voice sounds over involved lung
Musculoskeletal disorders	Variable	Aching	Short or long duration	Aggravated by movement; history of muscle exertion or injury	Tender to pressure or movement
Herpes zoster	Dermatomal in distribution	Burning, itching	Prolonged	None	Vesicular rash appears in area of discomfort
Esophageal reflux	Substernal, epigastric	Burning, visceral discomfort	10-60 min	Aggravated by large meal, postprandial recumbency; relief with antacid	Water brash
Peptic ulcer	Epigastric, substernal	Visceral burning, aching	Prolonged	Relief with food, antacid	
Gallbladder disease	Epigastric, right upper quadrant	Visceral	Prolonged	May be unprovoked or follow meals	Right upper quadrant tenderness may be present
Anxiety states	Often localized over precordium	Variable; location often moves from place to place	Varies; often fleeting	Situational	Sighing respirations, often chest wall tenderness

Modified from Andreoli TE, Carpenter CCJ, Griggs RC, et al. Evaluation of the patient with cardiovascular disease. In: *Cecil Essentials of Medicine*, 6th ed. Philadelphia: WB Saunders; 2004:34-35.

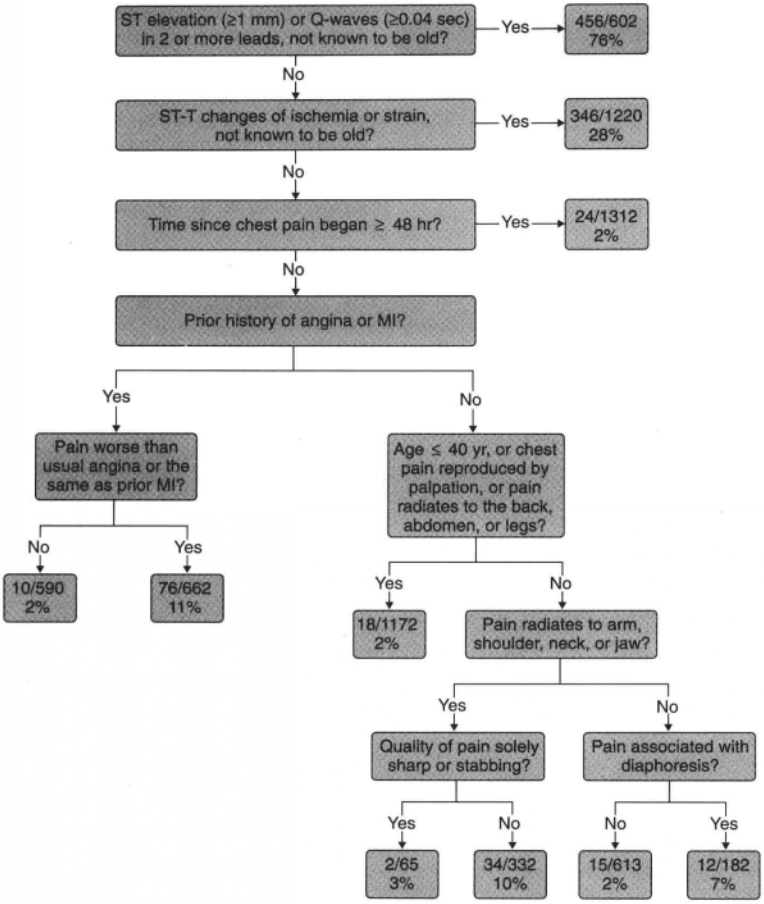

FIGURE 50-1. Flow diagram to estimate the risk for acute myocardial infarction in emergency departments in patients with acute chest pain. For each clinical subset, the numerator is the number of patients with the set of presenting characteristics who had a myocardial infarction; the denominator is the total number of patients presenting with that characteristic or set of characteristics. CHF = congestive heart failure; DVT = deep vein thrombosis. (Modified from Pearson SD, Goldman L, Garcia TB, et al. Physician response to a prediction rule for the triage of emergency department patients with chest pain. *J Gen Intern Med.* 1994;9:241-247.)

TABLE 50-3

PROTOCOL	STAGE	DURATION (min)	GRADE (%)	RATE (mph)	METABOLIC EQUIVALENTS AT COMPLETION	FUNCTIONAL CLASS
Modified Bruce protocol†	1	3	0	1.7	2.5	III
	2	3	10	1.7	5	II
	3	3	12	2.5	7	I
	4	3	14	3.4	10	I
	5	3	16	4.2	13	I
Naughton protocol‡	0	2	0	2	2	III
	1	2	3.5	2	3	III
	2	2	7	2	4	III
	3	2	10.5	2	5	II
	4	2	14	2	6	II
	5	2	17.5	2	7	I

*Ramp protocols in which the workload is gradually increased on the basis of the patient's estimated functional capacity to achieve maximal effort in approximately 10 minutes are also useful.
†Commonly used in ambulatory patients.
‡Commonly used in patients with recent myocardial infarction, unstable angina, or other conditions that are expected to limit exercise.
Modified from Braunwald E, Goldman L, eds. *Primary Cardiology*, 2nd ed. Philadelphia: WB Saunders; 2003.

TABLE 50-4 AMERICAN HEART ASSOCIATION/AMERICAN COLLEGE OF CARDIOLOGY DIAGNOSTIC TESTS IN PATIENTS WITH PALPITATIONS*

AMBULATORY ELECTROCARDIOGRAPHY	
Class I	Palpitations, syncope, dizziness
Class II	Shortness of breath, chest pain, or fatigue (not otherwise explained, episodic, and strongly suggestive of an arrhythmia as the cause because of a relation of the symptom with palpitation)
Class III	Symptoms not reasonably expected to be due to arrhythmia
ELECTROPHYSIOLOGIC STUDY	
Class I	Patients with palpitations who have a pulse rate documented by medical personnel as inappropriately rapid and in whom electrocardiographic recordings fail to document the cause of the palpitations Patients with palpitations preceding a syncopal episode
Class II	Patients with clinically significant palpitations, suspected to be of cardiac origin, in whom symptoms are sporadic and cannot be documented; studies are performed to determine the mechanisms of arrhythmias, to direct or provide therapy or to assess prognosis
Class III	Patients with palpitations documented to be due to extracardiac causes (e.g., hyperthyroidism)
ECHOCARDIOGRAPHY	
Class I	Arrhythmias with evidence of heart disease Family history of genetic disorder associated with arrhythmias
Class II	Arrhythmias commonly associated with, but without evidence of, heart disease Atrial fibrillation or flutter
Class III	Palpitations without evidence of arrhythmias Minor arrhythmias without evidence of heart disease

*Class I, general agreement the test is useful and indicated; class II, frequently used, but there is a divergence of opinion with respect to its utility; class III, general agreement the test is not useful.
From Braunwald E, Goldman L., eds. *Primary Cardiology*, 2nd ed. Philadelphia: WB Saunders; 2003:132.

TABLE 50-5 A COMPARISON OF THREE METHODS OF ASSESSING CARDIOVASCULAR FUNCTIONAL CLASS

CLASS	NEW YORK HEART ASSOCIATION FUNCTIONAL CLASSIFICATION	CANADIAN CARDIOVASCULAR SOCIETY FUNCTIONAL CLASSIFICATION	SPECIFIC ACTIVITY SCALE
I	Patients with cardiac disease but without resulting limitations of physical activity Ordinary physical activity does not cause undue fatigue, palpitation, dyspnea, or anginal pain.	Ordinary physical activity, such as walking and climbing stairs, does not cause angina. Angina with strenuous or rapid or prolonged exertion at work or recreation	Patients can perform to completion any activity requiring ≥7 metabolic equivalents, e.g., can carry 24 lb up 8 steps; carry objects that weigh 80 lb; do outdoor work (shovel snow, spade soil); do recreational activities (skiing, basketball, squash, handball, jog or walk 5 mph)
II	Patients with cardiac disease resulting in slight limitation of physical activity They are comfortable at rest. Ordinary physical activity results in fatigue, palpitations, dyspnea, or anginal pain.	Slight limitation of ordinary activity Walking or climbing stairs rapidly, walking uphill, walking or stair climbing after meals, in cold, in wind, or when under emotional stress, or only during the few hours after awakening Walking >2 blocks on the level and climbing >1 flight of ordinary stairs at a normal pace and in normal conditions	Patient can perform to completion any activity requiring ≥5 metabolic equivalents but cannot and does not perform to completion activities requiring ≥7 metabolic equivalents, e.g., have sexual intercourse without stopping, garden, rake, weed, roller skate, dance foxtrot, walk at 4 mph on level ground
III	Patients with cardiac disease resulting in marked limitation of physical activity They are comfortable at rest. Less than ordinary physical activity causes fatigue, palpitations, dyspnea, or anginal pain.	Marked limitation of ordinary physical activity Walking 1 or 2 blocks on the level and climbing >1 flight in normal conditions	Patient can perform to completion any activity requiring ≥2 metabolic equivalents but cannot and does not perform to completion any activities requiring ≥5 metabolic equivalents, e.g., shower without stopping, strip and make bed, clean windows, walk 2.5 mph, bowl, play golf, dress without stopping
IV	Patients with cardiac disease resulting in inability to carry on any physical activity without discomfort Symptoms of cardiac insufficiency or of the anginal syndrome may be present even at rest. If any physical activity is undertaken, discomfort is increased.	Inability to carry on any physical activity without discomfort—anginal syndrome may be present at rest	Patient cannot or does not perform to completion activities requiring ≥2 metabolic equivalents; cannot carry out activities listed above (Specific Activity Scale, class III)

From Goldman L, Hashimoto B, Cook EF, et al. Comparative reproducibility and validity of systems for assessing cardiovascular functional class: advantages of a new specific activity scale. *Circulation.* 1981;64:1227-1234. Reproduced by permission of the American Heart Association.

syncope. When the history, physical examination, and ECG do not provide helpful diagnostic information that points toward a specific cause of syncope, it is imperative that patients with heart disease or an abnormal ECG be tested with continuous ambulatory ECG monitoring to diagnose a possible arrhythmia (see Fig. 62-1 in Chapter 62); in selected patients, formal electrophysiologic testing may be indicated (Chapter 62). In patients with no evident heart disease, tilt testing (Chapter 62) can help detect reflex-mediated vasomotor instability.

Other Symptoms

Nonproductive *cough* (Chapter 83), especially a persistent cough (see Fig. 83-1 in Chapter 83), can be an early manifestation of elevated pulmonary venous pressure and otherwise unsuspected heart failure. *Fatigue* and *weakness* are common accompaniments of advanced cardiac disease and reflect an inability to perform normal activities. A variety of approaches have been used to classify the severity of cardiac limitations, ranging from class I (little or no limitation) to class IV (severe limitation) (Table 50-5). *Hemoptysis*

(Chapter 83) is a classic presenting finding in patients with pulmonary embolism, but it is also common in patients with mitral stenosis, pulmonary edema, pulmonary infections, and malignant neoplasms (see Table 83-5 in Chapter 83). *Claudication,* which is pain in the extremities with exertion, should alert the physician to possible peripheral arterial disease (Chapters 79 and 80).

Complete Medical History

The complete medical history should include a thorough review of systems, family history, social history, and past medical history (Chapter 14). The review of systems may reveal other symptoms that suggest a systemic disease as the cause of any cardiovascular problems. The family history should focus on premature atherosclerosis or evidence of familial abnormalities, such as may be found with various causes of the long QT syndrome (Chapter 65) or hypertrophic cardiomyopathy (Chapter 60).

The social history should include specific questioning about cigarette smoking, alcohol intake, and use of illicit drugs. The past medical history may reveal prior conditions or medications that suggest systemic diseases, ranging from chronic obstructive pulmonary disease, which may explain a complaint of dyspnea, to hemochromatosis, which may be a cause of restrictive cardiomyopathy. A careful history to inquire about recent dental work or other procedures is crucial if bacterial endocarditis is part of the differential diagnosis.

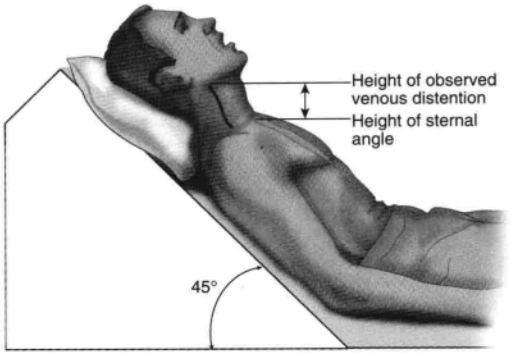

FIGURE 50-2. Jugular venous distention is defined by engorgement of the internal jugular vein more than 5 cm above the sternal angle at 45 degrees. The central venous pressure is the observed venous distention above the sternal angle plus 5 cm. (From American Academy of Family Physicians Online. http://www.aafp.org/afp/20000301/1319.html. Accessed June 9, 2010.)

PHYSICAL EXAMINATION FOR DETECTION OF SIGNS OF CARDIOVASCULAR DISEASE

The cardiovascular physical examination, which is a subset of the complete physical examination, provides important clues to the diagnosis of asymptomatic and symptomatic cardiac disease and may reveal cardiovascular manifestations of noncardiovascular diseases. The cardiovascular physical examination begins with careful measurement of the pulse and blood pressure (Chapter 7). If aortic dissection (Chapter 78) is a consideration, blood pressure should be measured in both arms and, preferably, in at least one leg. When coarctation of the aorta is suspected (Chapter 69), blood pressure must be measured in at least one leg and in the arms. Discrepancies in blood pressure between the two arms also can be caused by atherosclerotic disease of the great vessels. Pulsus paradoxus, which is more than the usual 10 mm Hg drop in systolic blood pressure during inspiration, is typical of pericardial tamponade (Chapter 77).

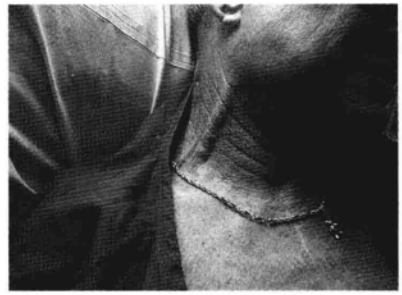

FIGURE 50-3. Typical distention of the internal jugular vein. (From http://courses.cvcc.vccs.edu/WisemanD/jugular_vein_distention.htm.)

General Appearance

The respiratory rate may be increased in patients with heart failure. Patients with pulmonary edema are usually markedly tachypneic and may have labored breathing. Patients with advanced heart failure may have Cheyne-Stokes respirations.

Systemic diseases, such as hyperthyroidism (Chapter 233), hypothyroidism (Chapter 233), rheumatoid arthritis (Chapter 272), scleroderma (Chapter 275), and hemochromatosis (Chapter 219), may be suspected from the patient's general appearance. Marfan syndrome (Chapter 268), Turner's syndrome (Chapter 243), Down syndrome (Chapter 40), and a variety of congenital anomalies also may be readily apparent.

Ophthalmologic Examination

Examination of the fundi may show diabetic (see Fig. 431-24 in Chapter 431) or hypertensive retinopathy (see Fig. 67-11 in Chapter 67) or Roth's spots (see Fig. 431-28 in Chapter 431) typical of infectious endocarditis. Beading of the retinal arteries is typical of severe hypercholesterolemia. Osteogenesis imperfecta, which is associated with blue sclerae, also is associated with aortic dilation and mitral valve prolapse. Retinal artery occlusion (see Fig. 431-29 in Chapter 431) may be caused by an embolus from clot in the left atrium or left ventricle, a left atrial myxoma, or atherosclerotic debris from the great vessels. Hyperthyroidism may present with exophthalmos and typical stare (see Fig. 431-6 in Chapter 431), whereas myotonic dystrophy, which is associated with atrioventricular block and arrhythmia, often is associated with ptosis and an expressionless face (see Fig. 429-2 in Chapter 429).

Jugular Veins

The external jugular veins help in assessment of mean right atrial pressure, which normally varies between 5 and 10 cm H_2O; the height (in centimeters) of the central venous pressure is measured by adding 5 cm to the height of the observed jugular venous distention above the sternal angle of Louis (Fig. 50-2). The normal jugular venous pulse, best seen in the internal jugular vein (and not seen in the external jugular vein unless insufficiency of the jugular venous valves is present), includes an *a* wave, caused by right atrial contraction; a *c* wave, reflecting carotid artery pulsation; an *x* descent; a *v* wave, which corresponds to isovolumetric right ventricular contraction and is more marked in the presence of tricuspid insufficiency; and a *y* descent, which occurs as the tricuspid valve opens and ventricular filling begins (Fig. 50-3). Abnormalities of the jugular venous pressure (Fig. 50-4) and arterial pulse are useful in detecting conditions such as heart failure, pericardial disease, tricuspid valve disease, and pulmonary hypertension (Table 50-6).

Carotid Pulse

The carotid pulse should be examined in terms of its volume and contour. The carotid pulse (Fig. 50-5) may be increased in frequency and may be more intense than normal in patients with a higher stroke volume secondary to aortic regurgitation, arteriovenous fistula, hyperthyroidism, fever, or anemia. In aortic regurgitation or arteriovenous fistula, the pulse may have a bisferious quality. The carotid upstroke is delayed in patients with valvular aortic stenosis (Chapter 75) and has a normal contour but diminished amplitude in any cause of reduced stroke volume.

Cardiac Inspection and Palpation

Inspection of the precordium may reveal the hyperinflation of obstructive lung disease or unilateral asymmetry of the left side of the chest because of right ventricular hypertrophy before puberty. Palpation may be performed with the patient either supine or in the left lateral decubitus position; the latter position moves the left ventricular apex closer to the chest wall and increases the ability to palpate the point of maximal impulse and other

phenomena. Low-frequency phenomena, such as systolic heaves or lifts from the left ventricle (at the cardiac apex) or right ventricle (parasternal in the third or fourth intercostal space), are felt best with the heel of the palm. With the patient in the left lateral decubitus position, this technique also may allow palpation of an S_3 gallop in cases of advanced heart failure or an S_4 gallop in cases of poor left ventricular distensibility during diastole. The left ventricular apex is more diffuse and sometimes may be frankly dyskinetic in patients with advanced heart disease. The distal palm is best for feeling thrills, which are the tactile equivalent of cardiac murmurs. By definition, a thrill denotes a murmur of grade 4/6 or louder. Higher-frequency events may be felt best with the fingertips; examples include the opening snap of mitral stenosis or the loud pulmonic second sound of pulmonary hypertension.

Auscultation

The first heart sound (Fig. 50-6), which is largely produced by closure of the mitral and—to a lesser extent—the tricuspid valves, may be louder in patients with mitral valve stenosis and intact valve leaflet movement and less audible in patients with poor closure due to mitral regurgitation (Chapter 75). The second heart sound is caused primarily by closure of the aortic valve, but closure of the pulmonic valve is also commonly audible. In normal individuals, the louder aortic closure sound occurs first, followed by pulmonic closure. With expiration, the two sounds are virtually superimposed. With inspiration, by comparison, the increased stroke volume of the right ventricle commonly leads to a discernible splitting of the second sound. This splitting may be fixed in patients with an atrial septal defect (Chapter 69) or a right bundle

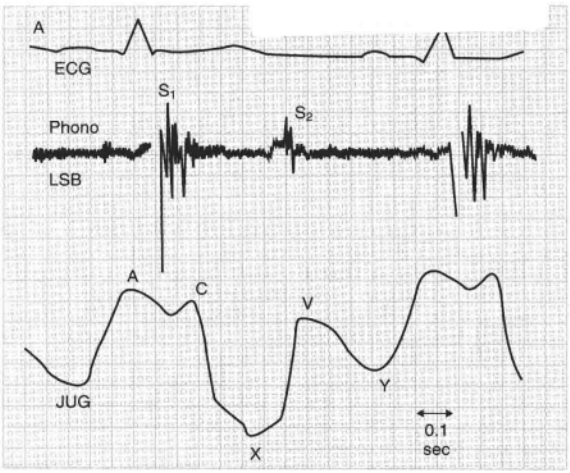

FIGURE 50-4. Normal jugular venous pulse. ECG = electrocardiogram; JUG = jugular vein; LSB = left sternal border; phono = phonocardiogram; S_1 = first heart sound; S_2 = second heart sound.

TABLE 50-6

Positive hepatojugular reflux	Suspect heart failure, particularly left ventricular systolic dysfunction (echocardiography recommended)
Elevated systemic venous pressure without obvious *x* or *y* descent, quiet precordium, and pulsus paradoxus	Suspect cardiac tamponade (echocardiography recommended)
Elevated systemic venous pressure with sharp *y* descent, Kussmaul's sign, and quiet precordium	Suspect constrictive pericarditis (cardiac catheterization and MRI or CT recommended)
Elevated systemic venous pressure with a sharp brief *y* descent, Kussmaul's sign, and evidence of pulmonary hypertension and tricuspid regurgitation	Suspect restrictive cardiomyopathy (cardiac catheterization and MRI or CT recommended)
A prominent *a* wave with or without elevation of mean systemic venous pressure	Exclude tricuspid stenosis, right ventricular hypertrophy due to pulmonary stenosis, and pulmonary hypertension (echo-Doppler study recommended)
A prominent *v* wave with a sharp *y* descent	Suspect tricuspid regurgitation (echo-Doppler or cardiac catheterization to determine etiology)

CT = computed tomography; MRI = magnetic resonance imaging.
From Braunwald E, ed. *Heart Disease: A Textbook of Cardiovascular Medicine*, 5th ed. Philadelphia: WB Saunders; 1997.

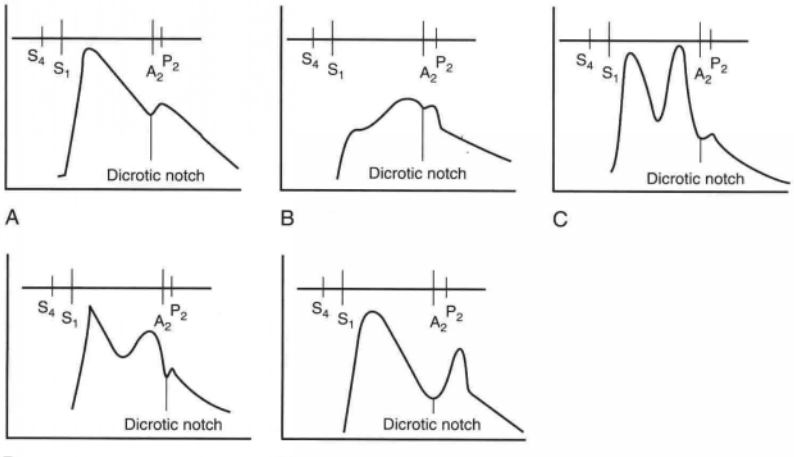

FIGURE 50-5. Schematic diagrams of the configurational changes in the carotid pulse and their differential diagnosis. Heart sounds also are illustrated. **A,** Normal. **B,** Anacrotic pulse with slow initial upstroke. The peak is close to the second heart sound. These features suggest fixed left ventricular outflow obstruction, such as valvular aortic stenosis. **C,** Pulsus bisferiens, with percussion and tidal waves occurring during systole. This type of carotid pulse contour is observed most frequently in patients with hemodynamically significant aortic regurgitation or combined aortic stenosis and regurgitation with dominant regurgitation. It rarely is observed in patients with mitral valve prolapse or in normal individuals. **D,** Pulsus bisferiens in hypertrophic obstructive cardiomyopathy. This finding rarely is appreciated at the bedside by palpation. **E,** Dicrotic pulse results from an accentuated dicrotic wave and tends to occur in sepsis, severe heart failure, hypovolemic shock, and cardiac tamponade and after aortic valve replacement. A_2 = aortic component of the second heart sound; P_2 = pulmonary component of the second heart sound); S_1 = first heart sound; S_4 = atrial sounds. (From Chatterjee K. Bedside evaluation of the heart: the physical examination. In: Chatterjee K, Chetlin MD, Karliner J, et al, eds. *Cardiology: An Illustrated Text/Reference*. Philadelphia: JB Lippincott; 1991:3.11-3.51.)

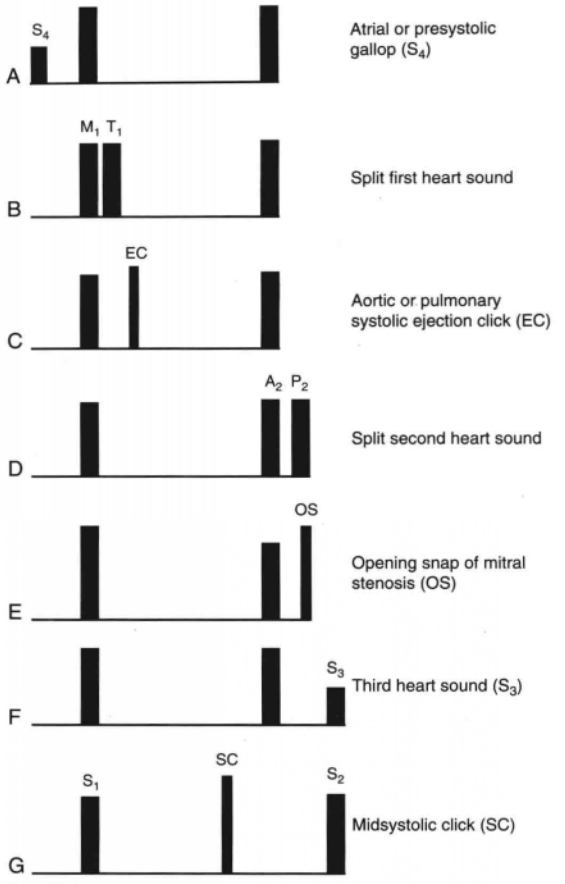

FIGURE 50-6. Timing of the different heart sounds and added sounds. (Modified from Wood P. *Diseases of the Heart and Circulation*, 3rd ed. Philadelphia: JB Lippincott; 1968.)

branch block. The split may be paradoxical in patients with left bundle branch block or other causes of delayed left ventricular emptying. The aortic component of the second sound is increased in intensity in the presence of systemic hypertension and decreased in intensity in patients with aortic stenosis. The pulmonic second sound is increased in the presence of pulmonary hypertension.

Early systolic ejection sounds are related to forceful opening of the aortic or pulmonic valve. These sounds are common in congenital aortic stenosis, with a mobile valve; in hypertension, with forceful opening of the aortic valve; and in healthy young individuals, especially when cardiac output is increased. Midsystolic or late systolic clicks are caused most commonly by mitral valve prolapse (Chapter 75). Clicks are relatively high-frequency sounds that are heard best with the diaphragm of the stethoscope.

An S_3 corresponds to rapid ventricular filling during early diastole. It may occur in normal children and young adults, especially if stroke volume is increased. After about 40 years of age, however, an S_3 should be considered abnormal; it is caused by conditions that increase the volume of ventricular filling during early diastole (e.g., mitral regurgitation) or that increase pressure in early diastole (e.g., advanced heart failure). A left ventricular S_3 gallop is heard best at the apex, whereas the right ventricular S_3 gallop is heard best at the fourth intercostal space at the left parasternal border; both are heard best with the bell of the stethoscope. An S_4 is heard rarely in young individuals but is common in adults older than 40 or 50 years because of reduced ventricular compliance during atrial contraction; it is a nearly ubiquitous finding in patients with hypertension, heart failure, or ischemic heart disease.

The opening snap of mitral and, less commonly, tricuspid stenosis (Chapter 75) occurs at the beginning of mechanical diastole, before the onset of the rapid phase of ventricular filling. An opening snap is high pitched and is heard best with the diaphragm; this differential frequency should help distinguish an opening snap from an S_3 on physical examination. An opening snap commonly can be distinguished from a loud pulmonic component of the second heart sound by the differential location (mitral opening snap at the apex, tricuspid opening snap at the left third or fourth intercostal space, pulmonic second sound at the left second intercostal space) and by the longer interval between S_2 and the opening snap.

Heart murmurs may be classified as systolic, diastolic, or continuous (Table 50-7). Murmurs are graded by intensity on a scale of 1 to 6. Grade 1 is faint and appreciated only by careful auscultation; grade 2, readily audible; grade 3, moderately loud; grade 4, loud and associated with a palpable thrill; grade 5, loud and audible with the stethoscope only partially placed on the chest; and grade 6, loud enough to be heard without the stethoscope on the chest. Systolic ejection murmurs usually peak in early to mid systole when left ventricular ejection is maximal; examples include fixed valvular, supravalvular, or infravalvular aortic stenosis and pulmonic stenosis. The murmur of hypertrophic obstructive cardiomyopathy has a similar ejection quality, although its peak may be later in systole when dynamic obstruction is maximal (Chapter 60). Pansystolic murmurs are characteristic of mitral or tricuspid regurgitation or with a left-to-right shunt from conditions such as a ventricular septal defect (left ventricle to right ventricle). A late systolic murmur is characteristic of mitral valve prolapse (Chapter 75) or ischemic papillary muscle dysfunction. Ejection quality murmurs also may be heard in patients with normal valves but increased flow, such as occurs with marked anemia, fever, or bradycardia secondary to congenital complete heart block; they also may be heard across a valve that is downstream from increased flow because of an intracardiac shunt. Maneuvers such as inspiration, expiration, standing, squatting, and hand gripping can be especially useful in the differential diagnosis of a murmur; however, echocardiography commonly is required to make a definitive diagnosis of cause and severity (Table 50-8).

High-frequency, early diastolic murmurs are typical of aortic regurgitation and pulmonic regurgitation from a variety of causes. The murmurs of mitral and tricuspid stenosis begin in early to mid-diastole and tend to diminish in intensity later in diastole in the absence of effective atrial contraction, but they tend to increase in intensity in later diastole if effective atrial contraction is present.

Continuous murmurs may be caused by any abnormality that is associated with a pressure gradient in systole and diastole. Examples include a patent ductus arteriosus, ruptured sinus of Valsalva aneurysm, arteriovenous fistula (of the coronary artery, pulmonary artery, or thoracic artery), and a mammary soufflé. In some situations, murmurs of two coexistent conditions (e.g., aortic stenosis and regurgitation; atrial septal defect with a large shunt and resulting flow murmurs of relative mitral and pulmonic stenosis) may mimic a continuous murmur.

Abdomen

The most common cause of hepatomegaly in patients with heart disease is hepatic engorgement from elevated right-sided pressures associated with right ventricular failure of any cause. Hepatojugular reflux is elicited by pressing on the liver and showing an increase in the jugular venous pressure; it indicates advanced right ventricular failure or obstruction to right ventricular filling. Evaluation of the abdomen also may reveal an enlarged liver caused by a systemic disease, such as hemochromatosis (Chapter 219) or sarcoidosis (Chapter 95), which also may affect the heart. In more severe cases, splenomegaly and ascites also may be noted. Large, palpable, polycystic kidneys (Chapter 129) commonly are associated with hypertension. A systolic bruit suggestive of renal artery stenosis (Chapter 127) or an enlarged abdominal aorta (Chapter 78) is a clue of atherosclerosis.

Extremities

Extremities should be evaluated for peripheral pulses, edema, cyanosis, and clubbing. Diminished peripheral pulses suggest peripheral arterial disease (Chapters 79 and 80). Delayed pulses in the legs are consistent with coarctation of the aorta and are seen after aortic dissection.

TABLE 50-7 SOME COMMON CAUSES OF HEART MURMURS*

	USUAL LOCATION	COMMON ASSOCIATED FINDINGS
SYSTOLIC		
Holosystolic		
Mitral regurgitation	Apex → axilla	↑ with handgrip; S_3 if marked mitral regurgitation; left ventricular dilation common
Tricuspid regurgitation	LLSB	↑ with inspiration; right ventricular dilation common
Ventricular septal defect	LLSB → RLSB	Often with thrill
Early–mid systolic		
Aortic valvular stenosis	RUSB	
Fixed supravalvular or subvalvular	RUSB	Ejection click if mobile valve; soft or absent A_2 if valve immobile; later peak associated with more severe stenosis
Dynamic infravalvular	LLSB → apex + axilla	Hypertrophic obstructive cardiomyopathy; murmur louder if left ventricular volume lower or contractility increased, softer if left ventricular volume increased†; can be later in systole if obstruction delayed
Pulmonic valvular stenosis	LUSB	↑ with inspiration
Infravalvular (infundibular)	LUSB	↑ with inspiration
Supravalvular	LUSB	↑ with inspiration
"Flow murmurs"	LUSB	Anemia, fever, increased flow of any cause‡
Mid–late systolic		
Mitral valve prolapse	LLSB or apex → axilla	Preceded by click; murmur lengthens with maneuvers that decrease left ventricular volume†
Papillary muscle dysfunction	Apex → axilla	Ischemic heart disease
DIASTOLIC		
Early diastolic		
Aortic regurgitation	RUSB, LUSB	High-pitched, blowing quality; endocarditis, diseases of the aorta, associated aortic valvular stenosis; signs of low peripheral vascular resistance
Pulmonic valve regurgitation	LUSB	Pulmonary hypertension as a causative factor
Mid–late diastolic		
Mitral stenosis, tricuspid stenosis	Apex, LLSB	Low pitched; in rheumatic heart disease, opening snap commonly precedes murmur; can be due to increased flow across normal valve‡
Atrial myxomas	Apex (L), LLSB (R)	"Tumor plop"
Continuous		
Venous hum	Over jugular or hepatic vein or breast	Disappears with compression of vein or pressure of stethoscope
Patent ductus arteriosus	LUSB	
Arteriovenous fistula		
Coronary	LUSB	
Pulmonary, bronchial, chest wall	Over fistula	
Ruptured sinus of Valsalva aneurysm	RUSB	Sudden onset

*See also Chapters 69 and 75.
†Left ventricular volume is decreased by standing or during prolonged, forced expiration against a closed glottis (Valsalva maneuver); it is increased by squatting or by elevation of the legs; contractility is increased by adrenergic stimulation or in the beat after an extrasystolic beat.
‡Including a left-to-right shunt through an atrial septal defect for tricuspid or pulmonic flow murmurs, and a ventricular septal defect for pulmonic or mitral flow murmurs.
LLSB = left lower sternal border (4th intercostal space); LUSB = left upper sternal border (2nd-3rd intercostal spaces); RLSB = right lower sternal border (4th intercostal space); RUSB = right upper sternal border (2nd-3rd intercostal spaces).

Edema (Fig. 50-7) is a cardinal manifestation of right-sided heart failure. When it is caused by heart failure, pericardial disease, or pulmonary hypertension, the edema is usually symmetrical and progresses upward from the ankles; each of these causes of cardiac edema commonly is associated with jugular venous distention and often with hepatic congestion. Unilateral edema suggests thrombophlebitis or proximal venous or lymphatic obstruction (Fig. 50-8). Edema in the absence of evidence of right-sided or left-sided heart failure suggests renal disease, hypoalbuminemia, myxedema, or other noncardiac causes. Among unselected patients with bilateral edema, about 40% have an underlying cardiac disease, about 40% have an elevated pulmonary blood pressure, about 20% have bilateral venous disease, about 20% have renal disease, and about 25% have idiopathic edema.

Cyanosis (Fig. 50-9) is a bluish discoloration caused by reduced hemoglobin exceeding about 5 g/dL in the capillary bed. Central cyanosis is seen in patients with poor oxygen saturation resulting from a reduced inspired oxygen concentration or inability to oxygenate the blood in the lungs (e.g., as a result of advanced pulmonary disease, pulmonary edema, pulmonary arteriovenous fistula, or right-to-left shunting); it also may be seen in patients with marked erythrocytosis. Methemoglobinemia (Chapter 161) also can present with cyanosis. Peripheral cyanosis may be caused by reduced blood flow to the extremities secondary to vasoconstriction, heart failure, or shock.

Clubbing (Fig. 50-10), which is loss of the normal concave configuration of the nail as it emerges from the distal phalanx, is seen in patients with pulmonary abnormalities such as lung cancer (Chapter 197) and in patients with cyanotic congenital heart disease (Chapter 69).

Examination of the Skin

Examination of the skin may reveal bronze pigmentation typical of hemochromatosis (Chapter 219); jaundice (see Fig. 149-2 in Chapter 149) characteristic of severe right-sided heart failure or hemochromatosis; or capillary hemangiomas typical of Osler-Weber-Rendu disease (see Fig. 176-1 in Chapter 176), which also is associated with pulmonary arteriovenous fistulas and cyanosis. Infectious endocarditis may be associated with Osler's nodes (see Fig. 76-2 in Chapter 76), Janeway's lesions, or splinter hemorrhages (Fig. 50-11) (Chapter 76). Xanthomas (Fig. 50-12) are subcutaneous deposits of cholesterol seen on the extensor surfaces of the extremities or on the palms and digital creases; they are found in patients with severe hypercholesterolemia.

Laboratory Studies

All patients with known or suspected cardiac disease should have an ECG and chest radiograph. The ECG (Chapter 54) helps identify rate, rhythm,

TABLE 50-8 SENSITIVITY AND SPECIFICITY OF BEDSIDE MANEUVERS IN THE IDENTIFICATION OF SYSTOLIC MURMURS

MANEUVER	RESPONSE	MURMUR	SENSITIVITY (%)	SPECIFICITY (%)
Inspiration	↑	RS	100	88
Expiration	↓	RS	100	88
Valsalva maneuver	↑	HC	65	96
Squat to stand	↑	HC	95	84
Stand to squat	↓	HC	95	85
Leg elevation	↓	HC	85	91
Handgrip	↓	HC	85	75
Handgrip	↑	MR and VSD	68	92
Transient arterial occlusion	↑	MR and VSD	78	100

HC = hypertrophic cardiomyopathy; MR = mitral regurgitation; RS = right sided; VSD = ventricular septal defect.

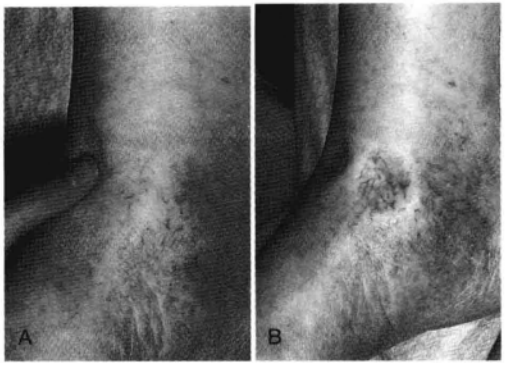

FIGURE 50-7. Pitting edema in a patient with cardiac failure. A depression ("pit") remains in the edema for some minutes after firm fingertip pressure is applied. (From Forbes CD, Jackson WD. *Color Atlas and Text of Clinical Medicine*, 3rd ed. London: Mosby; 2003.)

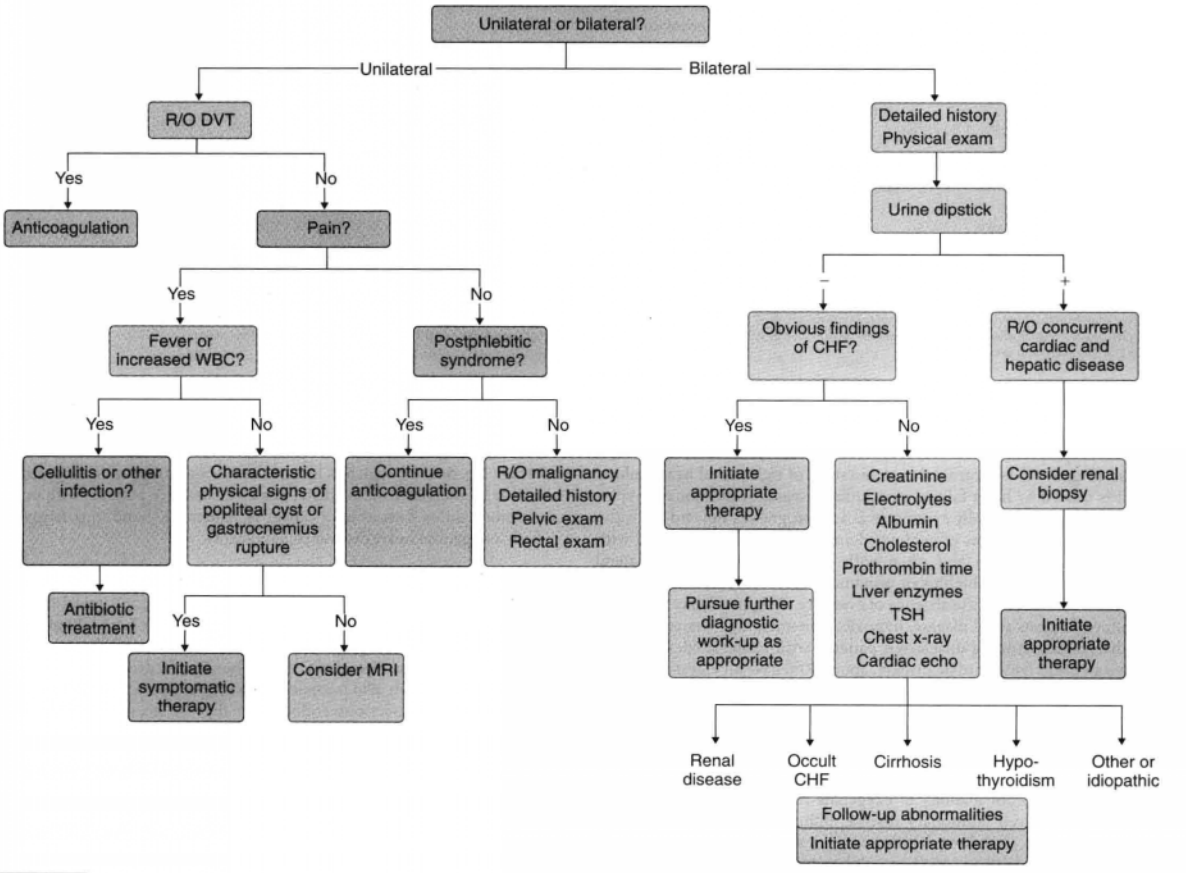

FIGURE 50-8. Diagnostic approach to patients with edema. CHF = congestive heart failure; DVT = deep venous thrombosis; MRI = magnetic resonance imaging; R/O = rule out; TSH = thyroid-stimulating hormone; WBC = white blood cell count. (From Chertow G. Approach to the patient with edema. In: Braunwald E, Goldman L, eds. *Primary Cardiology*, 2nd ed. Philadelphia: WB Saunders; 2003.)