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心脏病学

BRAUNWALD

HEART DISEASE

A Textbook of Cardiovascular Medicine

5TH EDITION

VOLUME 1

科学出版社
Harcourt Asia
W.B.SAUNDERS

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第 5 版

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EUGENE BRAUNWALD

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Part I
Examination of the Patient

Chapter 1
The History

EUGENE BRAUNWALD

IMPORTANCE OF THE HISTORY

Specialized examinations of the cardiovascular system, presented in Chapters 3 to 11, provide a large portion of the data base required to establish a specific anatomical diagnosis of cardiac disease and to determine the extent of functional impairment of the heart. The development and application of these methods represent one of the triumphs of modern medicine. However, their appropriate use is to supplement but not to supplant a careful clinical examination. The latter remains the cornerstone of the assessment of the patient with known or suspected cardiovascular disease. There is a temptation in cardiology, as in many other areas of medicine, to carry out expensive, uncomfortable, and occasionally hazardous procedures to establish a diagnosis when a detailed and thoughtful history and physical examination are sufficient. Obviously, it is undesirable to subject patients to the unnecessary risks and expenses inherent in many specialized tests when a diagnosis can be made on the basis of an adequate clinical examination or when management will not be altered significantly as a result of these tests.[1] Intelligent selection of investigative procedures from the ever-increasing array of tests now available requires far more sophisticated decision-making than was necessary when the choices were limited to electrocardiography and chest roentgenography; some of the principles in such decision-making are dealt with in Chapters 11 and 53. The history and physical examination provide the critical information necessary for most of these decisions.

THE ROLE OF THE HISTORY. The overreliance on laboratory tests has increased as physicians attempt to utilize their time more efficiently by delegating responsibility for taking the history to a physician's assistant or nurse or even by limiting the history to a questionnaire—an approach that I consider to be an undesirable trend insofar as the patient with known or suspected heart disease is concerned.[2] First, it must be appreciated that the history remains the richest source of information concerning the patient's illness,[3,4] and any practice that might diminish the quality or quantity of information provided by the history is likely ultimately to impair the quality of care. Second, the physician's attentive and thoughtful taking of a history establishes a bond with the patient that may be valuable later in securing the patient's compliance in following a complex treatment plan, undergoing hospitalization for an intensive diagnostic work-up or a hazardous operation, and, in some instances, accepting that heart disease is not present at all.

Taking a history also permits the physician to evaluate the results of diagnostic tests that have strong subjective components, such as the determination of exercise capacity (Chap. 5). Perhaps most importantly, a careful history allows the physician to evaluate the impact of the disease, or the fear of the disease, on the various aspects of the patient's life and to assess the patient's personality, affect, and stability; often it provides a glimpse of the patient's responsibilities, fears, aspirations, and threshold for discomfort as well as the likelihood of compliance with one or another therapeutic regimen. Whenever possible, the physician should question not only the patient but also relatives or close friends to obtain a clearer understanding of the extent of the patient's disability and a broader perspective concerning the impact of the disease on both the patient and the family. (For example, the patient's spouse is much more likely than the patient to provide a history of Cheyne-Stokes [periodic] respiration.)

The combination of the widespread fear of cardiovascular disorders and the deep-seated emotional, symbolic, and sometimes even religious connotations surrounding the heart may, on the one hand, provoke symptoms that mimic those of organic heart disease in persons with normal cardiovascular systems. On the other, they cause so much fear that serious symptoms are repressed or denied by patients with established heart disease.

TECHNIQUE. Several approaches can be employed successfully in obtaining a medical history. I believe that pa-

tients should first be given the opportunity to relate their experiences and complaints in their own way. Although time-consuming and likely to include much seemingly irrelevant information, this technique has the advantage of providing considerable information concerning the patient's intelligence, emotional make-up, and attitude toward his or her complaints, as well as providing the patient with the satisfaction that he has been "heard" by the physician, rather than merely having had a few questions thrown at him and then been exposed to a battery of laboratory examinations. After the patient has given an account of the illness, the physician should direct the discussion and obtain information concerning the onset and chronology of symptoms; their location, quality, and intensity; the precipitating, aggravating, and alleviating factors, the setting in which the symptoms occur, and any associated symptoms; and the response to therapy.

Of course, a detailed general medical history including the personal past history, occupational history, nutritional history, and review of systems must be obtained. Of particular interest is a history of thyroid disease, recent dental extractions or manipulations, catheterization of the bladder, and earlier examinations that showed abnormalities of the cardiovascular system as reflected in restriction from physical activity at school and in rejection for life insurance, employment, or military service. Personal habits such as exercise, cigarette smoking, alcohol intake, and parenteral use of drugs—illicit and otherwise—should be ascertained. The exact nature of the patient's work, including the physical and emotional stresses, should be assessed. The increasing appreciation of the importance of genetic influences in many forms of heart disease (Chap. 49) underscores the importance of the family history.

A wide variety of disorders including, but not limited to, neurological (Chap. 60), endocrine (Chap. 61), and rheumatic (Chap. 56) may have important effects on the cardiovascular system; it is vital to ascertain the presence of these and other conditions that are not primarily cardiovascular. A history of the risk factors for ischemic heart disease—the history of cigarette smoking, hypertension, hypercholesterolemia, diabetes mellitus, artificial or early menopause, and long-term contraceptive pill ingestion, as well as the family history of ischemic heart disease (Chap. 35)—should always be sought.

Myocardial or coronary function that may be adequate at rest is often inadequate during exertion; therefore, specific attention should be directed to the influence of activity on the patient's symptoms. Thus, a history of chest discomfort and/or undue shortness of breath that appears only during activity is characteristic of heart disease, whereas the opposite pattern, i.e., the appearance of symptoms at rest and their remission during exertion, is almost never observed in patients with heart disease but is more characteristic of functional disorders. In attempting to assess the severity of functional impairment, both the *extent* of activity and the *rate* at which it is performed before symptoms develop should be determined and related to a detailed consideration of the therapeutic regimen. For example, the development of dyspnea after walking slowly up a flight of stairs in a patient receiving intensive treatment of heart failure denotes far more severe functional disability than does a similar symptom occurring in an untreated patient who has run up a flight of stairs.

As the patient relates the history, important nonverbal clues are often provided. The physician should observe the patient's attitude, reactions, and gestures while being questioned, as well as his or her choice of words or emphasis. Tumulty has aptly likened obtaining a meaningful clinical history to playing a game of chess:[5] "The patient makes a statement and based upon its content, and mode of expression, the physician asks a counter-question. One answer stimulates yet another question until the clinician is convinced that he understands precisely all of the circumstances of the patient's illness."

CARDINAL SYMPTOMS OF HEART DISEASE

The cardinal symptoms of heart disease include dyspnea, chest pain or discomfort, syncope, collapse, palpitation, edema, cough, hemoptysis, and excess fatigue. Cyanosis is more often a sign rather than a symptom, but it may be a key feature of the history, particularly in patients with congenital heart disease. Without doubt, history-taking is the most valuable technique available for determining whether or not these symptoms are caused by heart disease. Examples of the manner in which these symptoms may serve as a guide to diagnosis are given in the following pages, and reference is made to other portions of the book that contain more detailed information.

Dyspnea

(See also pp. 450 and 464)

Dyspnea is defined as an abnormally uncomfortable awareness of breathing; it is one of the principal symptoms of cardiac and pulmonary disease.[6] Since dyspnea is regularly caused by strenuous exertion in healthy, well-conditioned subjects and by only moderate exertion in those who are normal but unaccustomed to exercise, it should be regarded as abnormal only when it occurs at rest or at a level of physical activity not expected to cause this symptom. Dyspnea is associated with a wide variety of diseases of the heart and lungs, chest wall, and respiratory muscles as well as with anxiety[7–10]; the history is the most valuable means of establishing the etiology.[11,12] Table 1–1 provides a list of the various syndromes that may cause dyspnea and the primary pathophysiological mechanisms that are responsible.[13] Borg and Noble have developed a scale that is useful in quantitating the severity of dyspnea.[14]

The *sudden* development of dyspnea suggests pulmonary embolism, pneumothorax, acute pulmonary edema, pneumonia, or airway obstruction.[10] In contrast, in most forms of *chronic* heart failure, dyspnea progresses slowly over weeks or months. Such a protracted course may also occur in a variety of unrelated conditions, including obesity, pregnancy, and bilateral pleural effusion. *Inspiratory dyspnea* suggests obstruction of the upper airways, whereas *expiratory dyspnea* characterizes obstruction of the lower airways. Exertional dyspnea suggests the presence of organic diseases, such as left ventricular failure (Chap. 15) or chronic obstructive lung disease (Chap. 47), whereas dyspnea developing at rest may occur in pneumothorax, pulmonary embolism (Chap. 46), or pulmonary edema (Chap. 15), or it may be functional. Dyspnea that occurs only at rest and is absent on exertion is almost always functional. A *functional origin* is also suggested when dyspnea, or simply a heightened awareness of breathing, is accompanied by brief stabbing pain in the region of the cardiac apex or by prolonged (more than 2 hours) dull chest pain. It is often associated with difficulty in getting enough air into the lungs, claustrophobia, and sighing respirations that are relieved by exertion, by taking a few deep breaths, or by sedation. Dyspnea in patients with panic attacks is usually accompanied by hyperventilation. A history of relief of dyspnea by bronchodilators and corticosteroids suggests asthma as the etiology, whereas relief of dyspnea by rest, diuretics, and digitalis suggests left heart failure. Dyspnea accompanied by wheezing may be secondary to left ventricular failure (*cardiac* asthma) or primary bronchial constriction (*bronchial* asthma).

In patients with *chronic heart failure*, dyspnea is a clinical expression of pulmonary venous and capillary hyper-

TABLE 1–1 DISORDERS CAUSING DYSPNEA AND LIMITING EXERCISE PERFORMANCE; PATHOPHYSIOLOGY; AND DISCRIMINATING MEASUREMENTS

DISORDERS	PATHOPHYSIOLOGY	MEASUREMENTS THAT DEVIATE FROM NORMAL
Pulmonary		
Airflow limitation	Mechanical limitation to ventilation, mismatching of $\dot{V}_A/\dot{Q}$, hypoxic stimulation to breathing	$\dot{V}_E$ max/MVV, expiratory flow pattern, V_D, V_T; $\dot{V}_{O_2}$ max, $\dot{V}_E/\dot{V}_{O_2}$, $\dot{V}_E$ response to hyperoxia, $(A - a)P_{O_2}$
Restrictive	Mismatching $\dot{V}_A/\dot{Q}$, hypoxic stimulation to breathing	
Chest wall	Mechanical limitation to ventilation	$\dot{V}_E$ max/MVV, P_{ACO_2}, $\dot{V}_{O_2}$ max
Pulmonary circulation	Rise in physiological dead space as fraction of V_T, exercise hypoxemia	V_D/V_T, work-rate–related hypoxemia, $\dot{V}_{O_2}$ max, $\dot{V}_E/\dot{V}_{O_2}$, $(a - ET)P_{CO_2}$, O_2-pulse
Cardiac		
Coronary	Coronary insufficiency	ECG, $\dot{V}_{O_2}$ max, anaerobic threshold $\dot{V}_{O_2}$, $\dot{V}_E/\dot{V}_{O_2}$, O_2-pulse, BP (systolic, diastolic, pulse)
Valvular	Cardiac output limitation (decreased effective stroke volume)	
Myocardial	Cardiac output limitation (decreased ejection fraction and stroke volume)	
Anemia	Reduced O_2-carrying capacity	O_2-pulse, anaerobic threshold $\dot{V}_{O_2}$, $\dot{V}_{O_2}$ max, $\dot{V}_E/\dot{V}_{O_2}$
Peripheral circulation	Inadequate O_2 flow to metabolically active muscle	Anaerobic threshold $\dot{V}_{O_2}$, $\dot{V}_{O_2}$ max
Obesity	Increased work to move body; if severe, respiratory restriction and pulmonary insufficiency	$\dot{V}_{O_2}$-work-rate relationship, P_{AO_2}, P_{ACO_2}, $\dot{V}_{O_2}$ max
Psychogenic	Hyperventilation with precisely regular respiratory rate	Breathing pattern, P_{CO_2}
Malingering	Hyperventilation and hypoventilation with irregular respiratory rate	Breathing pattern, P_{CO_2}
Deconditioning	Inactivity or prolonged bed rest; loss of capability for effective redistribution of systemic blood flow	O_2-pulse, anaerobic threshold $\dot{V}_{O_2}$, $\dot{V}_{O_2}$ max

$\dot{V}_A$ = alveolar ventilation; $\dot{Q}$ = pulmonary blood flow; $\dot{V}_E$ = minute ventilation; MVV = maximum voluntary ventilation; V_D/V_T = physiological dead space/tidal volume ratio; O_2 = oxygen; $\dot{V}_{O_2}$ = O_2 consumption; $(A - a)P_{O_2}$ = alveolar-arterial P_{O_2} difference; $(a - ET)P_{CO_2}$ = arterial-end tidal P_{CO_2} difference.
Modified from Wasserman, D.: Dyspnea on exertion: Is it the heart or the lungs? JAMA *248*:2042, 1982. Copyright 1982 the American Medical Association.

tension (see p. 453). It occurs either during exertion or, in resting patients, in the recumbent position, and it is relieved promptly by sitting upright or standing *(orthopnea)*. Patients with left ventricular failure soon learn to sleep on two or more pillows to avoid this symptom. In patients with heart failure, dyspnea is often accompanied by edema, upper abdominal pain (due to congestive hepatomegaly), and nocturia. The *sudden* occurrence of dyspnea in a patient with a history of mitral valve disease suggests the development of atrial fibrillation, rupture of chordae tendineae, or pulmonary embolism.

Paroxysmal nocturnal dyspnea is due to interstitial pulmonary edema and sometimes intraalveolar edema and is most commonly secondary to left ventricular failure (see p. 450). This condition, beginning usually 2 to 4 hours after the onset of sleep and often accompanied by cough, wheezing, and sweating, is quite frightening to the patient. Paroxysmal nocturnal dyspnea is often ameliorated by the patient's sitting on the side of the bed or getting out of bed; relief is not instantaneous but usually requires 15 to 30 minutes. Although paroxysmal nocturnal dyspnea secondary to left ventricular failure is usually accompanied by coughing, a careful history often discloses that the dyspnea *precedes* the cough, not vice versa. In contrast, patients with *chronic pulmonary disease* may also awaken at night, but cough and expectoration usually precede the dyspnea. These patients also often have a long history of smoking and a chronic cough with sputum production and wheezing and may be able to breathe more easily while leaning forward. Nocturnal dyspnea associated with pulmonary disease is usually relieved after the patient rids himself or herself of secretions rather than specifically by sitting up. Details of the value and limitations of the history of dyspnea in differentiating between primary diseases of the heart and lungs[15,16] are presented on page 451.

Patients with *pulmonary embolism* usually experience sudden dyspnea that may be associated with apprehension, palpitation, hemoptysis, or pleuritic chest pain (Chap. 46). The development or intensification of dyspnea, sometimes associated with a feeling of faintness, may be the only complaint of the patient with pulmonary emboli. *Pneumothorax* and *mediastinal emphysema* also cause dyspnea acutely, accompanied by sharp chest pain. Dyspnea accompanying thoracic pain occurs in *acute myocardial infarction*. Dyspnea is a common *"anginal equivalent"* (see p. 1290), i.e., a symptom secondary to myocardial ischemia that occurs in place of typical anginal discomfort.[16] This form of dyspnea may or may not be associated with a sensation of tightness in the chest, is present on exertion or emotional stress, is relieved by rest (more often in the sitting than in the recumbent position), is similar to angina in duration (i.e., 2 to 10 minutes), and is usually responsive to or prevented by nitroglycerin. The sudden development of severe dyspnea while sitting rather than lying, or whenever a particular position is assumed, suggests the possibility of a myxoma (see p. 1467) or ball-valve thrombus in the left atrium. When dyspnea is relieved by squatting, it is caused most commonly by tetralogy of Fallot or a variant thereof (see p. 929).

Chest Pain or Discomfort

(See also p. 1290)

Elucidation of the cause of chest pain is one of the key tasks of physicians, and this symptom is responsible for many cardiac consultations. The history remains the most

important technique for distinguishing among the many causes of chest discomfort. Although chest pain or discomfort is one of the cardinal manifestations of cardiac disease, it is crucial to recognize that the pain may originate not only in the heart but also in (1) a variety of noncardiac intrathoracic structures, such as the aorta, pulmonary artery, bronchopulmonary tree, pleura, mediastinum, esophagus, and diaphragm; (2) the tissues of the neck or thoracic wall, including the skin, thoracic muscles, cervicodorsal spine, costochondral junctions, breasts, sensory nerves, and spinal cord; and (3) subdiaphragmatic organs such as the stomach, duodenum, pancreas, and gallbladder (Table 1–2). Pain of functional origin or factitious pain may also occur in the chest. Although a wide variety of laboratory tests is available to aid in the differential diagnosis of chest pain, without question the history remains the most valuable mode of examination. In obtaining the history of a patient with chest pain it is helpful to have a mental checklist and to ask the patient to describe the location, radiation, and character of the discomfort; what causes and relieves it; time relationships, including the duration, frequency, and pattern of recurrence of the discomfort; the setting in which it occurs; and associated symptoms. It is also particularly useful to observe the patient's gestures. Clenching the fist in front of the sternum while describing the sensation (Levine's sign) is a strong indication of an ischemic origin of the pain.

QUALITY. *Angina pectoris* may be defined as a discomfort in the chest and/or adjacent area associated with myocardial ischemia but without myocardial necrosis.[17–20] It is important to recognize that angina means *choking*, not pain. Thus, the discomfort of angina often is described not as pain at all but rather as an unpleasant sensation; "pressing," "squeezing," "strangling," "constricting," "bursting," and "burning" are some of the adjectives commonly used to describe this sensation (Table 1–3). "A band across the chest" and "a weight in the center of the chest" are other frequent descriptors. It is characteristic of angina that the intensity of effort required to incite it may vary from day to day and throughout the day in the same patient, but often a careful history will uncover explanations for this, such as meals ingested, weather, emotions, and the like. The anginal threshold is lower in the morning than at any other time of day; thus patients note frequently that activities that may cause angina in the morning or when first undertaken do not do so later in the day. When the threshold for angina is quite variable, defies any pattern, and is prominent at rest, the possibility that myocardial ischemia is caused by coronary spasm should be considered (see p. 1189). Thus, a careful history not only may indicate the cause of the pain (i.e., myocardial ischemia) but can also provide a clue to the mechanism of the ischemia (spasm vs. organic obstruction).

A history of prolonged, severe anginal chest discomfort accompanied by profound fatigue often signifies acute myocardial infarction.[21] There is some relationship between location of the chest pain and the site of coronary artery occlusion[22]; patients with ischemic heart disease who complain of substernal or left chest pain with radiation to the left arm often have heart disease involving the left coronary artery, while those with epigastric pain radiating to the neck or jaw may *not* have disease of the left anterior descending coronary artery.

When dyspnea is an "anginal equivalent," the patient may describe the midchest as the site of the shortness of breath, whereas true dyspnea is usually not well localized. Other anginal equivalents are discomfort limited to areas that are ordinarily sites of secondary radiation, such as the ulnar aspect of the left arm and forearm, lower jaw, teeth, neck, or shoulders, and the development of gas and belching, nausea, "indigestion," dizziness, and diaphoresis. Anginal equivalents above the mandible or below the umbilicus are quite uncommon. It is useful to determine whether the patient has symptoms or complications caused by atherosclerosis of other vascular beds, e.g., intermittent claudication, transient ischemic attacks, or stroke. In patients with suspected angina, a history of one of these manifestations of extracardiac atherosclerosis lends weight to the diagnosis of myocardial ischemia.

The chest discomfort of *pulmonary hypertension* (see p. 788) may be identical to that of typical angina[23,24]; it is caused by right ventricular ischemia or dilation of the pul-

TABLE 1–2 DIFFERENTIAL DIAGNOSIS OF EPISODIC CHEST PAIN RESEMBLING ANGINA PECTORIS

	DURATION	QUALITY	PROVOCATION	RELIEF	LOCATION	COMMENT
Effort angina	5–15 minutes	Visceral (pressure)	During effort or emotion	Rest, nitroglycerin	Substernal, radiates	First episode vivid
Rest angina	5–15 minutes	Visceral (pressure)	Spontaneous (? with exercise)	Nitroglycerin	Substernal, radiates	Often nocturnal
Mitral prolapse	Minutes to hours	Superficial (rarely visceral)	Spontaneous (no pattern)	Time	Left anterior	No pattern, variable character
Esophageal reflux	10 minutes to 1 hour	Visceral	Recumbency, lack of food	Food, antacid	Substernal, epigastric	Rarely radiates
Esophageal spasm	5–60 minutes	Visceral	Spontaneous, cold liquids, exercise	Nitroglycerin	Substernal, radiates	Mimics angina
Peptic ulcer	Hours	Visceral, burning	Lack of food, "acid" foods	Foods, antacids	Epigastric, substernal	
Biliary disease	Hours	Visceral (waxes and wanes)	Spontaneous, food	Time, analgesia	Epigastric, ? radiates	Colic
Cervical disc	Variable (gradually subsides)	Superficial	Head and neck movement, palpation	Time, analgesia	Arm, neck	Not relieved by rest
Hyperventilation	2–3 minutes	Visceral	Emotion, tachypnea	Stimulus removal	Substernal	Facial paresthesia
Musculoskeletal	Variable	Superficial	Movement, palpation	Time, analgesia	Multiple	Tenderness
Pulmonary	30 minutes +	Visceral (pressure)	Often spontaneous	Rest, time, bronchodilator	Substernal	Dyspneic

Reproduced with permission from Christie, L.G., Jr., and Conti, C.R.: Systematic approach to the evaluation of angina-like chest pain. Am. Heart J. *102*:897, 1981.

TABLE 1–3 SOME FEATURES DIFFERENTIATING CARDIAC FROM NONCARDIAC CHEST PAIN

FAVORING ISCHEMIC ORIGIN	AGAINST ISCHEMIC ORIGIN
Character of Pain	
Constricting	Dull ache
Squeezing	"Knife-like," sharp, stabbing
Burning	"Jabs" aggravated by respiration
"Heaviness," "heavy feeling"	
Location of Pain	
Substernal	In the left submammary area
Across mid-thorax, anteriorly	In the left hemithorax
In both arms, shoulders	
In the neck, cheeks, teeth	
In the forearms, fingers	
In the interscapular region	
Factors Provoking Pain	
Exercise	Pain *after* completion of exercise
Excitement	Provoked by a specific body motion
Other forms of stress	
Cold weather	
After meals	

From Selzer, A.: Principles and Practice of Clinical Cardiology. 2nd ed. Philadelphia, W.B. Saunders Company, 1983, p. 17.

monary arteries. The chest discomfort of *unstable angina* and *acute myocardial infarction* (see p. 1198) is similar in quality to that of angina pectoris in location and character; however, it usually radiates more widely than does angina, is more severe, and therefore is generally referred to by the patient as true *pain* rather than *discomfort*. This pain generally develops unrelated to unusual effort or emotional stress, often with the patient at rest or even sleeping. Usually nitroglycerin does not provide complete or lasting relief.

Acute pericarditis (see p. 1481) is frequently preceded by a history of a viral upper respiratory infection. The inflammation causes pain that is sharper than is anginal discomfort, is more left-sided than central, and is often referred to the neck. The pain of pericarditis lasts for hours and is little affected by effort but is often aggravated by breathing, turning in bed, swallowing, or twisting the body; unlike angina, the pain of acute pericarditis may lessen when the patient sits up and leans forward.

Aortic dissection (see p. 1554) is suggested by the sudden development of persistent, severe pain with radiation to the back and into the lumbar region in a patient with a history of hypertension. An expanding *thoracic aortic aneurysm* may erode the vertebral bodies and cause localized, severe, boring pain that may be worse at night. An aneurysmally enlarged left atrium in patients with mitral valve disease rarely causes chest pain; instead, patients commonly complain of discomfort in the back or right side of the chest that intensifies on exertion.

Chest-wall pain due to *costochondritis* or *myositis* is common in patients who present with fear of heart disease.[25] It is associated with both local costochondral and muscle tenderness, which may be aggravated by moving or coughing. Chest-wall pain[26] may also accompany chest injury, or *Tietze syndrome* (i.e., discomfort localized in swelling of the costochondral and costosternal joints, which are painful on palpation). When *herpes zoster* affects the left chest it may mimic myocardial infarction. However, its persistence, its localization to a dermatome, the extreme sensitivity of the skin to touch, and the appearance of the characteristic vesicles allow recognition of this condition. Pain in the chest wall is quite common following cardiac or thoracic surgery and may be confused with myocardial ischemia. Postsurgical pain is usually localized to the incision or the site of insertion of a chest tube.

The pain of *pulmonary embolism* (Ch. 46) usually commences suddenly, and, when it occurs at rest, is seen in patients at high risk for this condition (heart failure, venous disease, the postoperative state), and is accompanied by shortness of breath. It is typically described as tightness in the chest and is accompanied or followed by *pleuritic* chest pain, i.e., sharp pain in the side of the chest that is intensified by respiration or cough. Chest pain associated with *spontaneous pneumothorax* develops suddenly, is associated with acute dyspnea, and is located in the lateral area of the chest. The chest pain associated with *mediastinal emphysema* also commences suddenly and is accompanied by dyspnea, sometimes severe; it is located in the center of the chest.

Functional or *psychogenic chest* pain may be one feature of an anxiety state called Da Costa syndrome or neurocirculatory asthenia.[27–29] It is localized typically to the cardiac apex and consists of a dull, persistent ache that lasts for hours and is often accentuated by or alternates with attacks of sharp, lancinating stabs of inframammary pain of 1 or 2 seconds' duration. The condition may occur with emotional strain and fatigue, bears little relation to exertion, and may be accompanied by precordial tenderness. Attacks may be associated with palpitation, hyperventilation, numbness and tingling in the extremities, sighing, dizziness, dyspnea, generalized weakness, and a history of panic attacks and other signs of emotional instability or depression. The pain may not be completely relieved by any medication other than analgesics, but it is often attenuated by many types of interventions, including rest, exertion, tranquilizers, and placebos. Therefore, in contrast to ischemic discomfort, functional pain is more likely to show variable responses to interventions on different occasions. Since functional chest pain is often preceded by hyperventilation, which in turn may cause increased muscle tension and be responsible for diffuse chest tightness, some instances of so-called functional chest pain may, in fact, have an organic basis. Chest pain is common in patients with prolapse of the mitral valve (see p. 1029). The nature of the pain varies considerably among patients with this condition; it may be similar to that of classic angina pectoris or may resemble the chest pain of neurocirculatory asthenia described above.

LOCATION. Embryologically the heart is a midline viscus; thus, cardiac ischemia produces anginal symptoms that are characteristically felt substernally or across both sides of the chest (Figs. 1–1 and 1–2). Some patients complain of discomfort only to the left or less commonly only to the right of the midline. If the pain or discomfort can be localized to the skin or superficial structures and can be reproduced by localized pressure, it generally arises from the chest wall. If the patient can point directly to the site of discomfort, and if that site is quite small (< 3 cm in diameter), it is usually not angina pectoris. Like other symptoms arising in deeper structures, angina tends to be diffuse and

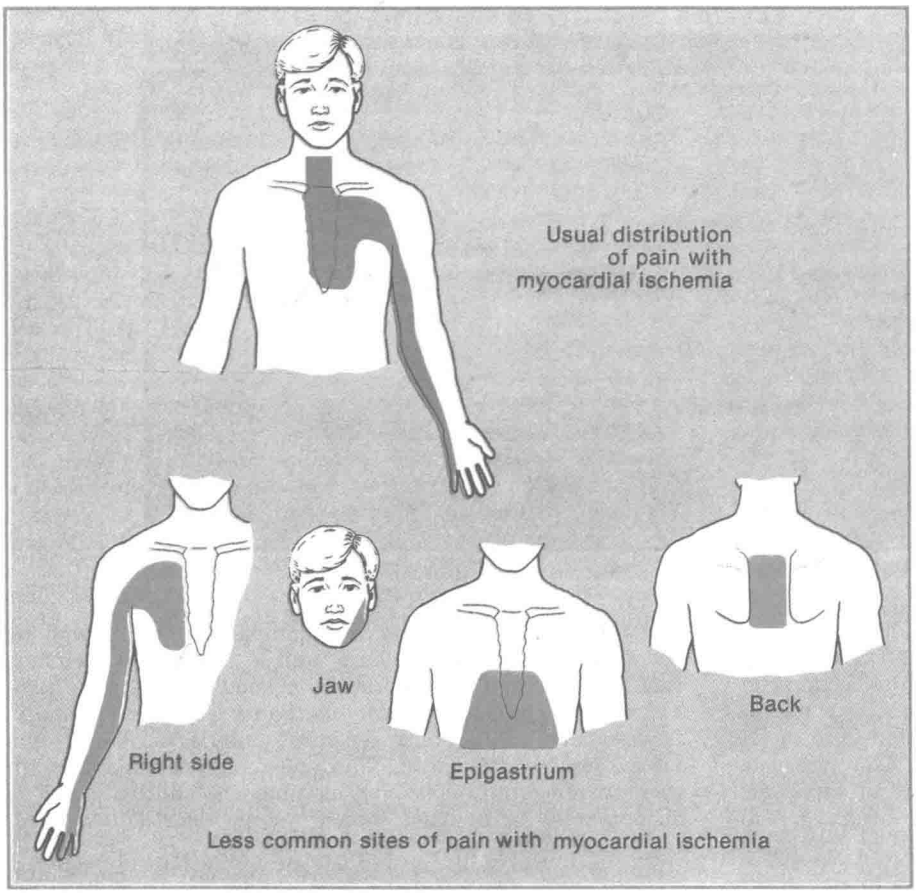

FIGURE 1–1. Pain patterns with myocardial ischemia. The usual distribution is referral to all or part of the sternal region, the left side of the chest, and the neck and down the ulnar side of the left forearm and hand. With severe ischemic pain, the right chest and right arm are often involved as well, although isolated involvement of these areas is rare. Other sites sometimes involved, either alone or together with other sites, are the jaw, epigastrium, and back. (From Horwitz, L. D.: Chest pain. *In* Horwitz, L. D., and Groves, B. M. [eds.]: Signs and Symptoms in Cardiology. Philadelphia, J. B. Lippincott, 1985, p. 9.)

eludes precise localization. Pain that is localized to the region of or under the left nipple or that radiates to the right lower chest[30] is usually noncardiac in origin and may be functional or due to osteoarthritis, gaseous distention of the stomach, or the splenic flexure syndrome. Although pain due to myocardial ischemia often radiates to the arm, especially the ulnar aspect of the left arm, wrist, epigastrium, or left shoulder, such radiation may also occur in pericarditis and disorders of the cervical spine. Radiation of pain from the chest to the neck and jaws is typical of myocardial infarction. Chest pain that radiates to the neck and jaw occurs in pericarditis as well as in myocardial ischemia. Dissection of the aorta or enlargement of an aortic aneurysm usually produces pain in the back in addition to the front of the chest.

DURATION. The duration of the pain is important in de-

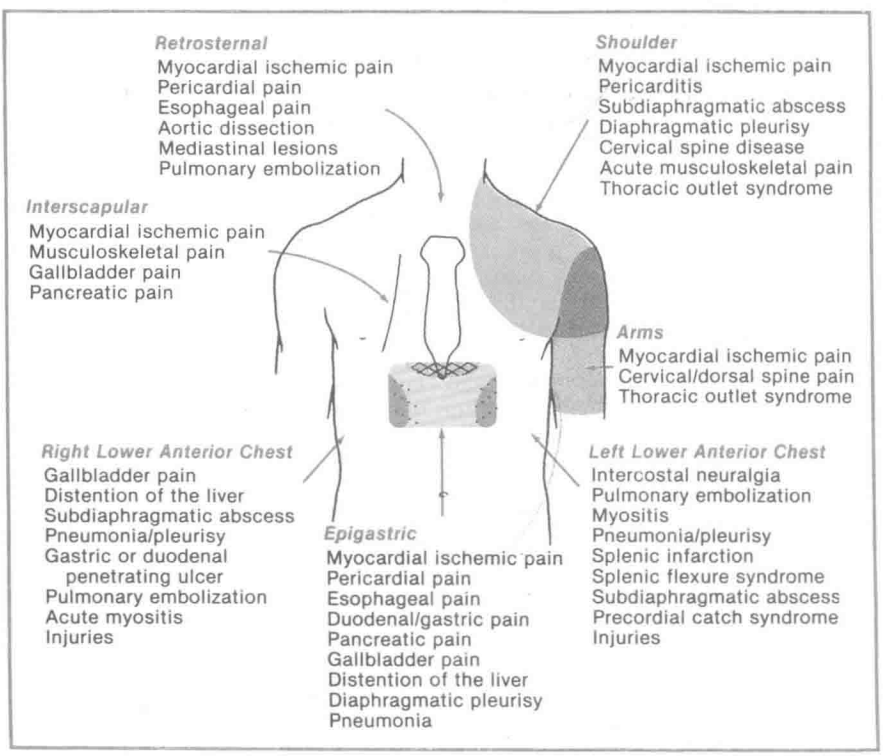

FIGURE 1–2. Differential diagnosis of chest pain according to location where pain starts. Serious intrathoracic or subdiaphragmatic diseases are usually associated with pains that begin in the left anterior chest, left shoulder, or upper arm, the interscapular region, or the epigastrium. The scheme is not all-inclusive; e.g., intercostal neuralgia occurs in locations other than the left, lower anterior chest area. (From Miller, A. J.: Diagnosis of Chest Pain. New York, Raven Press, 1988, p. 175.)

termining its etiology. Angina pectoris is relatively short, usually lasting from 2 to 10 minutes. However, if the pain is very brief, i.e., a momentary, lancinating, sharp pain, "stitch," or other discomfort that lasts less than 15 seconds, angina can usually be excluded; such a short duration points instead to musculoskeletal pain, pain due to hiatal hernia, or functional pain. Chest pain that is otherwise typical of angina but that lasts for more than 10 minutes or occurs at rest is typical of unstable angina. Chest pain lasting for hours may be seen with acute myocardial infarction, pericarditis, aortic dissection, musculoskeletal disease, herpes zoster, and anxiety.

PRECIPITATING AND AGGRAVATING FACTORS. Angina pectoris occurs characteristically on exertion, particularly when the patient is hurrying or walking up an incline. Thus, the development of chest discomfort or pain during walking, typically in the cold and against a wind, and after a heavy meal, is characteristic of angina pectoris. Angina may be precipitated by strong emotion or fright, by a nightmare, by working with the arms over the head, by cold exposure, or smoking a cigarette. Prinzmetal's (variant) angina characteristically occurs at rest (see p. 1340), and may or may not be affected by exertion; however, it must be remembered that (nonvariant) angina, although most often precipitated by effort, not uncommonly may be experienced at rest, as in unstable angina (see p. 1331); in these patients exertion intensifies the discomfort.

DIFFERENTIAL DIAGNOSIS. Chest pain that occurs after protracted vomiting may be due to the Mallory-Weiss syndrome, i.e., a tear in the lower portion of the esophagus. Pain that occurs while the patient is bending over is often radicular and may be associated with osteoarthritis of the cervical or upper thoracic spine. Chest pain occurring on moving the neck may be due to a herniated intervertebral disc.

Esophageal Pain. Substernal and epigastric discomfort during swallowing may be due to esophageal spasm or esophagitis, often with acid reflux, with or without a hiatal hernia. These conditions may also be associated with substernal or epigastric burning pain that is brought on by eating or lying down after meals and that may be relieved by antacids. Pain due to esophageal spasm has many of the features of and may be difficult to differentiate from angina pectoris.[30] Indeed, it is a common cause of chest pain considered atypical of angina pectoris.[31–34] A history of acid reflux into the mouth (water brash) and/or dysphagia[35] may be a useful diagnostic clue pointing to esophageal disease.[36] The chest discomfort secondary to esophageal reflux is most common after meals and occurs in the supine position or on bending. The difficulty in distinguishing angina from esophageal disease is compounded by the frequent coexistence of these two common conditions, by the observation that esophageal reflux lowers the threshold for the development of angina,[37] and by the observation that esophageal spasm may be precipitated by ergonovine and relieved by nitroglycerin. Esophageal pain radiates to the back more frequently than does angina pectoris.[32]

The discomfort produced by *peptic ulcer disease* is characteristically located in the midepigastrium. It may also resemble angina pectoris, but its characteristic relationship to food ingestion and its relief by antacids are important differentiating features. While the pain of *acute pancreatitis* may mimic acute myocardial infarction, with the former there is usually a history of alcoholism or biliary tract disease. The pain of pancreatitis, like that of myocardial infarction, may be predominant in the epigastrium. However, unlike the pain of myocardial infarction, it is usually transmitted to the back, is position-sensitive, and may be relieved in part by leaning forward.[30] Chest pain aggravated by coughing may be due to pericarditis, bronchitis, or pleurisy or may be of radicular origin. *Congenital absence of the pericardium* (see p. 1522) produces chest pain that is relieved by changing position in bed, is brought on by lying on the left side, and lasts a few seconds. Pain due to the *scalenus anticus (thoracic outlet) syndrome* may be confused with angina because it is often associated with paresthesias along the ulnar distribution of the arm and forearm. However, in contrast to angina, not only is it typically precipitated by abduction of the arm or lifting a weight, but it is not brought on by walking.

RELIEF OF PAIN. Rest and nitroglycerin characteristically relieve the discomfort of angina in approximately 1 to 5 minutes. If more than 10 minutes transpire before relief, the diagnosis of chronic stable angina becomes questionable and instead may be unstable angina, acute myocardial infarction, or pain not caused by myocardial ischemia at all. Although nitroglycerin commonly relieves the pain of angina pectoris, the discomfort of esophageal spasm and esophagitis may also be relieved by this drug. Angina pectoris is alleviated by quiet standing or sitting; sometimes resting in the recumbent position does not relieve angina. Chest pain secondary to *acute pericarditis* is characteristically relieved by leaning forward, whereas pain that is relieved by food or antacids may be due to *peptic ulcer disease* or esophagitis. Pain that is alleviated by holding the breath in deep expiration is commonly due to pleurisy. Some patients with upper gastrointestinal disease or anxiety report relief of symptoms after belching.

ACCOMPANYING SYMPTOMS. The physician should always be concerned about the patient who reports the presence of chest pain and profuse sweating. This combination of symptoms frequently signals a serious disorder, most often acute myocardial infarction, but also acute pulmonary embolism or aortic dissection. Severe chest pain accompanied by nausea and vomiting is also often due to myocardial infarction. The latter diagnosis, as well as pneumothorax, pulmonary embolism, or mediastinal emphysema, is suggested by pain associated with shortness of breath. Chest pain accompanied by palpitation may be due to the acute myocardial ischemia that results from a tachyarrhythmia-induced increase in myocardial oxygen consumption in the presence of coronary artery disease. Chest pain accompanied by hemoptysis suggests pulmonary embolism with infarction or lung tumor, whereas pain accompanied by fever occurs in pneumonia, pleurisy, and pericarditis. Functional pain is commonly accompanied by frequent sighing, anxiety, or depression.

Cyanosis

Cyanosis, both a symptom and a physical sign, is a bluish discoloration of the skin and mucous membranes resulting from an increased quantity of reduced hemoglobin or of abnormal hemoglobin pigments in the blood perfusing these areas[38,39] (see also pp. 885 and 891). There are two principal forms of cyanosis: (1) central cyanosis, characterized by decreased arterial oxygen saturation due to right-to-left shunting of blood or impaired pulmonary function, and (2) peripheral cyanosis, most commonly secondary to cutaneous vasoconstriction due to low cardiac output or exposure to cold air or water; if peripheral cyanosis is confined to an extremity, localized arterial or venous obstruction should be suspected. A history of cyanosis localized to the hands suggests Raynaud's phenomenon. Patients with central cyanosis due to congenital heart disease or pulmonary disease characteristically report that it worsens during exertion, whereas the resting peripheral cyanosis of congestive heart failure may be accentuated only slightly, if at all, during exertion.

Central cyanosis usually becomes apparent at a mean capillary concentration of 4 gm/dl reduced hemoglobin (or 0.5 gm/dl methemoglobin). In general, a history of cyanosis in light-skinned people is rarely elicited unless arterial saturation is 85 per cent or less; in pigmented races arterial saturation has to drop far lower before cyanosis is perceptible.

Although a history of cyanosis beginning in infancy suggests a congenital cardiac malformation with a right-to-left shunt, hereditary methemoglobinemia is another, albeit rare, cause of congenital cyanosis; the diagnosis of this condition is supported by a family history of cyanosis in the absence of heart disease.

A history of cyanosis limited to the neonatal period suggests the diagnosis of atrial septal defect with transient right-to-left shunting or, more commonly, pulmonary parenchymal disease or central nervous system depression. Cyanosis beginning at age 1 to 3 months may be reported when spontaneous closure of a patent ductus arteriosus causes a reduction of pulmonary blood flow in the presence of right-sided obstructive cardiac anomalies, most commonly tetralogy of Fallot. If cyanosis appears at age 6 months or later in childhood, it may be due to the development or progression of obstruction to right ventricular outflow in patients with ventricular septal defect. A history of the development of cyanosis in a patient with congenital heart disease between 5 and 20 years of age suggests an Eisenmenger reaction with right-to-left shunting as a consequence of a progressive increase in pulmonary vascular resistance (see p. 903). Cyanosis secondary to a pulmonary arteriovenous fistula also usually appears first in childhood.

Syncope

Syncope, which may be defined as a loss of consciousness (see also Chap. 28), results most commonly from reduced perfusion of the brain. The history is extremely valuable in the differential diagnosis of syncope (Table 1–4). Several daily attacks of loss of consciousness suggest (1) Stokes-Adams attacks, i.e., transient asystole or ventricular fibrillation in the presence of atrioventricular block; (2) other cardiac arrhythmias; or (3) a seizure disorder, i.e., petit mal epilepsy. These diagnoses are suggested when the loss of consciousness is abrupt and occurs over 1 or 2 seconds; a more gradual onset suggests vasodepressor syncope (i.e., the common faint) or syncope due to hyperventilation or, much less commonly, hypoglycemia.

CARDIAC SYNCOPE. This condition is usually of rapid onset without aura and is usually not associated with convulsive movements, urinary incontinence, and a postictal confusional state. Syncope in aortic stenosis[40,41] is usually precipitated by effort. Patients with epilepsy often have a prodromal aura preceding the seizure. Injury from falling is common, as are urinary incontinence and a postictal confusional state, associated with headache and drowsiness. Unconsciousness developing gradually and lasting for a few seconds suggests vasodepressor syncope or syncope secondary to postural hypotension, whereas a longer period suggests aortic stenosis or hyperventilation. Hysterical fainting is usually not accompanied by any untoward display of anxiety or change in pulse, blood pressure, or skin color, and there may be a question whether any true loss of consciousness occurred. It is often associated with paresthesias of the hands or face, hyperventilation, dyspnea, chest pain, and feelings of acute anxiety.

A history of syncope independent of body position suggests Stokes-Adams attacks, hyperventilation, or a convulsive disorder, whereas syncope of other etiology usually occurs in the upright position. Syncope occurring upon bending, leaning, or assuming a particular body position should raise the possibility of a left atrial myxoma (see p. 1467) or a ball-valve thrombus. Since syncope is an unusual feature of mitral stenosis, when it does occur in a patient thought to have this condition, the possibility of left atrial myxoma or ball-valve thrombus should be considered. A history of syncope occurring during or immediately following exertion suggests aortic stenosis, hypertrophic obstructive cardiomyopathy, or primary pulmonary hypertension. Syncope is rare in patients with angina pectoris unless the latter is secondary to one of the aforementioned conditions. Syncope following insulin administration suggests a hypoglycemic etiology; a history of syncope occurring several hours after eating is characteristic of reactive hypoglycemia. Loss of consciousness following an emotional stress suggests that it is vasodepressor syncope or secondary to hyperventilation.

Patients with vasodepressor syncope often have a history of recurrent fainting, commonly associated with emotional or painful stimuli. This, the most common form of syncope, may be precipitated by the sight or loss of blood or by physical or emotional stress; it can be averted by promptly lying down, and it is characteristically preceded by symptoms of autonomic hyperactivity such as dim vision, giddiness, yawning, sweating, and nausea. A history of syncope in the erect position may also be elicited in patients who have become hypovolemic as a consequence of overly vigorous diuresis. Syncope secondary to *cerebrovascular disturbance* is often preceded by aphasia, unilateral weakness, or confusion. A history of fainting following sudden movements of the head, shaving the neck, or wearing a tight collar suggests carotid sinus syncope. Syncope associated with chest pain may be secondary to massive acute myocardial infarction or infarction associated with arrhythmias; occasionally, following recovery of consciousness, the associated chest pain may be forgotten, and the infarction may be recognized only by the characteristic changes in serum enzymes and on the electrocardiogram. A history of syncope following chest pain may also occur in patients with *acute pulmonary embolism.*

REGAINING CONSCIOUSNESS. Consciousness is usually regained quite promptly in syncope of cardiovascular origin, but more slowly in patients with convulsive disorders. When consciousness is regained after vasodepressor syncope, the patient is often pale and diaphoretic with a slow heart rate, whereas after a Stokes-Adams attack, the face is often flushed and there may be cardiac acceleration. Patients who report an injury when falling during a fainting spell usually have epilepsy or occasionally syncope of cardiac origin, but patients who have unconsciousness related to emotional disturbance rarely sustain physical trauma.

DIFFERENTIAL DIAGNOSIS. A family history of syncope or near-syncope can often be elicited in patients with hypertrophic cardiomyopathy (see p. 1414) or ventricular tachyarrhythmias associated with Q-T

TABLE 1–4 CLUES FROM THE HISTORY IN ELUCIDATING THE CAUSE OF SYNCOPE

PRECEDING EVENTS	
Drugs:	Orthostatic hypotension (antihypertensives), hypoglycemia (insulin)
Severe pain, emotional stress:	Vasovagal syncope, hyperventilation
Movement of head and neck:	Carotid sinus hypersensitivity
Exertion:	Any form of obstruction to left ventricular outflow, Takayasu's arteritis
Upper extremity exertion:	Subclavian "steal"
TYPE OF ONSET	
Sudden:	Neurological (seizure disorder); arrhythmia (ventricular tachycardia or fibrillation, Stokes-Adams)
Rapid with premonition:	Vasovagal, neurological (aura)
Gradual:	Hyperventilation, hypoglycemia
POSITION AT ONSET	
Arising:	Orthostatic hypotension
Prolonged standing:	Vasovagal
Any position:	Arrhythmias, neurological, hypoglycemia, hyperventilation
POST-SYNCOPAL CLEARING OF SENSORIUM	
Slow:	Neurological
Rapid:	All others
ASSOCIATED EVENTS	
Incontinence, tongue biting, injury:	Neurological

Modified from Lindenfeld, J. A.: Syncope. *In* Horwitz, L. D., and Groves, B. M. (eds.): Signs and Symptoms in Cardiology. Philadelphia, J. B. Lippincott, 1985, 506 pp.

prolongation (see p. 685). A family history of epilepsy is positive in approximately 4 per cent of patients with convulsive disorders. A history of syncope associated with progressive intensification of cyanosis in an infant or child with cyanotic congenital heart disease is likely to be due to cerebral anoxia as a consequence of an increase in the right-to-left shunt, secondary to an increase in the obstruction to right ventricular outflow or a reduction in systemic vascular resistance (see p. 884). A history of syncope during childhood suggests the possibility of a cardiovascular anomaly obstructing left ventricular outflow—valvular, supravalvular, or subvalvular aortic stenosis. In patients with hypertrophic cardiomyopathy, syncope may be post-tussive and occurs characteristically in the erect position, when arising suddenly, after standing erect for long periods, and during or immediately after cessation of exertion.

Patients with syncope secondary to orthostatic hypotension may have a history of drug therapy for hypertension or of abnormalities of autonomic function, such as impotence, disturbances of sphincter function, peripheral neuropathy, and anhidrosis (see p. 865). When syncope is secondary to hypovolemia, there is often a history of melena, anemia, menorrhagia, or treatment with anticoagulants. Syncope due to cerebrovascular insufficiency is frequently associated with a history of unilateral blindness, weakness, paresthesias, or memory defects.

PALPITATION

This common symptom is defined as an unpleasant awareness of the forceful or rapid beating of the heart. It may be brought about by a variety of disorders involving changes in cardiac rhythm or rate, including all forms of tachycardia, ectopic beats, compensatory pauses, augmented stroke volume due to valvular regurgitation, hyperkinetic (high cardiac output) states, and the sudden onset of bradycardia. In the case of premature contractions the patient is more commonly aware of the postextrasystolic beat than of the premature beat itself, and it appears that it is the increased motion of the heart within the chest that is perceived. This explains why palpitation is not a characteristic feature of aortic or pulmonic stenosis or of severe systemic or pulmonary hypertension, conditions characterized by an increased force of cardiac contraction.

When episodes of palpitation last for an instant, they are described as "skipped beats" or a "flopping sensation" in the chest and most commonly are due to extrasystoles. On the other hand, the sensation that the heart has "stopped beating" often correlates with the compensatory pause following a premature contraction.

DIFFERENTIAL DIAGNOSIS. Palpitation characterized by a slow heart rate may be due to atrioventricular block or sinus node disease. When palpitation begins and ends abruptly, it is often due to a paroxysmal tachycardia such as paroxysmal atrial or junctional tachycardia, atrial flutter, or atrial fibrillation, whereas a gradual onset and cessation of the attack suggest sinus tachycardia and/or an anxiety state. A history of chaotic, rapid heart action suggests the diagnosis of atrial fibrillation; fleeting and repetitive palpitation suggests multiple ectopic beats. A history of multiple paroxysms of tachycardia followed by palpitation that occurs only with effort or excitement suggests paroxysmal atrial fibrillation that has become permanent—the palpitation being experienced only when the ventricular rate rises.

Some patients have taken their pulse during palpitation or have asked a companion to do so. A regular rate between 100 and 140 beats/min suggests sinus tachycardia, a regular rate of approximately 150 beats/min suggests atrial flutter, and a regular rate exceeding 160 beats/min suggests paroxysmal supraventricular tachycardia. As an adjunct to the history, it may be possible to ascertain the rhythm responsible for the palpitation by tapping the finger on the patient's chest in a variety of rhythms and asking the patient to identify the pattern that most closely resembles the abnormal feeling. Alternatively, patients can be asked to reproduce the arrhythmia by tapping their fingers on a tabletop at the rate and rhythm they perceived during palpitation. As described on p. 640, these maneuvers may provide important clues to the etiology of the responsible arrhythmia.

A history of palpitation during strenuous physical activity is normal, whereas palpitation during mild exertion suggests the presence of heart failure, atrial fibrillation, anemia, or thyrotoxicosis, or that the individual is severely "out of condition." A feeling of forceful heart action accompanied by throbbing in the neck suggests aortic regurgitation. When palpitation can be relieved suddenly by stooping, breath-holding, or induced gagging or vomiting, i.e., by vagal maneuvers, the diagnosis of paroxysmal supraventricular tachycardia is suggested. A history of syncope *following* an episode of palpitation suggests either asystole or severe bradycardia following the termination of a tachyarrhythmia or a Stokes-Adams attack. A history of palpitation associated with anxiety, a lump in the throat, dizziness, and tingling in the hands and face suggests sinus tachycardia accompanying an anxiety state with hyperventilation. Palpitation followed by angina suggests that myocardial ischemia has been precipitated by increased oxygen demands induced by the rapid heart rate.

A directed history is also useful (Table 1–5). Is there a history of cocaine or amphetamine abuse? Thyrotoxicosis? Anemia? Do the palpitations occur after heavy cigarette smoking or caffeine ingestion? Is there a family history of syncope, arrhythmia, or sudden death?

In many individuals no obvious cause for palpitation emerges despite careful work-up, including a correlation between episodes of palpitation with a simultaneously recorded ambulatory electrocardiogram (see p. 578) or an electrocardiogram recorded by transtelephonic transmission. Anxiety is responsible for the symptom in many such patients, some of whom are known to have heart disease and may be receiving a vasodilator for the treatment of hypertension or nifedipine for the treatment of myocardial ischemia. In these patients palpitation may be due to postural hypotension resulting in reflex cardiac acceleration.

Edema

LOCALIZATION. This is helpful in elucidating the etiology of edema.[42,43] Thus a history of edema of the legs that is most pronounced in the evening is characteristic of heart failure or bilateral chronic venous insufficiency. Inability to fit the feet into shoes is a common early complaint. In most patients any visible edema of both lower extremities is preceded by a weight gain of at least 7 to 10 lb. Cardiac edema is generally symmetrical. As it progresses, it usually ascends to involve the legs, thighs, genitalia, and abdominal wall. In patients with heart failure who are confined largely to bed, the edematous fluid localizes particularly in the sacral area. Edema affecting both the abdomen and the legs is observed in heart failure and hepatic cirrhosis. Edema may be generalized (anasarca) in the nephrotic syndrome, severe heart failure, and hepatic cirrhosis. A history of edema around the eyes and face is characteristic of the nephrotic syndrome, acute glomerulonephritis, angioneurotic edema, hypoproteinemia, and myxedema. A history of edema limited to the face, neck, and upper arms may be associated with obstruction of the superior vena cava, most commonly by carcinoma of the lung, lymphoma, or aneurysm of the aortic arch. A history of edema restricted to one extremity is usually due to venous thrombosis or lymphatic blockage of that extremity.

ACCOMPANYING SYMPTOMS. A history of dyspnea asso-

TABLE 1–5 ITEMS TO BE COVERED IN HISTORY OF PATIENT WITH PALPITATION

DOES THE PALPITATION OCCUR:	IF SO, SUSPECT:
As isolated "jumps" or "skips"?	Extrasystoles
In attacks, known to be of abrupt beginning, with a heart rate of 120 beats per minute or over, with regular or irregular rhythm?	Paroxysmal rapid heart action
Independent of exercise or excitement adequate to account for the symptom?	Atrial fibrillation, atrial flutter, thyrotoxicosis, anemia, febrile states, hypoglycemia, anxiety state
In attacks developing rapidly though not absolutely abruptly, unrelated to exertion or excitement?	Hemorrhage, hypoglycemia, tumor of the adrenal medulla
In conjunction with the taking of drugs?	Tobacco, coffee, tea, alcohol, epinephrine, ephedrine, aminophylline, atropine, thyroid extract, monoamine oxidase inhibitors
On standing?	Postural hypotension
In middle-aged women, in conjunction with flushes and sweats?	Menopausal syndrome
When the rate is known to be normal and the rhythm regular?	Anxiety state

From Goldman, L., and Braunwald, E.: Chest discomfort and palpitation. *In* Isselbacher, K. J., Braunwald, E., et al. (eds.): Harrison's Principles of Internal Medicine. 13th ed. New York, McGraw-Hill, 1994.

ciated with edema is most frequently due to heart failure, but may also be observed in patients with large bilateral pleural effusions, elevation of the diaphragm due to ascites, angioneurotic edema with laryngeal involvement, and pulmonary embolism. When dyspnea precedes edema, the underlying disorder is usually left ventricular dysfunction, mitral stenosis, or chronic lung disease with cor pulmonale. A history of jaundice suggests that edema may be of hepatic origin, whereas edema associated with a history of ulceration and pigmentation of the skin of the legs is most commonly due to chronic venous insufficiency or postphlebitic syndrome. When cardiac edema is not associated with orthopnea, it may be due to tricuspid stenosis or regurgitation or constrictive pericarditis; in these conditions edema is not always most prominent in the lower extremities but may be generalized and may even involve the face. A history of leg edema after prolonged sitting (particularly in the elderly in wheelchairs) may be due to stasis and not be associated with disease at all.

A history of ascites preceding edema suggests cirrhosis, whereas a history of ascites following edema suggests cardiac or renal disease. Angioneurotic edema occurs intermittently, particularly after emotional stress or eating certain foods. Idiopathic cyclic edema is associated with menstruation. A history of edema on prolonged standing is observed in patients with chronic venous insufficiency.

Cough

Cough, one of the most frequent of all cardiorespiratory symptoms, may be defined as an explosive expiration that provides a means of clearing the tracheobronchial tree of secretions and foreign bodies.[44–46] It can be caused by a variety of infectious, neoplastic, or allergic disorders of the lungs and tracheobronchial tree. Cardiovascular disorders most frequently responsible for cough include those that lead to pulmonary venous hypertension, interstitial and alveolar pulmonary edema, pulmonary infarction, and compression of the tracheobronchial tree (aortic aneurysm). Cough due to pulmonary venous hypertension secondary to left ventricular failure or mitral stenosis tends to be dry, irritating, spasmodic, and nocturnal. When cough accompanies exertional dyspnea, it suggests either chronic obstructive lung disease or heart failure, whereas in a patient with a history of allergy and/or wheezing, cough is often a concomitant of bronchial asthma. A history of cough associated with expectoration for months or years occurs in chronic obstructive lung disease and/or chronic bronchitis.

The character of the sputum may be helpful in the differential diagnosis. Thus, a cough producing frothy, pink-tinged sputum occurs in pulmonary edema; clear, white, mucoid sputum suggests viral infection or longstanding bronchial irritation; thick, yellowish sputum suggests an infectious cause; rusty sputum suggests pneumococcal pneumonia; blood-streaked sputum suggests tuberculosis, bronchiectasis, carcinoma of the lung, or pulmonary infarction.

A history of a combination of cough with hoarseness without upper respiratory disease may be due to pressure of a greatly enlarged left atrium on an enlarged pulmonary artery compressing the recurrent laryngeal nerve.

HEMOPTYSIS

The expectoration of blood or of sputum, either streaked or grossly contaminated with blood, may be due to (1) escape of red cells into the alveoli from congested vessels in the lungs (acute pulmonary edema); (2) rupture of dilated endobronchial vessels that form collateral channels between the pulmonary and bronchial venous systems (mitral stenosis); (3) necrosis and hemorrhage into the alveoli (pulmonary infarction); (4) ulceration of the bronchial mucosa or the slough of a caseous lesion (tuberculosis); minor damage to the tracheobronchial mucosa, produced by excessive coughing of any cause, can result in mild hemoptysis; (5) vascular invasion (carcinoma of the lung); or (6) necrosis of the mucosa with rupture of pulmonary-bronchial venous connections (bronchiectasis).

The history is often decisive in pinpointing the etiology of hemoptysis.[46] Recurrent episodes of minor bleeding are observed in patients with chronic bronchitis, bronchiectasis, tuberculosis, and mitral stenosis. Rarely, these conditions result in the expectoration of large quantities of blood, i.e., more than one-half cup. Massive hemoptysis may also be due to rupture of a pulmonary arteriovenous fistula; exsanguinating hemoptysis may occur with rupture of an aortic aneurysm into the bronchopulmonary tree.[47,48]

Hemoptysis associated with a history of expectoration of clear, gray sputum suggests chronic obstructive lung disease and of yellowish-green sputum, pulmonary infection. Hemoptysis associated with shortness of breath suggests mitral stenosis; in this condition the hemoptysis is often precipitated by sudden elevations in left atrial pressure during effort or pregnancy and is attributable to rupture of small pulmonary or bronchopulmonary anastomosing veins. Blood-tinged sputum in patients with mitral stenosis may also be due to transient pulmonary edema; in these circumstances it is usually associated with severe dyspnea.

A history of hemoptysis associated with acute pleuritic chest pain suggests pulmonary embolism with infarction. Recurrent hemoptysis in a young, otherwise asymptomatic woman favors the diagnosis of bronchial adenoma. Hemoptysis associated with congenital heart disease and cyanosis suggests Eisenmenger syndrome (see p. 799). A history of recurrent hemoptysis with chronic excessive sputum production suggests the diagnosis of bronchiectasis. Hemoptysis associated with the production of putrid sputum occurs in lung abscess, whereas hemoptysis associated with weight loss and anorexia in a male smoker suggests carcinoma of the lung. When blunt trauma to the chest is followed by hemoptysis, lung contusion is the probable cause.

A history of drug ingestion may be helpful in elucidating the etiology of hemoptysis; e.g., anticoagulants and immunosuppressive drugs can cause bleeding. A history of ingestion of contraceptive pills may be a risk factor for the development of deep vein thrombosis and subsequent pulmonary embolism and infarction.

FATIGUE AND OTHER SYMPTOMS

Cardiovascular disorders can cause symptoms emanating from every organ system. Several of these are mentioned here primarily to point out how detailed the history should be in providing a comprehensive evaluation of a patient suspected of having cardiovascular disease; fuller discussions are found elsewhere in this text.

FATIGUE. This is among the most common symptoms in patients with impaired cardiovascular function. However, it is also one of the most nonspecific of all symptoms in clinical medicine; in patients with impaired systemic circulation as a consequence of a depressed cardiac output, it may be associated with muscular weakness. In other patients with heart disease, fatigue may be caused by drugs, such as beta-adrenoceptor blocking agents. It may be the result of excessive blood pressure reduction in patients treated too vigorously for hypertension or heart failure. In patients with heart failure, fatigue may also be caused by excessive diuresis and by diuretic-induced hypokalemia. Extreme fatigue sometimes precedes or accompanies acute myocardial infarction.[20]

OTHER SYMPTOMS. *Nocturia* is a common early complaint in patients with congestive heart failure. *Anorexia,* abdominal fullness, right upper quadrant discomfort, weight loss, and cachexia are symptoms of advanced heart failure (see p. 452). Anorexia, *nausea, vomiting,* and *visual changes* are important signs of digitalis intoxication (see p. 484). Nausea and vomiting occur frequently in patients with acute myocardial infarction. Hoarseness may be caused by compression of the recurrent laryngeal nerve by an aortic aneurysm, a dilated pulmonary artery, or a greatly enlarged left atrium. A history of *fever* and *chills* is common in patients with infective endocarditis (see p. 1084).

The aforementioned symptoms are examples of the wide variety of symptoms that are not obviously associated with abnormalities of the cardiovascular system but that can be of critical importance in differential diagnosis when they are elicited in patients known to have or suspected of having heart disease. They serve to reemphasize that the physician whose responsibility it is to care for patients with heart disease must be first and foremost a broadly based clinician.

THE HISTORY IN SPECIFIC FORMS OF HEART DISEASE

Just as the history is of central importance in determining whether or not a specific symptom is caused by heart disease, it is equally valuable in elucidating the etiology of recognized heart disease. A few examples are given below; considerably greater detail is provided in later chapters that deal with each specific disease entity.

Heart Disease in Infancy and Childhood

The history is particularly helpful in establishing a diagnosis of congenital heart disease. In view of the familial incidence of certain congenital malformations (Chaps. 29 and 49), a history of congenital heart disease, cyanosis, or heart murmur in the family should be ascertained. Rubella in the first 2 months of pregnancy is associated with a number of congenital cardiac malformations (patent ductus arteriosus, atrial and ventricular septal defect, tetralogy of Fallot, and supravalvular aortic stenosis [see p. 919]). A maternal viral illness in the last trimester of pregnancy may be responsible for neonatal myocarditis. Syncope on exertion in a child with congenital heart disease suggests a lesion in which the cardiac output is fixed, such as aortic or pulmonic stenosis. Exertional angina in a child suggests severe aortic stenosis, pulmonary stenosis, primary pulmonary hypertension, or anomalous origin of the left coronary artery. A history of syncope or faintness with straining and associated with cyanosis suggests tetralogy of Fallot (see p. 929).

In infants or children with cardiac murmurs, it is important to ascertain as precisely as possible when the murmur was first detected. Murmurs due to either aortic or pulmonic stenosis are usually audible within the first 48 hours of life, whereas those produced by a ventricular septal defect are usually apparent a few days or weeks later. On the other hand, the murmur produced by an atrial septal defect often is not heard until age 2 to 3 months.

Frequent episodes of pneumonia early in infancy suggest a large left-to-right shunt, and a history of excessive diaphoresis occurs in left ventricular failure, most commonly due to ventricular septal defect in this age group. A history of squatting is most frequently associated with tetralogy of Fallot or tricuspid atresia (see p. 932). Dysphagia in early infancy suggests the presence of an aortic arch anomaly such as double aortic arch or an anomalous origin of the right subclavian artery passing behind the esophagus. A history of headaches, weakness of the legs, and intermittent claudication is compatible with the diagnosis of coarctation of the aorta (see p. 965). Weakness or lack of coordination in a child with heart disease suggests cardiomyopathy associated with Friedreich's ataxia or muscular dystrophy (Chap. 60). Recurrent bleeding from the nose, lips, or mouth, associated with dizziness and visual disturbances, and a family history of bleeding in a cyanotic child suggest hereditary hemorrhagic telangiectasia (Osler-Weber-Rendu disease) with pulmonary arteriovenous fistula(s) (see p. 461). A cerebrovascular accident in a cyanotic patient may be due to cerebral thrombosis or abscess or paradoxical embolization (see p. 885).

MYOCARDITIS AND CARDIOMYOPATHY

Rheumatic fever (Chap. 55) is suggested by a history of sore throat followed by symptoms including rash and chorea (St. Vitus dance). This is manifested as a period of twitching or clumsiness for a few months in childhood, as well as by frequent episodes of epistaxis and growing pains, i.e., nocturnal pains in the legs. In patients suspected of having myocarditis or cardiomyopathy, a history of Raynaud's phenomenon, dysphagia, or tight skin suggests scleroderma (see p. 1781). A history of dyspnea following an influenza-like illness with myalgia suggests acute myocarditis. Pain in the hip or lower back that awakens the patient in the morning and is followed by morning back stiffness suggests rheumatoid spondylitis, which is often associated with aortic valve disease (see p. 1780). *Carcinoid heart disease* is associated with a history of diarrhea, bronchospasm, and flushing of the upper chest and head (see p. 1434). A history of diabetes, particularly if resistant to insulin and associated with bronzing of the skin, suggests *hemochromatosis* (see p. 1790), which may be associated with heart failure due to cardiac infiltration. *Amyloid heart disease* (see p. 1427) is often associated with a history of postural hypotension and peripheral neuropathy. *Hypertrophic cardiomyopathy* (see p. 1414) is often associated with a family history of this condition and sometimes with a family history of sudden death. The characteristic symptoms are angina, dyspnea, and syncope, which are often intensified paradoxically by digitalis and which occur during or immediately after exercise.

HIGH-OUTPUT HEART FAILURE AND COR PULMONALE

Patients with symptoms of heart failure (breathlessness and excess fluid accumulation) with warm extremities often have *high-output heart failure* (see p. 460). They should be questioned about a history of anemia and of its common causes and accompaniments, such as menorrhagia, melena, peptic ulcer, hemorrhoids, sickle cell disease, and the neurological manifestations of vitamin B_{12} deficiency. Also, in such patients an attempt should be made to elicit a history of thyrotoxicosis (see pp. 461 and 1894) (weight loss, polyphagia, diarrhea, diaphoresis, heat intolerance, nervousness, breathlessness, muscle weakness, and goiter). Patients with beriberi heart disease responsible for high-output heart failure often have a history characteristic of peripheral neuritis, alcoholism, poor eating habits, fad diets, or upper gastrointestinal surgery.

Patients with chronic cor pulmonale (see Chap. 47) frequently have a history of smoking, chronic cough and sputum production, dyspnea, and wheezing relieved by bronchodilators. Alternatively, they may have a history of pulmonary emboli, phlebitis, and the sudden development of dyspnea at rest with palpitations, pleuritic chest pain, and, in the case of massive infarction, syncope.

PERICARDITIS AND ENDOCARDITIS

In patients in whom *pericarditis* or *cardiac tamponade* is suspected (Chap. 43), an attempt should be made to elicit a history of chest trauma, a recent viral infection, recent cardiac surgery, neoplastic disease of the chest with or without extensive radiation therapy, myxedema, scleroderma, tuberculosis, or contact with tuberculous patients. The *sequence of development* of abdominal swelling, ankle edema, and dyspnea should be determined, since in patients with chronic constrictive pericarditis, ascites often precedes edema, which in turn usually precedes exertional dyspnea. A history of joint symptoms with a face rash suggests the possibility of systemic lupus erythematosus (SLE), an important cause of pericarditis, and it should be recalled that procainamide, hydralazine, and isoniazid can produce an SLE-like syndrome (see p. 604).

The diagnosis of infective endocarditis is suggested by a history of fever, severe night sweats, anorexia, and weight loss and embolic phenomena expressed as hematuria, back pain, petechiae, tender finger pads, and a cerebrovascular accident (see p. 1084).

Drug-Induced Heart Disease

Since a wide variety of cardiac abnormalities can be induced by drugs,[49] a meticulous history of drug intake is of great importance. Table 1–6 summarizes the major drugs responsible for various cardiovascular manifestations.

Catecholamines, whether administered exogenously or secreted by a pheochromocytoma (see p. 1897), may produce myocarditis and arrhythmias. *Digitalis glycosides* can be responsible for a variety of tachyarrhythmias and bradyarrhythmias as well as gastrointestinal, visual, and central nervous system disturbances (see p. 499). *Quinidine* may cause Q-T prolongation, ventricular tachycardia of the torsades de pointes variety, syncope, and sudden death, presumably due to ventricular fibrillation (see p. 602). Paradoxically, the administration of antiarrhythmic drugs is one of the major causes of serious cardiac arrhythmias (see p. 600).

Disopyramide (see p. 604), *beta-adrenoceptor blockers* (see p. 610), and the calcium channel antagonists *diltiazem* and *verapamil* (see p. 616) may depress ventricular performance, and in patients with ventricular dysfunction these drugs may intensify heart failure. *Alcohol* is also a potent myocardial depressant and may be responsible for the development of cardiomyopathy (see p. 1412), arrhythmias, and sudden death. *Tricyclic antidepressants* may cause orthostatic hypotension and arrhythmias. *Lithium,* also used in the treatment of psychiatric disorders, can aggravate preexisting cardiac arrhythmias, particularly in patients with heart failure in whom the renal clearance of this ion is impaired. *Cocaine* can cause coronary spasm with resultant myocardial ischemia, myocardial infarction, and sudden death.[50,51]

The *anthracycline compounds* doxorubicin (Adriamycin) and daunorubicin, which are widely used because of their broad spectrum of activity against various tumors, may cause or intensify left ventricular failure, arrhythmias, myocarditis, and pericarditis (see p. 1800). *Cyclophosphamide,* an antineoplastic alkylating agent, may also cause

TABLE 1–6 CARDIOVASCULAR MANIFESTATIONS OF ADVERSE REACTIONS TO DRUGS

Acute Chest Pain
(nonischemic)
Bleomycin
Angina Exacerbation
Alpha blockers
Beta-blocker withdrawal
Ergotamine
Excessive thyroxine
Hydralazine
Methysergide
Minoxidil
Nifedipine
Oxytocin
Vasopressin

Arrhythmias
Adriamycin
Antiarrhythmic drugs
Astemizole
Atropine
Anticholinesterases
Beta blockers
Daunorubicin
Digitalis
Emetine
Erythromycin
Guanethidine
Lithium
Papaverine
Phenothiazines, particularly thioridazine
Sympathomimetics
Terfenadine
Theophylline
Thyroid hormone
Tricyclic antidepressants
Verapamil
AV Block
Clonidine
Methyldopa
Verapamil
Cardiomyopathy
Adriamycin
Daunorubicin
Emetine
Lithium
Phenothiazines
Sulfonamides
Sympathomimetics

Fluid Retention/Congestive Heart Failure/Edema
Beta blockers
Calcium blockers
Carbenoxolone
Diazoxide
Estrogens
Indomethacin
Mannitol
Minoxidil
Phenylbutazone
Steroids
Verapamil
Hypotension (see also Arrhythmias)
Amiodarone (perioperative)
Calcium channel blockers, e.g., nifedipine
Citrated blood
Diuretics
Interleukin-2
Levodopa
Morphine
Nitroglycerin
Phenothiazines
Protamine
Quinidine

Hypertension
Clonidine withdrawal
Corticotropin
Cyclosporine
Glucocorticoids
Monoamine oxidase inhibitors with sympathomimetics
NSAIDs (some)
Oral contraceptives
Sympathomimetics
Tricyclic antidepressants with sympathomimetics
Pericarditis
Emetine
Hydralazine
Methysergide
Procainamide
Pericardial Effusion
Minoxidil
Thromboembolism
Oral contraceptives

From Wood, A. J.: Adverse reactions to drugs. *In* Isselbacher, K. J., Braunwald, E., et al. (eds.): Harrison's Principles of Internal Medicine. 13th ed. New York, McGraw-Hill, 1994.

TABLE 1–7 A COMPARISON OF THREE METHODS OF ASSESSING CARDIOVASCULAR DISABILITY

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 eight steps; carry objects that weigh 80 lb; do outdoor work (shovel snow, spade soil); do recreational activities (skiing, basketball, squash, handball, jog/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, palpitation, 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 more than two blocks on the level and climbing more than one flight of ordinary stairs at a normal pace and in normal conditions.	Patients can perform to completion any activity requiring ≤ 5 metabolic equivalents, e.g., have sexual intercourse without stopping, garden, rake, weed, roller skate, dance fox trot, walk at 4 mph on level ground, but cannot and do not perform to completion activities requiring ≥ 7 metabolic equivalents.
III	Patients with cardiac disease resulting in marked limitation of physical activity. They are comfortable at rest. Less than ordinary physical activity causes fatigue, palpitation, dyspnea, or anginal pain.	Marked limitation of ordinary physical activity. Walking one to two blocks on the level and climbing more than one flight in normal conditions.	Patients can perform to completion any activity requiring ≤ 2 metabolic equivalents, e.g., shower without stopping, strip and make bed, clean windows, walk 2.5 mph, bowl, play golf, dress without stopping, but cannot and do not perform to completion any activities requiring ≥ 5 metabolic equivalents.
IV	Patient 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.	Patients cannot or do not perform to completion activities requiring ≥ 2 metabolic equivalents. *Cannot* carry out activities listed above (Specific Activity Scale, Class III).

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left ventricular dysfunction, while 5-fluorouracil and its derivatives (see p. 1803) may be responsible for angina secondary to coronary spasm (see p. 1189). Radiation therapy to the chest may cause acute and chronic pericarditis (see p. 1799), pancarditis, or coronary artery disease; further, it may enhance the aforementioned cardiotoxic effects of the anthracyclines.

Assessing Cardiovascular Disability

(Table 1–7)

One of the greatest values of the history is in categorizing the degree of cardiovascular disability, so that a patient's status can be followed over time, the effects of a therapeutic intervention assessed, and patients compared with one another. The Criteria Committee of the New York Heart Association has provided a widely used classification that relates functional activity to the ability to carry out "ordinary" activity.[52] The term "ordinary," of course, is subject to widely varying interpretation, as are terms such as "undue fatigue" that are used in this classification, and this has limited its accuracy and reproducibility. More recently, this Heart Association changed its evaluation from functional activity to a broader one, called Cardiac Status, which takes account of symptoms and other data gathered from the patient.[52] Cardiac status is classified as: (1) uncompromised, (2) slightly compromised, (3) moderately compromised, and (4) severely compromised.

Somewhat more detailed and specific criteria were provided by the Canadian Cardiovascular Society,[53] but this classification is limited to patients with angina pectoris. Goldman et al.[54] developed a specific activity scale in which classification is based on the estimated metabolic cost of various activities. This scale appears to be more reproducible and to be a better predictor of exercise tolerance than either the New York Heart Association Classification or the Canadian Cardiovascular Society Criteria.

A key element of the history is to determine whether the patient's disability is stable or progressive. A useful way to accomplish this is to inquire whether a specific task which now causes symptoms, e.g., dyspnea after climbing two flights of stairs, did so 3, 6, and 12 months previously. Precise questioning on this point is important since a gradual reduction of ordinary activity as heart disease progresses may lead to an underestimation of the apparent degree of disability.[55]

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