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The History and Physical Examination

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The History and Physical Examination

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The ability to determine whether disease is present or absent—and how that patient should be treated—is the ultimate goal for clinicians evaluating patients with suspected heart disease. Despite the number of diagnostic tests available, never has the importance of a careful history and physical examination been greater. Opportunities for error in judgment are abundant, and screening patients for coronary risk using a broad and unfocused panel of laboratory and noninvasive tests can lead to incorrect diagnoses and unnecessary testing. Selection of the most appropriate test and therapeutic approach for each patient is based on a skillfully performed history and physical examination. Furthermore, interpretation of any test results is based on the prior probability of disease, which again is based on the history and physical. While entire texts have been written on cardiac history and physical examination, this chapter specifically focuses on features of the cardiac history and the cardiovascular physical examination that help discern the presence or absence of heart disease.

THE CONCEPT OF PRIOR PROBABILITY

The history and physical examination should allow the clinician to establish the prior probability of heart disease—that is, the likelihood that the symptoms reported by the patient result from heart disease. A reasonable goal is to establish a patient's risk of heart disease as "low," "intermediate," or "high." One demonstration of this principle in clinical medicine is the assessment of patients with chest pain, in which the power of exercise stress testing to accurately diagnose coronary heart disease (CHD) depends on the prior probability of disease. In patients with a very low risk of CHD based on clinical findings, exercise stress testing resulted in a large number of false-positive test results. Because up to 15% of exercise stress tests produce positive results in individuals without CHD, use of this test in a low-risk population can result in an adverse ratio of false-positive to true-positive test results and unnecessary cardiac catheterizations. Conversely, in patients with a very high risk of CHD based on clinical findings, exercise stress testing can result in false-negative test results—an equally undesirable outcome, because patients with significant coronary artery disease (CAD) and their physicians may be falsely reassured that no further evaluation or treatment is necessary.

Emphasis is increasing on quantifying prior probability to an even greater degree using various mathematical models. This is a useful approach in teaching and may be clinically feasible in some diseases. However, for the majority of patients with suspected heart disease, categorizing risk as low, intermediate, and high is appropriate, reproducible, and feasible in a busy clinical practice. Therefore, obtaining the history and physical examination represents a key step before any testing, to minimize use of inappropriate diagnostic procedures.

THE HISTORY

A wealth of information is available to clinicians who carefully assess the patient's history. Key components are assessment of the chief complaint; careful questioning for related, often subtle symptoms that may further define the chief complaint; and determination of other factors that help categorize the likelihood of disease. Major symptoms of heart patients include chest discomfort, dyspnea, palpitations, and syncope or presyncope.

Chest Discomfort

Determining whether chest discomfort results from a cardiac cause is often a challenge. The most common cause of chest discomfort is myocardial ischemia, which produces angina pectoris. Many causes of angina exist, and the differential diagnosis for chest discomfort is extensive (Box 1-1). Angina that is reproducible and constant in frequency and severity is often referred to as *stable angina*. For the purposes of this chapter, stable angina is a condition that occurs when CAD is present and coronary blood flow cannot be increased to accommodate for increased myocardial demand. However, as discussed in Chapters 12 through 14, there are many causes of myocardial ischemia, including fixed coronary artery stenoses and endothelial dysfunction, which leads to reduced vasodilatory capacity.

A description of chest discomfort can help establish whether the pain is angina or of another origin. First, characterization of the quality and location of the discomfort is essential (Fig. 1-1). Chest discomfort because of myocardial ischemia may be described as pain, a tightness, a heaviness, or simply an uncomfortable and difficult-to-describe feeling. The discomfort can be localized to the mid-chest or epigastric area or may be characterized as pain in related areas, including the left arm, both arms, the jaw, or the back. The radiation of chest discomfort to any of these areas increases the likelihood of the discomfort being angina. Second, the duration of discomfort is important, because chest discomfort due to cardiac causes generally lasts minutes. Therefore, pain of very short duration ("seconds" or "moments"), regardless of how typical it may be of angina, is less likely to be of cardiac origin. Likewise, pain that lasts for hours, on many occasions, in the absence of objective evidence of myocardial infarction (MI), is not likely to be of coronary origin. Third, the presence of accompanying symptoms should be considered. Chest discomfort may be accompanied by other symptoms (including dyspnea, diaphoresis, or nausea), any of which increase the likelihood that the pain is cardiac in origin. However, the presence of accompanying symptoms is not needed to define the discomfort as angina. Fourth, factors that precipitate or relieve the discomfort should be evaluated. Angina typically occurs during physical exertion, during emotional stress, or in

Box 1-1 Differential Diagnosis of Chest Discomfort

Cardiovascular

Ischemic
- Hyperthyroidism
- Tachycardia (e.g., atrial fibrillation)
- Coronary spasm
- Coronary atherosclerosis (angina pectoris)
- Acute coronary syndrome
- Aortic stenosis
- Hypertrophic cardiomyopathy
- Aortic regurgitation
- Mitral regurgitation
- Severe systemic hypertension
- Severe right ventricular/pulmonary hypertension
- Severe anemia/hypoxia

Nonischemic
- Aortic dissection
- Pericarditis
- Mitral valve prolapse syndrome: autonomic dysfunction

Gastrointestinal
- Gastroesophageal reflux disease
- Esophageal spasm
- Esophageal rupture
- Hiatal hernia
- Cholecystitis

Pulmonary
- Pulmonary embolus
- Pneumothorax
- Pneumonia
- Chronic obstructive pulmonary disease
- Pleurisy

Neuromusculoskeletal
- Thoracic outlet syndrome
- Degenerative joint disease of the cervical or thoracic spine
- Costochondritis
- Herpes zoster

Psychogenic
- Anxiety
- Depression
- Cardiac psychosis
- Self-gain

other circumstances of increased myocardial oxygen demand. When exercise precipitates chest discomfort, relief after cessation of exercise substantiates the diagnosis of angina. Sublingual nitroglycerin also relieves angina, generally over a period of minutes. Instant relief or relief after longer periods lessens the likelihood that the chest discomfort was angina.

Although the presence of symptoms during exertion is important in assessing CHD risk, individuals, especially sedentary ones, may have angina-like symptoms that are not related to exertion. These include postprandial and nocturnal angina or angina that occurs while the individual is at rest. As described herein, "rest-induced angina," or the new onset of angina, connotes a pathophysiology different from effort-induced angina. Angina can also occur in persons with fixed CAD and increased myocardial oxygen demand due to anemia, hyperthyroidism, or similar conditions (Box 1-2). Angina occurring at rest, or with minimal exertion, may denote a different pathophysiology, one involving platelet aggregation and clinically termed "unstable angina" or "acute coronary syndrome" (see Chapters 13 and 14).

Patients with heart disease need not present with chest pain at all. Anginal equivalents include dyspnea during exertion, abdominal discomfort, fatigue, or decreased exercise tolerance. Clinicians must be alert to and specifically ask about these symptoms. Often, a patient's family member or spouse notices subtle changes in endurance in the patient or that the individual no longer performs functions that require substantial physical effort. Sometimes patients may be unable to exert themselves due to comorbidities. For instance, the symptoms of myocardial ischemia may be absent in patients with severe peripheral vascular disease who have limiting claudication. One should also be attuned to subtle or absent symptoms in individuals with diabetes mellitus (including type 1 and type 2 diabetes), a "coronary risk equivalent" as defined by the Framingham Risk Calculator.

When considering the likelihood that CHD accounts for a patient presenting with chest discomfort or any of the aforementioned variants, assessment of the cardiac risk factor profile is important. The Framingham Study first codified the concept of cardiac risk factors, and over time, quantification of risk using these factors has become an increasingly useful tool in clinical medicine. Cardiac risk factors determined by the Framingham Study include a history of cigarette smoking, diabetes mellitus, hypertension, or hypercholesterolemia; a family history of CHD (including MI, sudden cardiac death, and first-degree relatives having undergone coronary revascularization); age; and sex (male). Although an attempt has been made to rank these risk factors, all are important, with a history of diabetes mellitus being perhaps the single most important factor. Subsequently, a much longer list of potential predictors of cardiac risk has been made (Box 1-3). An excellent, easy-to-use model for predicting risk is the Framingham Risk Calculator, as described in the Adult Treatment Panel III guidelines from the National Heart, Lung and Blood Institute (see "Evidence" section).

Symptoms suggestive of vascular disease require special attention. Peripheral vascular disease may mask CHD, because the individual may not be able to exercise sufficiently to provoke angina. A history of stroke, transient ischemic attack, or atheroembolism in any vascular distribution is usually evidence of significant vascular disease. Sexual dysfunction in men is not an uncommon presentation of peripheral vascular disease. The presence of Raynaud's-type symptoms should also be elicited, because such symptoms suggest abnormal vascular tone and function, and increase the risk that CHD is present.

Determining whether the patient has stable or unstable angina is as important as making the diagnosis of angina. Stable angina is important to evaluate and treat, but does not necessitate emergent intervention. Unstable angina, or acute coronary syndrome, however, carries a significant risk of MI or death in the immediate future. The types of symptoms reported by patients with stable and unstable angina differ little, and the risk factors for both are identical. Indeed, the severity of symptoms is not necessarily greater in patients with unstable angina, just as a lack of chest discomfort does not rule out significant CHD. The important distinction between stable and unstable coronary syndromes rests in whether the onset is new or recent and/or progressive (e.g., occurring more frequently or with less

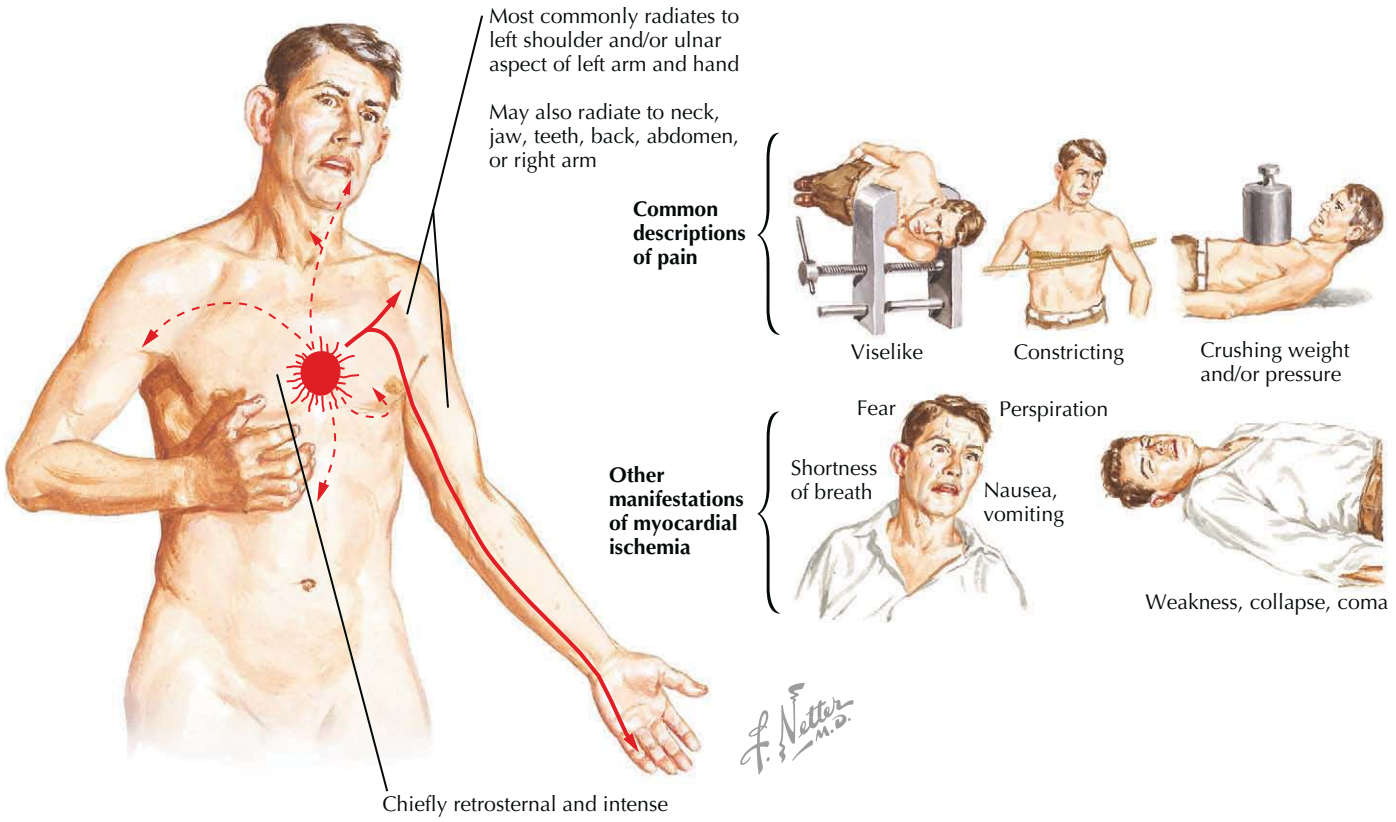

Figure 1-1 *Pain of myocardial ischemia.*

exertion). The initial presentation of angina is, by definition, unstable angina; although for a high percentage of individuals this may merely represent the first recognizable episode of angina. For those with unstable angina, the risk of MI in the near future is markedly increased. Likewise, when the patient experiences angina in response to decreased levels of exertion or when exertional angina has begun to occur at rest, these urgent circumstances require immediate therapy. The treatment of stable angina and acute coronary syndrome is discussed in Chapters 12, 13, and 14. The Canadian Cardiovascular Society Functional Classification of Angina Pectoris is a useful guide for everyday patient assessment (Box 1-4). Categorizing patients according to their class of symptoms is rapid and precise and can be used in follow-up. Class IV describes the typical patient with acute coronary syndrome.

Box 1-2 Conditions that Cause Increased Myocardial Oxygen Demand

- Hyperthyroidism
- Tachycardia of various etiologies
- Hypertension
- Pulmonary embolism
- Pregnancy
- Psychogenic
- Central nervous system stimulants
- Exercise
- Psychological stress
- Fever

Finally, it is important to distinguish those patients who have noncoronary causes of chest discomfort from those with CHD. Patients with gastroesophageal reflux disease (GERD) often present with symptoms that are impossible to distinguish from angina. In numerous studies, GERD is the most common diagnosis in patients who undergo diagnostic testing for angina and are found not to have CHD. The characteristics of the pain can be identical. Because exercise can increase intra-abdominal pressure, GERD may be exacerbated with exercise, especially after meals. Symptoms from GERD can also be relieved with use of sublingual nitroglycerin. GERD can also result in

Box 1-3 Cardiac Risk Factors

- Diabetes
- Smoking
- Hypertension
- High cholesterol
- Hyperlipidemia
- Sedentary lifestyle
- High-fat diet
- Stress
- "Metabolic syndrome"
- Family history of CHD (including history of MI, sudden cardiac death, and first-degree relatives who underwent coronary revascularization)
- Age
- Male sex
- Obesity

CHD, coronary heart disease; MI, myocardial infarction.

Box 1-4 Canadian Cardiovascular Society Classification of Angina Pectoris

- **I** Ordinary physical activity, for example, walking or climbing stairs, does not cause angina; angina occurs with strenuous, rapid, or prolonged exertion at work or recreation.
- **II** Slight limitation of ordinary activity; for example, angina occurs when walking or stair climbing after meals, in cold, in wind, under emotional stress, or only during the few hours after awakening, when walking more than two blocks on the level, or when climbing more than one flight of ordinary stairs at a normal pace and during normal conditions.
- **III** Marked limitation of ordinary activity; for example, angina occurs when walking one or two blocks on the level or when climbing one flight of stairs during normal conditions and at a normal pace.
- **IV** Inability to carry on any physical activity without discomfort; angina syndrome may be present at rest.

From Campeau L. Grading of angina pectoris [letter]. *Circulation.* 1976;54:522–523.

early-morning awakening (as can unstable angina) but tends to awaken individuals 2 to 4 hours after going to sleep, rather than 1 to 2 hours before arising, as is the case with unstable angina. Other causes (see Box 1-1) of angina-like pain can be benign, or suggestive of other high-risk syndromes, such as aortic dissection or pulmonary embolus. Many of these "coronary mimics" can be ruled out by patient history, but others, such as valvular aortic stenosis, can be confirmed or excluded by physical examination. The goal of taking the history is to alert the clinician to entities that can be confirmed or excluded by physical examination, or that necessitate further diagnostic testing.

Dyspnea, Edema, and Ascites

Dyspnea can accompany angina pectoris or it can be an anginal equivalent. Dyspnea can also reflect congestive heart failure (CHF) or occur because of noncardiac causes. The key to understanding the etiology of dyspnea is a clear patient history, which is then confirmed by a targeted physical examination.

Dyspnea during exertion that quickly resolves at rest or with use of nitroglycerin may be a result of myocardial ischemia. It is important to establish the amount of activity necessary to provoke dyspnea, the reproducibility of these symptoms, and the duration of recovery. As with angina, dyspnea as an anginal equivalent or an accompanying symptom tends to occur at a given workload or stress level; dyspnea occurring one day at low levels of exertion but not prompted by vigorous exertion on another day is less likely to be an anginal equivalent.

In patients with CHF, dyspnea generally reflects increased left ventricular (LV) filling pressures (Fig. 1-2). Although most commonly LV systolic dysfunction is the cause of the dyspnea, dyspnea also occurs in individuals with preserved LV systolic function and severe diastolic dysfunction. These two entities present differently, however, and physical examination can

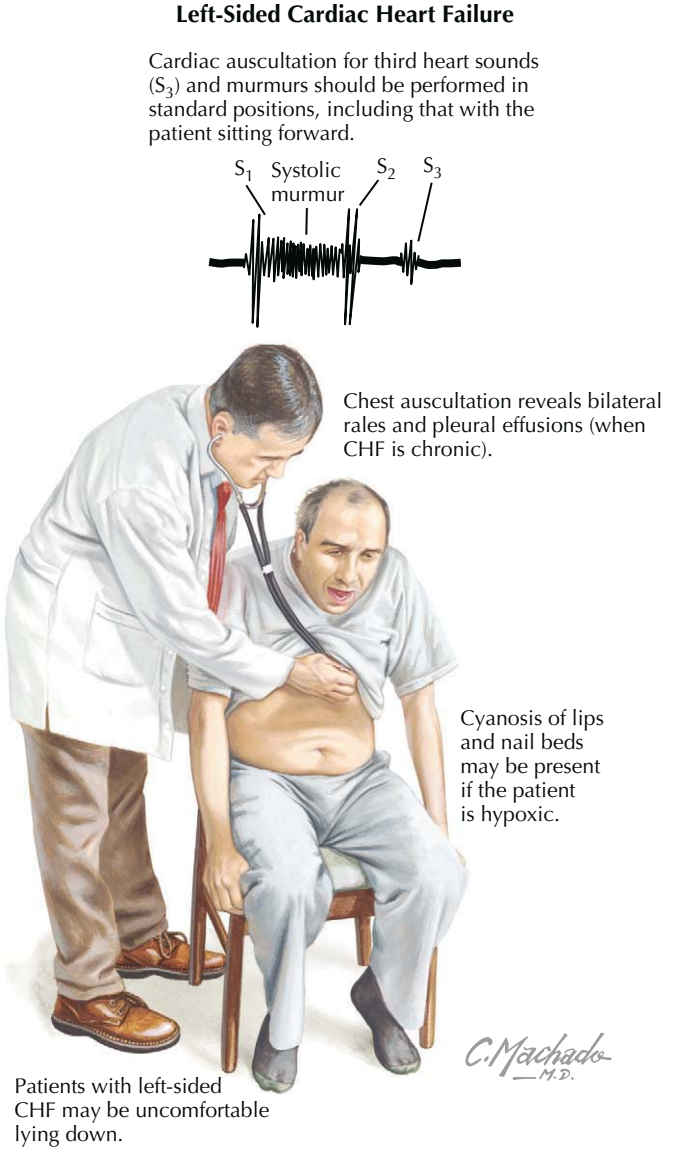

Figure 1-2 *Physical examination.* CHF, congestive heart failure.

distinguish them. With LV systolic dysfunction, dyspnea tends to gradually worsen, and its exacerbation is more variable than that of exertional dyspnea resulting from myocardial ischemia, although both are due to fluctuations in pulmonary arterial volume and left atrial filling pressures. Typically, patients with LV systolic dysfunction do not recover immediately after exercise cessation or use of sublingual nitroglycerin, and the dyspnea may linger for longer periods. Orthopnea, the occurrence of dyspnea when recumbent, or paroxysmal nocturnal edema provides further support for a presumptive diagnosis of LV systolic dysfunction. Patients with LV diastolic dysfunction tend to present abruptly with severe dyspnea that resolves more rapidly in response to diuretic therapy than does dyspnea caused by LV systolic dysfunction. The New York Heart Association (NYHA) Classification for CHF (Table 1-1) is extremely useful in following patients with CHF and provides a simple and rapid means for longitudinal assessment. The NYHA Classification

Table 1-1 Comparison of the ACC/AHA and the NYHA Classifications of Heart Failure

ACC/AHA Stage		*NYHA Functional Class*	
Stage	**Description**	**Class**	**Description**
A	Patients without structural heart disease and without symptoms of heart failure but who are at high risk for the development of heart failure	No comparable functional class	
B	Patients with structural heart disease that is strongly associated with the development of heart failure but who have never shown signs or symptoms of heart failure	I (Mild)	No limitation of physical activity. Ordinary physical activity does not cause undue fatigue, palpitation, or dyspnea.
C	Patients who have current or prior symptoms of heart failure and underlying structural heart disease	II (Mild)	Slight limitation of physical activity. Comfortable at rest, but ordinary physical activity results in fatigue, palpitation, or dyspnea.
		III (Moderate)	Marked limitation of physical activity. Comfortable at rest, but less than ordinary activity causes fatigue, palpitation, or dyspnea.
D	Patients with advanced structural heart disease and symptoms of heart failure at rest despite maximal medical therapy	IV (Severe)	Unable to carry out any physical activity without discomfort. Symptoms of cardiac insufficiency at rest. If any physical activity is undertaken, discomfort is increased.

ACC/AHA, American College of Cardiology/American Heart Association; NYHA, New York Heart Association. NYHA data from the Criteria Committee of the New York Heart Association. *Diseases of the Heart and Blood Vessels: Nomenclature and Criteria for Diagnosis.* Boston: Brown; 1964. ACC/AHA data from ACC/AHA 2005 Guideline Update for the Diagnosis and Management of Chronic Heart Failure in the Adult. *Circulation.* 2005:112:e154–e235.

also correlates well with prognosis. Patients who are NYHA class I have a low risk of death or hospital admission within the following year. In contrast, the annual mortality rate of those with NYHA class IV symptoms exceeds 30%.

As with chest discomfort, the differential diagnosis of dyspnea is broad, encompassing many cardiac and noncardiac causes (Box 1-5). Congenital heart disease, with or without pulmonary hypertension, can cause exertional dyspnea. Patients with significant intra- or extracardiac shunts and irreversible pulmonary hypertension (Eisenmenger's syndrome) are dyspneic during minimal exertion and often at rest. It is also possible to have dyspnea because of acquired valvular heart disease, usually from aortic or mitral valve stenosis or regurgitation. All of these causes should be easily distinguished from CHD or CHF by physical examination. Primary pulmonary causes of dyspnea must be considered, with chronic obstructive pulmonary disease (COPD) and reactive airways disease (asthma) being most common. Again, a careful history for risk factors (e.g., cigarette smoking, industrial exposure, allergens) associated with these entities and an accurate physical examination should distinguish primary pulmonary causes from dyspnea due to CHD or CHF.

Peripheral edema and ascites are physical examination findings consistent with pulmonary hypertension and/or right ventricular (RV) failure. These findings are included in the history because they may be part of the presentation. Although patients often comment on peripheral edema, with careful questioning they may also identify increasing abdominal girth consistent with ascites. Important questions on lower extremity edema include determination of whether the edema is symmetric (unilateral edema suggests alternate diagnoses) and whether the edema improves or resolves with elevation of the lower extremities. The finding of "no resolution overnight" argues against RV failure as an etiology. In addition, for peripheral edema and ascites, it is important to ask questions directed toward determining the presence of anemia, hypoproteinemia, or other

Box 1-5 Differential Diagnosis of Dyspnea

Pulmonary
- Reactive airways disease (asthma)
- Chronic obstructive pulmonary disease
- Emphysema
- Pulmonary edema
- Pulmonary hypertension
- Lung transplant rejection
- Infection
- Interstitial lung disease
- Pleural disease
- Pulmonary embolism
- Respiratory muscle failure
- Exercise intolerance

Cardiac
- Ischemic heart disease/angina pectoris
- Right-sided heart failure
- Aortic stenosis or regurgitation
- Arrhythmias
- Dilated cardiomyopathy
- Hypertrophic cardiomyopathy
- Congestive heart failure
- Mitral regurgitation or stenosis
- Mediastinal abnormalities
- Pericardial tuberculosis
- Transposition of the great arteries

Other
- Blood transfusion reaction
- Measles

causes. The differential diagnosis of edema is broad and beyond the scope of this chapter.

Palpitations and Syncope

It is normal to be aware of the sensation of the heart beating, particularly during or immediately after exertion or emotional stress. *Palpitations* refer to an increased awareness of the heart beating. Patients use many different descriptions, including a "pounding or racing of the heart," the feeling that their heart is "jumping" or "thumping" in their chest, the feeling that the heart "skips beats" or "races," or countless other descriptions. A history showing that palpitations have begun to occur during or immediately after exertion, and not at other times, raises the concern that these sensations reflect ventricular ectopy associated with myocardial ischemia. It is more difficult to assess the significance of palpitations occurring at other times. Supraventricular and ventricular ectopy may occur at any time and may be benign or morbid. As discussed in Chapters 29, 30, and 31, ventricular ectopy is worrisome in patients with a history of MI or cardiomyopathy. Lacking this information, clinicians should be most concerned if lightheadedness or presyncope accompanies palpitations.

Syncope generally indicates an increased risk for sudden cardiac death and is usually a result of cardiovascular disease and arrhythmias. If a syncopal episode is a presenting complaint, the patient should be admitted for further assessment. In approximately 85% of patients, the cause of syncope is cardiovascular. In patients with syncope, one must assess for CHD, cardiomyopathy, and congenital or valvular heart disease. In addition, neurocardiogenic causes represent a relatively common and important possible etiology for syncope. Box 1-6 shows the differential diagnosis for syncope. It is critical to determine whether syncope really occurred. A witness to the episode and documentation of an intervening period are very helpful. In addition, with true syncope, injuries related to the sudden loss of consciousness are common. However, an individual who reports recurrent syncope (witnessed or unwitnessed) but has never injured himself or herself may not be experiencing syncope. This is not to lessen the concern that a serious underlying medical condition exists but instead to reaffirm that the symptoms fall short of syncope, with its need for immediate evaluation.

Box 1-6 Differential Diagnosis for Syncope

Cardiogenic

- Mechanical
  - Outflow tract obstruction
  - Pulmonary hypertension
  - Congenital heart disease
  - Myocardial disease: low-output states
- Electrical
  - Bradyarrhythmias
  - Tachyarrhythmias
- Neurocardiogenic
  - Vasovagal (vasodepression)
  - Orthostatic hypotension

Other

- Peripheral neuropathy
- Medications
- Primary autonomic insufficiency
- Intravascular volume depletion
- Reflex
- Cough
- Micturition
- Acute pain states
- Carotid sinus hypersensitivity

THE PHYSICAL EXAMINATION

There are several advantages to obtaining a patient's history before the physical examination. First, the information gained in the history directs the clinician to pay special attention to aspects of the physical examination. For instance, a history consistent with CHD necessitates careful inspection for signs of vascular disease; a history suggestive of CHF should make the clinician pay particular attention to the presence of a third heart sound. Second, the history allows the clinician to establish a rapport with patients, to assure patients that he or she is interested in their well-being, and that the physical examination is an important part of a complete evaluation. In this light, the therapeutic value of the physical examination to the patient should not be underestimated. Despite the emphasis on technology today, even the most sophisticated patients expect to be examined, to have their hearts listened to, and to be told whether worrisome findings exist or the examination results were normal.

General Inspection and Vital Signs

Much useful information can be gained by an initial "head-to-toe" inspection and assessment of vital signs. For instance, truncal obesity may signal the presence of type 2 diabetes or the metabolic syndrome. Cyanosis of the lips and nail beds may indicate underlying cyanotic heart disease. Hairless, dry-skinned lower extremities or distal ulceration may indicate peripheral vascular disease. Other findings are more specific (Fig. 1-3). Abnormalities of the digits are found in atrial septal defect; typical findings of Down's syndrome indicate an increased incidence of ventricular septal defect or more complex congenital heart disease; hyperextensible skin and lax joints are suggestive of Ehlers-Danlos syndrome; and tall individuals with arachnodactyly, lax joints, pectus excavatum, and an increased arm length-to-height ratio may have Marfan's syndrome. These represent some of the more common morphologic phenotypes in individuals with heart disease. Vital signs can also be helpful. Although normal vital signs do not rule out CHD, marked hypertension may signal cardiac risk, whereas tachycardia, tachypnea, and/or hypotension at rest suggest CHF.

Important Components of the Cardiovascular Examination

The clinician should focus efforts on those sites that offer a window into the heart and vasculature. Palpation and careful inspection of the skin for secondary changes because of vascular disease or diabetes is important. Lips, nail beds, and fingertips

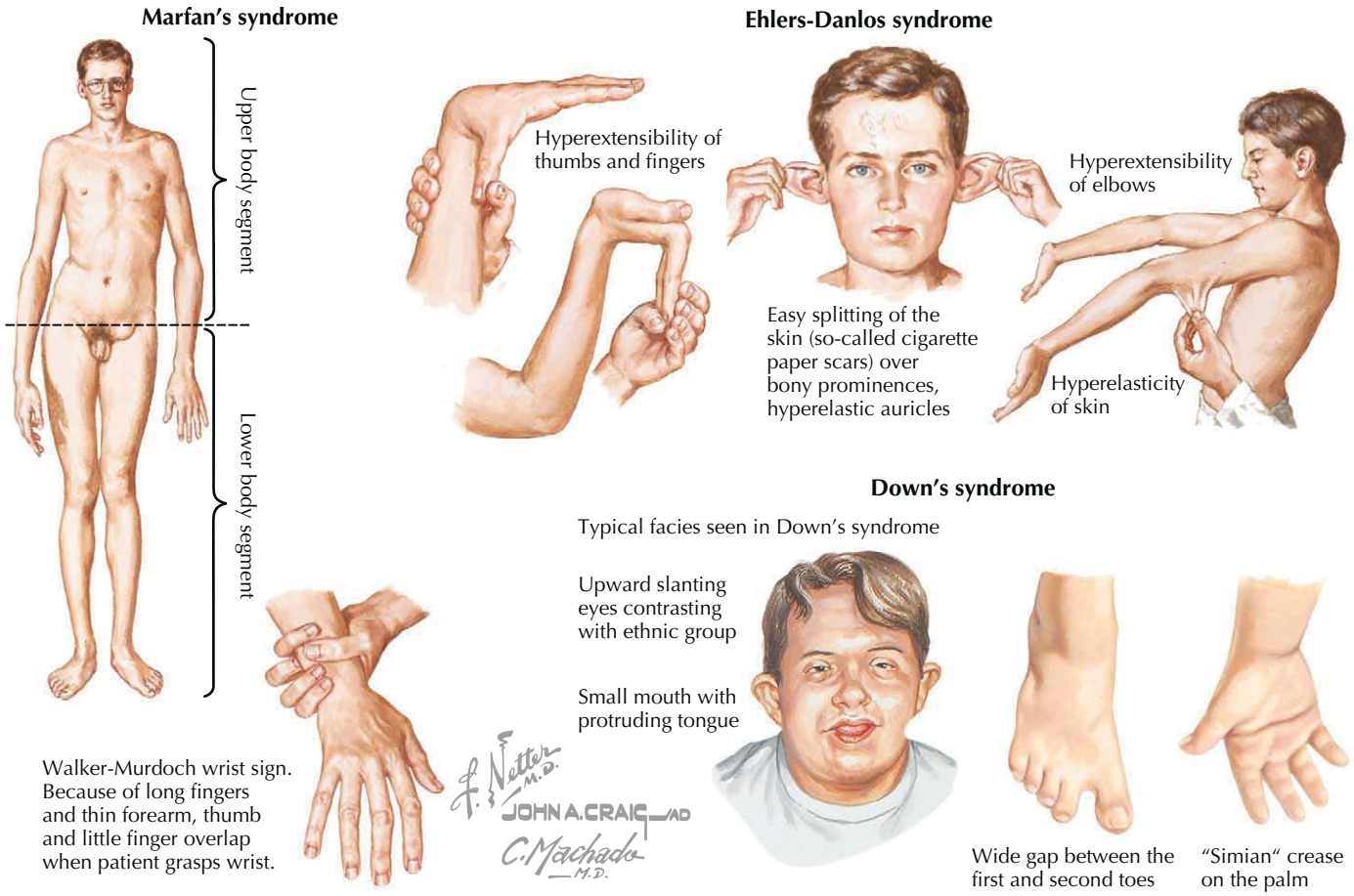

Figure 1-3 *Physical examination: general inspection.*

should be examined for cyanosis (including clubbing of the fingernails) and, when indicated, for signs of embolism. Examination of the retina using an ophthalmoscope can reveal evidence of long-standing hypertension, diabetes, or atheroembolism, denoting underlying vascular disease. Careful examination of the chest, including auscultation, can help to differentiate causes of dyspnea. The presence of dependent rales is consistent with left-sided heart failure. Pleural effusions can result from long-standing LV dysfunction or noncardiac causes and can be present with predominantly right-sided heart failure, representing transudation of ascites into the pleural space. Hyperexpansion with or without wheezing suggests a primary pulmonary cause of dyspnea, such as COPD or reactive airways disease. The presence of wheezing rather than rales does not rule out left-sided heart failure. It is not uncommon to hear wheezing with left-sided CHF. Most commonly, wheezing from left-sided CHF is primarily expiratory. Inspiratory and expiratory wheezing, particularly with a prolonged inspiratory-to-expiratory ratio, is more likely to be caused by intrinsic lung disease.

The vascular examination is an important component of a complete evaluation. The quality of the pulses, in particular the carotid and the femoral pulses, can identify underlying disease (Fig. 1-4). Diminished or absent distal pulses indicate peripheral vascular disease. The examiner should also auscultate for bruits over both carotids, over the femoral arteries, and in the abdomen. Abdominal auscultation should be performed, carefully listening for aortic or renal bruits, in the mid-abdominal area before abdominal palpation, which can stimulate increased bowel sounds. Distinguishing bruits from transmitted murmurs in the carotid and abdominal areas can be challenging. When this is a concern, carefully marching out from the heart using the stethoscope can be helpful. If the intensity of the murmur or bruit continually diminishes farther from the heart, it becomes more likely that this sound originates from the heart, rather than from a stenosis in the peripheral vasculature. Much information is available about the peripheral vascular examination, but by following the simple steps outlined herein, the examiner can gather the majority of the accessible clinical information.

Examination of the jugular venous pulsations is a commonly forgotten step. Jugular venous pressure, which correlates with right atrial pressure and RV diastolic pressure, should be estimated initially with the patient lying with the upper trunk elevated 30 degrees. In this position, at normal jugular venous pressure, no pulsations are visible. This correlates roughly to a jugular venous pressure less than 6 to 10 cm. The absence of jugular vein pulsations with the patient in this position can be confirmed by occluding venous return by placing a fingertip parallel to the clavicle in the area of the sternocleidomastoid muscle. The internal and external jugular veins should partially

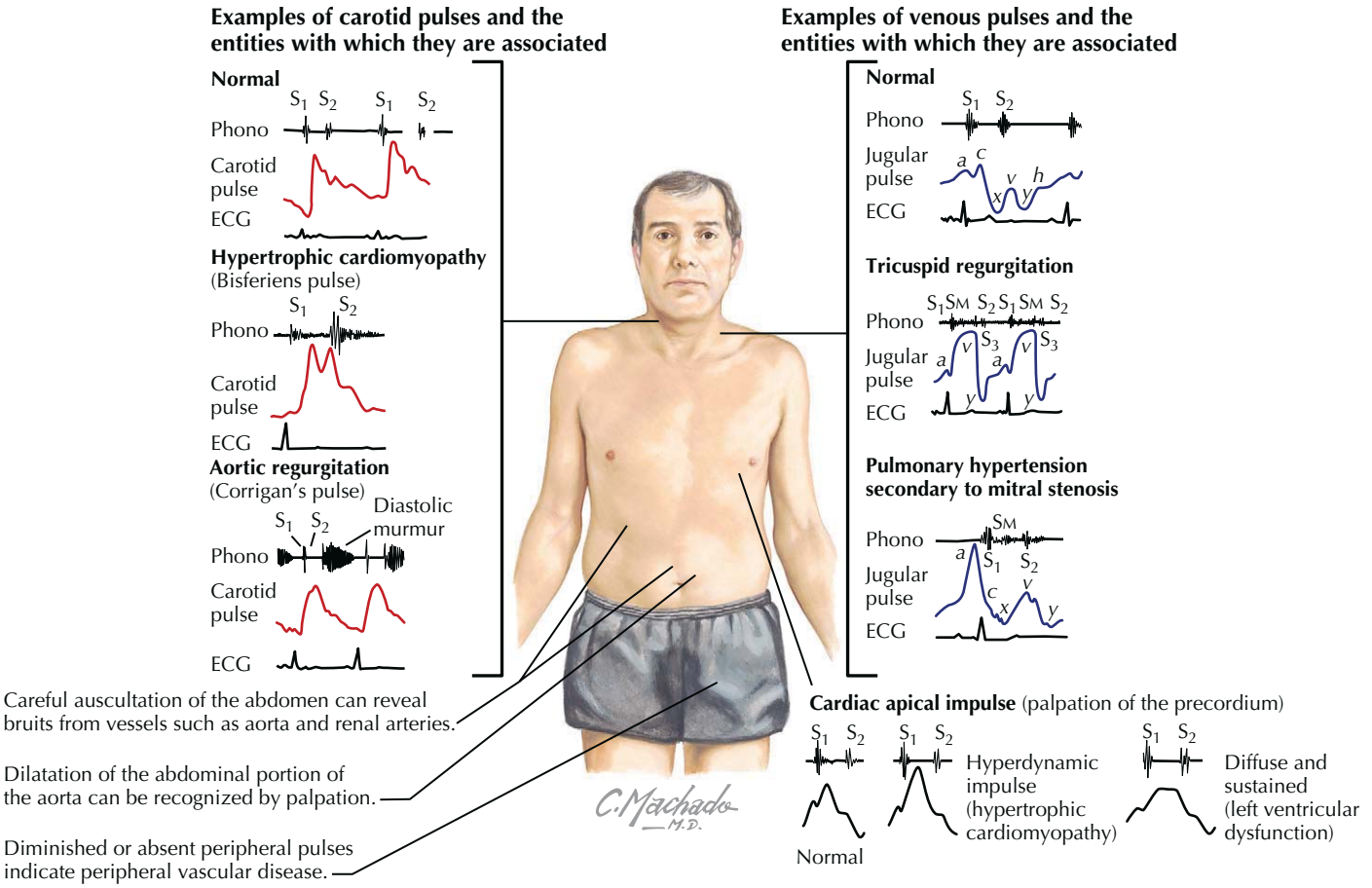

Figure 1-4 *Important components of cardiac examination.* ECG, electrocardiogram.

fill. Although with normal jugular venous pressure examination of the waveforms is less important, the head of the examination table can be lowered until the jugular venous pulsations are evident. When the jugular venous pulsations are visible at 30 degrees, the examiner should note the waveforms. It is possible to observe and time the *a* and *v* waves by simultaneously timing the cardiac apical impulse or the carotid impulse on the contralateral side. An exaggerated *a* wave is consistent with increased atrial filling pressures because of tricuspid valve stenosis or increased RV diastolic pressure. A large *v* wave generally indicates tricuspid valve regurgitation, a finding easily confirmed by auscultation.

Finally, before cardiac auscultation it is important to palpate the precordium. This is the easiest way to identify dextrocardia. Characteristics of the cardiac impulse can also yield important clues about underlying disease. Palpation of the precordium is best performed from the patient's right side with the patient lying flat. The cardiac apical impulse is normally located in the fifth intercostal space along the midclavicular line. Most examiners use the fingertips to palpate the apical impulse. It is often possible to palpate motion corresponding to a third or fourth heart sound. Use of the fingertips offers fine detail on the size and character of the apical impulse. A diffuse and sustained apical impulse is consistent with LV systolic dysfunction. Patients with hypertrophic cardiomyopathy, in contrast, often have a hyperdynamic apical impulse. Thrills, palpable vibrations from loud murmurs or bruits, can also be palpated.

The RV impulse, if identifiable, is located along the left sternal border. Many clinicians prefer to palpate the RV impulse with the base of the hand, lifting the fingertips off the chest wall. In RV hypertrophy, a sustained impulse can be palpated, and the fingertips then can be placed at the LV impulse to confirm that the two are distinct. In patients with a sustained RV impulse, the examiner should again look for prominent *a* and *v* waves in the jugular venous pulsations.

Cardiac Auscultation

Hearing and accurately describing heart sounds is arguably the most difficult part of the physical examination. For this reason and because of the commonplace use of echocardiography, many clinicians perform a cursory examination. The strongest arguments for performing cardiac auscultation carefully are to determine whether further diagnostic testing is necessary; to correlate findings of echocardiography with the clinical examination so that, in longitudinal follow-up, the clinician can determine progression of disease without repeating echocardiography at each visit; and because the more a clinician makes these correlations, the better his or her skills in auscultation will become and the better his or her patients will be served. It should also

be noted that, with normal general cardiac physical examination results, the absence of abnormal heart sounds, and a normal electrocardiogram, the use of echocardiography for evaluation of valvular or congenital heart disease is not indicated. Furthermore, if there are no symptoms of CHF or evidence of hemodynamic compromise, echocardiography is not indicated for assessment of LV function. Practice guidelines from cardiologists and generalists agree on this point, as do third-party insurers. It is neither appropriate nor feasible to replace a careful cardiovascular examination using auscultation with more expensive testing.

The major impact of echocardiography has been in quantitative assessment of cardiovascular hemodynamics—that is, the severity of valvular and congenital heart disease. No longer is it necessary for the clinician to make an absolute judgment on whether an invasive assessment (cardiac catheterization) is needed to further define hemodynamic status or whether the condition is too advanced to allow surgical intervention based on history and physical examination. But instead of diminishing the role of cardiac auscultation, the advent of echocardiography has redefined it. Auscultation remains important as a screening technique for significant hemodynamic abnormalities, as an independent technique to focus and verify the echocardiographic study, and an important means by which the physician can longitudinally follow patients with known disease.

There are several keys to excellence in auscultation. Foremost is the ability to perform a complete general cardiac physical examination, as described. The findings help the examiner focus on certain auscultatory features. Second, it is important to use a high-quality stethoscope. Largely dictated by individual preference, clinicians should select a stethoscope that has bell and diaphragm capacity both (for optimal appreciation of low- and high-frequency sounds, respectively) and that fits the ears comfortably and is well insulated so that external sounds are minimized. Third, it is important to perform auscultation in a quiet environment. Particularly as skills in auscultation are developing, trying to hone these in the hall of a busy emergency department or on rounds while others are speaking is time spent poorly. Additionally, taking the time to return to see a patient with interesting findings detected during auscultation, and repetition, are keys to becoming competent in auscultation.

The patient should be examined while he or she is in several positions: while recumbent, while in the left lateral decubitus position, and while sitting forward. Every patient is different and, using all three positions, the examiner can optimize the chance that soft heart sounds can be heard. Likewise, it is important to listen carefully at the standard four positions on the chest wall (Fig. 1-5), as well as over the apical impulse and RV impulse (if present). It is also best to isolate different parts of the examination in time. Regardless of the intensity of various sounds, it is best always to perform the examination steps in the same chronologic order, so that the presence of a loud murmur, for instance, does not result in failure to listen to the other heart sounds.

Listen for S_1 (the first heart sound) first. As with jugular venous pulsations, the heart sounds can be timed by simultaneously palpating the cardiac apical impulse or the carotid upstroke. Even the most experienced clinician occasionally needs to time the heart sounds. Is a single sound present, or is the first heart sound split? Is a sound heard before S_1, indicating an S_4? Next, listen to the second heart sound. Normally the first component (A_2, the aortic valve closing sound) is louder than the second component (P_2, the pulmonic valve closing sound). A louder second component may indicate increased pulmonary pressure. A more subtle finding is a reversal of A_2 and P_2 timing that occurs with left bundle branch block and in some other circumstances. Additionally, it is important to assess whether A_2 and P_2 are normally split or whether they are widely split with no respiratory variation—a finding suggestive of an atrial septal defect. The examiner should then listen carefully for a third heart sound. An S_3 is often best heard over the tricuspid or mitral areas and is a low-frequency sound. It is heard best with the bell and is often not heard with the diaphragm.

After characterizing these heart sounds, it is time to listen carefully for murmurs. Murmurs are classified according to their intensity, their duration, their location, and their auscultatory characteristics: crescendo, decrescendo, blowing, among others. It is also important to note the site where the murmur is loudest and whether the murmur radiates to another area of the precordium or to the carotids. All of these features contribute to determining the origin of the murmur, the likelihood that it represents an acute or chronic process, and how it affects the diagnostic and therapeutic approaches. Most importantly, it is necessary for clinicians to judge whether a murmur represents cardiac disease or is innocent. Innocent murmurs, also termed "flow murmurs," are common in children. More than 60% of children have innocent murmurs. Innocent murmurs become less common in adults; however, an innocent murmur can still be found into the fourth decade of life. Alterations in hemodynamics induced by pregnancy, anemia, fever, hyperthyroidism, or any state of increased cardiac output can produce an innocent murmur. These murmurs are generally midsystolic, heard over the tricuspid or pulmonic areas, and do not radiate extensively. They are often loudest in thin individuals. Innocent murmurs do not cause alterations in the carotid pulse and do not coexist with abnormal cardiac impulses or with other abnormalities, such as extra heart sounds (S_3 and S_4), in adults. In elderly individuals a common finding is a systolic murmur that shares auditory characteristics with the murmur of aortic stenosis; however, carotid upstrokes are normal. This finding, aortic sclerosis, may necessitate confirmation by echocardiography. It represents sclerosis of the aortic leaflets but without significant hemodynamic consequence.

The characteristics of the most common and hemodynamically important murmurs are shown in Figure 1-5. As noted, the murmur is defined not only by its auditory characteristics but also by the company it keeps. Often the key to excellence in auscultation is being thorough in all aspects of the cardiovascular examination.

Maneuvers

No discussion of cardiac auscultation would be complete without the use of maneuvers to accentuate auscultatory findings. As shown in Figure 1-6, patient positioning can alter peripheral vascular resistance or venous return, accentuating murmurs that are modulated by these changes. Murmurs associated with fixed

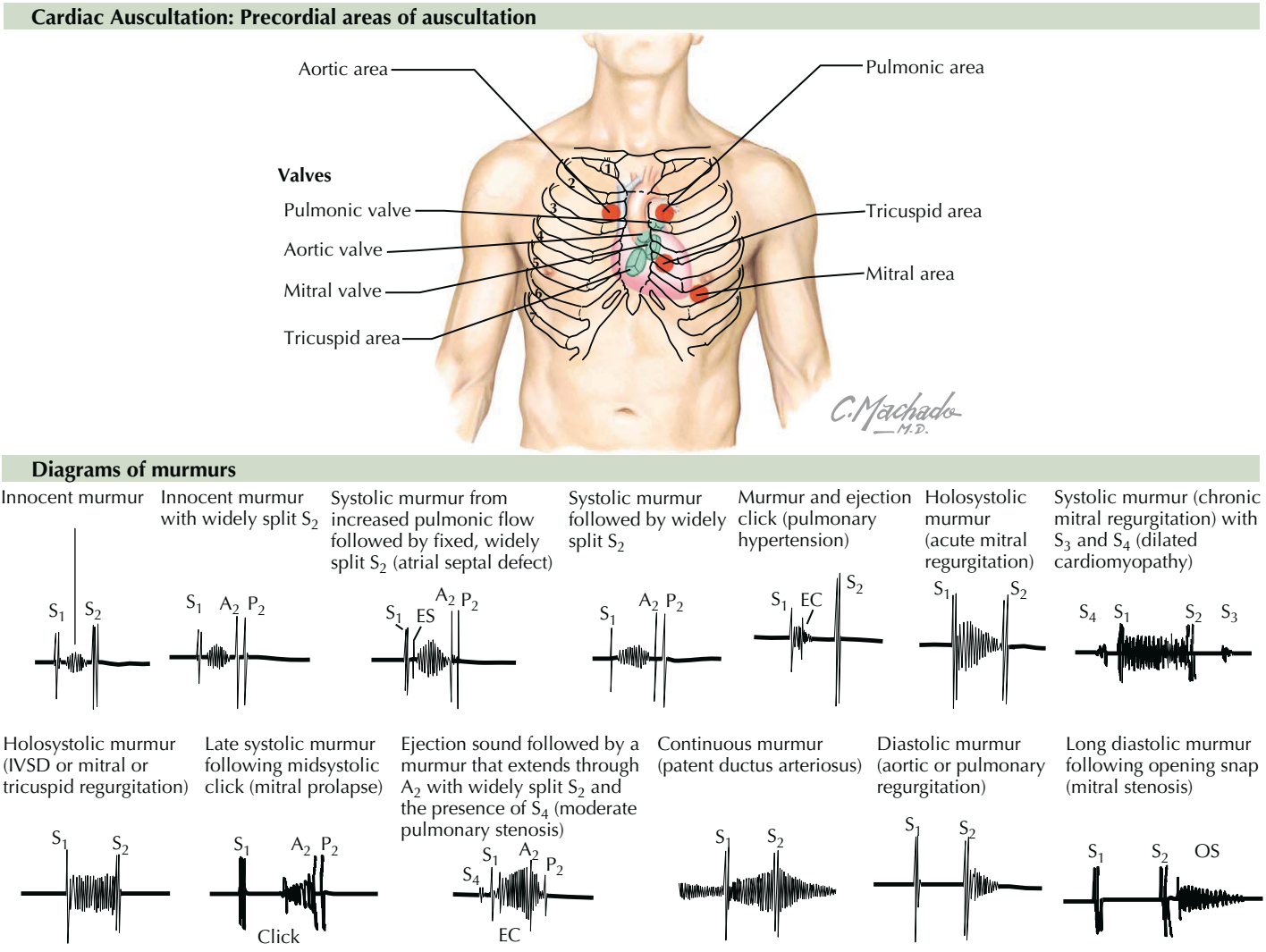

Figure 1-5 *Cardiac auscultation: Correlation of murmurs and other adventitious sounds with underlying pathophysiology.*

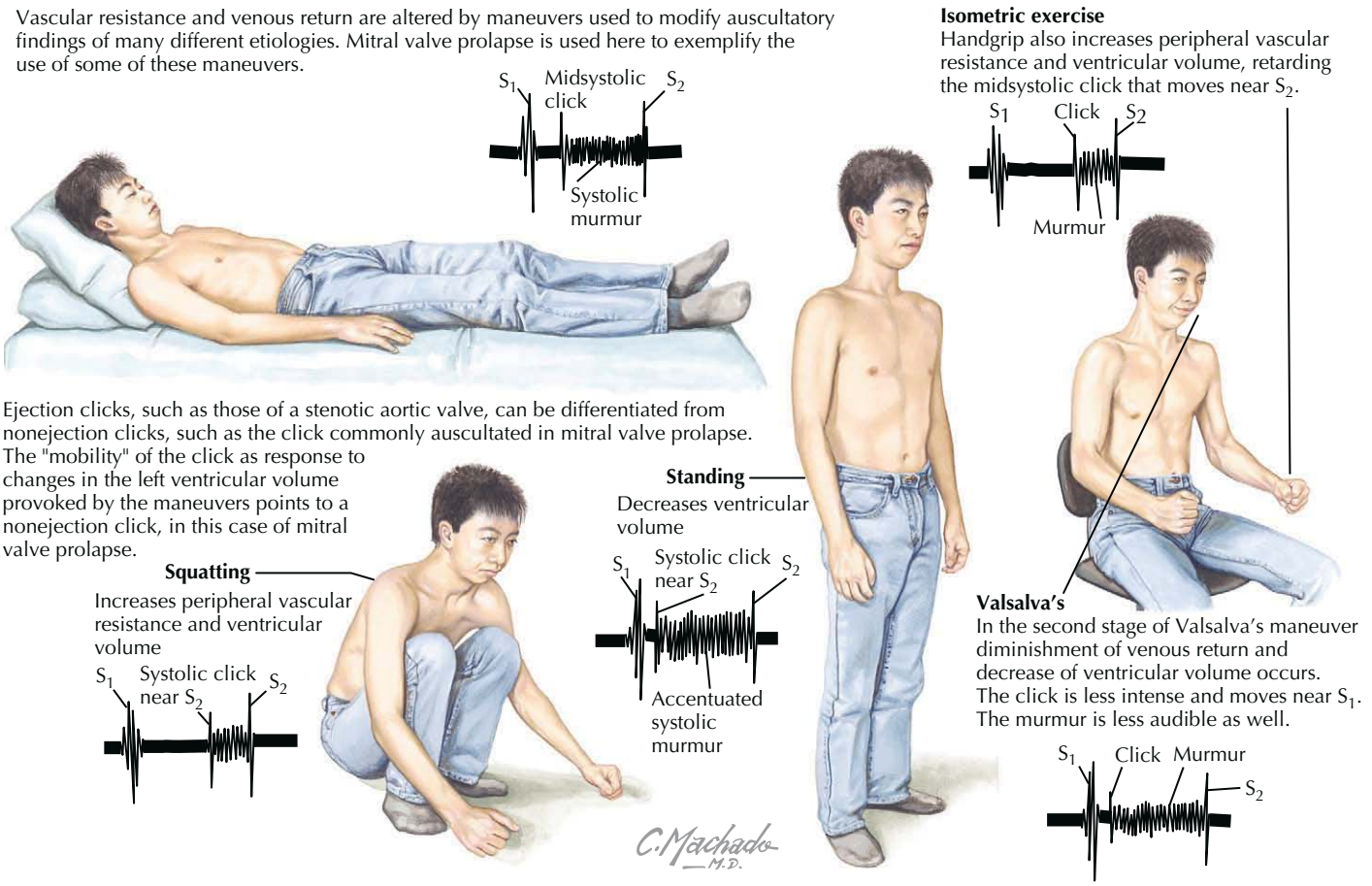

Figure 1-6 *Maneuvers.*

valvular lesions change little with changes in position or the maneuvers illustrated in Figure 1-6. Thus, these maneuvers are most useful for diagnosing entities in which hemodynamic status affects murmurs. The two classic examples are the click and murmur of mitral valve prolapse, as shown, and the aortic outflow murmur of hypertrophic cardiomyopathy (not shown).

FUTURE DIRECTIONS

Handheld echocardiography machines can be carried on the shoulder and have a small transducer that can obtain echocardiographic images of sufficient quality to quantify murmurs and assess LV dysfunction. Although these portable echocardiographic machines have advantages and have been incorporated in medical school curricula at many institutions, they have not yet replaced the stethoscope, nor are they likely to do so.

The roles of cardiac history and physical examination have changed. Before the noninvasive testing of today, astute clinicians were the arbiters of whether invasive diagnostic testing was needed, based largely on examination findings alone. Today it is believed that the role of the clinician is to use physical examination findings to establish the prior probability of cardiovascular disease, whether CHD, valvular heart disease, or congenital heart disease, thereby determining the need for further testing. In the continual quest for improved noninvasive testing, it is likely that a clinician's skill will continue to evolve as the interplay between history taking, physical examination, and diagnostic testing further develops.

ADDITIONAL RESOURCES

ACC/AHA Joint Guidelines. <http://www.americanheart.org/presenter.jhtml?identifier=3004542>; Accessed 22.02.10.

Guidelines outlining the current opinion of experts from the American College of Cardiology and the American Heart Association for managing cardiovascular diseases.

American Heart Association. Heart Profilers. Available at: <http://www.americanheart.org/presenter.jhtml?identifier=3000416>; Accessed 22.02.10.

Provides individual specific information based on your risk profile.

National Heart, Lung and Blood Institute. National Cholesterol Education Program. Available at: <http://hp2010.nhlbihin.net/atpiii/calculator.asp?usertype=prof>; Accessed 22.02.10.

A website where you can enter patient-specific data to calculate the 10-year risk of a cardiac event based on Framingham data (Framingham Risk Calculator).

EVIDENCE

Diamond GA, Forrester JS. Analysis of probability as an aid in the clinical diagnosis of coronary-artery disease. *N Engl J Med.* 1979; 300:1350–1358.

A classic discussion of the importance of pre-test and post-test probabilities in interpreting any diagnostic testing.

Harvey WP. *Cardiac Pearls [video recording].* Atlanta: Emory Medical Television Network; 1981.

This video recording is a timeless example of Dr. Harvey—a master clinician—and his approach to the evaluation of patients with cardiovascular disease.

Hurst JW, Morris DC. *Chest Pain.* Armonk, NY: Futura Publishing; 2001.

Drs. Hurst and Morris provide a sophisticated summary on the evaluation of patients with chest pain.

National Heart, Lung and Blood Institute. Third Report of the Expert Panel on Detection, Evaluation, and Treatment of High Blood Cholesterol in Adults (Adult Treatment Panel III) and *ATP III Update 2004: Implications of Recent Clinical Trials for the ATP III Guidelines*. Available at: <http://www.nhlbi.nih.gov/guidelines/cholesterol>; Accessed 10.11.09.

An overview of the current recommendations regarding treatment of elevated lipids.