HERRICK AND HEART DISEASE

At the 1912 meeting of the Association of American Physicians, James B. Herrick read a paper entitled “Clinical Features of Sudden Obstruction of the Coronary Arteries,” which is now considered a classic. For Herrick, however, the event was a disaster:

In 1912 when I arose to read my paper at the Association, I was elated, for I knew I had a substantial contribution to present. I read it, and it fell flat as a pancake. No one discussed it except Emanuel Libman, and he discussed every paper read there that day. I was sunk in disappointment and despair. (Means, 1961)

It was not until Herrick readdressed the same association in 1919 that his views finally received proper attention.

Born in Oak Park, Illinois, in 1861, Herrick graduated from the University of Michigan in 1882 and taught English, Latin, and Greek for several years in a high school. He graduated from Rush Medical College in 1888 and interned at Cook County Hospital. He entered practice in Chicago in 1890 and was appointed to the staffs of Cook County Hospital and Presbyterian Hospital. His first book, A Manual of Medical Diagnosis, was published in 1895 when he was age 34. In 1900 he decided to engage only in consulting and became widely known as a master clinician and teacher. Aside from his accomplishments in cardiology, Herrick called attention to anemia secondary to chronic blood loss in 1902, and wrote the first description of sickle cell anemia in 1910.

The prevailing opinion of coronary obstruction in Herrick’s early years was that it usually resulted in sudden death because the coronary arteries were believed to be end arteries, lacking sufficient anastomoses. Conheim lent support to this pessimistic view of coronary obstruction; Herrick disagreed, writing:
Obstruction of a coronary artery or any of its large branches has long been regarded as a serious accident. Several events contributed toward the prevalence of the view that this condition was almost always suddenly fatal. . . . But there are reasons for believing that even large branches of the coronary arteries may be occluded—at times acutely occluded—without resulting death, at least without death in the immediate future. Even the main trunk may at times be obstructed and the patient live. It is the object of this paper to present a few facts along this line, and particularly to describe some of the clinical manifestations of sudden yet not immediately fatal cases of coronary obstruction. (1912)

Herrick's medical experience at the bedside enabled him to differentiate clinical manifestations of coronary obstruction when no one had before. His classification consisted of the following four events: (1) instantaneous, perhaps painless, death; (2) severe angina followed minutes later by shock and death; (3) mild, nonfatal angina; and (4) severe angina that is usually eventually fatal, but not immediately. Herrick concluded with an insight concerning a therapeutic dilemma that remains today: "The hope for the damaged myocardium lies in the direction of securing a supply of blood through friendly neighboring vessels so as to restore so far as possible its functional integrity". Herrick advocated use of digitalis instead of nitrates in the treatment of sudden coronary obstruction and recommended bed rest for 6 weeks.

In 1930 Herrick received the Kober Medal for distinguished research in medicine conferred by the Association of American Physicians, and in 1939 he received a Distinguished Service Medal from the American Medical Association. His bibliography includes at least 163 medical articles and 9 papers presented to the Chicago Literary Club. Apart from his classical scholarship, Herrick was interested in medical history. In 1942 he published A Short History of Cardiology, which covered the subject from Harvey to Roentgen. In a review of the book, Paul Dudley White wrote:

The next to the last chapter, devoted to coronary disease, is of particular interest in its attempt to explain the lapse in interest in the subject between 1900 and 1912. . . . Sudden deaths and temporary invalidism from heart disease, were occurring commonly throughout that century, pathologists were finding scars in the heart and other evidences of serious coronary disease, but there was no clinician to put all the facts together until Herrick himself analyzed the problem. The occasional references in the literature to which he himself refers, although interesting, and of course accumulative, nevertheless do not take away in my mind at all from the luster of Herrick's own accomplishment in his classic paper in 1912. The only outstanding omission in the book as I see it is the lack of recognition of the importance of his own contribution. It undoubtedly is from sincere modesty. (1942)

Herrick described much of his life in his Memories of Eighty Years, in which he mentions his first encounter with Osler in 1898: "A more inspiring, delightful hour I had never spent. I had fallen victim—a willing one—to the charm of the Osler personality." Herrick died in 1954, having read everything written by Chaucer, his favorite author.

—Charles Stewart Roberts

REFERENCES
An Overview of the Cardiovascular System

JOEL M. FELNER

The evaluation of the cardiovascular system includes a thorough medical history, a detailed examination of the heart and the peripheral arterial and venous circulations, and appropriate laboratory studies. In addition to the electrocardiogram and chest x-ray, the availability of sophisticated noninvasive techniques (e.g., echocardiography and nuclear cardiology) and the continued improvement of cardiac catheterization and angiography have significantly enhanced the clinical work-up of the patient with a cardiovascular problem. A careful assessment will enable the clinician to identify the etiologic, anatomic, and physiologic components of a specific cardiovascular disorder, as well as to determine overall cardiac function.

History

The medical history in a patient with a cardiac problem is usually centered on symptoms due to myocardial ischemia, dysrhythmias, and reduction in ventricular function. The majority of these individuals will consult a physician because of chest pain, dyspnea, palpitations, ankle edema, or syncope. Any or all of these symptoms may also have extra-cardiac causes. Because symptoms of heart disease may be absent at rest and appear only during stress, the medical history has unique diagnostic importance. The patient's daily activities should be assessed for their role in precipitating the symptoms and in identifying these symptoms as cardiac in origin. A purely symptom-based classification of heart disease has major limitations, however, since functional abnormalities are often more extensive than those represented by symptoms alone. In addition, the anatomic and physiologic disturbances may develop to advanced stages before symptoms appear. Examples of the manner in which the principal symptoms of heart disease may serve as a guide to diagnosis will be highlighted.

Table 7.1

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<tr>
<th>Symptom</th>
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<td>Chest pain or discomfort</td>
<td>9</td>
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<td>Dyspnea</td>
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<td>Palpitations</td>
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<td>Syncope</td>
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<td>Peripheral edema</td>
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<td>Claudication</td>
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Chest pain or discomfort (Chapter 9) has numerous cardiac causes (e.g., myocardial ischemia, pericarditis, pulmonary embolism, aortic dissection) as well as noncardiac etiologies (e.g., anxiety, cholecystitis, pneumonia). The pain of myocardial ischemia, characterized by a squeezing, strangling, or burning sensation, must be differentiated from pleuritic pain, which is sharp, stabbing, intensified by inspiration, and relieved by sitting up. Among the causes of myocardial ischemia are angina pectoris and myocardial infarction. Pleuritic pain usually accompanies pericarditis and pulmonary embolism.

Dyspnea (shortness of breath) (Chapter 11) of cardiac origin must be distinguished from dyspnea due to pulmonary disease. Cardiac dyspnea, including paroxysmal nocturnal dyspnea (breathlessness at night) and orthopnea (dyspnea precipitated by assuming the recumbent position), is characteristically related to effort until the advanced stages of heart disease when it may become present at rest. Rapid progression of an episode of respiratory distress may result in a very severe form of dyspnea, acute pulmonary edema, i.e., "asthmatic" wheezes and a pink, frothy sputum.

Palpitations (Chapter 10) describe an awareness of the heartbeat. Although the underlying disturbance usually requires electrocardiographic confirmation, occasionally the cadence of the palpitations may be ascertained at the bedside. Palpitations may often be of no consequence.

Syncope of cardiac origin (Chapter 12) may be due either to an inability of the heart to maintain adequate cardiac output for a given level of activity or to a dysrhythmia that results in sudden loss of cardiac output. Left ventricular outflow tract obstruction (e.g., aortic stenosis or hypertrophic cardiomyopathy) commonly causes effort syncope, whereas syncopal episodes due to dysrhythmias can occur either at rest or during activity.

Edema (Chapter 29), a detectable excess of fluid in the interstitial spaces, is most commonly located in the ankles and feet and is referred to as peripheral or ankle edema. When due to cardiac disease, it is usually a late sign of congestive heart failure, specifically, right heart failure.

Additional symptoms that may herald a cardiovascular problem include claudication (extertional cramping of the muscles) (Chapter 13), most often of the lower extremities, fatigue, and hemoptysis (Table 7.1).

Physical Examination

Instruments needed for the cardiovascular examination are listed in Table 7.2. The examination involves inspection, palpation, and auscultation of the heart, arteries, and veins. The cardiac examination consists of evaluation of (1) the carotid arterial pulse and auscultation for carotid bruits; (2) the

Table 7.2

<table>
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<th>Instruments Needed for Cardiovascular Examination</th>
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<td>Stethoscope</td>
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<td>Penlight</td>
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<td>Centimeter ruler and tape measure</td>
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<td>Sphygmomanometer</td>
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jugular venous pulse and auscultation for cervical venous humps; (3) the precordial impulses and palpation for heart sounds and murmurs; and (4) auscultation of the heart. The evaluation of the peripheral arteries, the aortic pulsation, elicitation of pulsus alternans, and a search for thrombo-flebitis completes the cardiovascular examination (Table 7.3).

Special attention should be given to the patient’s general appearance, since it can reflect the state of the circulation as well as noncardiac diseases that may involve the heart. The patient’s color (pale, flushed, or cyanotic), facial features, body build, and obvious pulsations should be noted. The blood pressure and heart rate and rhythm are obtained with the vital signs, but must be integrated with the findings of the cardiovascular examination to arrive at the proper diagnosis.

Examination of the Heart

CAROTID ARTERIES

Begin the cardiovascular examination by assessing the carotid arterial pulse (Chapter 20). They are ordinarily examined while the patient is breathing normally and reclining with the trunk of the body elevated about 15 to 30 degrees (Figure 7.1). In order to examine the carotid arteries, the sternocleidomastoid (SCM) muscle should be relaxed and the head rotated slightly toward the examiner. The examiner places the forefinger or thumb, depending on individual preference, slightly over the artery in the groove just lateral to the trachea. Care should be taken always to palpate in the lower half of the neck in order to avoid the area of the carotid bulb, lest a hypersensitive carotid sinus reflex be evoked with resultant bradycardia and hypotension. It is important that the carotid pulses be palpated using light pressure, one side at a time, since bilateral carotid compression may produce cerebral ischemia and syncope; extreme caution should be exercised in patients who have a history of syncope or transient neurologic symptoms.

Listen to the heart with the stethoscope in order to identify the first (S₁) and second (S₂) heart sounds while simultaneously palpating the carotid artery. The heart sounds are used as reference points for the cardiac examination in order to determine which events occur in systole (between S₁ and S₂) and which in diastole (between S₂ and S₃). While listening to the heart sounds, the examiner carefully and slowly applies more and more pressure to the carotid artery until the maximum pulse is felt; palpation should be continued for 5 to 8 seconds. The examiner should then slowly release the pressure on the artery while attempting to form a mental image of the pulse wave. It may be possible to detect an anacrotic notch (halt on the upstroke) or a bisferiens pulse (bifid or double peak) more easily with light pressure than with heavy pressure. In patients with evidence of carotid arterial disease, palpation of the vessels obviously should be gentle.

Divide the carotid pulse wave into three parts: (1) ascending limb or upstroke; (2) peak; and (3) descending limb. Initially concentrate on the amplitude (size) of the carotid pulse and note whether it is normal, decreased, or increased. If necessary, use your own carotid pulse as a control. Next, direct attention to analysis of the pulse contour and note if there is a single or double peak, a shudder or thrill, and the location of the peak within systole (i.e., early, mid, or late). Then concentrate on the upstroke and note whether it is normal, rapid, or slow. Finally, concentrate on the downstroke of the pulse, which is difficult to palpate reliably. If there is a rapid fall-off, for instance, the majority of the downstroke will be completed during systole.

The normal carotid arterial pulse wave is illustrated in Figure 7.2 together with the heart sounds. The upstroke of the carotid tracing is moderately rapid and smooth, and begins just after the initial component of the first heart sound. The summit of the carotid pulse is smooth and dome shaped, and occurs approximately in the middle of systole. The descending limb from the systolic peak is usually less steep than the ascending limb. In most normal individuals,
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Figure 7.2
These graphics represent the normal cardiac pulsations and heart sounds. The jugular venous pulsation normally has 3 positive waves—the a, c, and v waves and 2 negative troughs—x and y troughs. The “a” wave is approximately synchronous with the first heart sound and just precedes the carotid upstroke. The “v” wave coincides approximately with the second heart sound. The normal carotid artery pulsation has a single positive wave during systole, followed by the dicrotic notch (about the time of the second heart sound). The apex impulse represents the normal brief, palpable systolic impulse occurring at the time of the first heart sound. In young normal individuals there may be a palpable early diastolic filling wave representing the rapid filling phase of ventricular diastole and corresponding to the normal third heart sound. Auscultation at the aortic area reveals a normal first heart sound (S1) and second heart sound (S2). S2 is normally louder than S1 in this area. At the pulmonary area there is normal inspiratory (physiologic) splitting of the second sound due to asynchronous aortic and pulmonic closure. The aortic component of the second heart sound (A2) normally precedes the pulmonic component (P2). At the tricuspid area there is normal splitting of the first heart sound due to asynchronous mitral and tricuspid closure. The mitral component of the first sound (M1) normally precedes the tricuspid component (T1). At this area, physiologic splitting of the second sound may also be appreciated. At the mitral area, the first and second heart sounds are normal. The first sound is normally louder than the second heart sound and only the aortic component of the second heart sound is normally appreciated. Occasionally a third heart sound is normal, reflecting deceleration of blood into the left ventricle during the rapid filling phase of early diastole. Children and young adults often have normal or physiologic third heart sounds.

The carotid incisura or dicrotic notch is not palpable; however, one can usually sense a change to a less steep downslope.

There are a variety of abnormal arterial pulses including the hypokinetic pulse commonly seen with left ventricular failure; the hyperkinetic pulse commonly seen with mitral or aortic regurgitation; and the bisferiens pulse seen with aortic regurgitation or hypertrophic cardiomyopathy. During routine palpation of the carotid pulse, pay particular attention to the amplitude of the pulse following any premature beat. A diminished pulse amplitude following a premature beat is suggestive of hypertrophic cardiomyopathy.

Auscultation should be performed along the course of the carotid artery in order to detect any bruits. (See Figure 7.3 and Chapter 18). The location of maximum intensity of the bruit should be noted, as well as the pitch and duration of the sound. It is necessary for the patient to stop breathing during auscultation to eliminate the harsh sounds of tracheal breathing that could mask a low-pitched carotid bruit.

JUGULAR VEINS
The jugular venous pulse (Chapter 19) is usually examined next. It includes observation of venous wave form, assessment of the response of the venous pressure to abdominal compression, estimation of the central venous pressure (CVP), and auscultation for cervical venous hums. Venous pulsations are examined by inspection of either the external or internal jugular veins, although the latter are generally more reliable because they more directly reflect right atrial hemodynamics.

The position of the patient is extremely important for the examination of the jugular veins (Figure 7.4). Relax the neck muscles by placing a small pillow behind the neck. The head should not be rotated more than a few degrees, since
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Figure 7.4
Anatomy of the blood vessels in the neck.

Evaluation of the jugular venous pulse and the carotid artery are best accomplished with the patient supine, the neck muscles relaxed by placing a small pillow under the head and the trunk elevated until the maximal internal jugular venous pulsations are visible. Venous pulsations are usually most prominent in the suprasternal notch, the supravacular fossa or just below the ear lobes.

The common carotid artery is located deep to the sternocleidomastoid muscle at the root of the neck. It divides into external and internal branches. The terminal portion of the common carotid and the root of the internal carotid artery become dilated at the level of the carotid sinus. The internal carotid artery continues to the base of the skull where it enters the cranium.

The internal jugular vein ends below and behind the sternoclavicular. Throughout its course, the internal jugular vein lies at the side of the carotid artery and crosses the artery as it passes behind the sternocleidomastoid muscle. Since this vein lies deep to the muscle usually only pulsations transmitted to the surrounding soft tissues are visible. Superiorly, the internal jugular vein is posterolateral to the common carotid and at the root of the neck it lies anterior to the artery. The external jugular vein is a superficial vessel that crosses the sternocleidomastoid muscle perpendicularly as it descends towards the middle of the clavicle.

Generally, internal and external jugular venous pulsations may be eliminated by applying gentle pressure below the point of observation. This procedure also may produce increased distention of the vein by blocking the flow of blood to the heart. The visible venous pulsations in the neck are slower and more undulating than the brisk, forceful arterial pulse waves. Respiration may produce marked changes in venous pulsations, whereas arterial pulses normally change relatively little. Under normal circumstances, inspiration decreases intrathoracic pressure and increases return of blood to the heart from the peripheral veins. The result is to reduce the mean level of venous pulsation and distention; the opposite occurs during expiration.

Abdominal pressure may also be used to distinguish venous from arterial neck pulsations. This test is best performed with the patient lying comfortably in bed at the optimal...
angle for observing the internal jugular venous pulsations. The patient is instructed to continue normal breathing in order to avoid performance of a Valsalva maneuver. Moderately firm pressure is then slowly applied for about 30 seconds with the palm of the hand pressing on the abdomen, usually on the right side (over the liver). Normally this maneuver produces no visible change in the arterial pulse, but a slight increase in the prominence of the jugular venous pulsations. In the presence of heart failure the jugular venous pulsations may be markedly increased. The response of jugular venous pulse to abdominal compression is known as the hepato-jugular reflux test. It is useful not only for distinguishing venous from arterial pulsations but also in unmasking occult abnormalities of circulatory function.

The normally visible jugular venous pulsations consist of two outward pulsations or positive waves ("a" and "v") and two descents or collapses or negative waves ("x" and "y") as shown in Figure 7.2. The "c" wave, a positive wave that follows the "a" wave, may be recorded, but is seldom seen at the bedside. The carotid pulse may be used to time venous pulsations, but the heart sounds generally are preferable at the bedside. The "a" wave, the larger of the two visible positive waves, begins before S, and precedes the upstroke of the carotid pulse. The negative "x" and "y" waves occupy systole and diastole, respectively. The "v" wave occurs in late systole virtually synchronous with S,. Frequently, it is easier to visualize jugular descents because they are the largest and fastest movements.

The internal jugular veins are also used to estimate central venous pressure. The technique is similar to that for evaluation of the internal jugular venous wave form (Figure 7.5). The patient is examined at the optimum degree of trunk elevation for inspection of the venous pulse. While the patient is breathing gently or preferably at the end of a normal expiration, the highest point of visible pulsation of the internal jugular vein is determined (Figure 7.5). The CVP can then be estimated by measuring the vertical distance between the mid-right atrium and the top of the column of venous blood in relation to a fixed reference point, the sternal angle (of Louis). The vertical distance between the top of the column of venous blood and the level of the sternal angle is normally 2 cm. For convenience at the bedside, the sternal angle is chosen as a reference point because it has a relatively constant relationship, in all positions, to the midpoint of the right atrium (i.e., 5 cm above the mid-right atrium).

![Figure 7.5](image)

**Figure 7.5**

_Estimation of the central venous pressure (CVP)._ Place the patient in the supine position with the head slightly elevated on a pillow to relax the sternocleidomastoid muscle. Elevate the trunk by adjusting the head of the bed so as to maximize the internal jugular venous pulsations and make them visible above the clavicles. Since the internal jugular vein lies deep to the sternocleidomastoid muscle, the vein itself is not seen, but watch instead for the pulsations transmitted through the surrounding soft tissue.

With the patient breathing quietly or preferably at the end of normal expiration, identify the highest point at which pulsations of the internal jugular vein can be seen. With a centimeter ruler positioned at the sternal angle of Louis (felt as a transverse ridge formed by the angulation of the sternum at the articulation of the second ribs) measure the vertical distance using a cotton-swab between this point and the venous pulsations. The sternal angle bears a constant relation to the mean CVP with the patient in a supine, upright or any intermediate position and is 5 cm above the mid-right atrium. If the highest point of the venous pulsation is below the sternal angle, hold the centimeter ruler at the neck rather than at the sternal angle and use the cotton swab to establish the horizontal and vertical relationships that underlie this measurement. Record the distance in centimeters, above or below the sternal angle, together with angle at which the patient is lying.
The normal CVP is less than or equal to 7 cm H$_2$O (i.e., 5 + 2 cm). The most common cause of an elevated CVP is failure of the right ventricle secondary to failure of the left ventricle. Earlobe and rarely eyeball pulsations are evidence of markedly elevated venous pressure.

Central venous pressure may also be estimated by examining the veins of the dorsum of the hand. To perform this determination, the patient should be in either a sitting or lying position at a 30-degree elevation or greater, and the hand should be kept below the level of the heart long enough for the veins of the dorsum of the hand to become distended. The arm is then slowly and passively raised while the physician observes the veins. Care should be taken that the arm is not flexed excessively at either the shoulder or the elbow and that the upper arm is not constricted by clothing. Normally, the veins will be seen to collapse when the level of the dorsum of the hand reaches the sternal angle or the level of the suprasternal notch. The vertical distance above the sternal angle at which the veins collapse should be recorded as well as the position of the patient during the test.

Auscultation at the base of the neck, just above the clavicle and lateral to the clavicular attachment of the SCM muscle, with the patient sitting, enables one to determine if a **cervical venous hum** is present (Chapter 18). The cervical venous hum is a continuous whining or roaring noise throughout systole and diastole produced by the flow of blood through the internal jugular veins. It occurs more frequently on the right than on the left, but may be present bilaterally. It is loudest with the patient sitting, during inspiration, and in diastole. It may be increased by turning the head away from the side being auscultated. It is obliterated by applying light pressure directly above the point of auscultation, the Valsalva maneuver, compression of the internal jugular vein, or lying down. An arterial bruit, in contrast, is loudest in systole, unaffected by light pressure, the Valsalva maneuver, and the patient's changing position. The venous hum is a frequent finding in normal individuals, but may also be a clue to high-output states (e.g., thyrotoxicosis).

**PRECORDIAL MOVEMENTS AND THRILLS**

The precordial examination, performed next, consists of inspection and palpation of the anterior chest wall. **Precordial movements** (Chapter 21) should be evaluated at the apex (left ventricle), lower left parasternal edge (right ventricle), upper left (pulmonary artery) and upper right (aorta) parasternal edges, and epigastric and sternoclavicular areas (Figure 7.6). It is best to examine the precordium with the patient supine because if the patient is turned on the left side, the apical region of the heart is displaced against the chest wall by positioning yourself on the patient's right side and looking tangentially across the fourth, fifth, and sixth intercostal spaces. Ask the patient to take a deep breath and then to exhale slowly as you look for a discrete area of apical movement. The following are the factors to be considered about any precordial movement that can be seen or felt: (1) location; (2) amplitude; (3) duration; (4) time of the impulse in the cardiac cycle; and (5) contour.

**Locate the apex impulse** by placing the palm and fingers of your right hand over the left precordium in the fourth, fifth, and sixth intercostal spaces near the midclavicular line (Figure 7.7). If unable to palpate an impulse, move your hand laterally to the anterior axillary line. If still unable to locate the impulse, ask the patient to roll onto the left side and then to exhale slowly as you look for a discrete area of the left precordium. On rare occasions the impulse may be more important than its location and size; therefore, state whether the impulse is of normal, increased, or diminished force and whether it occupies half or more than half of systole. If necessary, palpate your own apex impulse for comparison. Try to detect by both inspection and palpation any abnormal pulsations that the underlying cardiac chambers and great vessels may produce. Auscultation is routinely performed over each of these areas. The aortic area is the second intercostal space to the right of the sternum. The pulmonary area is the second left intercostal space to the left of the sternum. The parasternal or right ventricular area is the lower half of the sternum on the left. The apical or left ventricular area is the fifth intercostal space at the mid clavicular line. The epigastric area is just below the xiphisternum (subxyphoid area).

Figure 7.6
**Anteroposterior view of the chest indicating the major precordial areas to be examined.**

Try to detect by both inspection and palpation any abnormal pulsations that the underlying cardiac chambers and great vessels may produce. Auscultation is routinely performed over each of these areas. The aortic area is the second intercostal space to the right of the sternum. The pulmonary area is the second left intercostal space to the left of the sternum. The parasternal or right ventricular area is the lower half of the sternum on the left. The apical or left ventricular area is the fifth intercostal space at the mid clavicular line. The epigastric area is just below the xiphisternum (subxyphoid area).

and attempt to palpate the apex as just described (Figure 7.8). Always state in which patient position the apex impulse was identified because the left lateral decubitus position distorts a normal apex and makes it appear or feel unduly sustained. If still unable to locate the apex impulse, inspect the right precordium in a manner similar to that used for the left precordium. On rare occasions the impulse may be visible in the right chest, providing the initial clue to the presence of dextrocardia.

Record the location of the apex impulse by noting its distance from either the midsternal or midclavicular line and the intercostal space in which it is felt. Also record the approximate diameter (cm) of the apex. The amplitude and duration of the apical pulsation may be more important than its location and size; therefore, state whether the impulse is of normal, increased, or diminished force and whether it occupies half or more than half of systole. If necessary, palpate your own apex impulse for comparison. In addition, if a single pulsation is palpated, determine whether its force is uniform throughout systole or whether there is a late systolic accentuation or bulge.

In a normal individual the apex impulse is a tapping, early systolic outward thrust that occurs in the fifth intercostal space in the midclavicular line. It is localized to a small area not more than 1 to 2 cm (dime-sized) in diameter. The outward thrust is brief, lasting about one-half of systole, and is of minimal amplitude. Normally, diastolic pulsations are not palpable. Left ventricular hypertrophy, for instance, results in exaggeration of the normal left ventricular thrust in both systole and diastole. It is nondisplaced, sustained (occupying more than one-half of systole), and 2 to...
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Figure 7.7
Examination of the apex impulse in the supine position.
Place the patient in the supine position elevating the trunk approximately 30–45 degrees. Position yourself on the patient’s right side and place the flat part of your right hand over the left precordium in the fourth, fifth, and sixth intercostal spaces near the mid clavicular line. First locate the apex impulse with the distal half of the palm of your hand and its adjacent proximal phalanges. The full extent of a broad apex impulse is best appreciated with the palm. Use the tips of the first and second fingers placed directly on the impulse to make detailed observations of the impulse. Assess the location, diameter and duration of the impulse.

3 cm (quarter-sized). Left ventricular dilation, in contrast, results in downward and lateral displacement of the apex below the fifth interspace.

The lower left parasternal region of the chest is best evaluated by looking at the chest from the side and by placing the heel of the hand over or just to the left of the sternum (Figure 7.9). If a left parasternal impulse or lift is appreciated, determine if the impulse is sustained throughout systole, vigorous or slight, and begins with or after the onset of the apex impulse. Determine its onset by placing your right hand over the apex impulse and your left palm over the left parasternal impulse. The right ventricle is really an anterior structure, and when enlarged, it may lift the anterior portion of the chest including the sternum. In normal individuals the parasternal region usually retracts (moves inward) during ejection and is not palpable.

Right ventricular hypertrophy and/or dilation results in a sustained systolic lift of the lower parasternal region of the chest. This lift is present in patients with high right ventricular pressure (pulmonary arterial hypertension or pulmonic stenosis) or volume overload (tricuspid regurgitation or atrial septal defect), but is infrequently seen in chronic lung disease if right-sided heart failure is not present. The pulsations of the main pulmonary artery are also often visible and palpable, especially in patients with parasternal pulsations.

Patients with acute myocardial infarction or with angina pectoris may have an outward paradoxical precordial movement that often can be seen or palpated at the apex, the anterior precordium, or in an “ectopic” area. The impulse is usually sustained throughout systole, frequently with a second systolic bulge, and is often difficult to distinguish from that of left ventricular hypertrophy by palpation alone if the bulge occurs in the region of the apex. In order to time systolic and diastolic cardiac movements accurately, it is essential to inspect and palpate the precordium while actually listening to the heart sounds.

The early diastolic and late diastolic (presystolic) precordial movements are the visible and palpable counterparts of the third (S₃) and fourth (S₄) heart sounds, respectively. They may be felt at the cardiac apex with the patient in the left lateral decubitus position. Careful inspection of the precordium with the naked eye or observing the motions of a wooden stick taped over the cardiac apex best demonstrates any precordial impulses that correlate with audible S₃ and S₄ sounds. The right-sided S₄, seen and felt at the lower left sternal edge, occurs slightly earlier in diastole than the left-sided S₃. It often correlates with a very prominent jugular “a” wave and a sustained outward movement (lift) in the lower left parasternal area.

Heart murmurs that can be palpated are referred to as thrills (Chapters 26 and 27). The diastolic rumble of mitral stenosis and the systolic murmur of mitral regurgitation may be palpated at the cardiac apex. The harsh systolic murmur of aortic stenosis may cut across the palm of the hand toward the right side of the neck, while the thrill of...
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Figure 7.8
Examination of the apex impulse with the patient in the left lateral decubitus position.
If the apex impulse cannot be located with the patient supine, ask the patient to roll to his
left side and attempt to palpate the apex with the fingers of your right hand placed over the
left precordium in the fourth, fifth, and sixth intercostal spaces near the mid clavicular line.
This maneuver helps find the apical impulse, but since it displaces the impulse laterally, it
should not be used to identify its true location. When the impulse is palpable, access its quality
by making finer observations with your fingertips; this position, however, also occasionally
distorts a normal apex into feeling unduly sustained.

Pulmonic stenosis cuts across the palm of the hand to the
left side of the neck. The thrill due to ventricular septal
defect is usually located in the third and fourth intercostal
spaces near the left sternal border. Heart sounds may also
be palpable. For instance, the loud S, of mitral stenosis may
be palpated at the apex.

HEART SOUNDS AND MURMURS

Auscultation (Chapters 22–28) of the heart is performed
after examining the jugular venous pulse, carotid pulse, and
precordial movements because acoustic events can best be
interpreted after the other components of the cardiac ex-
amination have been evaluated. Auscultation of the heart
should, therefore, not be performed as an isolated event
because heart sounds, murmurs and pulse tracings must all
be integrated in order to understand normal and altered
cardiac physiology and anatomy. Attempt to study two and
occasionally three aspects of the cardiac examination si-
multaneously. Then pictorially display the heart sounds and
correlate them, by use of a diagram, with any murmurs
heard, the jugular venous wave form, the carotid pulse, and
the apex impulse to best understand the patient’s cardiac
problem (Figure 7.2).

Proper use of a quality stethoscope is essential for an
accurate auscultatory examination. The important parts of
the stethoscope are the ear pieces, the tubing, and the chest
pieces. The ear pieces must fit the ear canal snugly without
going to an uncomfortable depth. The tubing should be as
short as possible, but long enough to be comfortable and
convenient for the user; 10 to 12 inches is an ideal length.
The chest pieces, the bell, and the diaphragm should be
combined into one housing. A diaphragm pressed firmly
on the chest filters out low-frequency vibrations and ampli-
ifies high-frequency vibrations. It is routinely used to hear
the first and second heart sounds, systolic murmurs, and
the diastolic murmur of aortic regurgitation. The bell should
be applied to the chest with very light pressure, barely cre-
ating an air seal, so low-frequency sounds and murmurs are
appreciated. It is used to hear the third and fourth heart
sounds and the diastolic murmur (rumble) of mitral ste-
nosis. A trumpet-shaped bell is much better than a shallow
one.

Auscultation usually begins at the aortic area (upper right
sternal edge). The stethoscope is then moved sequentially
to the pulmonary (upper left sternal edge), tricuspid (lower
left sternal edge), and mitral (apex) areas. It is helpful to
palpate the carotid pulse or apex impulse simultaneously
to time the acoustic events as systolic or diastolic. A finger
on the carotid artery will sense the systolic thrust that is
virtually coincident with S,. Use of a more distant artery for
this purpose leads to error because of the time it takes the
pulse wave to reach the periphery.

The heart sounds are usually the first auscultatory events
identified. They are brief, discrete, auditory vibrations of
varying intensity (loudness) and frequency (pitch). The first
heart sound (Chapter 22) identifies the onset of systole. It is
normally split into mitral (M,) and tricuspid (T,) com-
ponents that are temporally related to closure of the respec-
tive atrioventricular (A-V) valves. The second heart sound (Chap-
ter 23) identifies the end of systole and the onset of diastole.
It is normally split into aortic (A2) and pulmonic (P2) com-
ponents that are temporally related to closure of the re-
7. AN OVERVIEW OF THE CARDIOVASCULAR SYSTEM

Figure 7.9
Palpation of the left parasternal impulse.
Place the patient in the supine position with the trunk elevated 30–45 degrees. Place the heel of your right hand, with the hand slightly cocked up, together with downward pressure (arrow) of your left hand over the lower left sternal area to best feel the slight left parasternal upward movement that usually reflects right ventricular action. If no movement is felt, ask the patient to take a deep breath, exhale slowly and then hold the breath during expiration, before repeating the hand maneuvers.

Have the patient exercise (5 to 10 sit-ups), or cough if myocardial infarction is suspected, to "bring out" the murmur of mitral stenosis and the $S_2$ and $S_3$ gallop sounds. The third and fourth heart sounds (Chapters 24 and 25) are low pitched and may be audible as well as palpable. They are best heard at the cardiac apex when generated from the left ventricle and at the lower left sternal edge when generated from the right ventricle.

The technique known as inching may help determine whether extra cardiac sounds are systolic or diastolic. The examiner moves the stethoscope inch by inch from the aortic area to the apex while focusing attention on $S_2$. Determine whether the extra sound precedes (systolic) or follows (diastolic) $S_2$. The acoustic events can best be appreciated by a careful review of the hemodynamic curves of the cardiac cycle.

Several additional areas should be ausculted (Figure 7.10), including (1) the inferior edge of the sternum and epigastrium, especially in patients with chronic obstructive lung disease, in whom heart sounds and murmurs are difficult to hear in the more conventional listening areas; (2) the first and second intercostal spaces below the left midclavicular area for the continuous (systolic and diastolic) murmur of patent ductus arteriosus; (3) the interscapular area of the back for the systolic murmur of coarctation of the aorta; (4) over the lung areas; (5) over the head, eyes, liver, sacrum, abdomen, as well as over tumors or bony overgrowths; and (6) over all scars to identify the continuous murmur of a peripheral A-V fistula.

spective semilunar valves. The second sound is louder than $S_1$ in the aortic listening area; $S_2$ is louder at the apex.

In the aortic area, listen for the first and second heart sounds, the aortic ejection sound, and the murmurs of aortic stenosis and aortic regurgitation with the stethoscope diaphragm. Then move the diaphragm to the second and third left intercostal spaces in order to appreciate the aortic and pulmonic components of $S_2$, the pulmonary ejection sound, and the murmurs of pulmonic stenosis and pulmonary and aortic regurgitation. Continue to the fourth and fifth intercostal spaces along the left sternal border and listen for the murmurs of tricuspid regurgitation and tricuspid stenosis and the murmur produced by ventricular septal defect. Right ventricular $S_2$ and $S_3$ sounds (gallops) can best be heard in this area with the stethoscope bell. The diaphragm should be moved to the third and fourth intercostal spaces to the right of the sternum to judge whether a murmur of aortic regurgitation is louder along the left or right sternal border.

The diaphragm should then be positioned at the cardiac apex where $S_1$ and $S_2$ should be studied again. An aortic ejection sound, a mid to late systolic click, the opening snap of mitral stenosis, the systolic murmur of mitral regurgitation, and occasionally the high-pitched murmur of aortic regurgitation are also heard in this area with the diaphragm.

The bell of the stethoscope should then be positioned over the apical impulse with the patient turned to the left lateral position to listen for low-frequency diastolic sounds and murmurs.
Examination of the Peripheral Arteries

The evaluation of the peripheral arterial pulses (Chapter 30) is an integral part of the cardiovascular examination. Palpation of the peripheral arteries may yield the following information: (1) frequency and regularity of the pulsations; (2) condition and patency of the peripheral arteries; and (3) characteristics of the arterial pulse wave. All of the following pulses should be examined thoroughly by palpation: temporalis, brachials, radials, aortic, femorals, popliteals, posterior tibials, and the dorsalis pedis (Figure 7.11). When applicable, pulses should be examined bilaterally and graded as to quality of the impulse on a scale of 0 to 3 with 2 being normal, 0 absent, 1 decreased, and 3 increased. In addition, hair distribution should be checked on the toes and feet.

To examine the brachial artery, palpate along the course of the artery just medial to the biceps tendon and lateral to the medial epicondyle of the humerus. To examine the radial artery, palpate along the radial—volar aspect of the subject’s forearm at the wrist.

The abdominal aorta is an upper abdominal, retroperitoneal structure that is best palpated by applying firm pressure with the flattened fingers of both hands to indent the epigastrium toward the vertebral column. The normal aortic pulse should be less than 6 cm in diameter. Auscultation should be performed over the aorta and along both iliac arteries into the lower abdominal quadrants to detect bruits.

The femoral artery is best palpated with the fingertips of the examining hand pressed firmly into the groin. Auscultation should be performed in this area as well.

The popliteal artery passes vertically through the deep portion of the popliteal space. It may be difficult or impossible to palpate in obese or very muscular individuals. The pulse is detected by pressing deeply into the popliteal space with the supporting fingertips.

The posterior tibial artery lies just posterior to the medial malleolus and is felt most readily by curling the fingers of the examining hand around the ankle.

The dorsalis pedis artery usually lies near the center of the long axis of the foot. Place the fingertips transversely across the dorsum of the forefoot near the ankle. This pulse often requires some searching and is congenitally absent in approximately 10% of normal individuals.

Information should be obtained by simultaneously palpating the radial and femoral arteries and noting the relative time of onset of the pulse at the two locations. Normally, the pulse wave arrives in these locations simultaneously. In a patient with coarctation of the aorta, both onset and peak of the weak femoral pulses are delayed. Compared to the carotid pulse, the pulse wave in these more peripheral vessels arrives later and is characterized by a steeper initial wave that reaches a higher systolic peak, whereas the diastolic and mean arterial pressures are lower.

Palpating peripheral arteries is the most important single maneuver in establishing whether or not chronic occlusive arterial disease is present. One may obtain an impression of thickness and hardness of the walls of the brachial, radial, and dorsalis pedis arteries by “rolling” the compressed vessel against the underlying tissue. Auscultation of the peripheral arteries is also very important. A systolic bruit over the abdominal aorta, iliac, or femoral arteries when the patient has been supine for more than 10 minutes usually signifies intimal disease, but not necessarily significant occlusion.

Pulsus alternans refers to a characteristic pulse pattern in which the beats occur at regular intervals, but with an alternation of the height of the pulse. This valuable sign should be searched for by palpating the femoral artery; less often, the radial artery may disclose that alternate pulses vary in amplitude. Have the patient hold his or her breath while...
you feel the pulse to make certain that the alternation of pulse volume is independent of respiration. The pulse rhythm should be regular, since alternation of the strength of cardiac beats commonly results from bigeminal rhythm. Rarely, pulsus alternans may feel irregular because of a slight delay in sensing the weaker beat.

When pulsus alternans is prominent, it may be confirmed and quantified by use of a sphygmomanometer. As the pressure is lowered in the sphygmomanometer, the examiner should routinely observe whether every Korotkoff sound is heard with equal intensity. As the cuff pressure is lowered further, the frequency of the sounds may suddenly double as the weaker beats also become audible. In most instances, the weak pulses are only slightly weaker than the strong beats. On rare occasions, a weak beat may be so small that no palpable pulse is detected at the periphery, so-called total alternans. Pulsus alternans is produced by an alternation in left ventricular contractile force associated with an alternation of left ventricular fiber length; it is a very valuable sign of left ventricular dysfunction. In most instances, it is found in association with a third heart sound.

Examination for Thrombophlebitis
The presence or absence of thrombophlebitis (Chapter 30) is an integral part of the cardiovascular database. Thrombophlebitis refers to venous inflammation with secondary thrombosis of the involved vein. It is most commonly noted in the deep veins of the leg and superficial veins of the arm. Superficial thrombophlebitis can be seen and felt, making the diagnosis easy in most cases. Most deep vein thromboses are clinically silent and cannot be detected by routine examination. When thrombophlebitis is confined to small venous channels beneath the subcutaneous tissue or in the pelvis, the lesions are neither visible nor palpable. Mild pain in the calf may be the only symptom, and tenderness may be the only sign.

The examiner should also compare the skin temperature in the two calves, since active phlebitis may create local warmth. Calf circumference should also be determined in all suspected cases. Pain is a prominent feature of muscular, synovial, or vascular leg diseases, and various tests have been used to identify the specific etiology. Homans' test (dorsiflexion sign) is used to detect irritability of the posterior leg muscles through which inflamed or thrombosed veins course. The Lowenberg cuff test is another helpful clinical maneuver for detection of calf vein thrombosis.

A summary of only the most common conditions associated with an abnormality on the cardiovascular examination is shown in Table 7.4.
### Table 7.4
Common Conditions Associated with an Abnormality on the Cardiovascular Examination

<table>
<thead>
<tr>
<th>Abnormality</th>
<th>Condition</th>
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<tr>
<td>Hypokinetis</td>
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<td>Hypokinetis</td>
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<tr>
<td>Deep “x” and “y” descents</td>
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<td>Mitral regurgitation</td>
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<td></td>
<td>Coarctation of aorta</td>
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<td>Left ventricular dysfunction</td>
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<tr>
<td></td>
<td>Thrombophlebitis</td>
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</table>

**Conclusion**

An orderly process of history taking and physical examination together with selective application of modern laboratory technology should enable the clinician to arrive at an accurate diagnosis, estimate the degree of severity, formulate a logical plan of treatment, and better understand the pathophysiologic abnormalities in the patient with a cardiovascular problem.