CHAPTER 8

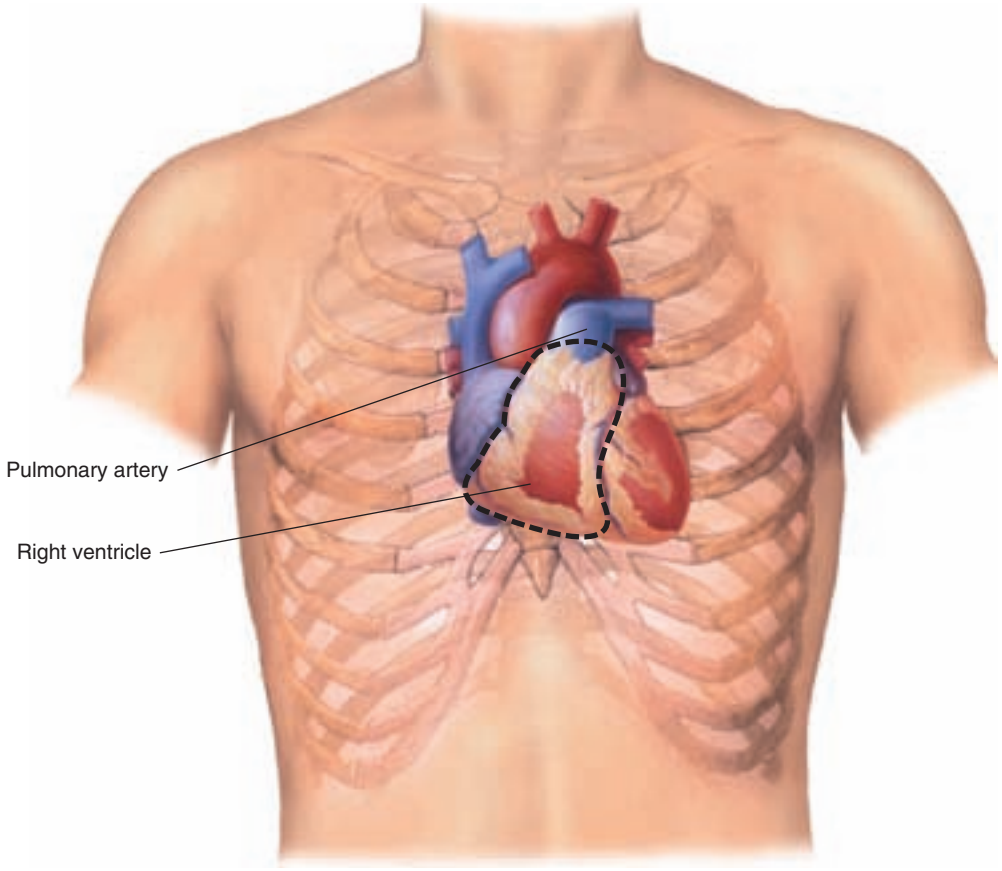
The Cardiovascular System

ANATOMY AND PHYSIOLOGY

SURFACE PROJECTIONS OF THE HEART AND GREAT VESSELS

Understanding cardiac anatomy and physiology is particularly important in the examination of the cardiovascular system. Learn to visualize the underlying structures of the heart as you examine the anterior chest.

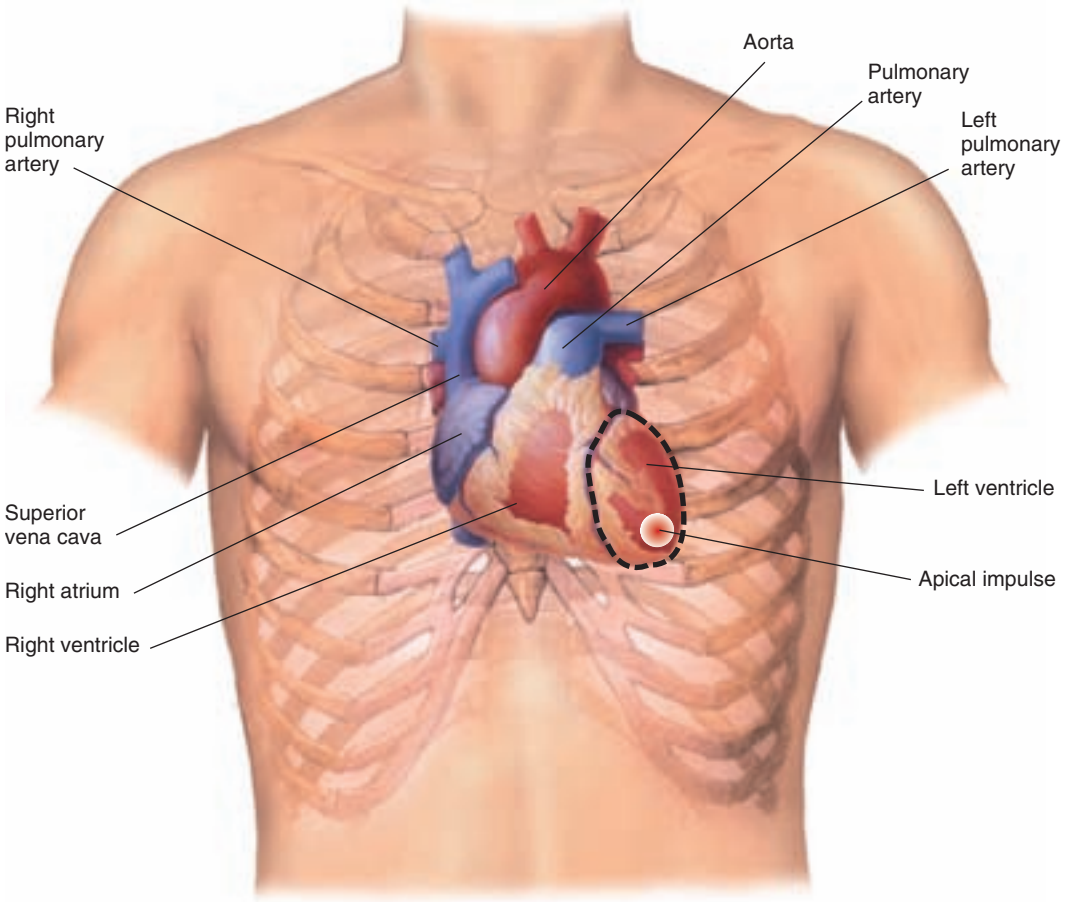
Note that the *right ventricle* occupies most of the anterior cardiac surface. This chamber and the pulmonary artery form a wedgelike structure behind and to the left of the sternum, outlined in red.



The inferior border of the right ventricle lies below the junction of the sternum and the xiphoid process. The right ventricle narrows superiorly and meets the pulmonary artery at the level of the sternum or "base of the heart"—a clinical term that refers to the proximal surface of the heart at the right and left 2nd interspaces close to the sternum.

The *left ventricle*, behind the right ventricle and to the left, as outlined, forms the left lateral margin of the heart. Its tapered inferior tip is often termed the cardiac "apex." It is clinically important because it produces the *apical impulse*, sometimes called the *point of maximal impulse*, or *PMI*. This impulse locates the left border of the heart and is usually found in the 5th interspace 7 cm to 9 cm lateral to the midsternal line. It is approximately the size of a quarter, roughly 1 to 2.5 cm in diameter. Because the most prominent cardiac impulse may not be apical, some authorities discourage use of the term PMI.

The right heart border is formed by the *right atrium*, a chamber not usually identifiable on physical examination. The *left atrium* is mostly posterior and cannot be examined directly, although its small atrial appendage may make up a segment of the left heart border between the pulmonary artery and the left ventricle.

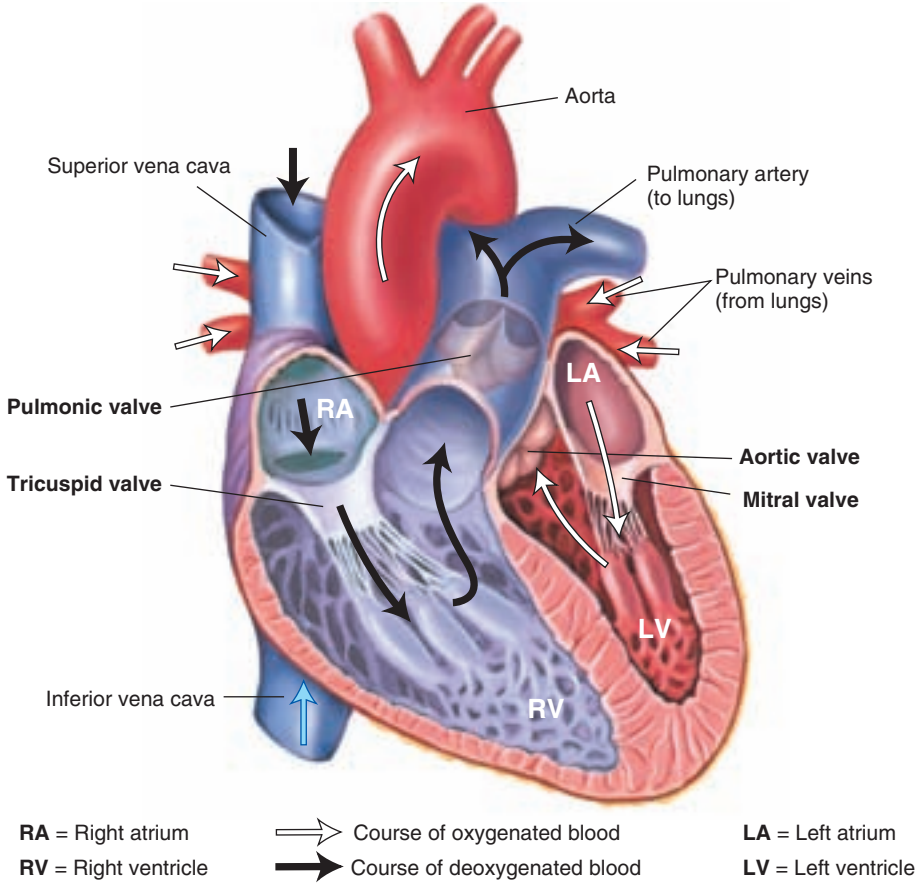


Above the heart lie the great vessels. The *pulmonary artery,* already mentioned, bifurcates quickly into its left and right branches. The *aorta* curves upward from the left ventricle to the level of the sternal angle, where it arches backward to the left and then down. On the right, the superior vena cava empties into the right atrium.

Although not illustrated, the inferior vena cava also empties into the right atrium. The *superior* and *inferior venae cavae* carry venous blood to the heart from the upper and lower portions of the body.

CARDIAC CHAMBERS, VALVES, AND CIRCULATION

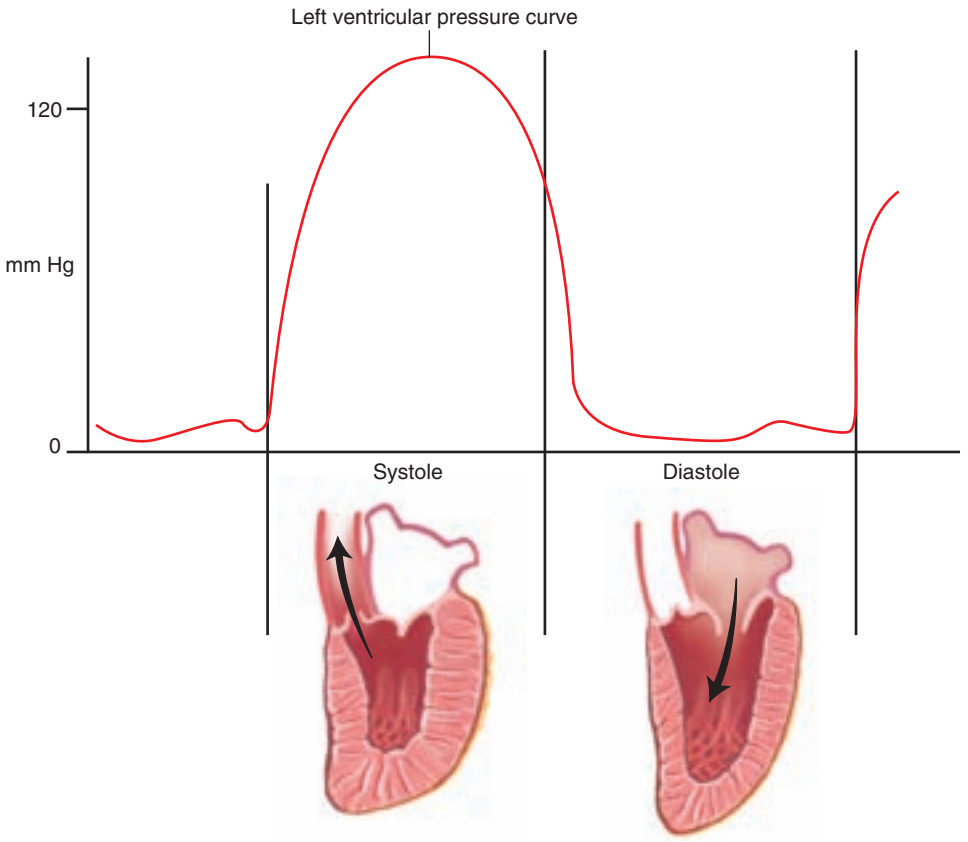
Circulation through the heart is shown in the diagram below, which identifies the cardiac chambers, valves, and direction of blood flow. Because of their positions, the *tricuspid* and *mitral valves* are often called *atrioventricular valves.* The *aortic* and *pulmonic valves* are called *semilunar valves* because each of their leaflets is shaped like a half moon. Although this diagram shows all valves in an open position, they do not open simultaneously in the living heart.



As the heart valves close, the heart sounds arise from vibrations emanating from the leaflets, the adjacent cardiac structures, and the flow of blood. It is essential to understand the positions and movements of the valves in relation to events in the cardiac cycle.

EVENTS IN THE CARDIAC CYCLE

The heart serves as a pump that generates varying pressures as its chambers contract and relax. *Systole is the period of ventricular contraction.* In the diagram below, pressure in the left ventricle rises from less than 5 mm Hg in its resting state to a normal peak of 120 mm Hg. After the ventricle ejects much of its blood into the aorta, the pressure levels off and starts to fall. *Diastole is the period of ventricular relaxation.* Ventricular pressure falls further to below 5 mm Hg, and blood flows from atrium to ventricle. Late in diastole, ventricular pressure rises slightly during inflow of blood from atrial contraction.

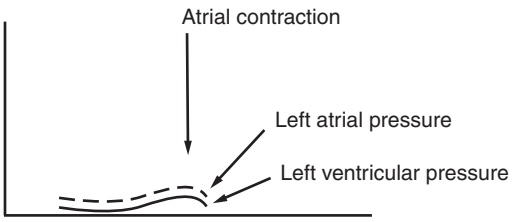


Note that during *systole* the aortic valve is open, allowing ejection of blood from the left ventricle into the aorta. The mitral valve is closed, preventing blood from regurgitating back into the left atrium. In contrast, during *diastole* the aortic valve is closed, preventing regurgitation of blood from the aorta back into the left ventricle. The mitral valve is open, allowing blood to flow from the left atrium into the relaxed left ventricle.

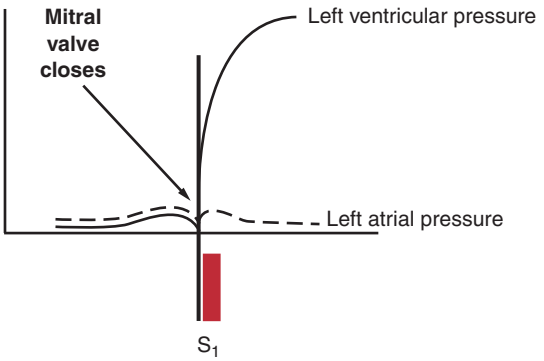
Understanding the interrelationships of the *pressure gradients* in these three chambers—left atrium, left ventricle, and aorta—together with the position and movement of the valves is fundamental to understanding heart sounds. Trace these changing pressures and sounds through one cardiac cycle. Note

that during auscultation the first and second heart sounds define the duration of *systole* and *diastole*. An extensive literature deals with the exact causes of heart sounds. Possible explanations include actual closure of valve leaflets, tensing of related structures, leaflet positions and pressure gradients at the time of atrial and ventricular systole, and the effects of columns of blood. The explanations given here are oversimplified but retain clinical usefulness.

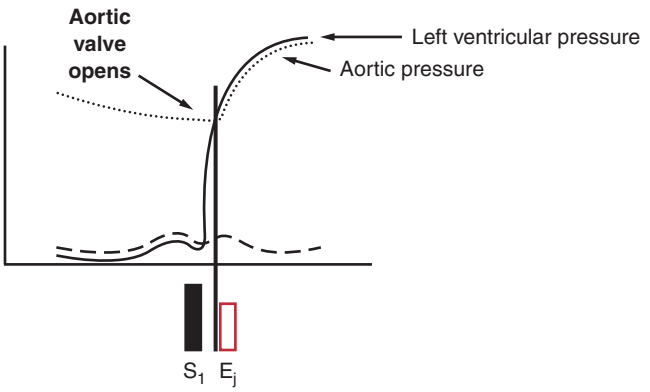
During *diastole*, pressure in the blood-filled left atrium slightly exceeds that in the relaxed left ventricle, and blood flows from left atrium to left ventricle across the open mitral valve. Just before the onset of ventricular systole, atrial contraction produces a slight pressure rise in both chambers.



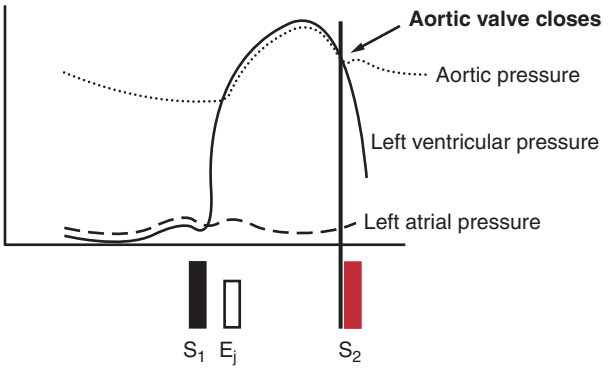
During *systole*, the left ventricle starts to contract and ventricular pressure rapidly exceeds left atrial pressure, thus shutting the mitral valve. *Closure of the mitral valve produces the first heart sound, S_1.*



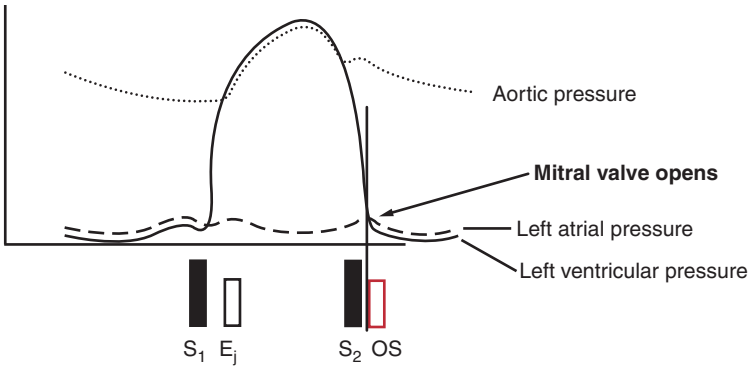
As left ventricular pressure continues to rise, it quickly exceeds the pressure in the aorta and forces the aortic valve open. In some pathologic conditions, opening of the aortic valve is accompanied by an early systolic ejection sound (Ej). *Normally, maximal left ventricular pressure corresponds to systolic blood pressure.*



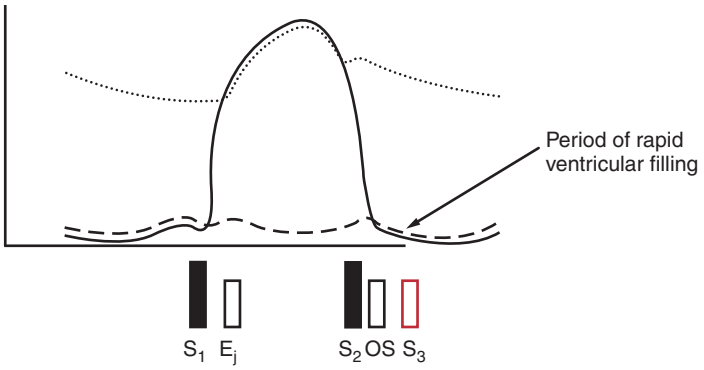
As the left ventricle ejects most of its blood, ventricular pressure begins to fall. When left ventricular pressure drops below aortic pressure, the aortic valve shuts. *Aortic valve closure produces the second heart sound, S_2,* and another diastole begins.



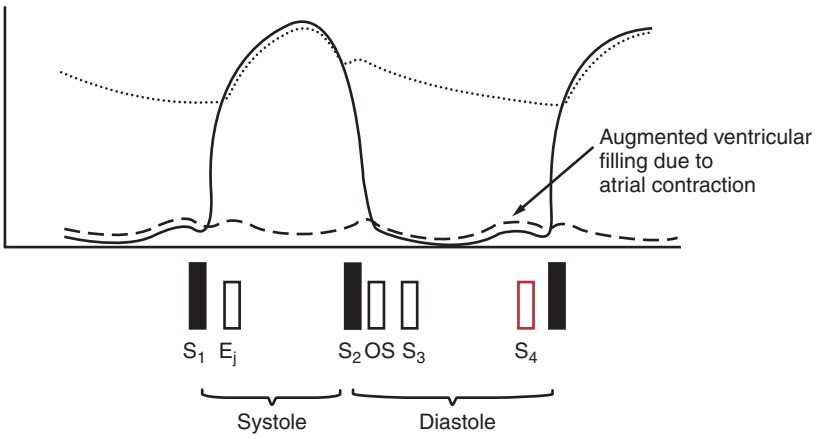
In *diastole*, left ventricular pressure continues to drop and falls below left atrial pressure. The mitral valve opens. This is usually a silent event, but may be audible as a pathologic opening snap (OS) if valve leaflet motion is restricted, as in mitral stenosis.



After the mitral valve opens, there is a period of rapid ventricular filling as blood flows early in diastole from left atrium to left ventricle. In children and young adults, a third heart sound, S_3, may arise from rapid deceleration of the column of blood against the ventricular wall. In older adults, an S_3, sometimes termed "an S_3 gallop," usually indicates a pathologic change in ventricular compliance.



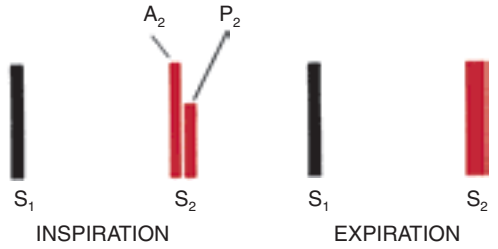
Finally, although not often heard in normal adults, a fourth heart sound, S_4, marks atrial contraction. It immediately precedes S_1 of the next beat, and also reflects a pathologic change in ventricular compliance.



THE SPLITTING OF HEART SOUNDS

While these events are occurring on the left side of the heart, similar changes are occurring on the right, involving the right atrium, right ventricle, tricuspid valve, pulmonic valve, and pulmonary artery. Right ventricular and pulmonary arterial pressures are significantly lower than corresponding pressures on the left side. Furthermore, right-sided events usually occur slightly later than those on the left. Instead of a single heart sound, you may hear two discernible components, the first from left-sided aortic valve closure, or A_2, and the second from right-sided closure of the pulmonic valve, or P_2.

Consider the second heart sound and its two components, A_2 and P_2, which come from closure of the aortic and pulmonic valves, respectively. During inspiration A_2 and P_2 separate slightly, and may split S_2 into its two audible components. During expiration, these two components are fused into a single sound, S_2.



Current explanations of inspiratory splitting cite increased capacitance in the pulmonary vascular bed during inspiration, which prolongs ejection of blood from the right ventricle, delaying closure of the pulmonic valve, or P_2. Ejection of blood from the left ventricle is comparatively shorter, so A_2 occurs slightly earlier.

Of the two components of the second heart sound, A_2 is normally louder, reflecting the high pressure in the aorta. It is heard throughout the precordium. P_2, in contrast, is relatively soft, reflecting the lower pressure in the pulmonary artery. It is heard best in its own area—the 2nd and 3rd left interspaces close to the sternum. It is here that you should search for splitting of the second heart sound.

S_1 also has two components, an earlier mitral and a later tricuspid sound. The mitral sound, its principal component, is much louder, again reflecting the high pressures on the left side of the heart. It can be heard throughout the precordium and is loudest at the cardiac apex. The softer tricuspid component is heard best at the lower left sternal border, and it is here that you may hear a split S_1. The earlier louder mitral component may mask the tricuspid sound, however, and splitting is not always detectable. Splitting of S_1 does not vary with respiration.

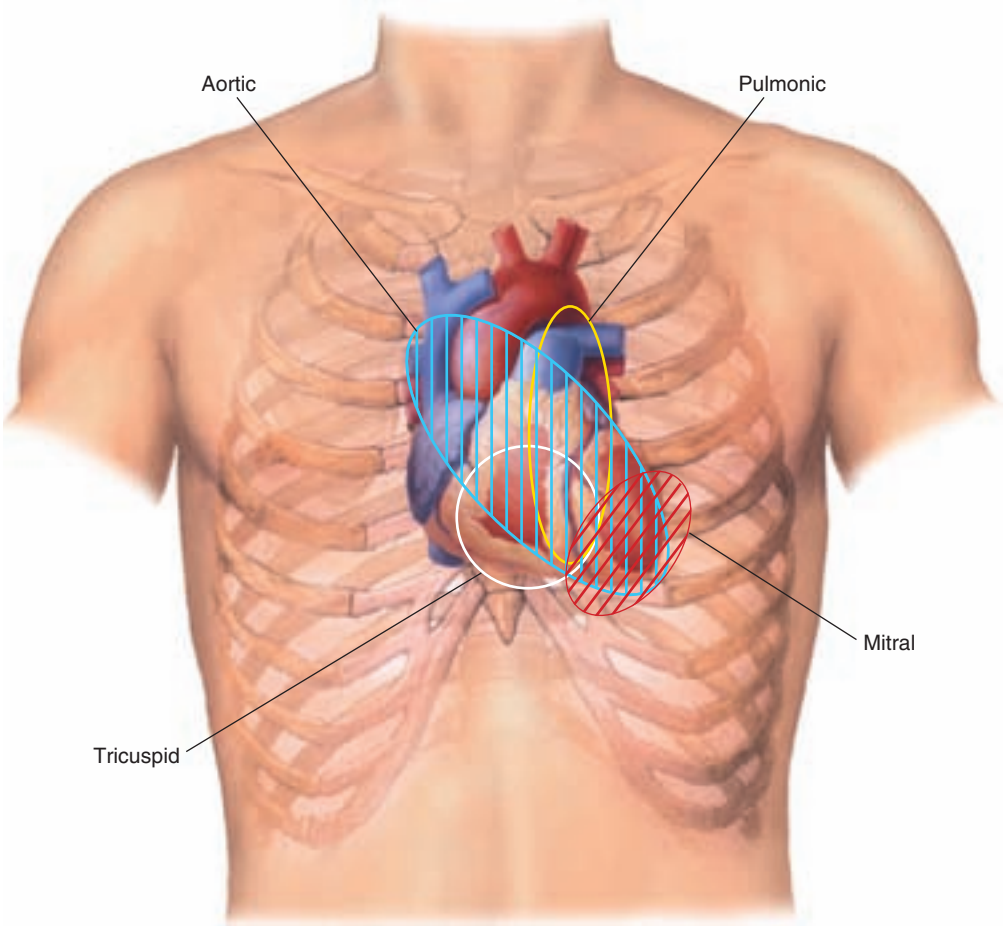
HEART MURMURS

Heart murmurs are distinguishable from heart sounds by their longer duration. They are attributed to turbulent blood flow and may be "innocent," as with flow murmurs of young adults, or diagnostic of valvular heart disease. A *stenotic valve* has an abnormally narrowed valvular orifice that obstructs blood flow, as in *aortic stenosis,* and causes a characteristic murmur. So does a valve that fails to fully close, as in *aortic regurgitation* or *insufficiency.* Such a valve allows blood to leak backward in a retrograde direction and produces a *regurgitant* murmur.

To identify murmurs accurately, you must learn to assess the chest wall location where they are best heard, their timing in systole or diastole, and their qualities. In the section on Techniques of Examination, you will learn to integrate several characteristics, including murmur intensity, pitch, duration, and direction of radiation (see pp. 316–319).

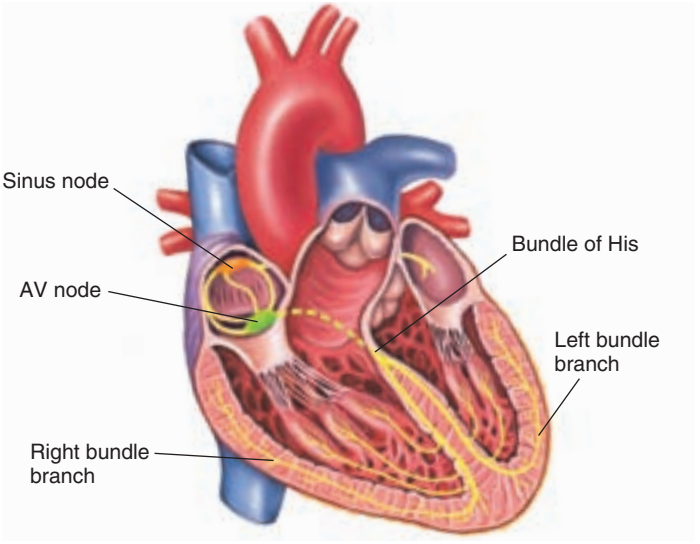
RELATION OF AUSCULTATORY FINDINGS TO THE CHEST WALL

The locations on the chest wall where you hear heart sounds and murmurs help to identify the valve or chamber where they originate. Sounds and murmurs arising from the mitral valve are usually heard best at and around the cardiac apex. Those originating in the tricuspid valve are heard best at or near the lower left sternal border. Murmurs arising from the pulmonic valve are usually heard best in the 2nd and 3rd left interspaces close to the sternum, but at times may also be heard at higher or lower levels, and those originating in the aortic valve may be heard anywhere from the right 2nd interspace to the apex. These areas overlap, as illustrated below, and you will need to correlate auscultatory findings with other portions of the cardiac examination to identify sounds and murmurs accurately.



THE CONDUCTION SYSTEM

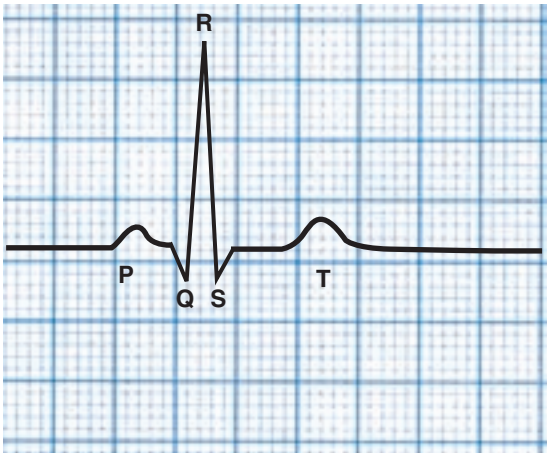
An electrical conduction system stimulates and coordinates the contraction of cardiac muscle.



Each normal electrical impulse is initiated in the *sinus node*, a group of specialized cardiac cells located in the right atrium near the junction of the vena cava. The sinus node acts as the cardiac pacemaker and automatically discharges an impulse about 60 to 100 times a minute. This impulse travels through both atria to the *atrioventricular node*, a specialized group of cells located low in the atrial septum. Here the impulse is delayed before passing down the bundle of His and its branches to the ventricular myocardium. Muscular contraction follows: first the atria, then the ventricles. The normal conduction pathway is diagrammed in simplified form above.

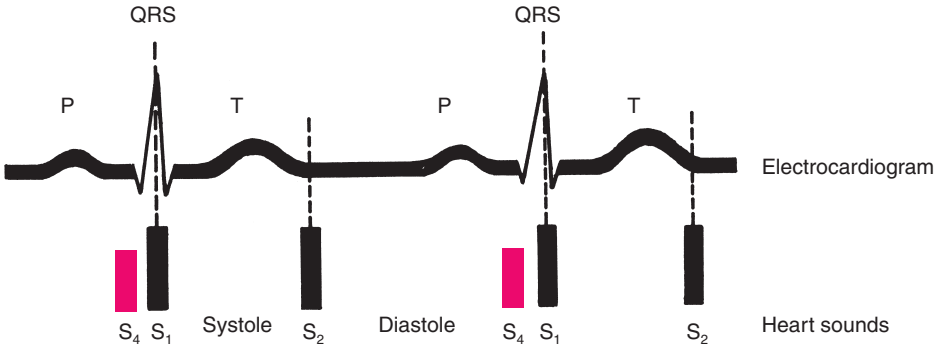
The electrocardiogram, or ECG, records these events. Contraction of cardiac smooth muscle produces electrical activity, resulting in a series of waves on the ECG. The components of the *normal ECG* and their duration are briefly summarized here, but you will need further instruction and practice to interpret recordings from actual patients. Note:

- The small *P wave* of atrial depolarization (duration up to 80 milliseconds; *PR interval* 120 to 200 milliseconds)



- The larger *QRS complex* of ventricular depolarization (up to 100 milliseconds), consisting of one or more of the following:
  - –the *Q wave*, a downward deflection from septal depolarization
  - –the *R wave*, an upward deflection from ventricular depolarization
  - –the *S wave*, a downward deflection following an R wave
- A *T wave* of ventricular repolarization, or recovery (duration relates to QRS).

The electrical impulse slightly precedes the myocardial contraction that it stimulates. The relation of electrocardiographic waves to the cardiac cycle is shown below.



THE HEART AS A PUMP

The left and right ventricles pump blood into the systemic and pulmonary arterial trees, respectively. *Cardiac output,* the volume of blood ejected from each ventricle during 1 minute, is the product of *heart rate* and *stroke volume*. Stroke volume (the volume of blood ejected with each heartbeat) depends in turn on preload, myocardial contractility, and afterload.

Preload refers to the load that stretches the cardiac muscle before contraction. The volume of blood in the right ventricle at the end of diastole, then, constitutes its preload for the next beat. Right ventricular preload is increased by increasing venous return to the right heart. Physiologic causes include inspiration and the increased volume of blood flow from exercising muscles. The increased blood volume in a dilated right ventricle of congestive heart failure also increases preload. Causes of decreased right ventricular preload include exhalation, decreased left ventricular output, and pooling of blood in the capillary bed or the venous system.

Myocardial contractility refers to the ability of the cardiac muscle, when given a load, to shorten. Contractility increases when stimulated by action of the sympathetic nervous system, and decreases when blood flow or oxygen delivery to the myocardium is impaired.

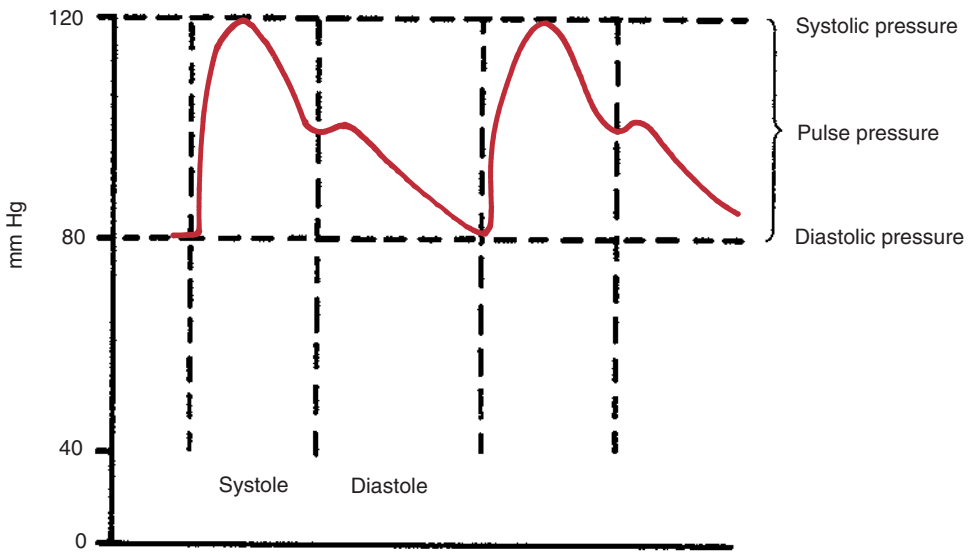
Afterload refers to the degree of vascular resistance to ventricular contraction. Sources of resistance to left ventricular contraction include the tone in the walls of the aorta, the large arteries, and the peripheral vascular tree (primarily the small arteries and arterioles), as well as the volume of blood already in the aorta.

Pathologic increases in preload and afterload, called *volume overload* and *pressure overload,* respectively, produce changes in ventricular function that may be clinically detectable. These changes include alterations in ventricular impulses, detectable by palpation, and in normal heart sounds. Pathologic heart sounds and murmurs may also develop.

ARTERIAL PULSES AND BLOOD PRESSURE

With each contraction, the left ventricle ejects a volume of blood into the aorta and on into the arterial tree. The ensuing pressure wave moves rapidly through the arterial system, where it is felt as the *arterial pulse*. Although the pressure wave travels quickly—many times faster than the blood itself—a palpable delay between ventricular contraction and peripheral pulses makes the pulses in the arms and legs unsuitable for timing events in the cardiac cycle.

Blood pressure in the arterial system varies during the cardiac cycle, peaking in systole and falling to its lowest trough in diastole. These are the levels that are measured with the blood pressure cuff, or sphygmomanometer. The difference between systolic and diastolic pressures is known as the *pulse pressure*.



The principal factors influencing arterial pressure are:

- Left ventricular stroke volume
- Distensibility of the aorta and the large arteries
- Peripheral vascular resistance, particularly at the arteriolar level
- Volume of blood in the arterial system.

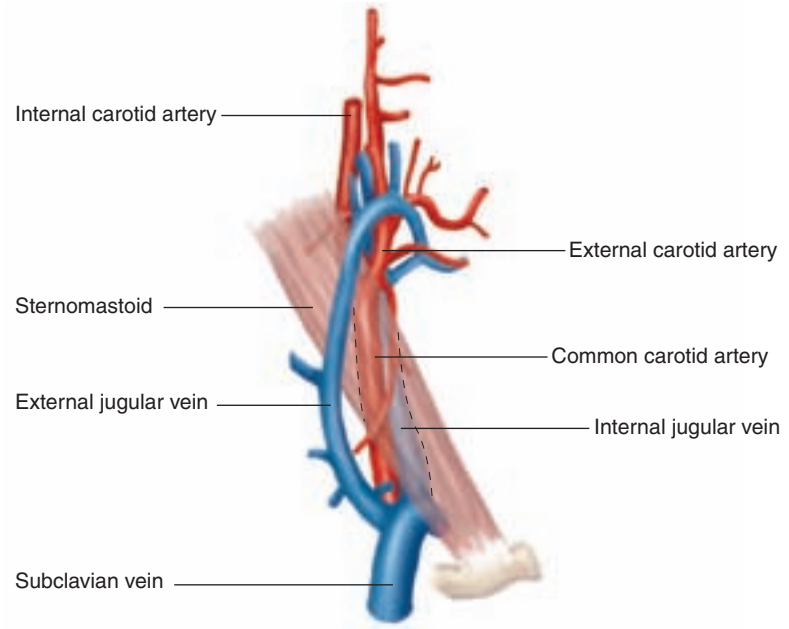
Changes in any of these four factors alter systolic pressure, diastolic pressure, or both. Blood pressure levels fluctuate strikingly through any 24-hour period, varying with physical activity; emotional state; pain; noise; environmental temperature; the use of coffee, tobacco, and other drugs; and even the time of day.

JUGULAR VENOUS PRESSURE (JVP)

Systemic venous pressure is much lower than arterial pressure. Although venous pressure ultimately depends on left ventricular contraction, much of this force is dissipated as blood flows through the arterial tree and the capillary bed. Walls of veins contain less smooth muscle than walls of arteries. This reduces venous tone and makes veins more distensible. Other important determinants of venous pressure include blood volume and the capacity of the right heart to eject blood into the pulmonary arterial system. Cardiac disease may alter these variables, producing abnormalities in central venous pressure. For example, venous pressure falls when left ventricular output or blood volume is significantly reduced; it rises when the right heart fails or when increased pressure in the pericardial sac impedes the return of blood to the right atrium. These venous pressure changes are reflected in the height of the venous column of blood in the internal jugular veins, termed the *jugular venous pressure* or *JVP*.

Pressure in the jugular veins reflects right atrial pressure, giving clinicians an important clinical indicator of cardiac function and right heart hemodynamics. Assessing the JVP is an essential, though challenging, clinical skill. The JVP is best estimated from the internal jugular vein, usually on the *right side,* because the right internal jugular vein has a more direct anatomic channel into the right atrium.[1]

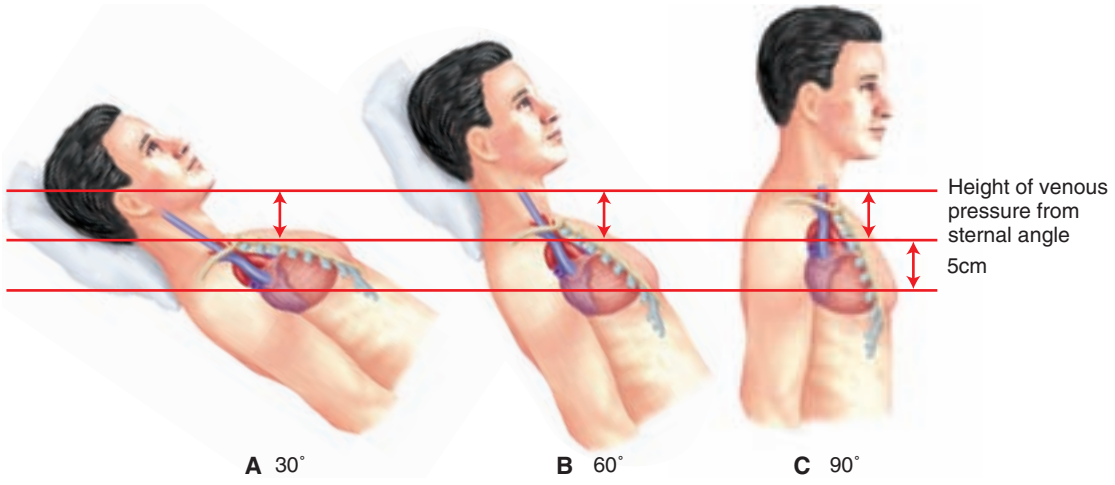
The internal jugular veins lie deep to the sternomastoid muscles in the neck and are not directly visible, so the clinician must learn to identify the *pulsations* of the internal jugular vein that are transmitted to the surface of the neck, making sure to carefully distinguish these venous pulsations from pulsations of the carotid artery. If pulsations from the internal jugular vein cannot be identified, those of the external jugular vein can be used, but they are less reliable.



To estimate the level of the JVP, you will learn to find the *highest point of oscillation in the internal jugular vein* or, if necessary, the point above which the external jugular vein appears collapsed. The JVP is usually measured in vertical distance above the *sternal angle*, the bony ridge adjacent to the second rib where the manubrium joins the body of the sternum.

Study carefully the illustrations below. Note that regardless of the patient's position, the sternal angle remains roughly 5 cm above the right atrium. In this patient, however, the pressure in the internal jugular vein is somewhat elevated.

- In *Position A,* the head of the bed is raised to the usual level, about 30°, but the JVP cannot be measured because the meniscus, or level of oscillation, is above the jaw and therefore not visible.
- In *Position B,* the head of the bed is raised to 60°. The "top" of the internal jugular vein is now easily visible, so the vertical distance from the sternal angle or right atrium can now be measured.
- In *Position C,* the patient is upright and the veins are barely discernible above the clavicle, making measurement untenable.

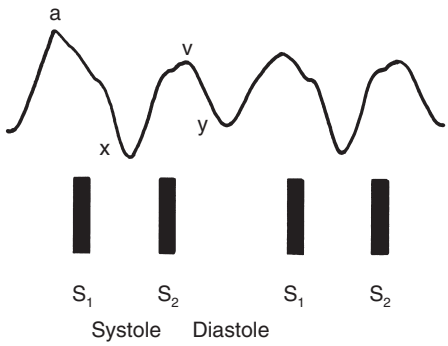


Note that the height of the venous pressure as measured from the sternal angle is the *same* in all three positions, but your ability to *measure* the height of the column of venous blood, or JVP, differs according to how you position the patient. Jugular venous pressure measured at more than 4 cm above the sternal angle, or more than 9 cm above the right atrium, is considered elevated or abnormal. The techniques for measuring the JVP are fully described in Techniques of Examination on pp. 302–304.

JUGULAR VENOUS PULSATIONS

The oscillations that you see in the internal jugular veins, and often in the externals, reflect changing pressures within the right atrium. The right internal jugular vein empties more directly into the right atrium and reflects these pressure changes best.

Careful observation reveals that the undulating pulsations of the internal jugular veins, and sometimes the externals, are composed of two quick peaks and two troughs.



The first elevation, the *a wave,* reflects the slight rise in atrial pressure that accompanies atrial contraction. It occurs just before the first heart sound and before the carotid pulse. The following trough, the *x descent,* starts with atrial relaxation. It continues as the right ventricle, contracting during systole, pulling the floor of the atrium downward. During ventricular systole, blood continues to flow into the right atrium from the venae cavae. The tricuspid valve is closed, the chamber begins to fill, and right atrial pressure begins to rise again, creating the second elevation, the *v wave.* When the tricuspid valve opens early in diastole, blood in the right atrium flows passively into the right ventricle, and right atrial pressure falls again, creating the second trough or *y descent.* To remember these four oscillations in an oversimplified way, think of the following sequence: atrial contraction, atrial relaxation, atrial filling, and atrial emptying. (You can think of the *a* wave as atrial contraction and the *v* wave as venous filling.)

To the naked eye, the two descents are the most obvious events in the normal jugular pulse. Of the two, the sudden collapse of the *x* descent late in systole is more prominent, occurring just before the second heart sound. The *y* descent follows the second heart sound early in diastole.

CHANGES OVER THE LIFE SPAN

Aging may affect the location of the apical impulse, the pitch of heart sounds and murmurs, the stiffness of the arteries, and blood pressure. For example, the *apical impulse* is usually felt easily in children and young adults; as the chest deepens in its anteroposterior diameter, the impulse gets harder to find. For the same reason, *splitting of the second heart sound* may be harder to hear in older people as its pulmonic component becomes less audible. Further, at some time over the life span, almost everyone has a *heart murmur.* Most murmurs occur without other evidence of cardiovascular abnormality and may therefore be considered innocent normal variants. These common murmurs vary with age, and familiarity with their patterns helps you to distinguish normal from abnormal. Turn to pp. 671–815, Chapter 18, Assessing Children:

Infancy Through Adolescence, and to pp. 817–838, Chapter 19, The Pregnant Woman, for information on how to distinguish these innocent murmurs.

Murmurs may originate in large blood vessels as well as in the heart. The *jugular venous hum,* which is very common in children, may still be heard through young adulthood (see p. 762). A second, more important example is the *cervical systolic murmur* or *bruit,* which may be innocent in children but suspicious for arterial obstruction in adults.

THE HEALTH HISTORY

Common or Concerning Symptoms

- Chest pain
- Palpitations
- Shortness of breath: dyspnea, orthopnea, or paroxysmal nocturnal dyspnea
- Swelling or edema

Chest pain or discomfort is one of the most important symptoms you will assess as a clinician. As you listen to the patient's story, you must always keep serious adverse events in mind, such as *angina pectoris, myocardial infarction,* or even a *dissecting aortic aneurysm.*[2–4] This section approaches chest symptoms from the *cardiac standpoint,* including chest pain, palpitations, orthopnea, paroxysmal nocturnal dyspnea (PND), and edema. For this complaint, however, it is wise to think through the range of possible cardiac, pulmonary, and extrathoracic etiologies. You should review the Health History section of Chapter 7, The Thorax and Lungs, which enumerates the various possible sources of chest pain: the myocardium, the pericardium, the aorta, the trachea and large bronchi, the parietal pleura, the esophagus, the chest wall, and extrathoracic structures such as the neck, gallbladder, and stomach. This review is important, because symptoms such as dyspnea, wheezing, cough, and even hemoptysis (see pp. 248–250) can be cardiac as well as pulmonary in origin.

See Table 7-1, Chest Pain, pp. 268–269.

Your initial questions should be broad . . . "Do you have any pain or discomfort in your chest?" Ask the patient to point to the pain and to describe all seven of its attributes. After listening closely to the patient's description, move on to more specific questions such as "Is the pain related to exertion?" and "What kinds of activities bring on the pain?" Also "How intense is the pain, on a scale of 1 to 10?" . . . "Does it radiate into the neck, shoulder, back, or down your arm?" . . . "Are there any associated symptoms like shortness of breath, sweating, palpitations, or nausea?" . . . "Does it ever wake you up at night?" . . . "What do you do to make it better?"

Exertional chest pain with radiation to the left side of the neck and down the left arm in *angina pectoris;* sharp pain radiating into the back or into the neck in *aortic dissection.*

Palpitations are an unpleasant awareness of the heartbeat. When reporting these sensations, patients use various terms such as skipping, racing, fluttering, pounding, or stopping of the heart. Palpitations may result from an irregular heartbeat, from rapid acceleration or slowing of the heart, or from increased forcefulness of cardiac contraction. Such perceptions, however, also depend on how patients respond to their own body sensations. Palpitations do not necessarily mean heart disease. In contrast, the most serious dysrhythmias, such as ventricular tachycardia, often do not produce palpitations.

See Tables 8-1 and 8-2 for selected heart rates and rhythms (pp. 324–325)

Symptoms or signs of irregular heart action warrant an electrocardiogram. Only *atrial fibrillation,* which is "irregularly irregular," can be reliably identified at the bedside.

You may ask directly about palpitations, but if the patient does not understand your question, reword it. "Are you ever aware of your heartbeat? What is it like?" Ask the patient to tap out the rhythm with a hand or finger. Was it fast or slow? Regular or irregular? How long did it last? If there was an episode of rapid heartbeats, did they start and stop suddenly or gradually? (For this group of symptoms, an electrocardiogram is indicated.)

Clues in the history include transient skips and flipflops (possible premature contractions); rapid regular beating of sudden onset and offset (possible paroxysmal supraventricular tachycardia); a rapid regular rate of less than 120 beats per minute, especially if starting and stopping more gradually (possible sinus tachycardia).

It is helpful to teach selected patients how to make serial measurements of their pulse rates in case they have further episodes.

Shortness of breath is a common patient concern and may represent dyspnea, orthopnea, or paroxysmal nocturnal dyspnea. *Dyspnea* is an uncomfortable awareness of breathing that is inappropriate to a given level of exertion. This complaint is often made by patients with cardiac or pulmonary problems, as discussed in Chapter 7, The Thorax and Lungs, p. 249.

Orthopnea is dyspnea that occurs when the patient is lying down and improves when the patient sits up. Classically, it is quantified according to the number of pillows the patient uses for sleeping, or by the fact that the patient needs to sleep sitting up. Make sure, however, that the reason the patient uses extra pillows or sleeps upright is shortness of breath when supine and not other causes.

Orthopnea suggests *left ventricular heart failure* or *mitral stenosis;* it may also accompany *obstructive lung disease.*

Paroxysmal nocturnal dyspnea, or *PND,* describes episodes of sudden dyspnea and orthopnea that awaken the patient from sleep, usually 1 or 2 hours after going to bed, prompting the patient to sit up, stand up, or go to a window for air. There may be associated wheezing and coughing. The episode usually subsides but may recur at about the same time on subsequent nights.

PND suggests *left ventricular heart failure* or *mitral stenosis* and may be mimicked by *nocturnal asthma* attacks.

Edema refers to the accumulation of excessive fluid in the interstitial tissue spaces and appears as swelling. Questions about edema are typically included in the cardiac history, but edema has many other causes, both local and general. Focus your questions on the location, timing, and setting of the swelling, and on associated symptoms. "Have you had any swelling anywhere? Where? . . . Anywhere else? When does it occur? Is it worse in the morning or at night? Do your shoes get tight?"

Dependent edema appears in the lowest body parts: the feet and lower legs when sitting, or the sacrum when bedridden. Causes may be cardiac (*congestive heart failure*), nutritional (*hypoalbuminemia*), or positional.

Continue with "Are the rings tight on your fingers? Are your eyelids puffy or swollen in the morning? Have you had to let out your belt?" Also, "Have your clothes gotten too tight around the middle?" It is useful to ask patients

Edema occurs in renal and liver disease: periorbital puffiness, tight rings in *nephrotic syndrome;*

who retain fluid to record daily morning weights, because edema may not be obvious until several liters of extra fluid have accumulated.

enlarged waistline from *ascites* and *liver failure.*

HEALTH PROMOTION AND COUNSELING

Important Topics for Health Promotion and Counseling

- Preventing hypertension
- Preventing cardiovascular disease and stroke
- Lowering cholesterol and low-density lipoprotein (LDL)
- Lifestyle modification and risk intervention, including healthy eating and counseling about weight and exercise

Despite improvements in risk factor modification, cardiovascular disease remains the leading cause of death for both men and women, accounting for approximately one third of all U.S. deaths. Both *primary prevention,* in those without evidence of cardiovascular disease, and *secondary prevention,* in those with known cardiovascular events such as angina or myocardial infarction, remain important priorities for the office, the hospital, and the nation's public health. Education and counseling will guide your patients to maintain optimal levels of blood pressure, cholesterol, weight, and exercise and reduce risk factors for cardiovascular disease and stroke.

Preventing Hypertension. According to the U.S. Preventive Services Task Force, hypertension accounts for "35% of all myocardial infarctions and strokes, 49% of all episodes of heart failure, and 24% of all premature deaths."[5] The Task Force strongly recommends *screening of adults 18 years and older for high blood pressure.* Recent long-term population-based studies have fueled a dramatic shift in national strategies to prevent and reduce blood pressure (BP). "The Seventh Report of the Joint National Committee on Prevention, Detection, Evaluation, and Treatment of High Blood Pressure," known as JNC 7, the National High Blood Pressure Education Program, and clinical investigators have issued several key messages (see box on next page)[6,7]: These findings underlie the tougher and simpler blood pressure classification of JNC 7 (see table on p. 109)[10]:

- The former six categories of blood pressure have been collapsed to four, with normal blood pressure defined as <120/80 mm Hg.
- Systolic blood pressures of 120 to 139 mm Hg and diastolic blood pressures of 80 to 89 mm Hg are no longer "high normal"; they are "prehypertension."

- Drug therapy should begin with stage 1 hypertension, namely systolic blood pressure of 140 to 159 mm Hg or diastolic blood pressure of 90 to 99 mm Hg.
- Adoption of healthy lifestyles by all people is now considered "indispensable."

KEY MESSAGES ABOUT HYPERTENSION

- "Individuals who are normotensive at 55 years have a 90% lifetime risk for developing hypertension."[6]
- "More than 1 of every 2 adults older than 60 years of age has hypertension,"[7] and only 34% of those with hypertension have achieved blood pressure goals.[6]
- "The relationship between pressure and risk of cardiovascular disease (CVD) events is continuous, consistent, and independent of other risk factors. . . . For individuals aged 40 to 70 years, each increment of 20 mm Hg in systolic BP or 10 mm Hg in diastolic BP doubles the risk of CVD across the entire BP range from 115/75 to 185/115 mm Hg."[6,8]
- Recent large population studies of cardiovascular risk factors reveal two striking findings[9]:
  1. Only approximately 4.8% to 9.9% of the young and middle-aged population is at low risk.
  2. The benefits of low-risk status are enormous: a 72% to 85% reduction in CVD mortality and a 40% to 58% reduction in mortality from all causes, leading to a gain of 5.8 to 9.5 years in life expectancy. This gain "holds for both African Americans and whites, and for those of lower and higher socioeconomic status."[9]
- Hence, identifying and treating people with risk factors are not enough. *A population-wide strategy is critical to prevent and reduce the magnitude of all the major risk factors* so that people develop favorable behaviors in childhood and *remain* at *low risk for life.*[9]

Risk factors for hypertension include physical inactivity, microalbuminuria or estimated GFR less than 60 mL/min, family history of premature CVD (<55 years for men and <65 years for women), excess intake of dietary sodium, insufficient intake of potassium, and excess consumption of alcohol.[6]

Preventing Cardiovascular Disease and Stroke. The American Heart Association (AHA) in its 2002 update placed the challenge for implementing risk factor reduction squarely on clinicians: "The challenge for health care professionals is to engage greater numbers of patients, at an earlier stage of their disease, in comprehensive cardiovascular risk reduction" to expand the benefits of primary prevention. "The continuing message is that adoption of healthy life habits remains the cornerstone of primary prevention." "The imperative is to prevent the first episode of coronary disease or

stroke or the development of aortic aneurysm and peripheral vascular disease because of the still-high rate of first events that are fatal or disabling."[11]

As a first step, clinicians need to identify not only elevated blood pressure but also other well-studied risk factors for coronary heart disease (CHD). In its "Guidelines for Primary Prevention of Cardiovascular Disease and Stroke," the AHA recommends *risk factor screening* for adults beginning at age 20, and *global absolute CHD risk estimation* for all adults 40 years and

RISK FACTORS AND SCREENING FREQUENCY FOR ADULTS BEGINNING AT AGE 20

Risk Factor	*Frequency*
Family history of coronary heart disease (CHD)	Update regularly
Smoking status Diet Alcohol intake Physical activity	At each routine visit
Blood pressure Body mass index Waist circumference Pulse (to detect atrial fibrillation)	At each routine visit (at least every 2 years)
Fasting lipoprotein profile Fasting glucose	At least every 5 years If risk factors for hyperlipidemia or diabetes present, every 2 years

Source: Pearson TA, Blair SN, Daniels SR, et al. AHA guidelines for primary prevention of cardiovascular disease and stroke: 2000 update. Circulation 106:388–391, 2002.

GLOBAL RISK ESTIMATION FOR 10-YEAR RISK FOR CHD FOR ADULTS > AGE 40

Establish multiple risk score for CHD based on:

- Age
- Sex
- Smoking status
- Systolic (and sometimes diastolic) blood pressure
- Total (and sometimes LDL) cholesterol
- HDL cholesterol
- Diabetes

For calculation of global CHD risk, use the risk calculators found at either of the Web sites below (or other equations):

http://www.americanheart.org/presenter.jhtml?identifier=3003499
http://hin.nhlbi.nih.gov/atpiii/calculator.asp?usertype=prof

Source: Pearson TA, Blair SN, Daniels SR, et al. AHA guidelines for primary prevention of cardiovascular disease and stroke: 2000 update. Circulation 106:388–391, 2002.

older.[11] The goal of global risk estimation is to help patients keep their risk as low as possible. Note that diabetes, or 10-year risk of more than 20%, is considered equivalent to established CHD risk equivalents.

Lowering Cholesterol and LDL. In 2001 the National Heart, Lung, and Blood Institute of the National Institutes of Health published the "Third Report of the National Cholesterol Education Program Expert Panel," known as ATP III.[12] Publication of the full NCEP report followed in 2002.[13] These reports provide evidence-based recommendations on the management of high cholesterol and related lipid disorders, and document that "epidemiological surveys have shown that serum cholesterol levels are continuously correlated with CHD risk over a broad range of cholesterol values," in many of the world's populations.[14] Key features of ATP III are as follows:

- Identifying LDL as the primary target of cholesterol-lowering therapy
- Classifying three risk categories:
  - *High risk (10-year risk > 20%):* established CHD and CHD risk equivalents
  - *Moderately high risk (10-year risk 10%–20%):* multiple, or 2+, risk factors
  - *Low risk (10-year risk < 10%):* zero to 1 risk factor

  Risk factors include cigarette smoking, BP >140/90 mm Hg or use of antihypertensive medication, HDL <40 mg/dL, family history of CHD in male first-degree relative <age 55 or female first-degree relative <age 65, and age ≥45 for men or ≥55 for women.

  CHD includes history of myocardial infarction, stable or unstable angina, coronary artery procedures such as angioplasty or bypass surgery, or evidence of significant myocardial ischemia.

  CHD risk equivalents include *noncoronary atherosclerotic disease,* such as peripheral arterial disease, abdominal aortic aneurysm, and carotid artery disease (transient ischemic attacks or stroke of carotid origin or > 50% obstruction of the carotid artery); *diabetes;* and *2+ risk factors with 10-year risk for CHD of > 20%.*
- Defining *high risk* as "all persons with CHD or CHD risk equivalents," with an *LDL goal for high-risk people of ≤ 100 mg/dL*

In July 2004, NCEP updated these reports based on the findings in five major clinical trials.[14] For *high-risk people,* NCEP now recommends an LDL goal of less than 70 mg/dL and intensive lipid therapy as a *therapeutic option*.[15] The NCEP cites data showing that high-risk patients benefit from a further 30% to 40% drop in LDL even when LDL is less than 100 mg/dL.

The U.S. Preventive Services Task Force recommends routine screening of LDL for men 35 years or older and for women 45 years or older.[16] Screening should begin at 20 years for those with risk factors for CHD.[17,18]

Counsel your patients to obtain a *fasting lipid profile* to determine levels of total and LDL cholesterol. Use the risk calculators on p. 297, or consult ATP III, to *establish your patient's 10-year risk category*. Use the 2004 guidelines below, which now have four risk groups, to plan your interventions regarding lifestyle change and lipid-lowering medications.

■ *Updated ATP III Guidelines*

10-year Risk Category	LDL Goal	Consider Drug Therapy if LDL:
High risk (>20%)	<100 mg/dL *Optional goal:* <70 mg/dL	≥100 mg/dL (<100 mg/dL: consider drug options, including further 30%–40% reduction in LDL)
Moderately high risk (10%–20%)	<130 mg/dL *Optional goal:* <100 mg/dL	≥130 mg/dL (100–129 mg/dL: consider drug options to achieve goal of <100 mg/dL)
Moderate risk (<10%)	<130 mg/dL	≥160 mg/dL
Lower risk (0–1 risk factor)	<160 mg/dL	≥190 mg/dL (160–189 mg/dL: drug therapy *optional*)

(Source: Adapted from Grundy SM, Cleeman JI, Merz NB, et al, for the Coordinating Committee of the National Cholesterol Education Program. Implications of recent clinical trials for the National Cholesterol Education Adult Treatment Panel III guidelines. Circulation 110(2):227–239, 2004.)

Lifestyle Modification and Risk Intervention. JNC VII, the National High Blood Pressure Education Program, and the AHA encourage a series of well-studied effective lifestyle modifications and risk interventions

LIFESTYLE MODIFICATIONS TO PREVENT OR MANAGE HYPERTENSION

- Optimal weight, or BMI of 18.5–24.9 kg/m^2
- Salt intake of less than 6 grams of sodium chloride or 2.4 grams of sodium per day
- Regular aerobic exercise such as brisk walking for at least 30 minutes per day, most days of the week
- Moderate alcohol consumption per day of 2 drinks or fewer for men and 1 drink or fewer for women (2 drinks = 1 oz ethanol, 24 oz beer, 10 oz wine, or 2–3 oz whiskey)
- Dietary intake of more than 3,500 mg of potassium
- Diet rich in fruits, vegetables, and low-fat dairy products with reduced content of saturated and total fat

Source: Whelton PK, He J, Appel LJ, et al. Primary prevention of hypertension. Clinical and Public Health Advisory from the National High Blood Pressure Education Program. JAMA 288(15):1882–1888, 2002.

to prevent hypertension, CVD, and stroke. Lifestyle modifications for hypertension can lower systolic blood pressure from 2 to 20 mm Hg.[6] Lifestyle modifications to reduce hypertension overlap with those recommended for reducing risk for CVD and stroke, as seen below.

RISK INTERVENTIONS TO PREVENT CARDIOVASCULAR DISEASE AND STROKE

- Complete cessation of smoking
- Optimal blood pressure control—see table for JNC VII guidelines on p. 109
- Healthy eating—see diet recommendations on previous page
- Lipid management—see table on p. 299
- Regular aerobic exercise—see previous page
- Optimal weight—see previous page
- Diabetes management so that fasting glucose level is below 110 mg/dL and HgA1C is less than 7%
- Conversion of atrial fibrillation to normal sinus rhythm or, if chronic, anticoagulation

Source: Pearson TA, Blair SN, Daniels SR, et al. AHA guidelines for primary prevention of cardiovascular disease and stroke: 2002 update. Circulation 106:388–391, 2002.

Healthy Eating. Begin with a dietary history (see pp. 92–93), then target low intake of cholesterol and total fat, especially less saturated and *trans* fat. Foods with monounsaturated fats, polyunsaturated fats, and omega-3 fatty acids in fish oils help to lower blood cholesterol. Review the food sources of these healthy and unhealthy fats.[19]

Sources of Unhealthy Fats

- *Foods high in cholesterol:* dairy products, egg yolks, liver and organ meats, high-fat meat and poultry
- *Foods high in saturated fat:* high-fat dairy products—cream, cheese, ice cream, whole and 2% milk, and sour cream; bacon, butter; chocolate; coconut oil; lard and gravy from meat drippings; high-fat meats like ground beef, bologna, hot dogs, and sausage
- *Foods high in* trans *fat:* snacks and baked goods with hydrogenated or partially hydrogenated oil, stick margarines, shortening, french fries

Sources of Healthy Fats

- *Foods high in monounsaturated fat:* nuts, such as almonds, pecans, and peanuts; sesame seeds; avocados; canola oil; olive and peanut oil; peanut butter
- *Foods high in polyunsaturated fat:* corn, safflower, cottonseed, and soy-

bean oil; walnuts; pumpkin or sunflower seeds; soft (tub) margarine; mayonnaise; salad dressings

- *Foods high in omega-3 fatty acids:* albacore tuna, herring, mackerel, rainbow trout, salmon, sardines

Counseling About Weight and Exercise. The January 2004 "Progress Review—Nutrition and Overweight" in Healthy People 2010 reports that "Dietary factors are associated with 4 of the 10 leading causes of death—coronary heart disease, some types of cancer, stroke, and type 2 diabetes—as well as with high blood pressure and osteoporosis. Overall, the data on the three Healthy People 2010 objectives for the weight status of adults and children reflect a trend for the worse."[20] More than 60% of all Americans are now obese or overweight, with a BMI greater than or equal to 25.

Counseling about weight has become a clinician imperative. Assess body mass index (BMI) as described in Chapter 4, pp. 90–92. Discuss the principles of healthy eating—patients with high fat intake are more likely to accumulate body fat than patients with high intake of protein and carbohydrate. Review the patient's eating habits and weight patterns in the family. Set realistic goals that will help the patient maintain healthy eating habits *for life*.

Regular exercise is the number one health indicator for Healthy People 2010. In its April 2004 "Progress Review—Physical Activity and Fitness," Healthy People 2010 states that "in 2000, poor diet coupled with lack of exercise was the second leading actual cause of death. The gap between this risk factor and tobacco use, the leading cause, has narrowed substantially over the past decade."[21] To reduce risk for CHD, counsel patients to pursue aerobic exercise, or exercise that increases muscle oxygen uptake, for at least 30 minutes on most days of the week. Spur motivation by emphasizing the immediate benefits to health and well-being. Deep breathing, sweating in cool temperatures, and pulse rates exceeding 60% of the maximum normal age-adjusted heart rate, or 220 minus the person's age, are markers that help patients recognize onset of aerobic metabolism. Be sure to evaluate any cardiovascular, pulmonary, or musculoskeletal conditions that present risks before selecting an exercise regimen.

TECHNIQUES OF EXAMINATION

As you begin the cardiovascular examination, review the blood pressure and heart rate recorded during the General Survey and Vital Signs at the start of the physical examination. If you need to repeat these measurements, or if they have not already been done, take the time to measure the blood pressure and heart rate using optimal technique (see Chapter 4, Beginning the Physical Examination: General Survey and Vital Signs, especially pp. 106–112).[22–26]

In brief, for *blood pressure,* after letting the patient rest for at least 5 minutes in a quiet setting, choose a correctly sized cuff and position the patient's arm at heart level, either resting on a table if seated or supported at midchest level if standing. Make sure the bladder of the cuff is centered over the brachial artery. Inflate the cuff about 30 mm Hg above the pressure at which the brachial or radial pulse disappears. As you deflate the cuff, listen first for the sounds of at least two consecutive heartbeats—these mark the *systolic* pressure. Then listen for the disappearance point of the heartbeats, which marks the *diastolic* pressure. For *heart rate,* measure the radial pulse using the pads of your index and middle fingers, or assess the apical pulse using your stethoscope (see p. 111).

Now you are ready to systematically assess the components of the cardiovascular system:

- The jugular venous pressure and pulsations
- The carotid upstrokes and presence or absence of bruits
- The point of maximal impulse (PMI) and any heaves, lifts, or thrills
- The first and second heart sounds, S_1 and S_2
- Presence or absence of extra heart sounds such as S_3 or S_4
- Presence or absence of any cardiac murmurs.

JUGULAR VENOUS PRESSURE AND PULSATIONS

Jugular Venous Pressure (JVP). Estimating the JVP is one of the most important and frequently used skills of physical examination. At first it will seem difficult, but with practice and supervision you will find that the JVP provides valuable information about the patient's volume status and cardiac function. As you have learned, the JVP reflects pressure in the right atrium, or central venous pressure, and is best assessed from pulsations in the right internal jugular vein. Note, however, that the jugular veins and pulsations are difficult to see in children younger than 12 years of age, so they are not useful for evaluating the cardiovascular system in this age group.

To assist you in learning this portion of the cardiac examination, steps for assessing the JVP are outlined on the next page. As you begin your assessment, take a moment to reflect on the patient's volume status and consider how you may need to alter the elevation of the head of the bed or examining table. The usual starting point for assessing the JVP is to elevate the head of the bed to 30°. Identify the external jugular vein on each side, then find the internal jugular venous pulsations transmitted from deep in the neck to

the overlying soft tissues. The JVP is the elevation at which the highest oscillation point, or meniscus, of the jugular venous pulsations is usually evident in euvolemic patients. In patients who are *hypovolemic*, you may anticipate that *the JVP will be low*, causing you to subsequently *lower the head of the bed*, sometimes even to 0°, to see the point of oscillation best. Likewise, in volume-overloaded or *hypervolemic* patients, you may anticipate that *the JVP will be high*, causing you to subsequently *raise the head of the bed*.

A hypovolemic patient may have to lie flat before you see the neck veins. In contrast, when jugular venous pressure is increased, an elevation up to 60° or even 90° may be required. In all these positions, the sternal angle usually remains about 5 cm above the right atrium, as diagrammed on p. 291.

STEPS FOR ASSESSING THE JUGULAR VENOUS PRESSURE (JVP)

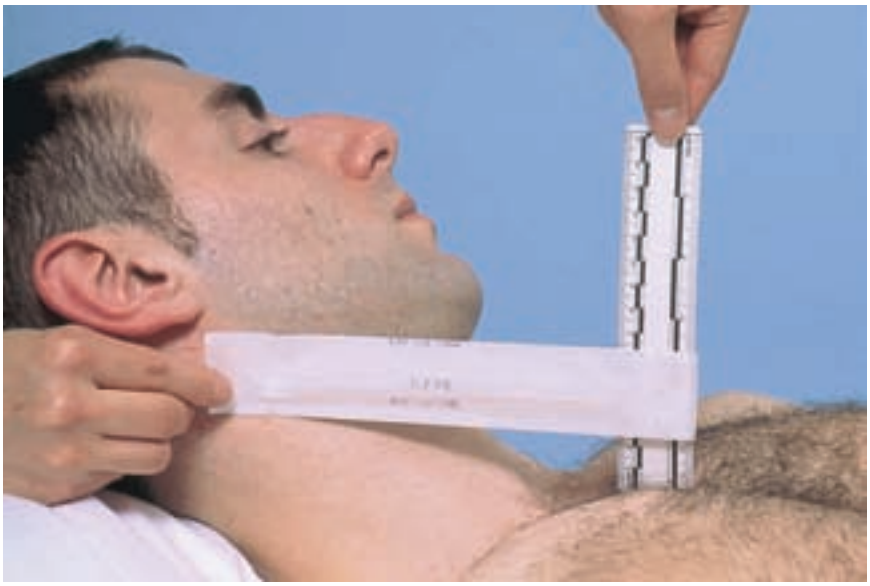
- Make the patient comfortable. *Raise the head slightly on a pillow* to relax the sternomastoid muscles.
- *Raise the head of the bed or examining table to about 30°. Turn the patient's head slightly away from the side you are inspecting.*
- Use *tangential lighting* and examine both sides of the neck. Identify the external jugular vein on each side, then find the internal jugular venous pulsations.
- *If necessary, raise or lower the head of the bed* until you can see the oscillation point or meniscus of the internal jugular venous pulsations in the lower half of the neck.
- Focus on the *right internal jugular vein.* Look for pulsations in the suprasternal notch, between the attachments of the sternomastoid muscle on the sternum and clavicle, or just posterior to the sternomastoid. The table below helps you distinguish internal jugular pulsations from those of the carotid artery.
- *Identify the highest point of pulsation in the right internal jugular vein.* Extend a long rectangular object or card horizontally from this point and a centimeter ruler vertically from the sternal angle, making an exact right angle. Measure the vertical distance in centimeters above the sternal angle where the horizontal object crosses the ruler. *This distance, measured in centimeters above the sternal angle or the right atrium, is the JVP.*

The following features help to distinguish jugular from carotid artery pulsations:[1]

Distinguishing Internal Jugular and Carotid Pulsations

Internal Jugular Pulsations	Carotid Pulsations
Rarely palpable	Palpable
Soft, rapid, undulating quality, usually with two elevations and two troughs per heart beat	A more vigorous thrust with a single outward component
Pulsations eliminated by light pressure on the vein(s) just above the sternal end of the clavicle	Pulsations not eliminated by this pressure
Level of the pulsations changes with position, dropping as the patient becomes more upright.	Level of the pulsations unchanged by position
Level of the pulsations usually descends with inspiration.	Level of the pulsations not affected by inspiration

Establishing the true vertical and horizontal lines to measure the JVP is difficult, much like the problem of hanging a picture straight when you are close to it. Place your ruler on the sternal angle and line it up with something in the room that you know to be vertical. Then place a card or rectangular object at an exact right angle to the ruler. This constitutes your horizontal line. Move it up or down—still horizontal—so that the lower edge rests at the top of the jugular pulsations, and read the vertical distance on the ruler. Round your measurement off to the nearest centimeter.



Venous pressure measured at greater than 3 cm or possibly 4 cm above the sternal angle, or more than 8 cm or 9 cm in total distance above the right atrium, is considered elevated *above normal.*

Increased pressure suggests *right-sided congestive heart failure* or, less commonly, *constrictive pericarditis, tricuspid stenosis,* or *superior vena cava obstruction.*[27–33]

In patients with obstructive lung disease, venous pressure may appear elevated on expiration only; the veins collapse on inspiration. This finding does not indicate congestive heart failure.

If you are unable to see pulsations in the internal jugular vein, look for them in the external jugular vein. If you see no pulsation, use *the point above which the external jugular veins appear to collapse.* Make this observation on each side of the neck. Measure the vertical distance of this point from the sternal angle.

Unilateral distention of the external jugular vein is usually caused by local kinking or obstruction. Occasionally, even bilateral distention has a local cause.

The highest point of venous pulsations may lie below the level of the sternal angle. Under these circumstances, venous pressure is not elevated and seldom needs to be measured.

Even though students may not see clinicians making these measurements very frequently in clinical settings, practicing exact techniques for measuring the JVP is important. Eventually, with experience, clinicians and cardiologists come to identify the JVP and estimate its height visually.

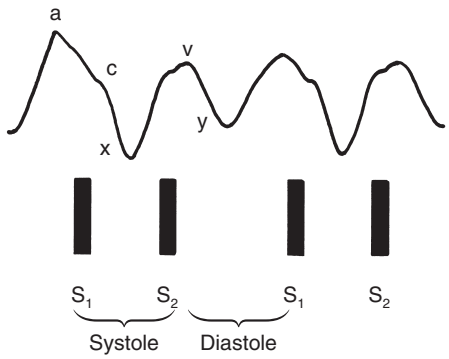
Jugular Venous Pulsations. *Observe the amplitude and timing of the jugular venous pulsations.* In order to time these pulsations, feel the left carotid artery with your right thumb or listen to the heart simultaneously. The *a* wave just precedes S_1 and the carotid pulse, the *x* descent can be seen

Prominent *a* waves indicate increased resistance to right atrial contraction, as in *tricuspid stenosis,* or, more commonly, the decreased

as a systolic collapse, the *v* wave almost coincides with S_2, and the *y* descent follows early in diastole. Look for absent or unusually prominent waves.

compliance of a hypertrophied right ventricle. The *a* waves disappear in atrial fibrillation. Larger *v* waves characterize tricuspid regurgitation.

Jugular venous pulsations



Jugular venous pressure curves

a = atrial contraction
c = carotid transmission not visible clinically
x = descent in right atrium following *a*
v = passive venous filling of atria from the vena cavae
y = descent during atrial resting phase before contraction

Considerable practice and experience are required to master jugular venous pulsations. A beginner is well-advised to concentrate primarily on jugular venous pressure.

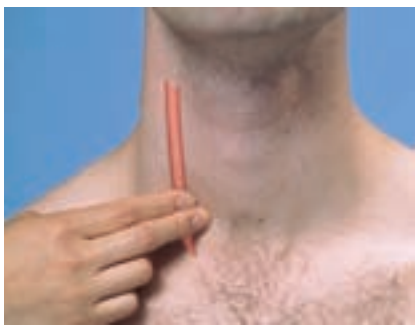
THE CAROTID PULSE

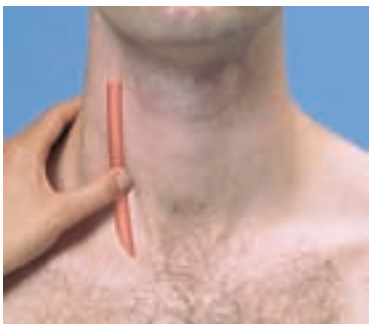
After you measure the JVP, move on to assessment of the *carotid pulse*. The carotid pulse provides valuable information about cardiac function and is especially useful for detecting stenosis or insufficiency of the aortic valve. Take the time to assess the quality of the carotid upstroke, its amplitude and contour, and presence or absence of any overlying *thrills* or *bruits*.

For irregular rhythms, see Table 8-1, Selected Heart Rates and Rhythms (p. 324), and Table 8-2, Selected Irregular Rhythms (p. 325).

To assess *amplitude and contour*, the patient should be lying down with the head of the bed still elevated to about 30°. When feeling for the carotid artery, first inspect the neck for carotid pulsations. These may be visible just medial to the sternomastoid muscles. Then place your left index and middle fingers (or left thumb) on the right carotid artery in the lower third of the neck, press posteriorly, and feel for pulsations.

A tortuous and kinked carotid artery may produce a unilateral pulsatile bulge.





Decreased pulsations may be caused by decreased stroke volume, but may also result from local factors in the artery such as atherosclerotic narrowing or occlusion.

Press just inside the medial border of a well-relaxed sternomastoid muscle, roughly at the level of the cricoid cartilage. Avoid pressing on the *carotid sinus,* which lies at the level of the top of the thyroid cartilage. For the left carotid artery, use your right fingers or thumb. Never press both carotids at the same time. This may decrease blood flow to the brain and induce syncope.

Pressure on the carotid sinus may cause a reflex drop in pulse rate or blood pressure.

Slowly increase pressure until you feel a maximal pulsation, then slowly decrease pressure until you best sense the arterial pressure and contour. Try to assess:

See Table 4-7, Abnormalities of the Arterial Pulse and Pressure Waves (p. 119).

- The *amplitude of the pulse*. This correlates reasonably well with the pulse pressure.

Small, thready, or weak pulse in *cardiogenic shock*; *bounding* pulse in *aortic insufficiency* (see p. 119).

- The *contour of the pulse wave,* namely the speed of the upstroke, the duration of its summit, and the speed of the downstroke. The normal upstroke is *brisk*. It is smooth, rapid, and follows S_1 almost immediately. The summit is smooth, rounded, and roughly midsystolic. The downstroke is less abrupt than the upstroke.

Delayed carotid upstroke in *aortic stenosis*

- Any *variations in amplitude,* either from beat to beat or with respiration.

Pulsus alternans (see p. 119), bigeminal pulse (beat-to-beat variation); paradoxical pulse (respiratory variation)

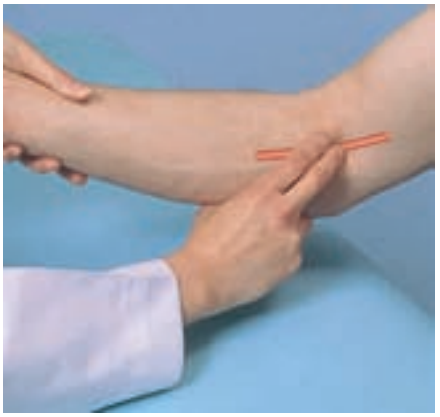
Thrills and Bruits. During palpation of the carotid artery, you may detect humming vibrations, or *thrills,* that feel like the throat of a purring cat. Routinely, but especially in the presence of a thrill, listen over both carotid arteries with the diaphragm of your stethoscope for a *bruit,* a murmur-like sound of vascular rather than cardiac origin.

You should also listen for carotid bruits if the patient is middle-aged or elderly or if you suspect cerebrovascular disease. Ask the patient to hold breathing for a moment so that breath sounds do not obscure the vascular sound, then listen with the bell.[34] Heart sounds alone do not constitute a bruit.

A carotid bruit with or without a thrill in a middle-aged or older person suggests but does not prove arterial narrowing. An aortic murmur may radiate to the carotid artery and sound like a bruit.

Further examination of arterial pulses is described in Chapter 14, The Peripheral Vascular System.

The Brachial Artery. The carotid arteries reflect aortic pulsations more accurately, but in patients with carotid obstruction, kinking, or thrills, they are unsuitable. If so, assess the pulse in the *brachial artery,* applying the techniques described above for determining amplitude and contour.



Use the index and middle fingers or thumb of your opposite hand. Cup

your hand under the patient's elbow and feel for the pulse just medial to the biceps tendon. The patient's arm should rest with the elbow extended, palm up. With your free hand, you may need to flex the elbow to a varying degree to get optimal muscular relaxation.

THE HEART

For most of the cardiac examination, the patient should be *supine* with the upper body raised by elevating the head of the bed or table to about 30°. Two other positions are also needed: (1) *turning to the left side*, and (2) *leaning forward*. These positions bring the ventricular apex and left ventricular outflow tract closer to the chest wall, enhancing detection of the point of maximal impulse and aortic insufficiency. *The examiner should stand at the patient's right side.*

The table below summarizes patient positions and a suggested sequence for the examination.

Sequence of the Cardiac Examination

Patient Position	Examination	Accentuated Findings
Supine, with the head elevated 30°	Inspect and palpate the precordium: the 2nd right and left interspaces; the right ventricle; and the left ventricle, including the apical impulse (diameter, location, amplitude, duration).	
Left lateral decubitus	Palpate the apical impulse if not previously detected. Listen at the apex with the *bell* of the stethoscope.	Low-pitched extra sounds (S_3, opening snap, diastolic rumble of mitral stenosis)
Supine, with the head elevated 30°	Listen at the tricuspid area with the *bell*.	
	Listen at all the auscultatory areas with the *diaphragm*.	
Sitting, leaning forward, after full exhalation	Listen along the left sternal border and at the apex with the *diaphragm*.	Soft decrescendo diastolic murmur of aortic insufficiency

During the cardiac examination, remember to correlate your findings with the patient's jugular venous pressure and carotid pulse. It is also important to identify both the anatomical location of your findings and their timing in the cardiac cycle.

- Note the *anatomical location* of sounds in terms of interspaces and their distance from the midsternal, midclavicular, or axillary lines. The midsternal line offers the most reliable zero point for measurement, but some feel that the midclavicular line accommodates the different sizes and shapes of patients.

- Identify the *timing of impulses or sounds* in relation to the cardiac cycle. Timing of sounds is often possible through auscultation alone. In most people with normal or slow heart rates, it is easy to identify the paired heart sounds by listening through a stethoscope. S_1 is the first of these sounds, S_2 is the second, and the relatively long diastolic interval separates one pair from the next.

The relative intensity of these sounds may also be helpful. S_1 is usually louder than S_2 at the apex; more reliably, S_2 is usually louder than S_1 at the base.

Even experienced clinicians are sometimes uncertain about the timing of heart sounds, especially extra sounds and murmurs. "Inching" can then be helpful. Return to a place on the chest—most often the base—where it is easy to identify S_1 and S_2. Get their rhythm clearly in mind. Then inch your stethoscope down the chest in steps until you hear the new sound.

Auscultation alone, however, can be misleading. The intensities of S_1 and S_2, for example, may be abnormal. At rapid heart rates, moreover, diastole shortens, and at about a rate of 120, the durations of systole and diastole become indistinguishable. *Use palpation of the carotid pulse or of the apical impulse to help determine whether the sound or murmur is systolic or diastolic.* Because both the carotid upstroke and the apical impulse occur in systole, right after S_1, sounds or murmurs coinciding with them are systolic; sounds or murmurs occurring after the carotid upstroke or apical impulse are diastolic.

For example, S_1 is decreased in *first-degree heart block*, and S_2 is decreased in *aortic stenosis.*

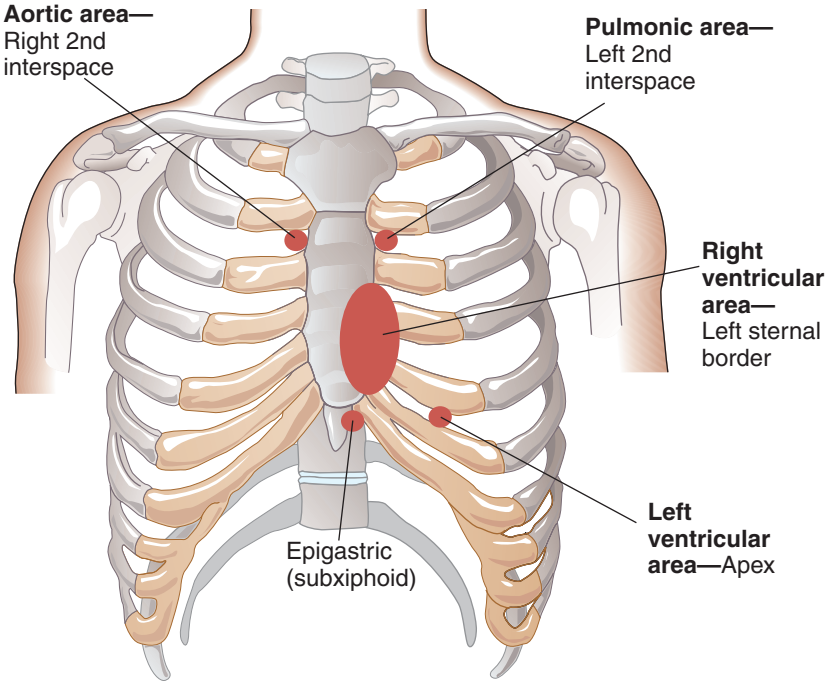
INSPECTION AND PALPATION

Overview. Careful *inspection* of the anterior chest may reveal the location of the *apical impulse* or *point of maximal impulse (PMI),* or less commonly, the ventricular movements of a left-sided S_3 or S_4. Tangential light is best for making these observations. Use *palpation* to confirm the characteristics of the apical impulse. Palpation is also valuable for detecting thrills and the ventricular movements of an S_3 or S_4.

Begin with general palpation of the chest wall. First palpate for heaves, lifts, or thrills using your *fingerpads.* Hold them flat or obliquely on the body surface, using light pressure for an S_3 or S_4, and firmer pressure for S_1 and S_2. Ventricular impulses may heave or lift your fingers. Then check for *thrills* by pressing the *ball of your hand* firmly on the chest. If subsequent auscultation reveals a loud murmur, go back and check for thrills over that area again. Be sure to assess the right ventricle by palpating the right ventricular area at the lower left sternal border and in the subxiphoid area, the pulmonary artery in the left 2nd interspace, and the aortic area in the right 2nd interspace. Review the diagram on the next page. *Note that the "areas" designated for the left and right ventricle, the pulmonary artery, and the aorta pertain to the majority of patients whose hearts are situated in the left chest, with normal anatomy of the great vessels.*

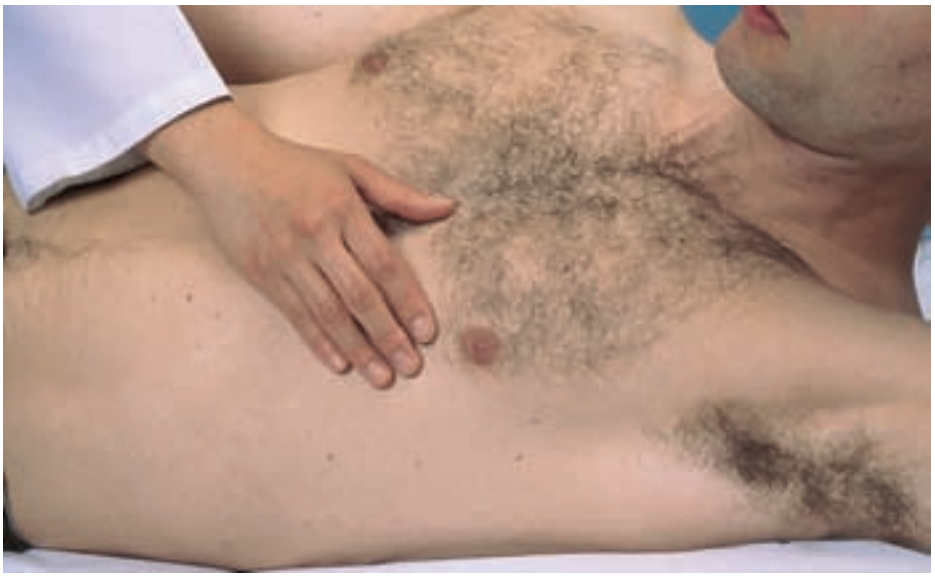
Thrills may accompany loud, harsh, or rumbling murmurs as in *aortic stenosis, patent ductus arteriosus, ventricular septal defect,* and, less commonly, *mitral stenosis.* They are palpated more easily in patient positions that accentuate the murmur.

On rare occasions, a patient has *dextrocardia*—a heart situated on the right side. The apical impulse



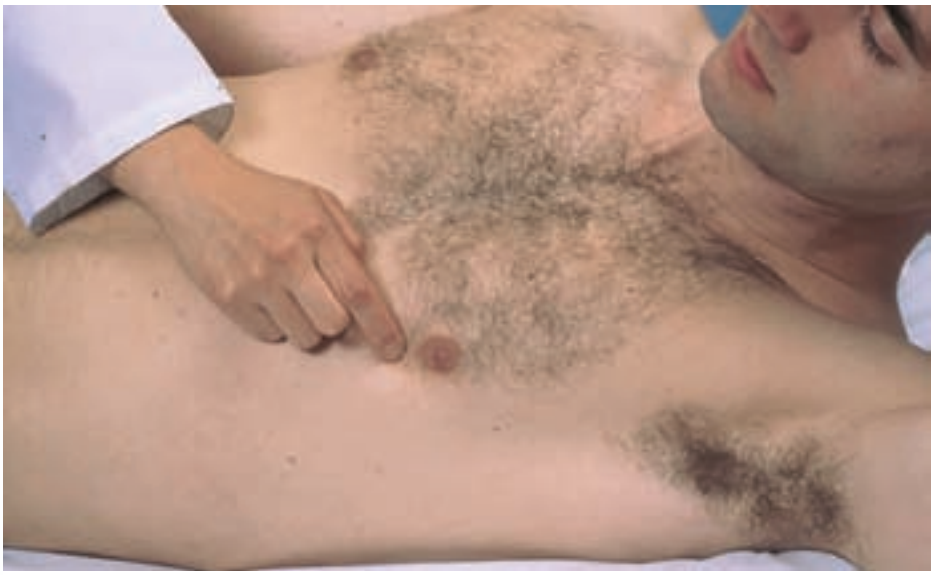
Left Ventricular Area—The Apical Impulse or Point of Maximal Impulse (PMI). The apical impulse represents the brief early pulsation of the left ventricle as it moves anteriorly during contraction and touches the chest wall. Note that in most examinations the apical impulse is the point of maximal impulse, or PMI; however, some pathologic conditions may produce a pulsation that is more prominent than the apex beat, such as an enlarged right ventricle, a dilated pulmonary artery, or an aneurysm of the aorta.

If you cannot identify the apical impulse with the patient supine, ask the patient to roll partly onto the left side—this is the *left lateral decubitus* position. Palpate again using the palmar surfaces of several fingers. If you cannot find will then be found on the right. If you cannot find an apical impulse, percuss for the dullness of heart and liver and for the tympany of the stomach. In *situs inversus,* all three of these structures are on opposite sides from normal. A right-sided heart with a normally placed liver and stomach is usually associated with congenital heart disease.



the apical impulse, ask the patient to exhale fully and stop breathing for a few seconds. When examining a woman, it may be helpful to displace the left breast upward or laterally as necessary; alternatively, ask her to do this for you.

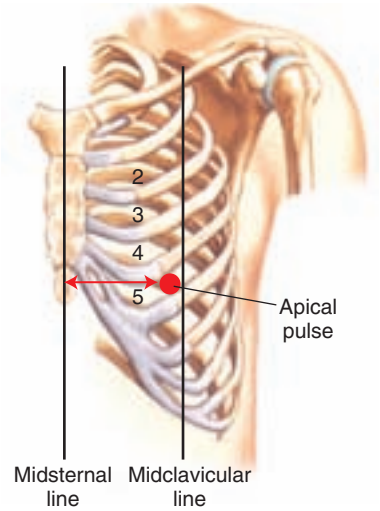
Once you have found the apical impulse, make finer assessments with your fingertips, and then with one finger.



With experience, you will learn to feel the apical impulse in a high percentage of patients. Obesity, a very muscular chest wall, or an increased anteroposterior diameter of the chest, however, may make it undetectable. Some apical impulses hide behind the rib cage, despite positioning.

Now assess the location, diameter, amplitude, and duration of the apical impulse. You may wish to have the patient breathe out and briefly stop breathing to check your findings.

- *Location*. Try to assess location with the patient *supine* because the left lateral decubitus position displaces the apical impulse to the left. Locate two points: the interspaces, usually the 5th or possibly the 4th, which give the vertical location; and the distance in centimeters from the midsternal line, which gives the horizontal location. Note that even though the apical impulse normally falls roughly at the midclavicular line, measurements from this line are less reproducible because clinicians vary in their estimates of the midpoint of the clavicle.



See Table 8-3, Variations and Abnormalities of the Ventricular Impulses (p. 326).

The apical impulse may be displaced upward and to the left by pregnancy or a high left diaphragm.

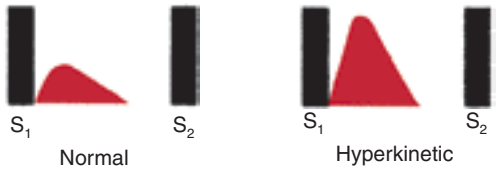
Lateral displacement from cardiac enlargement in *congestive heart failure, cardiomyopathy, ischemic heart disease.* Displacement in deformities of the thorax and mediastinal shift.

- *Diameter*. Palpate the diameter of the apical impulse. In the supine patient, it usually measures less than 2.5 cm and occupies only one interspace. It may feel larger in the left lateral decubitus position.

In the left lateral decubitus position, a diameter greater than 3 cm indicates left ventricular enlargement.

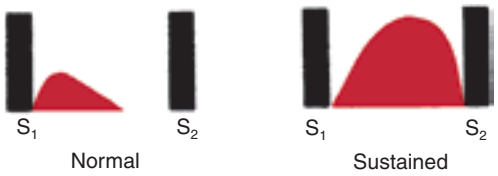
- *Amplitude*. Estimate the amplitude of the impulse. It is usually small and feels *brisk* and *tapping*. Some young people have an increased amplitude, or hyperkinetic impulse, especially when excited or after exercise; its duration, however, is normal.

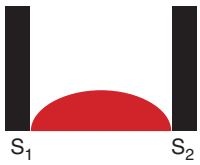
Increased amplitude may also reflect *hyperthyroidism, severe anemia,* pressure overload of the left ventricle (e.g., *aortic stenosis*), or volume overload of the left ventricle (e.g., *mitral regurgitation*).



- *Duration*. Duration is the most useful characteristic of the apical impulse for identifying hypertrophy of the left ventricle. To assess duration, listen to the heart sounds as you feel the apical impulse, or watch the movement of your stethoscope as you listen at the apex. Estimate the proportion of systole occupied by the apical impulse. Normally it lasts through the first two thirds of systole, and often less, but does not continue to the second heart sound.

A sustained, high-amplitude impulse that is normally located suggests left ventricular hypertrophy from pressure overload (as in hypertension). If such an impulse is displaced laterally, consider volume overload.





A sustained low-amplitude (hypokinetic) impulse may result from dilated cardiomyopathy.

S_3 and S_4. By inspection and palpation, you may detect ventricular movements that are synchronous with pathologic third and fourth heart sounds. For the left ventricular impulses, feel the apical beat gently with one finger. The patient should lie partly on the left side, breathe out, and briefly stop breathing. By inking an X on the apex, you may be able to see these movements.

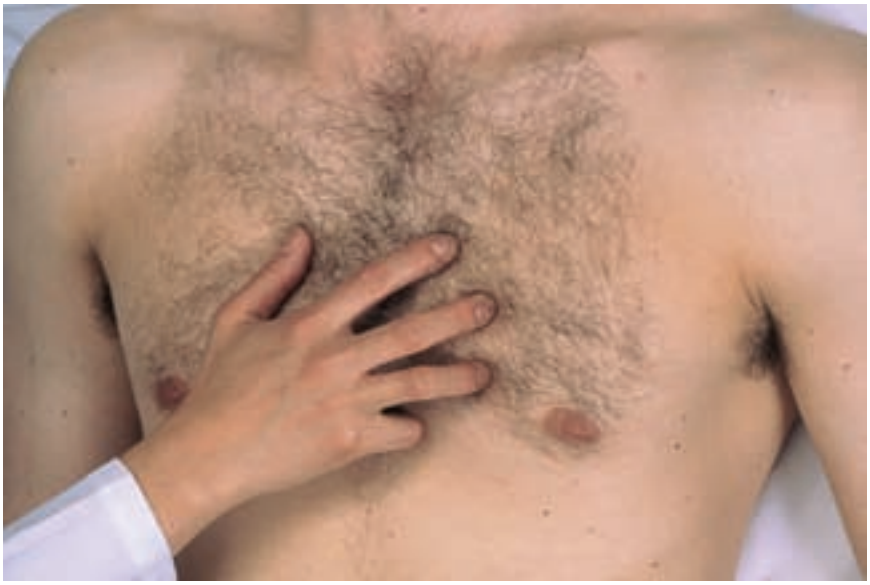
A brief middiastolic impulse indicates an S_3; an impulse just before the systolic apical beat itself indicates an S_4.

Right Ventricular Area—The Left Sternal Border in the 3rd, 4th, and 5th Interspaces. The patient should rest supine at 30°. Place the tips of your curved fingers in the 3rd, 4th, and 5th interspaces and try to feel the systolic impulse of the right ventricle. Again, asking the patient to breathe out and then briefly stop breathing improves your observation.

If an impulse is palpable, assess its location, amplitude, and duration. A brief systolic tap of low or slightly increased amplitude is sometimes felt in thin or shallow-chested people, especially when stroke volume is increased, as by anxiety.

A marked increase in amplitude with little or no change in duration occurs in chronic volume overload of the right ventricle, as from an *atrial septal defect.*

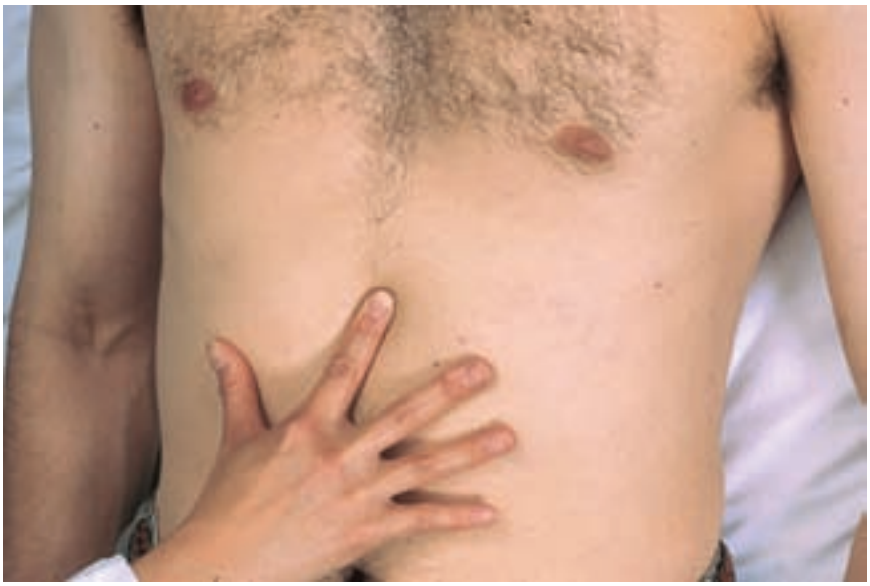
An impulse with increased amplitude and duration occurs with pressure overload of the right ventricle, as in *pulmonic stenosis* or *pulmonary hypertension.*



The diastolic movements of *right-sided third and fourth heart sounds* may be felt occasionally. Feel for them in the 4th and 5th left interspaces. Time them by auscultation or carotid palpation.

In patients with an increased anteroposterior (AP) diameter, palpation of the *right ventricle* in the *epigastric* or *subxiphoid area* is also useful. With your hand flattened, press your index finger just under the rib cage and up toward the left shoulder and try to feel right ventricular pulsations.

In obstructive pulmonary disease, hyperinflated lung may prevent palpation of an enlarged right ventricle in the left parasternal area. The impulse is felt easily, however, high in the epigastrium where heart sounds are also often heard best.



Asking the patient to inhale and briefly stop breathing is helpful. The inspiratory position moves your hand well away from the pulsations of the abdominal aorta, which might otherwise be confusing. The diastolic movements of S_3 and S_4, if present, may also be felt here.

Pulmonic Area—The Left 2nd Interspace. This interspace overlies the *pulmonary artery.* As the patient holds expiration, look and feel for an impulse and feel for possible heart sounds. In thin or shallow-chested patients, the pulsation of a pulmonary artery may sometimes be felt here, especially after exercise or with excitement.

A prominent pulsation here often accompanies dilatation or increased flow in the pulmonary artery. A palpable S_2 suggests increased pressure in the pulmonary artery (*pulmonary hypertension*).

Aortic Area—The Right 2nd Interspace. This interspace overlies the aortic outflow tract. Search for pulsations and palpable heart sounds.

A palpable S_2 suggests systemic *hypertension.* A pulsation here suggests a dilated or aneurysmal aorta.

PERCUSSION

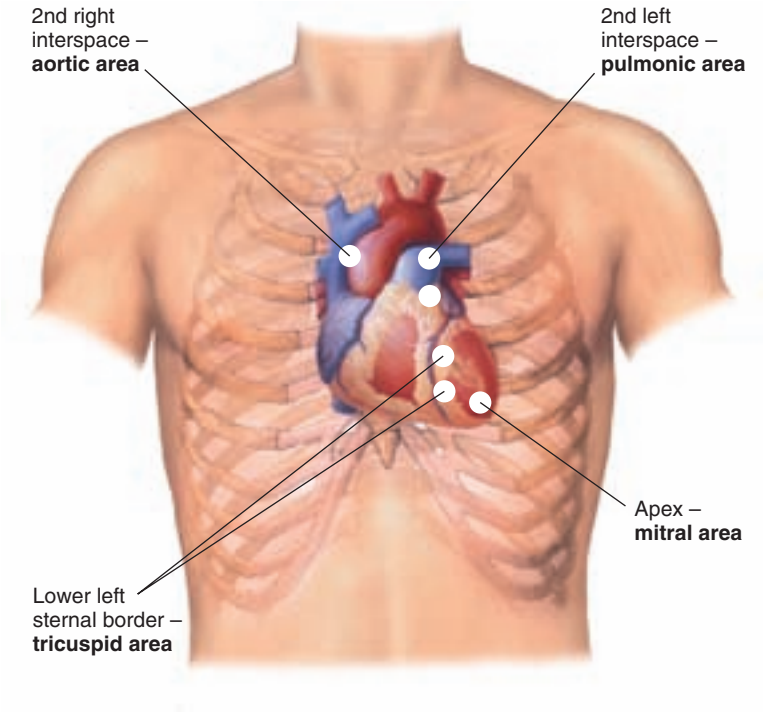
In most cases, palpation has replaced percussion in the estimation of cardiac size. When you cannot feel the apical impulse, however, percussion may suggest where to search for it. Occasionally, percussion may be your only tool. Under these circumstances, cardiac dullness often occupies a large area. Starting well to the left on the chest, percuss from resonance toward cardiac dullness in the 3rd, 4th, 5th, and possibly 6th interspaces.

A markedly dilated failing heart may have a hypokinetic apical impulse that is displaced far to the left. A large pericardial effusion may make the impulse undetectable.

AUSCULTATION

Overview. Auscultation of heart sounds and murmurs is a rewarding and important skill of physical examination that leads directly to several clinical diagnoses. In this section, you will learn the techniques for identifying S_1 and S_2, extra sounds in systole and diastole, and systolic and diastolic murmurs. Review the auscultatory areas on the next page with the following caveats: (1) some authorities discourage use of these names because murmurs of more than one origin may occur in a given area; and (2) these areas may not apply to patients with dextrocardia or anomalies of the great vessels. Also, if the heart is enlarged or displaced, your pattern of auscultation should be altered accordingly.

In a quiet room, listen to the heart with your stethoscope *in the right 2nd interspace* close to the sternum, *along the left sternal border* in each interspace from the 2nd through the 5th, and *at the apex.* Recall that the upper margins of the heart are sometimes termed the "base" of the heart. Some clinicians begin auscultation at the apex, others at the base. Either pattern is satisfactory. You should also listen in any area where you detect an abnormality and in areas adjacent to murmurs to determine where they are loudest and where they radiate.



Know your stethoscope! It is important to understand the uses of both the diaphragm and the bell.

- *The diaphragm.* The diaphragm is better for picking up the relatively high-pitched sounds of S_1 and S_2, the murmurs of aortic and mitral regurgitation, and pericardial friction rubs. *Listen throughout the precordium* with the diaphragm, pressing it firmly against the chest.

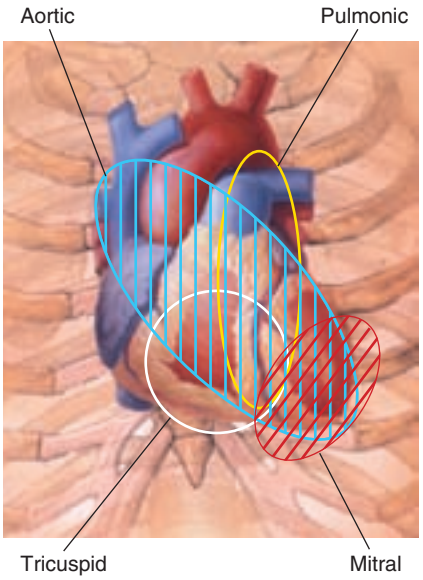
- *The bell.* The bell is more sensitive to the low-pitched sounds of S_3 and S_4 and the murmur of mitral stenosis. Apply the bell lightly, with just enough pressure to produce an air seal with its full rim. *Use the bell at the apex, then move medially along the lower sternal border.* Resting the heel of your hand on the chest like a fulcrum may help you to maintain light pressure.

  Pressing the bell firmly on the chest makes it function more like the diaphragm by stretching the underlying skin. Low-pitched sounds such as S_3 and S_4 may disappear with this technique—an observation that may help to identify them. In contrast, high-pitched sounds such as a midsystolic click, an ejection sound, or an opening snap, will persist or get louder.

  Listen to the entire precordium with the patient supine. For new patients and patients needing a complete cardiac examination, use two other important positions to listen for mitral stenosis and aortic regurgitation.

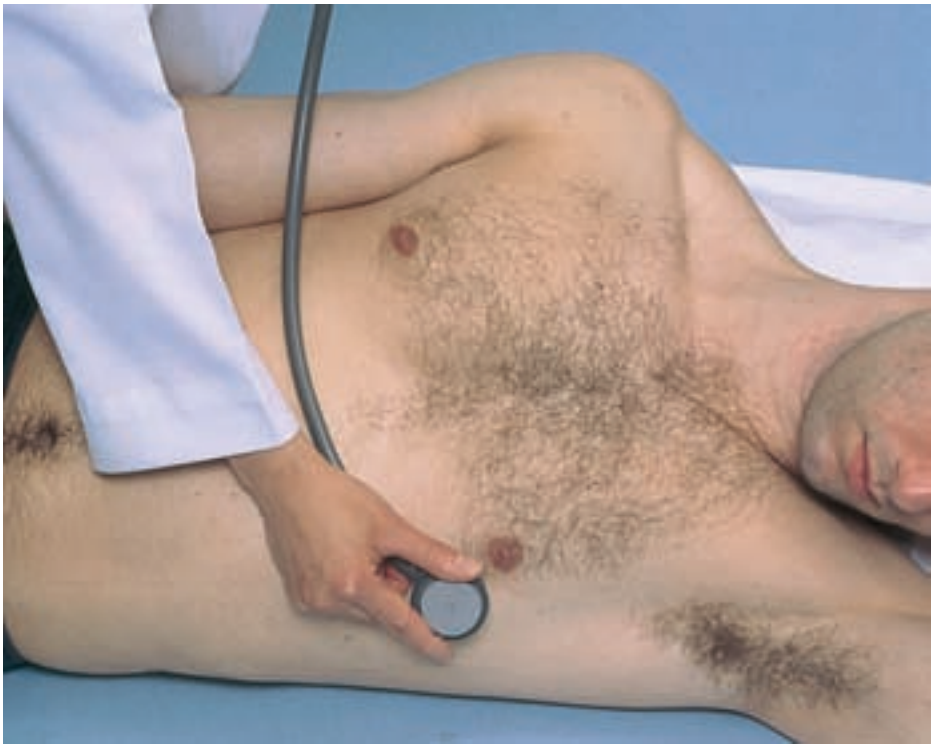
- Ask the patient to *roll partly onto the left side into the left lateral decubitus position,* bringing the left ventricle close to the chest wall. Place the bell of your stethoscope lightly on the apical impulse.

Heart sounds and murmurs that originate in the four valves are illustrated in the diagram below. Pulmonic sounds are usually heard best in the 2nd and 3rd left interspaces, but may extend further.



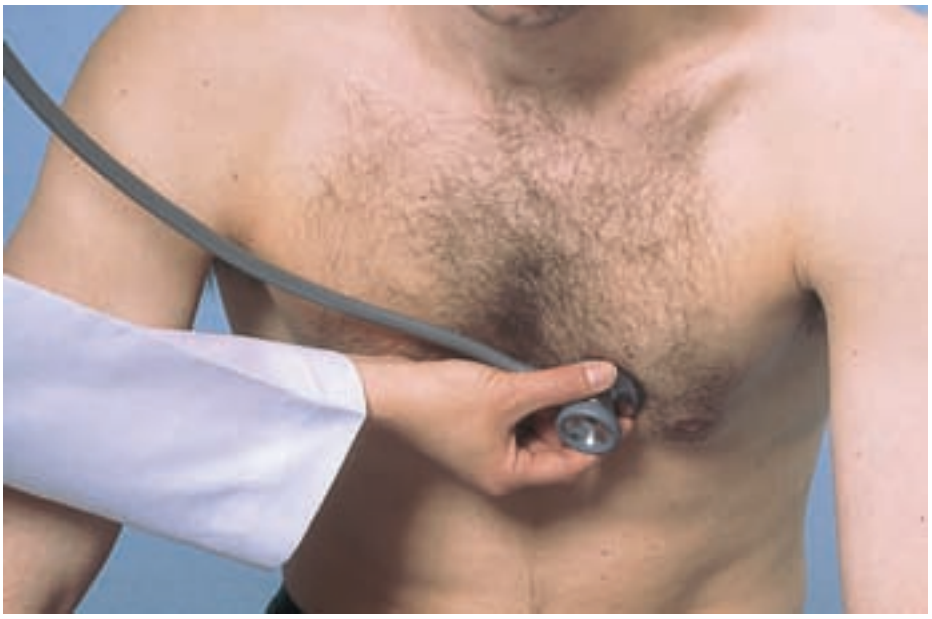
(Redrawn from Leatham A: Introduction to the Examination of the Cardiovascular System, 2nd ed. Oxford, Oxford University Press, 1979)

This position accentuates or brings out a left-sided S_3 and S_4 and mitral murmurs, especially *mitral stenosis.* You may otherwise miss these important findings.



- Ask the patient to *sit up, lean forward, exhale completely, and stop breathing in expiration*. Pressing the diaphragm of your stethoscope on the chest, listen along the left sternal border and at the apex, pausing periodically so the patient may breathe.

This position accentuates or brings out aortic murmurs. You may easily miss the soft diastolic murmur of *aortic regurgitation* unless you listen at this position.



Listening for Heart Sounds. Throughout your examination, take your time at each auscultatory area. Concentrate on each of the events in the cardiac cycle listed on the next page and sounds you may hear in systole and diastole.

Auscultatory Sounds

Heart Sounds	Guides to Auscultation	Examples of Abnormalities
S_1	Note its intensity and any apparent splitting. Normal splitting is detectable along the lower left sternal border.	See Table 8-4, Variations in the First Heart Sound—S_1 (p. 327).
S_2	Note its intensity.	
Split S_2	Listen for splitting of this sound in the 2nd and 3rd left interspaces. Ask the patient to breathe quietly, and then slightly more deeply than normal. Does S_2 split into its two components, as it normally does? If not, ask the patient to (1) breathe a little more deeply, or (2) sit up. Listen again. A thick chest wall may make the pulmonic component of S_1 inaudible.	See Table 8-5, Variations in the Second Heart Sound—S_2 (p. 328). When either A_2 or P_2 is absent, as in disease of the respective valves, S_2 is persistently single.
	Width of split. How wide is the split? It is normally quite narrow.	
	Timing of split. When in the respiratory cycle do you hear the split? It is normally heard late in inspiration.	Expiratory splitting suggests an abnormality (p. 328).
	Does the split disappear as it should, during exhalation? If not, listen again with the patient sitting up.	Persistent splitting results from delayed closure of the pulmonic valve or early closure of the aortic valve.
	Intensity of A_2 and P_2. Compare the intensity of the two components, A_2 and P_2. A_2 is usually louder.	A loud P_2 suggests pulmonary hypertension.
Extra Sounds in Systole	Such as ejection sounds or systolic clicks	The systolic click of mitral valve prolapse is the most common of these sounds. See Table 8-6, Extra Heart Sounds in Systole (p. 329).
	Note their location, timing, intensity, and pitch, and the effects of respiration on the sounds.	
Extra Sounds in Diastole	Such as S_3, S_4, or an opening snap	See Table 8-7, Extra Heart Sounds in Diastole (p. 330).
	Note the location, timing, intensity, and pitch, and the effects of respiration on the sounds. (An S_3 or S_4 in athletes is a normal finding.)	
Systolic and Diastolic Murmurs	Murmurs are differentiated from heart sounds by their longer duration.	See Table 8-8, Pansystolic (Holosystolic) Murmurs (p. 331), Table 8-9, Midsystolic Murmurs (pp. 332–333), and Table 8-10, Diastolic Murmurs (p. 334).

Attributes of Heart Murmurs. If you detect a heart murmur, you must learn to identify and describe its *timing, shape, location of maximal intensity, radiation* or transmission from this location, *intensity, pitch,* and *quality.*

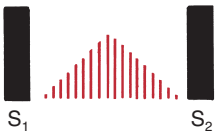
- *Timing.* First decide if you are hearing a *systolic murmur,* falling between S_1 and S_2, or a *diastolic murmur,* falling between S_2 and S_1. Palpating the carotid pulse as you listen can help you with timing. *Murmurs that coincide with the carotid upstroke are systolic.*

  Systolic murmurs are usually *midsystolic* or *pansystolic.* Late systolic murmurs may also be heard.

Diastolic murmurs usually indicate valvular heart disease. Systolic murmurs may indicate valvular disease, but often occur when the heart valves are entirely normal.

TECHNIQUES OF EXAMINATION

EXAMPLES OF ABNORMALITIES

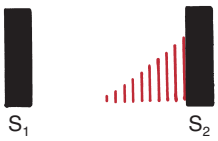


A *midsystolic murmur* begins after S_1 and stops before S_2. Brief gaps are audible between the murmur and the heart sounds. Listen carefully for the gap just before S_2. It is heard more easily and, if present, usually confirms the murmur as midsystolic, not pansystolic.

Midsystolic murmurs most often are related to blood flow across the semilunar (aortic and pulmonic) valves. See Table 8-9, Midsystolic Murmurs (p. 331).

A *pansystolic (holosystolic) murmur* starts with S_1 and stops at S_2, without a gap between murmur and heart sounds.

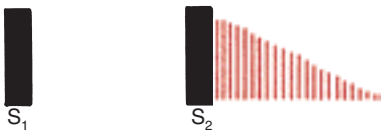
Pansystolic murmurs often occur with regurgitant (backward) flow across the atrioventricular valves. See Table 8-8, Pansystolic (Holosystolic) Murmurs (pp. 332–333).



A *late systolic murmur* usually starts in mid- or late systole and persists up to S_2.

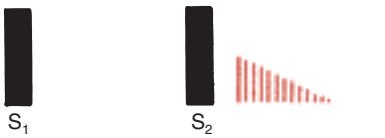
This is the murmur of mitral valve prolapse and is often, but not always, preceded by a systolic click (see p. 329).

Diastolic murmurs may be *early diastolic*, *middiastolic*, or *late diastolic*.



An *early diastolic murmur* starts right after S_2, without a discernible gap, and then usually fades into silence before the next S_1.

Early diastolic murmurs typically accompany regurgitant flow across incompetent semilunar valves.



A *middiastolic murmur* starts a short time after S_2. It may fade away, as illustrated, or merge into a late diastolic murmur.

Middiastolic and presystolic murmurs reflect turbulent flow across the atrioventricular valves. See Table 8-10, Diastolic Murmurs (p. 334).

A *late diastolic (presystolic) murmur* starts late in diastole and typically continues up to S_1.

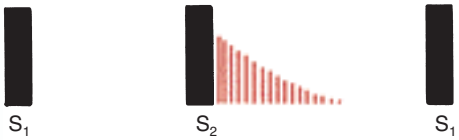
An occasional murmur, such as the murmur of a patent ductus arteriosus, starts in systole and continues without pause through S_2 into but not necessarily throughout diastole. It is then called a *continuous murmur*. Other cardiovascular sounds, such as pericardial friction rubs or venous hums, have *both systolic and diastolic components*. Observe and describe these sounds according to the characteristics used for systolic and diastolic murmurs.

See Table 8-11, Cardiovascular Sounds With Both Systolic and Diastolic Components (p. 335).

- *Shape*. The shape or configuration of a murmur is determined by its intensity over time.

A *crescendo murmur* grows louder.

The presystolic murmur of *mitral stenosis* in normal sinus rhythm

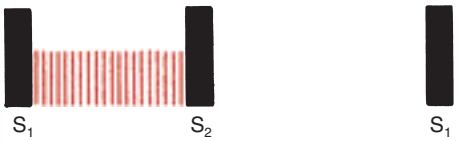


A *decrescendo murmur* grows softer.

The early diastolic murmur of *aortic regurgitation*

A *crescendo–decrescendo murmur* first rises in intensity, then falls.

The midsystolic murmur of *aortic stenosis* and *innocent flow murmurs*



A *plateau murmur* has the same intensity throughout.

The pansystolic murmur of *mitral regurgitation*

- *Location of Maximal Intensity*. This is determined by the site where the murmur originates. Find the location by exploring the area where you hear the murmur. Describe where you hear it best in terms of the interspace and its relation to the sternum, the apex, or the midsternal, the midclavicular, or one of the axillary lines.

For example, a murmur best heard in the 2nd right interspace (the aortic area) usually originates at or near the aortic valve.

- *Radiation or Transmission From the Point of Maximal Intensity*. This reflects not only the site of origin but also the intensity of the murmur and the direction of blood flow. Explore the area around a murmur and determine where else you can hear it.

A loud murmur of *aortic stenosis* often radiates into the neck (in the direction of arterial flow).

- *Intensity*. This is usually graded on a 6-point scale and expressed as a fraction. The numerator describes the intensity of the murmur wherever it is loudest, and the denominator indicates the scale you are using. Intensity is influenced by the thickness of the chest wall and the presence of intervening tissue.

An identical degree of turbulence would cause a louder murmur in a thin person than in a very muscular or obese person. Emphysematous lungs may diminish the intensity of murmurs.

Learn to grade murmurs using the 6-point scale below. Note that grades 4 through 6 require the added presence of a palpable thrill.

Gradations of Murmurs

Grade	Description
Grade 1	Very faint, heard only after listener has "tuned in"; may not be heard in all positions
Grade 2	Quiet, but heard immediately after placing the stethoscope on the chest
Grade 3	Moderately loud
Grade 4	Loud, with palpable thrill
Grade 5	Very loud, with thrill. May be heard when the stethoscope is partly off the chest
Grade 6	Very loud, with thrill. May be heard with stethoscope entirely off the chest

- *Pitch.* This is categorized as high, medium, or low.
- *Quality.* This is described in terms such as blowing, harsh, rumbling, and musical.

A fully described murmur might be: a "medium-pitched, grade 2/6, blowing decrescendo diastolic murmur, heard best in the 4th left interspace, with radiation to the apex" (aortic regurgitation).

Other useful characteristics of murmurs—and heart sounds too—include variation with respiration, with the position of the patient, or with other special maneuvers.

Murmurs originating in the right side of the heart tend to change more with respiration than left-sided murmurs.

INTEGRATING CARDIOVASCULAR ASSESSMENT

A good cardiovascular examination requires more than observation. You need to think about the possible meanings of your individual observations, fit them together in a logical pattern, and correlate your cardiac findings with the patient's blood pressure, arterial pulses, venous pulsations, jugular venous pressure, the remainder of your physical examination, and the patient's history.

In a 60-year-old person with angina, you might hear a harsh 3/6 midsystolic crescendo–decrescendo murmur in the right 2nd interspace radiating to the neck. These findings suggest *aortic stenosis,* but could arise from *aortic sclerosis* (leaflets sclerotic but not stenotic), a dilated aorta, or increased flow across a normal valve. Assess any delay in the carotid upstroke and the blood pressure for evidence of *aortic stenosis.* Check the apical impulse for left ventricular hypertrophy. Listen for *aortic regurgitation* as the patient leans forward and exhales.

Evaluating the common systolic murmur illustrates this point. In examining an asymptomatic teenager, for example, you might hear a grade 2/6 midsystolic murmur in the 2nd and 3rd left interspaces. Because this suggests a murmur of pulmonic origin, you should assess the size of the right ventricle by carefully palpating the left parasternal area. Because pulmonic stenosis and atrial septal defects can occasionally cause such murmurs, listen carefully to the splitting of the second heart sound and try to hear any ejection sounds. Listen to the murmur after the patient sits up. Look for evidence of anemia, hyperthyroidism, or pregnancy that could produce such a murmur by increasing the flow across the aortic or the pulmonic valve. If all your findings are normal, your patient probably has an *innocent murmur*—one with no pathologic significance.

Put all this information together to make a hypothesis about the origin of the murmur.

SPECIAL TECHNIQUES

Aids to Identify Systolic Murmurs. Elsewhere in this chapter you have learned how to improve your auscultation of heart sounds and murmurs by placing the patient in different positions. Two additional techniques help you distinguish the murmurs of mitral valve prolapse and hypertrophic cardiomyopathy from aortic stenosis.

(1) Standing and Squatting. When a person stands, venous return to the heart decreases, as does peripheral vascular resistance. Arterial blood pressure, stroke volume, and the volume of blood in the left ventricle all decline. When squatting, changes occur in the opposite direction. These changes help (1) to identify a prolapsed mitral valve, and (2) to distinguish hypertrophic cardiomyopathy from aortic stenosis.

Secure the patient's gown so that it will not interfere with your examination, and ready yourself for prompt auscultation. Instruct the patient to squat next to the examining table and hold on to it for balance. Listen to the heart with the patient in the squatting position and again in the standing position.

(2) Valsalva Maneuver. When a person strains down against a closed glottis, venous return to the right heart is decreased, and after a few seconds, left ventricular volume and arterial blood pressure both fall. Release of the effort has the opposite effects. These changes help to distinguish prolapse of the mitral valve and hypertrophic cardiomyopathy from aortic stenosis.

The patient should be lying down. Ask the patient to "bear down," or place one hand on the midabdomen and instruct the patient to strain against it. By adjusting the pressure of your hand you can alter the patient's effort to the desired level. Use your other hand to place your stethoscope on the patient's chest.

■ *Maneuvers to Identify Systolic Murmurs*

		Effect on Systolic Sounds and Murmurs		
Maneuver	**Cardiovascular Effect**	*Mitral Valve Prolapse*	*Hypertrophic Cardiomyopathy*	*Aortic Stenosis*
Standing; Strain Phase of Valsalva	**Decreased left ventricular volume** from ↓ venous return to heart	↑ prolapse of mitral valve	↑ outflow obstruction	↓ blood volume ejected into aorta
	Decreased vascular tone: ↓ arterial blood pressure	*Click moves earlier in systole* and *murmur lengthens*	↑ **intensity of murmur**	↓ **intensity of murmur**
Squatting; Release of Valsalva	**Increased left ventricular volume** from ↑ venous return to heart	↓ prolapse of mitral valve	↓ outflow obstruction	↑ blood volume ejected into aorta
	Increased vascular tone: ↑ arterial blood pressure; ↑ peripheral vascular resistance	*Delay of click* and *murmur shortens*	↓ **intensity of murmur**	↑ **intensity of murmur**

Pulsus Alternans. In *pulsus alternans,* the rhythm of the pulse remains regular, but the *force* of the arterial pulse alternates because of alternating strong and weak ventricular contractions. *Pulsus alternans* almost always indicates severe left-sided heart failure and is usually best felt by applying light pressure on the radial or femoral arteries.[35] Use a blood pressure cuff to confirm your finding. After raising the cuff pressure, lower it slowly to the systolic level—the initial Korotkoff sounds are the strong beats. As you lower the cuff, you will hear the softer sounds of the alternating weak beats.

Alternately loud and soft Korotkoff sounds or a sudden doubling of the apparent heart rate as the cuff pressure declines indicates a *pulsus alternans* (see p. 119).

The upright position may accentuate the alternation.

Paradoxical Pulse. If you have noted that the pulse varies in amplitude with respiration or if you suspect pericardial tamponade (because of increased jugular venous pressure, a rapid and diminished pulse, and dyspnea, for example), use a blood-pressure cuff to check for a *paradoxical pulse*. This is a greater than normal drop in systolic pressure during inspiration. As the

The level identified by first hearing Korotkoff sounds is the highest systolic pressure during the respiratory cycle. The level identified by hearing sounds throughout the cycle is the

patient breathes, quietly if possible, lower the cuff pressure slowly to the systolic level. Note the pressure level at which the first sounds can be heard. Then drop the pressure very slowly until sounds can be heard throughout the respiratory cycle. Again note the pressure level. The difference between these two levels is normally no greater than 3 or 4 mm Hg.

lowest systolic pressure. A difference between these levels of more than 10 mm Hg indicates a paradoxical pulse and suggests *pericardial tamponade,* possible *constrictive pericarditis,* but most commonly *obstructive airway disease* (see p. 119).

RECORDING YOUR FINDINGS

Note that initially you may use sentences to describe your findings; later you will use phrases. The style below contains phrases appropriate for most write-ups.

Recording the Physical Examination—The Cardiovascular Examination

"The jugular venous pulse (JVP) is 3 cm above the sternal angle with the head of bed elevated to 30°. Carotid upstrokes are brisk, without bruits. The point of maximal impulse (PMI) is tapping, 7 cm lateral to the midsternal line in the 5th intercostal space. Good S_1 and S_2. No murmurs or extra sounds."

OR

"The JVP is 5 cm above the sternal angle with the head of bed elevated to 50°. Carotid upstrokes are brisk; a bruit is heard over the left carotid artery. The PMI is diffuse, 3 cm in diameter, palpated at the anterior axillary line in the 5th and 6th intercostal spaces. S_1 and S_2 are soft. S_3 present. Harsh 2/6 holosystolic murmur best heard at the apex, radiating to the lower left sternal border (LLSB). No S_4 or diastolic murmurs."

Suggests *congestive heart failure* with possible *left carotid occlusion* and *mitral regurgitation.*[36–38]

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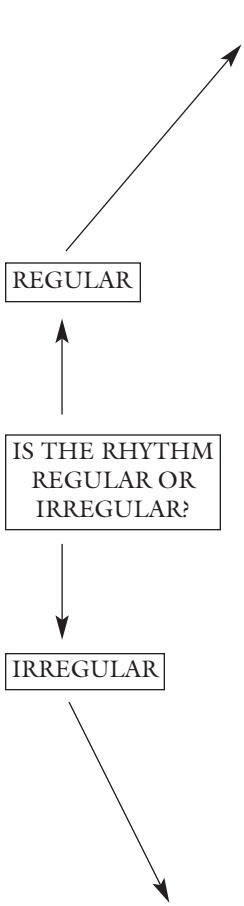
TABLE 8-1 Selected Heart Rates and Rhythms

Cardiac rhythms may be classified as *regular* or *irregular*. When rhythms are irregular or rates are fast or slow, obtain an ECG to identify the origin of the beats (sinus node, AV node, atrium, or ventricle) and the pattern of conduction. Note that with AV (atrioventricular) block, arrhythmias may have a fast, normal, or slow ventricular rate.



	ECG Pattern	Usual Resting Rate
FAST (>100)	Sinus tachycardia	100–180
	Supraventricular (atrial or nodal) tachycardia	150–250
	Atrial flutter with a regular ventricular response	100–175
	Ventricular tachycardia	110–250
OR		
NORMAL (60–100)	Normal sinus rhythm	60–100
	Second-degree AV block	60–100
	Atrial flutter with a regular ventricular response	75–100
OR		
SLOW (<60)	Sinus bradycardia	<60
	Second-degree AV block	30–60
	Complete heart block	<40
RHYTHMIC OR SPORADIC	With early beats, atrial or nodal (supraventricular) premature contraction OR ventricular premature contractions	See Table 8-2
	Sinus arrhythmia	
OR		
TOTAL	Atrial fibrillation	
	Atrial flutter with varying block	

TABLE 8-2 Selected Irregular Rhythms

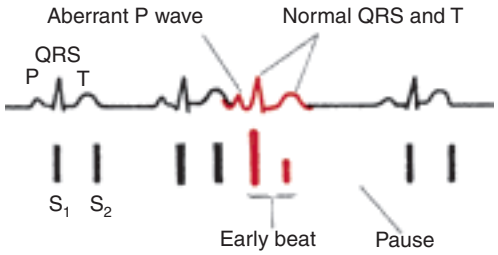
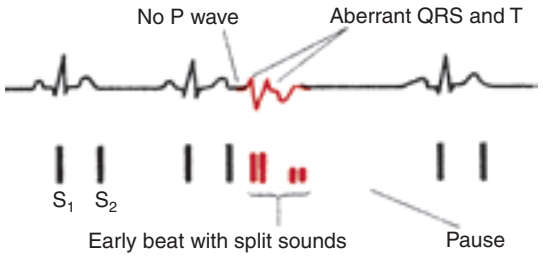
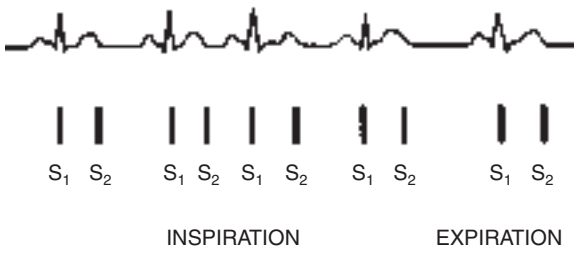
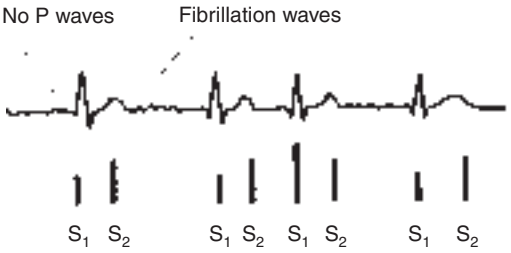
Type of Rhythm	ECG Waves and Heart Sounds	
Atrial or Nodal Premature Contractions (*Supraventricular*)		**Rhythm.** A beat of atrial or nodal origin comes earlier than the next expected normal beat. A pause follows, and then the rhythm resumes. **Heart Sounds.** S_1 may differ in intensity from the S_1 of normal beats, and S_2 may be decreased. Both sounds are otherwise similar to those of normal beats.
Ventricular Premature Contractions		**Rhythm.** A beat of ventricular origin comes earlier than the next expected normal beat. A pause follows, and the rhythm resumes. **Heart Sounds.** S_1 may differ in intensity from the S_1 of the normal beats, and S_2 may be decreased. Both sounds are likely to be split.
Sinus Arrhythmia		**Rhythm.** The heart varies cyclically, usually speeding up with inspiration and slowing down with expiration. **Heart Sounds.** Normal, although S_1 may vary with the heart rate.
Atrial Fibrillation and Atrial Flutter With Varying AV Block		**Rhythm.** The ventricular rhythm is totally irregular, although short runs of the irregular ventricular rhythm may seem regular. **Heart Sounds.** S_1 varies in intensity.

TABLE 8-3 Variations and Abnormalities of the Ventricular Impulses

In the healthy heart, the *left ventricular impulse* is usually the *point of maximal impulse,* or *PMI.* This brief impulse is generated by the movement of the ventricular apex against the chest wall during contraction. The *right ventricular impulse* is normally not palpable beyond infancy, and its characteristics are indeterminate. In contrast, learn the classical descriptors of the left ventricular PMI:

- *Location:* in the 4th or 5th interspace, ~7–10 cm lateral to the midsternal line, depending on the diameter of the chest
- *Diameter: discrete,* or ≤2 cm
- *Amplitude: brisk* and *tapping*
- *Duration:* ≤2/3 of systole

Careful examination of the ventricular impulse gives you important clues about underlying cardiovascular hemodynamics. The quality of the ventricular impulse changes as the left and right ventricles adapt to high-output states (anxiety, hyperthyroidism, and severe anemia) and to the more pathologic conditions of chronic pressure or volume overload. Note below the distinguishing features of three types of ventricular impulses: the *hyperkinetic ventricular impulse* from transiently increased stroke volume—this change does not necessarily indicate heart disease; the *sustained* ventricular impulse of ventricular hypertrophy from chronic pressure load, known as *increased afterload* (see p. 311); and the *diffuse* ventricular impulse of ventricular dilation from chronic volume overload, or *increased preload.*

	Left Ventricular Impulse			Right Ventricular Impulse		
	Hyperkinetic	*Pressure Overload*	*Volume Overload*	*Hyperkinetic*	*Pressure Overload*	*Volume Overload*
Examples of Causes	Anxiety, hyperthyroidism, severe anemia	Aortic stenosis, hypertension	Aortic or mitral regurgitation	Anxiety, hyperthyroidism, severe anemia	Pulmonic stenosis, pulmonary hypertension	Atrial septal defect
Location	Normal	Normal	Displaced to the left and possibly downward	3rd, 4th, or 5th left interspaces	3rd, 4th, or 5th left interspaces, also subxiphoid	Left sternal border, extending toward the left cardiac border, also subxiphoid
Diameter	~2 cm, though increased amplitude may make it seem larger	>2 cm	>2 cm	Not useful	Not useful	Not useful
Amplitude	More forceful tapping	More forceful tapping	*Diffuse*	Slightly more forceful	More forceful	Slightly to markedly more forceful
Duration	<2/3 systole	*Sustained* (up to S_2)	Often slightly sustained	Normal	Sustained	Normal to slightly sustained

TABLE 8-4 Variations in the First Heart Sound—S_1

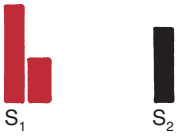
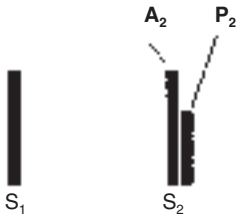
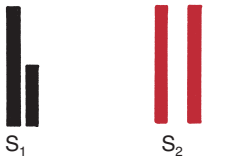
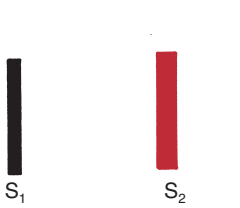
Normal Variations		S_1 is softer than S_2 at the *base* (right and left 2nd interspaces).
		S_1 is often but not always louder than S_2 at the *apex*.
Accentuated S_1		S_1 is accentuated in (1) tachycardia, rhythms with a short PR interval, and high cardiac output states (e.g., exercise, anemia, hyperthyroidism), and (2) mitral stenosis. In these conditions, the mitral valve is still open wide at the onset of ventricular systole, and then closes quickly.
Diminished S_1		S_1 is diminished in first-degree heart block (delayed conduction from atria to ventricles). Here the mitral valve has had time after atrial contraction to float back into an almost closed position before ventricular contraction shuts it. It closes less loudly. S_1 is also diminished (1) when the mitral valve is calcified and relatively immobile, as in mitral regurgitation, and (2) when left ventricular contractility is markedly reduced, as in congestive heart failure or coronary heart disease.
Varying S_1		S_1 varies in intensity (1) in complete heart block, when atria and ventricles are beating independently of each other, and (2) in any totally irregular rhythm (e.g., atrial fibrillation). In these situations, the mitral valve is in varying positions before being shut by ventricular contraction. Its closure sound, therefore, varies in loudness.
Split S_1		S_1 may be split normally along the lower left sternal border where the tricuspid component, often too faint to be heard, becomes audible. This split may sometimes be heard at the apex, but consider also an S_4, an aortic ejection sound, and an early systolic click. Abnormal splitting of both heart sounds may be heard in right bundle branch block and in premature ventricular contractions.

TABLE 8-5 Variations in the Second Heart Sound—S_2

	Inspiration	Expiration
Physiologic Splitting		Listen for *physiologic splitting* of S_2 in the *2nd or 3rd left interspace*. The pulmonic component of S_2 is usually too faint to be heard at the apex or aortic area, where S_2 is a single sound derived from aortic valve closure alone. Normal splitting is *accentuated by inspiration* and usually *disappears on expiration*. In some patients, especially younger ones, S_2 may not become single on expiration. It may merge when the patient sits up.
Pathologic Splitting *(involves splitting during expiration and suggests heart disease)*		*Wide splitting* of S_2 refers to an increase in the usual splitting that persists throughout the respiratory cycle. Wide splitting can be caused by delayed closure of the pulmonic valve (e.g., by pulmonic stenosis or right bundle branch block). As illustrated here, right bundle branch block also causes splitting of S_1 into its mitral and tricuspid components. Wide splitting can also be caused by early closure of the aortic valve, as in mitral regurgitation.
		*Fixed splitting* refers to wide splitting that does not vary with respiration. It occurs in atrial septal defect and right ventricular failure.
		*Paradoxical or reversed splitting* refers to splitting that appears on expiration and disappears on inspiration. Closure of the aortic valve is abnormally delayed so that A_2 follows P_2 in expiration. Normal inspiratory delay of P_2 makes the split disappear. The most common cause of paradoxical splitting is left bundle branch block.

Increased Intensity of A_2 in the Right Second Interspace (where only A_2 can usually be heard) occurs in systemic hypertension because of the increased pressure load. It also occurs when the aortic root is dilated, probably because the aortic valve is then closer to the chest wall.

Decreased or Absent A_2 in the Right Second Interspace is noted in calcific aortic stenosis because of valve immobility. If A_2 is inaudible, no splitting is heard.

Increased Intensity of P_2. When P_2 is equal to or louder than A_2, suspect pulmonary hypertension. Other causes include a dilated pulmonary artery and an atrial septal defect. When a split S_2 is heard widely, even at the apex and the right base, P_2 is accentuated.

Decreased or Absent P_2 is usually due to the increased anteroposterior diameter of the chest associated with aging. It can also result from pulmonic stenosis. If P_2 is inaudible, no splitting is heard.

TABLE 8-6 Extra Heart Sounds in Systole

There are two kinds of extra heart sounds in systole: (1) early ejection sounds, and (2) clicks, commonly heard in mid- and late systole.

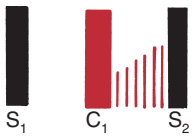
Early Systolic Ejection Sounds

Early systolic ejection sounds occur shortly after S_1, coincident with opening of the aortic and pulmonic valves. They are relatively high in pitch, have a sharp, clicking quality, and are heard better with the diaphragm of the stethoscope. An ejection sound indicates cardiovascular disease.

Listen for an *aortic ejection sound* at both the base and apex. It may be louder at the apex and usually does not vary with respiration. An aortic ejection sound may accompany a dilated aorta, or aortic valve disease from congenital stenosis or a bicuspid valve.

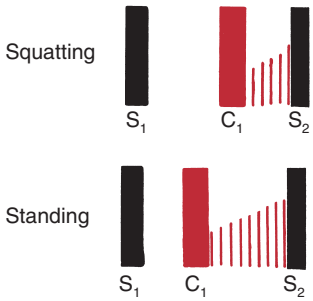
A *pulmonic ejection sound* is heard best in the 2nd and 3rd left interspaces. When S_1, usually relatively soft in this area, appears to be loud, you may be hearing a pulmonic ejection sound. Its intensity often *decreases with inspiration*. Causes include dilatation of the pulmonary artery, pulmonary hypertension, and pulmonic stenosis.

Systolic Clicks



Systolic clicks are usually due to *mitral valve prolapse*—an abnormal systolic ballooning of part of the mitral valve into the left atrium. The clicks are usually mid- or late systolic. Prolapse of the mitral valve is a common cardiac condition, affecting about 5% of the general population. There is equal prevalence in men and women.

The click is usually single, but you may hear more than one, usually *at or medial to the apex*, but also *at the lower left sternal border*. It is high-pitched, so listen with the diaphragm. The click is often followed by a late systolic murmur from mitral regurgitation—a flow of blood from left ventricle to left atrium. The murmur usually crescendos up to S_2. Auscultatory findings are notably variable. Most patients have only a click, some have only a murmur, and some have both. Systolic clicks may also be of extracardial or mediastinal origin.



Findings vary from time to time and often change with body position. Several positions are recommended to identify the syndrome: supine, seated, squatting, and standing. *Squatting delays the click and murmur; standing moves them closer to* S_1.

TABLE 8-7 Extra Heart Sounds in Diastole

Opening Snap

The *opening snap* is a very early diastolic sound usually produced by the opening of a *stenotic mitral valve*. It is heard best just medial to the apex and along the lower left sternal border. When it is loud, an opening snap radiates to the apex and to the pulmonic area, where it may be mistaken for the pulmonic component of a split S_2. Its high pitch and snapping quality help to distinguish it from an S_2. It is heard better with the diaphragm.

S_3

You will detect *physiologic* S_3 frequently in children and in young adults to the age of 35 or 40. It is common during the last trimester of pregnancy. Occurring early in diastole during rapid ventricular filling, it is later than an opening snap, dull and low in pitch, and heard best at the apex in the left lateral decubitus position. Use the bell of the stethoscope should be used with very light pressure.

A *pathologic* S_3 or *ventricular gallop* sounds just like a physiologic S_3. An S_3 in a person over age 40 (possibly a little older in women) is almost certainly pathologic. Causes include decreased myocardial contractility, congestive heart failure, and volume overloading of a ventricle, as in mitral or tricuspid regurgitation. A *left-sided* S_3 is heard typically at the apex in the left lateral decubitus position. A *right-sided* S_3 is usually heard along the lower left sternal border or below the xiphoid with the patient supine, and is louder on inspiration. The term *gallop* comes from the cadence of three heart sounds, especially at rapid heart rates, and sounds like "Kentucky."

S_4

An S_4 (*atrial sound* or *atrial gallop*) occurs just before S_1. It is dull, low in pitch, and heard better with the bell. An S_4 is heard occasionally in an apparently normal person, especially in trained athletes and older age groups. More commonly, it is due to increased resistance to ventricular filling following atrial contraction. This increased resistance is related to decreased compliance (increased stiffness) of the ventricular myocardium.

Causes of a left-sided S_4 include hypertensive heart disease, coronary artery disease, aortic stenosis, and cardiomyopathy. A *left-sided* S_4 is heard best at the apex in the left lateral position; it may sound like "Tennessee." The less common *right-sided* S_4 is heard along the lower left sternal border or below the xiphoid. It often gets louder with inspiration. Causes of a right-sided S_4 include pulmonary hypertension and pulmonic stenosis.

An S_4 may also be associated with delayed conduction between atria and ventricles. This delay separates the normally faint atrial sound from the louder S_1 and makes it audible. An S_4 is never heard in the absence of atrial contraction, which occurs with atrial fibrillation.

Occasionally, a patient has both an S_3 and an S_4, producing a *quadruple rhythm* of four heart sounds. At rapid heart rates, the S_3 and S_4 may merge into one loud extra heart sound, called a *summation gallop*.

TABLE 8-8 Pansystolic (Holosystolic) Murmurs

Pansystolic (holosystolic) murmurs are pathologic, arising from blood flow from a chamber with high pressure to one of lower pressure, through a valve or other structure that should be closed. The murmur begins immediately with S_1 and continues up to S_2.

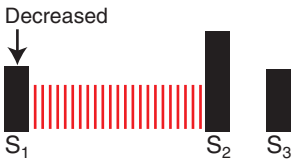
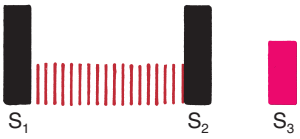
	Mitral Regurgitation	Tricuspid Regurgitation	Ventricular Septal Defect
			
Murmur	*Location.* Apex *Radiation.* To the left axilla, less often to the left sternal border *Intensity.* Soft to loud; if loud, associated with an apical thrill *Pitch.* Medium to high *Quality.* Harsh, holosystolic *Aids.* Unlike tricuspid regurgitation, it does not become louder in inspiration.	*Location.* Lower left sternal border *Radiation.* To the right of the sternum, to the xiphoid area, and perhaps to the left midclavicular line, but not into the axilla *Intensity.* Variable *Pitch.* Medium *Quality.* Blowing, holosystolic *Aids.* Unlike mitral regurgitation, the intensity may increase slightly with inspiration.	*Location.* 3rd, 4th, and 5th left interspaces *Radiation.* Often wide *Intensity.* Often very loud, with a thrill *Pitch.* High, holosystolic *Quality.* Often harsh
Associated Findings	S_1 is often decreased. An apical S_3 reflects volume overload on the left ventricle. The apical impulse is increased in amplitude and may be *sustained.*	The right ventricular impulse is increased in amplitude and may be *sustained.* An S_3 may be audible along the lower left sternal border. The jugular venous pressure is often elevated, and large *v* waves may be seen in the jugular veins.	A_2 may be obscured by the loud murmur. Findings vary with the severity of the defect and with associated lesions.
Mechanism	When the *mitral valve fails to close fully in systole,* blood regurgitates from left ventricle to left atrium, causing a murmur. This leakage creates volume overload on the left ventricle, with subsequent dilatation and hypertrophy. Several structural abnormalities cause this condition, and findings may vary accordingly.	When the *tricuspid valve fails to close fully in systole,* blood regurgitates from right ventricle to right atrium, producing a murmur. The most common cause is right ventricular failure and dilatation, with resulting enlargement of the tricuspid orifice. Either pulmonary hypertension or left ventricular failure is the usual initiating cause.	A ventricular septal defect is a congenital abnormality in which *blood flows from the relatively high-pressure left ventricle into the low-pressure right ventricle through a hole.* The defect may be accompanied by other abnormalities, but an uncomplicated lesion is described here.

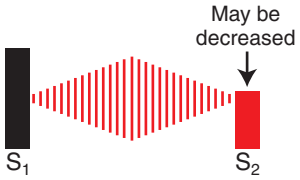
TABLE 8-9 Midsystolic Murmurs

Midsystolic ejection murmurs are the most common kind of heart murmur. They may be (1) *innocent*—without any detectable physiologic or structural abnormality; (2) *physiologic*—from physiologic changes in body metabolism; or (3) *pathologic*—arising from a structural abnormality in the heart or great vessels.[39,40] Midsystolic murmurs tend to peak near midsystole and usually stop before S_2. The crescendo–decrescendo or "diamond" shape is not always audible, but the gap between the murmur and S_2 helps to distinguish midsystolic from pansystolic murmurs.

	Innocent Murmurs	Physiologic Murmurs
		
Murmur	*Location.* 2nd to 4th left interspaces between the left sternal border and the apex *Radiation.* Little *Intensity.* Grade 1 to 2, possibly 3 *Pitch.* Soft to medium *Quality.* Variable *Aids.* Usually decreases or disappears on sitting	Similar to innocent murmurs
Associated Findings	None: normal splitting, no ejection sounds, no diastolic murmurs, and no palpable evidence of ventricular enlargement. Occasionally, both an innocent murmur and another kind of murmur are present.	Possible signs of a likely cause
Mechanism	Innocent murmurs result from turbulent blood flow, probably generated by ventricular ejection of blood into the aorta from the left and occasionally the right ventricle. Very common in children and young adults—may also be heard in older people. There is no underlying cardiovascular disease.	Turbulence due to a temporary increase in blood flow in predisposing conditions such as anemia, pregnancy, fever, and hyperthyroidism.

Pathologic Murmurs

Aortic Stenosis[41]



Location. Right 2nd interspace

Radiation. Often to the carotids, down the left sternal border, even to the apex

Intensity. Sometimes soft but often loud, with a thrill

Pitch. Medium, harsh; crescendo–decrescendo may be higher at the apex

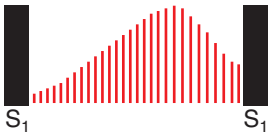
Quality. Often harsh; may be more musical at the apex

Aids. Heard best with the patient sitting and leaning forward

A_2 decreases as aortic stenosis worsens. A_2 may be delayed and merge with P_2 → single S_2 on expiration or paradoxical S_2 split. Carotid upstroke may be *delayed* with slow rise and small amplitude. Hypertrophied left ventricle may → *sustained* apical impulse and an S_4 from decreased compliance.

Significant aortic valve stenosis impairs blood flow across the valve, causing turbulence, and increases left ventricular afterload. Causes are congenital, rheumatic, and degenerative; findings may differ with each cause. Other conditions mimic aortic stenosis without obstructing flow: *aortic sclerosis,* a stiffening of aortic valve leaflets associated with aging; a *bicuspid aortic valve,* a congenital condition that may not be recognized until adulthood; *a dilated aorta,* as in arteriosclerosis, syphilis, or Marfan's syndrome; *pathologically increased flow across the aortic valve* during systole, as in aortic regurgitation.

Hypertrophic Cardiomyopathy



Location. 3rd and 4th left interspaces

Radiation. Down the left sternal border to the apex, possibly to the base, but not to the neck

Intensity. Variable

Pitch. Medium

Quality. Harsh

Aids. Decreases with squatting, increases with straining down from Valsalva

An S_3 may be present. An S_4 is often present at the apex (unlike mitral regurgitation). The apical impulse may be *sustained* and have two palpable components. The carotid pulse rises *quickly,* unlike the pulse in aortic stenosis.

Massive ventricular hypertrophy is associated with unusually rapid ejection of blood from the left ventricle during systole. Outflow tract obstruction to flow may coexist. Accompanying distortion of the mitral valve may cause mitral regurgitation.

Pulmonic Stenosis

Location. 2nd and 3rd left interspaces

Radiation. If loud, toward the left shoulder and neck

Intensity. Soft to loud; if loud, associated with a thrill

Pitch. Medium; crescendo–decrescendo

Quality. Often harsh

In severe stenosis, S_2 is widely split, and P_2 is diminished or inaudible. An early pulmonic ejection sound is common. May hear a right-sided S_4. Right ventricular impulse often increased in amplitude and *sustained*.

Pulmonic valve stenosis impairs flow across the valve, increasing right ventricular afterload. Congenital and usually found in children. In an *atrial septal defect,* the systolic murmur from pathologically increased flow across the pulmonic valve may mimic pulmonic stenosis.

TABLE 8-10 Diastolic Murmurs

Diastolic murmurs almost always indicate heart disease. There are two basic types. *Early decrescendo diastolic murmurs* signify regurgitant flow through an incompetent semilunar valve, more commonly the aortic. *Rumbling diastolic murmurs in mid- or late diastole* suggest stenosis of an atrioventricular valve, usually the mitral.

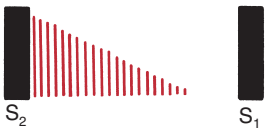
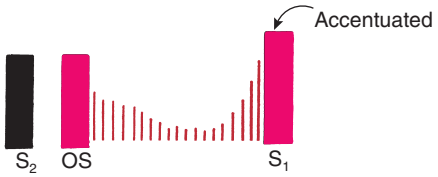
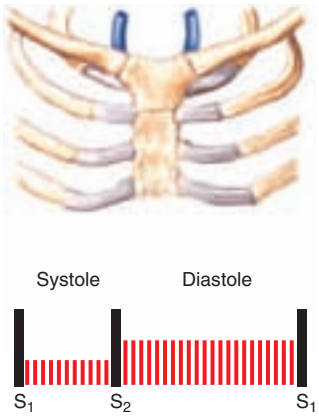
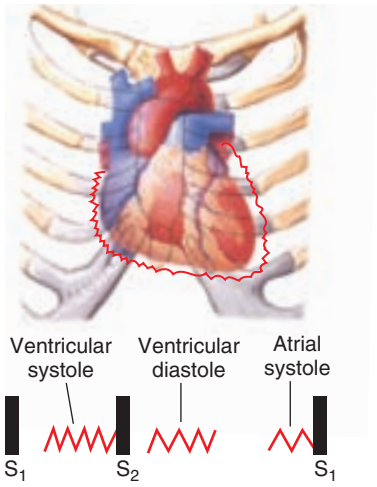
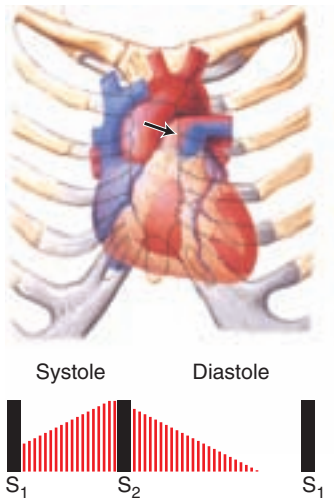
	Aortic Regurgitation[43,44]	Mitral Stenosis
		
Murmur	*Location.* 2nd to 4th left interspaces *Radiation.* If loud, to the apex, perhaps to the right sternal border *Intensity.* Grade 1 to 3 *Pitch.* High. *Use the diaphragm.* *Quality.* Blowing decrescendo; may be mistaken for breath sounds *Aids.* The murmur is heard best with the *patient sitting, leaning forward,* with breath held after exhalation.	*Location.* Usually limited to the apex *Radiation.* Little or none *Intensity.* Grade 1 to 4 *Pitch.* Decrescendo low-pitched rumble. *Use the bell.* *Aids.* Placing the bell exactly on the apical impulse, turning the patient into a *left lateral position,* and mild exercise all help to make the murmur audible. It is heard better in exhalation.
Associated Findings	An ejection sound may be present. An S_3 or S_4, if present, suggests severe regurgitation. Progressive changes in the apical impulse include increased amplitude, displacement laterally and downward, widened diameter, and increased duration. The pulse pressure increases, and *arterial pulses are often large and bounding.* A midsystolic flow murmur or an Austin Flint murmur suggests large regurgitant flow.	S_1 is accentuated and may be palpable at the apex. An opening snap (OS) often follows S_2 and initiates the murmur. If pulmonary hypertension develops, P_2 is accentuated, and the right ventricular impulse becomes palpable. Mitral regurgitation and aortic valve disease may be associated with mitral stenosis.
Mechanism	The leaflets of the aortic valve fail to close completely during diastole, and blood regurgitates from the aorta back into the left ventricle. Volume overload on the left ventricle results. Two other murmurs may be associated: (1) a midsystolic murmur from the resulting increased forward flow across the aortic valve, and (2) a mitral diastolic (*Austin Flint*) murmur, attributed to diastolic impingement of the regurgitant flow on the anterior leaflet of the mitral valve.	When the leaflets of the mitral valve thicken, stiffen, and become distorted from the effects of rheumatic fever, the *mitral valve fails to open sufficiently in diastole.* The resulting murmur has two components: (1) middiastolic (during rapid ventricular filling), and (2) presystolic (during atrial contraction). The latter disappears if atrial fibrillation develops, leaving only a middiastolic rumble.

TABLE 8-11 Cardiovascular Sounds With Both Systolic and Diastolic Components

Some cardiovascular sounds extend beyond one phase of the cardiac cycle. Three examples, further described below, are: (1) a *venous hum*, a benign sound produced by turbulence of blood in the jugular veins—common in children; (2) a *pericardial friction rub*, produced by inflammation of the pericardial sac; and (3) *patent ductus arteriosus*, a congenital abnormality in which an open channel persists between aorta and pulmonary artery. *Continuous murmurs* begin in systole and extend through S_2 into all or part of diastole, as in *patent ductus arteriosus*.

	Venous Hum	Pericardial Friction Rub	Patent Ductus Arteriosus
			
Timing	Continuous murmur without a silent interval. Loudest in diastole	May have three short components, each associated with friction from cardiac movement in the pericardial sac: (1) atrial systole, (2) ventricular systole, and (3) ventricular diastole. Usually the first two components are present; all three make diagnosis easy; only one (usually the systolic) invites confusion with a murmur.	Continuous murmur in both systole and diastole, often with a silent interval late in diastole. Loudest in late systole, obscures S_2, and fades in diastole
Location	Above the medial third of the clavicles, especially on the right	Variable, but usually heard best in the 3rd interspace to the left of the sternum	Left 2nd interspace
Radiation	1st and 2nd interspaces	Little	Toward the left clavicle
Intensity	Soft to moderate. Can be obliterated by pressure on the jugular veins	Variable. May increase when the patient leans forward, exhales, and holds breath (in contrast to pleural rub)	Usually loud, sometimes associated with a thrill
Quality	Humming, roaring	Scratchy, scraping	Harsh, machinery-like
Pitch	Low (heard better with a bell)	High (heard better with a diaphragm)	Medium