The cardiovascular system consists of the heart (a muscular pump) and the blood vessels. The blood vessels are arranged in two continuous loops, the pulmonary circulation and the systemic circulation (Fig. 19-1). When the heart contracts, it pumps blood simultaneously into both loops.

**POSITION AND SURFACE LANDMARKS**

The precordium is the area on the anterior chest directly overlying the heart and great vessels (Fig. 19-2). The great vessels are the major arteries and veins connected to the heart. The heart and the great vessels are located between the lungs in the middle third of the thoracic cage, called the mediastinum. The heart extends from the second to the fifth intercostal space and from the right border of the sternum to the left midclavicular line.

Think of the heart as an upside-down triangle in the chest. The “top” of the heart is the broader base, and the “bottom” is the apex, which points down and to the left (Fig. 19-3). During contraction, the apex beats against the chest wall, producing an apical impulse. This is palpable in most people, normally at the fifth intercostal space, 7 to 9 cm from the midsternal line.

Inside the body, the heart is rotated so that its right side is anterior and its left side is mostly posterior. Of the heart’s four chambers, the right ventricle forms the greatest area of
anterior cardiac surface. The left ventricle lies behind the right ventricle and forms the apex and slender area of the left border. The right atrium lies to the right and above the right ventricle and forms the right border. The left atrium is located posteriorly, with only a small portion, the left atrial appendage, showing anteriorly.

The great vessels lie bunched above the base of the heart. The superior and inferior vena cava return unoxygenated venous blood to the right side of the heart. The pulmonary artery leaves the right ventricle, bifurcates, and carries the venous blood to the lungs. The pulmonary veins return the freshly oxygenated blood to the left side of the heart, and the aorta carries it out to the body. The aorta ascends from the left ventricle, arches back at the level of the sternal angle, and descends behind the heart.

HEART WALL, CHAMBERS, AND VALVES

The heart wall has numerous layers. The pericardium is a tough, fibrous, double-walled sac that surrounds and protects the heart (see its cut edge in Fig. 19-4). It has two layers that contain a few milliliters of serous pericardial fluid. This ensures smooth, friction-free movement of the heart muscle. The pericardium is adherent to the great vessels, esophagus, sternum, and pleurae and is anchored to the diaphragm. The myocardium is the muscular wall of the heart; it does the pumping. The endocardium is the thin layer of endothelial tissue that lines the inner surface of the heart chambers and valves.

The common metaphor is to think of the heart as a pump. But consider that the heart is actually two pumps; the right side of the heart pumps blood into the lungs, and the left side of the heart simultaneously pumps blood into the body. The two pumps are separated by an impermeable wall, the septum.

© Pat Thomas, 2006.
Each side has an atrium and a ventricle. The atrium (Latin for “anteroom”) is a thin-walled reservoir for holding blood, and the thick-walled ventricle is the muscular pumping chamber. (It is common to use the following abbreviations to refer to the chambers: RA, right atrium; RV, right ventricle; LA, left atrium; and LV, left ventricle.)

The four chambers are separated by swinging-door–like structures, called valves, whose main purpose is to prevent backflow of blood. The valves are unidirectional; they can open only one way. The valves open and close passively in response to pressure gradients in the moving blood.
There are four valves in the heart (see Fig. 19-4). The two atrioventricular (AV) valves separate the atria and the ventricles. The right AV valve is the tricuspid, and the left AV valve is the bicuspid or mitral valve (so named because it resembles a bishop’s mitred cap). The valves’ thin leaflets are anchored by collagenous fibers (chordae tendineae) to papillary muscles embedded in the ventricle floor. The AV valves open during the heart’s filling phase, or diastole, to allow the ventricles to fill with blood. During the pumping phase, or systole, the AV valves close to prevent regurgitation of blood back up into the atria. The papillary muscles contract at this time, so that the valve leaflets meet and unite to form a perfect seal without turning themselves inside out.

The semilunar (SL) valves are set between the ventricles and the arteries. Each valve has three cusps that look like half moons. The SL valves are the pulmonic valve in the right side of the heart and the aortic valve in the left side of the heart. They open during pumping, or systole, to allow blood to be ejected from the heart.

Note: There are no valves between the vena cava and the right atrium nor between the pulmonary veins and the left atrium. For this reason, abnormally high pressure in the left side of the heart gives a person symptoms of pulmonary congestion, and abnormally high pressure in the right side of the heart shows in the neck veins and abdomen.

**DIRECTION OF BLOOD FLOW**

Think of an unoxygenated red blood cell being drained downstream into the vena cava. It is swept along with the flow of venous blood and follows the route illustrated in Fig. 19-5.

1. From liver to right atrium (RA) through inferior vena cava
   Superior vena cava drains venous blood from the head and upper extremities
   From RA, venous blood travels through tricuspid valve to right ventricle (RV)
2. From RV, venous blood flows through pulmonary valve to pulmonary artery
   Pulmonary artery delivers unoxygenated blood to lungs
3. Lungs oxygenate blood
   Pulmonary veins return fresh blood to left atrium (LA)
4. From LA, arterial blood travels through mitral valve to left ventricle (LV)
   LV ejects blood through aortic valve into aorta
5. Aorta delivers oxygenated blood to body

Remember that the circulation is a continuous loop. The blood is kept moving along by continually shifting pressure gradients. The blood flows from an area of higher pressure to one of lower pressure.

**CARDIAC CYCLE**

The rhythmic movement of blood through the heart is the cardiac cycle. It has two phases, diastole and systole. In diastole, the ventricles relax and fill with blood. This takes up two thirds of the cardiac cycle. The heart’s contraction is systole. During systole, blood is pumped from the ventricles and fills the pulmonary and systemic arteries. This is one third of the cardiac cycle.

**Diastole.** In diastole, the ventricles are relaxed and the AV valves (i.e., the tricuspid and mitral) are open (Fig. 19-6). (Opening of the normal valve is acoustically silent.) The pressure in the atria is higher than that in the ventricles, so blood pours rapidly into the ventricles. This first passive filling phase is called early or protodiastolic filling.

Toward the end of diastole, the atria contract and push the last amount of blood (about 25% of stroke volume) into the ventricles. This active filling phase is called presystole, or atrial systole, or sometimes the “atrial kick.” It causes a small rise in left ventricular pressure. (Note that atrial systole occurs during ventricular diastole, a confusing but important point.)

**Systole.** Now so much blood has been pumped into the ventricles that ventricular pressure is finally higher than that in the atria, so the mitral and tricuspid valves swing shut. The closure of the AV valves contributes to the first heart sound (S₁) and signals the beginning of systole. The AV valves close to prevent any regurgitation of blood back up into the atria during contraction.

For a very brief moment, all four valves are closed. The ventricular walls contract. This contraction against a closed system works to build pressure inside the ventricles to a high level (isometric contraction). Consider first the left side of the heart. When the pressure in the ventricle finally exceeds pressure in the aorta, the aortic valve opens and blood is ejected rapidly.
Pressure Changes in Left Heart

**Aortic pressure**

- Aortic valve opens
- Aortic valve closes

**Atrial pressure**

- AV valve opens
- AV valve closes

**Ventricular pressure**

**Heart Sounds**

- S1
- S2
- S3
- S4

**Electrocardiogram**

- P wave
- QRS complex
- ST segment
- T wave

THE CARDIAC CYCLE
Physical examination

After the ventricle’s contents are ejected, its pressure falls. When pressure falls below pressure in the aorta, some blood flows backward toward the ventricle, causing the aortic valve to swing shut. This closure of the semilunar valves causes the second heart sound ($S_2$) and signals the end of systole.

Diastole Again. Now all four valves are closed and the ventricles relax (called isometric or isovolumic relaxation). Meanwhile, the atria have been filling with blood delivered from the lungs. Atrial pressure is now higher than the relaxed ventricular pressure. The mitral valve drifts open, and diastolic filling begins again.

Events in the Right and Left Sides. The same events are happening at the same time in the right side of the heart, but pressures in the right side of the heart are much lower than those of the left side because less energy is needed to pump blood to its destination, the pulmonary circulation. Also, events occur just slightly later in the right side of the heart because of the route of myocardial depolarization. As a result, two distinct components to each of the heart sounds exist, and sometimes you can hear them separately. In the first heart sound, the mitral component ($M_1$) closes just before the tricuspid component ($T_1$). And with $S_2$, aortic closure ($A_2$) occurs slightly before pulmonic closure ($P_2$).

HEART SOUNDS

Events in the cardiac cycle generate sounds that can be heard through a stethoscope over the chest wall. These include normal heart sounds and, occasionally, extra heart sounds and murmurs (Fig. 19-7).

Normal Heart Sounds

The first heart sound ($S_1$) occurs with closure of the AV valves and thus signals the beginning of systole. The mitral component of the first sound ($M_1$) slightly precedes the tricuspid component ($T_1$), but you usually hear these two components fused as one sound. You can hear $S_1$ over all the precordium, but usually it is loudest at the apex.

The second heart sound ($S_2$) occurs with closure of the semilunar valves and signals the end of systole. The aortic component of the second sound ($A_2$) slightly precedes the pulmonic component ($P_2$). Although it is heard over all the precordium, $S_2$ is loudest at the base.

Effect of Respiration. The volume of right and left ventricular systole is about equal, but this can be affected by respiration. To learn this, consider the phrase:

More to the Right heart,
Less to the Left

That means that during inspiration, intrathoracic pressure is decreased. This pushes more blood into the vena cava, increasing venous return to the right side of the heart, which increases right ventricular stroke volume. The increased volume prolongs right ventricular systole and delays pulmonic valve closure.

Meanwhile, on the left side, a greater amount of blood is sequestered in the lungs during inspiration. This momentarily decreases the amount returned to the left side of the heart, decreasing left ventricular stroke volume. The decreased volume shortens left ventricular systole and allows the aortic valve to close a bit earlier. When the aortic valve closes significantly earlier than the pulmonic valve, you can hear the two components separately. This is a split $S_2$.

Extra Heart Sounds

Third Heart Sound ($S_3$). Normally, diastole is a silent event. However, in some conditions, ventricular filling creates vibrations that can be heard over the chest. These vibrations are $S_3$. The $S_3$ occurs when the ventricles are resistant to filling
during the early rapid filling phase (protodiastole). This occurs immediately after \( S_2 \), when the AV valves open and atrial blood first pours into the ventricles. (See a complete discussion of \( S_2 \) in Table 19-7 on pp. 490-491.)

**Fourth Heart Sound (\( S_4 \)).** The \( S_4 \) occurs at the end of diastole, at presystole, when the ventricle is resistant to filling. The atria contract and push blood into a noncompliant ventricle. This creates vibrations that are heard as \( S_4 \). The \( S_4 \) occurs just before \( S_1 \).

**Murmurs**

Blood circulating through normal cardiac chambers and valves usually makes no noise. However, some conditions create turbulent blood flow and collision currents. These result in a murmur, much like a pile of stones or a sharp turn in a stream creates a noisy water flow. A murmur is a gentle, blowing, swooshing sound that can be heard on the chest wall. Conditions resulting in a murmur are as follows:

1. Velocity of blood increases (flow murmur) (e.g., in exercise, thyrotoxicosis)
2. Viscosity of blood decreases (e.g., in anemia)
3. Structural defects in the valves (narrowed valve, incompetent valve) or unusual openings occur in the chambers (dilated chamber, wall defect)

**Characteristics of Sound**

All heart sounds are described by:

1. Frequency (pitch)—heart sounds are described as high pitched or low pitched, although these terms are relative because all are low-frequency sounds, and you need a good stethoscope to hear them
2. Intensity (loudness)—loud or soft
3. Duration—very short for heart sounds; silent periods are longer
4. Timing—systole or diastole

**CONDUCTION**

Of all organs, the heart has a unique ability—automaticity. The heart can contract by itself, independent of any signals or stimulation from the body. The heart contracts in response to an electrical current conveyed by a conduction system (Fig. 19-8). Specialized cells in the sinoatrial (SA) node near the superior vena cava initiate an electrical impulse. (Because the SA node has an intrinsic rhythm, it is the “pacemaker.”) The current flows in an orderly sequence, first across the atria to the AV node low in the atrial septum. There it is delayed slightly so that the atria have time to contract before the ventricles are stimulated. Then the impulse travels to the bundle of His, the right and left bundle branches, and then through the ventricles.

The electrical impulse stimulates the heart to do its work, which is to contract. A small amount of electricity spreads to the body surface, where it can be measured and recorded on the electrocardiograph (ECG). The ECG waves are arbitrarily labeled \( PQRST \), which stand for the following elements:

- **P wave**—depolarization of the atria
- **PR interval**—from the beginning of the \( P \) wave to the beginning of the QRS complex (the time necessary for atrial depolarization plus time for the impulse to travel through the AV node to the ventricles)
- **QRS complex**—depolarization of the ventricles
- **T wave**—repolarization of the ventricles
Electrical events slightly precede the mechanical events in the heart. The ECG juxtaposed on the cardiac cycle is illustrated in Figure 19-6.

**PUMPING ABILITY**

In the resting adult, the heart normally pumps between 4 and 6 L of blood per minute throughout the body. This cardiac output equals the volume of blood in each systole (called the stroke volume) times the number of beats per minute (rate). This is described as:

\[
\text{CO} = \text{SV} \times \text{R}
\]

The heart can alter its cardiac output to adapt to the metabolic needs of the body. Preload and afterload affect the heart’s ability to increase cardiac output.

**Preload** is the venous return that builds during diastole. It is the length to which the ventricular muscle is stretched at the end of diastole just before contraction (Fig. 19-9).

When the volume of blood returned to the ventricles is increased (as when exercise stimulates skeletal muscles to contract and force more blood back to the heart), the muscle bundles are stretched beyond their normal resting state to accommodate. The force of this switch is the preload. According to the Frank-Starling law, the greater the stretch, the stronger is the heart’s contraction. This increased contractility results in an increased volume of blood ejected (increased stroke volume).

**Afterload** is the opposing pressure the ventricle must generate to open the aortic valve against the higher aortic pressure. It is the resistance against which the ventricle must pump its blood. Once the ventricle is filled with blood, the ventricular end diastolic pressure is 5 to 10 mm Hg, whereas that in the aorta is 70 to 80 mm Hg. To overcome this difference, the ventricular muscle tenses (isovolumic contraction). After the aortic valve opens, rapid ejection occurs.

**THE NECK VESSELS**

Cardiovascular assessment includes the survey of vascular structures in the neck—the carotid artery and the jugular veins (Fig. 19-10). These vessels reflect the efficiency of cardiac function.

**The Carotid Artery Pulse**

Chapter 9 describes the pulse as a pressure wave generated by each systole pumping blood into the aorta. The carotid artery is a central artery—that is, it is close to the heart. Its timing closely coincides with ventricular systole. (Assessment of the peripheral pulses is found in Chapter 20, and blood pressure assessment is found in Chapter 9.)

The carotid artery is located in the groove between the trachea and the sternomastoid muscle, medial to and alongside that muscle. Note the characteristics of its waveform (Fig. 19-11): a smooth rapid upstroke, a summit that is rounded and smooth, and a downstroke that is more gradual and that has a dicrotic notch caused by closure of the aortic valve (marked D in the figure).

**Jugular Venous Pulse and Pressure**

The jugular veins empty unoxygenated blood directly into the superior vena cava. Because no cardiac valve exists to separate the superior vena cava from the right atrium, the jugular veins give information about activity on the right side of the heart. Specifically, they reflect filling pressure and volume changes. Because volume and pressure increase when the right side of the heart fails to pump efficiently, the jugular veins expose this.

Two jugular veins are present in each side of the neck (see Fig. 19-10). The larger internal jugular lies deep and medial to the sternomastoid muscle. It is usually not visible, although its diffuse pulsations may be seen in the sternal notch when the person is supine. The external jugular vein is more superficial; it lies lateral to the sternomastoid muscle, above the clavicle.

Although an arterial pulse is caused by a forward propulsion of blood, the jugular pulse is different. The jugular pulse results from a backwash, a waveform moving backward caused by events upstream. The jugular pulse has five components, as shown in Fig 19-12.
The five components of the jugular venous pulse occur because of events in the right side of the heart. The A wave reflects atrial contraction because some blood flows backward to the vena cava during right atrial contraction. The C wave, or ventricular contraction, is backflow from the bulging upward of the tricuspid valve when it closes at the beginning of ventricular systole (not from the neighboring carotid artery pulsation). Next, the X descent shows atrial relaxation when the right ventricle contracts during systole and pulls the bottom of the atria downward. The V wave occurs with passive atrial filling because of the increasing volume in the right atria and increased pressure. Finally, the Y descent reflects passive ventricular filling when the tricuspid valve opens and blood flows from the RA to the RV.
Infants and Children

The fetal heart functions early; it begins to beat at the end of 3 weeks' gestation. The lungs are nonfunctional, but the fetal circulation compensates for this (Fig. 19-13). Oxygenation takes place at the placenta, and the arterial blood is returned to the right side of the fetal heart. There is no point in pumping all this freshly oxygenated blood through the lungs, so it is rerouted in two ways. First, about two thirds of it is shunted through an opening in the atrial septum, the foramen ovale, into the left side of the heart, where it is pumped out through the aorta. Second, the rest of the oxygenated blood is pumped by the right side of the heart out through the pulmonary artery, but it is detoured through the ductus arteriosus to the aorta. Because they are both pumping into the systemic circulation, the right and left ventricles are equal in weight and muscle wall thickness.

Inflation and aeration of the lungs at birth produces circulatory changes. Now the blood is oxygenated through the lungs rather than through the placenta. The foramen ovale closes within the first hour because of the new lower pressure in the right side of the heart than in the left side. The ductus arteriosus closes later, usually within 10 to 15 hours of birth. Now the left ventricle has the greater workload of pumping into the systemic circulation, so that when the baby has reached 1 year of age, the left ventricle's mass increases to reach the adult ratio of 2:1, left ventricle to right ventricle.

The heart's position in the chest is more horizontal in the infant than in the adult; thus the apex is higher, located at the fourth left intercostal space (Fig. 19-14). It reaches the adult position when the child reaches age 7 years.

The Pregnant Woman

Blood volume increases by 30% to 40% during pregnancy, with the most rapid expansion occurring during the second
trimester. This creates an increase in stroke volume and cardiac output and an increased pulse rate of 10 to 15 beats per minute. Despite the increased cardiac output, arterial blood pressure decreases in pregnancy as a result of peripheral vasodilation. The blood pressure drops to its lowest point during the second trimester and then rises after that. The blood pressure varies with the person’s position, as described on p. 509.

The Aging Adult

It is difficult to isolate the “aging process” of the cardiovascular system per se because it is so closely interrelated with lifestyle, habits, and diseases. We now know that lifestyle is a modifying factor in the development of cardiovascular disease; smoking, diet, alcohol use, exercise patterns, and stress have an influence on coronary artery disease. Lifestyle also affects the aging process; cardiac changes once thought to be due to aging are partially due to the sedentary lifestyle accompanying aging (Fig. 19-15). What is left to be attributed to the aging process alone?

Hemodynamic Changes with Aging

- With aging, there is an increase in systolic blood pressure (BP). This is due to stiffening of the large arteries, which in turn is due to calcification of vessel walls (atherosclerosis). This stiffening creates an increase in pulse wave velocity because the less compliant arteries cannot store the volume ejected.
- The overall size of the heart does not increase with age, but left ventricular wall thickness increases. This is an adaptive mechanism to accommodate the vascular stiffening mentioned earlier that creates an increased workload on the heart.
- No significant change in diastolic pressure occurs with age. A rising systolic pressure with a relatively constant diastolic pressure increases the pulse pressure (the difference between the two).
- No change in resting heart rate occurs with aging.
- Cardiac output at rest is not changed with aging.
- There is a decreased ability of the heart to augment cardiac output with exercise. This is shown by a decreased maximum heart rate with exercise and diminished sympathetic response. Noncardiac factors also cause a decrease in maximum work performance with aging: decrease in skeletal muscle performance, increase in muscle fatigue, increased sense of dyspnea. Chronic exercise conditioning will modify many of the aging changes in cardiovascular function.
Dysrhythmias. The presence of supraventricular and ventricular dysrhythmias increases with age. Ectopic beats are common in aging people; although these are usually asymptomatic in healthy older people, they may compromise cardiac output and blood pressure when disease is present. Tachydyssrhythmias may not be tolerated as well in older people. The myocardium is thicker and less compliant, and early diastolic filling is impaired at rest. Thus it may not tolerate a tachycardia as well because of shortened diastole. Also, tachydyssrhythmias may further compromise a vital organ whose function has already been affected by aging or disease. For example, a ventricular tachycardia produces a 40% to 70% decrease in cerebral blood flow. Although a younger person may tolerate this, an older person with cerebrovascular disease may experience syncope.

ECG. Age-related changes in the ECG occur as a result of histologic changes in the conduction system. These changes include:

- Prolonged P-R interval (first-degree AV block) and prolonged Q-T interval, but the QRS interval is unchanged.
- Left axis deviation from age-related mild LV hypertrophy and fibrosis in left bundle branch.
- Increased incidence of bundle branch block.

Although the hemodynamic changes associated with aging alone do not seem severe or portentous, the fact remains that the incidence of cardiovascular disease increases with age. The incidence of coronary artery disease increases sharply with advancing age and accounts for about half of the deaths of older people. Hypertension (systolic >140 mm Hg and/or diastolic >90 mm Hg) and heart failure also increase with age. Certainly, lifestyle habits (smoking, chronic alcohol use, lack of exercise, diet) play a significant role in the acquisition of heart disease. Also, increasing the physical activity of older adults—even at a moderate level—is associated with a reduced risk of death from cardiovascular diseases and respiratory illnesses. Both points underscore the need for health teaching as an important treatment parameter.

CULTURE AND GENETICS

Prevalence is an estimate of how many people in a stated geographic location have a disease at a given point in time. In the United States, an estimated 81 million people (more than 1 in 3) have one or more forms of cardiovascular heart disease (CVD). The annual rates of first CVD event increase with age. For women, comparable rates occur 10 years later in life than for men, but this gap narrows with advancing age.

Causes of CVD include an interaction of genetic, environmental, and lifestyle factors. However, evidence shows potentially modifiable risk factors contribute to the overwhelming majority of cardiac risk. For example, myocardial infarction (MI) is an important type of CVD. The INTERHEART study covering 52 countries indicated that nine potentially modifiable risk factors accounted for 90% of the population attributable risk for MI in men and 94% in women. These nine modifiable risk factors include abnormal lipids, smoking, hypertension, diabetes, abdominal obesity, psychosocial factors, consumption of fruits and vegetables, alcohol use, and regular physical activity.

High Blood Pressure (HBP). Although all adults have some potential CVD risk, some groups (defined by race, ethnicity, gender, socioeconomic status, educational level) carry an excess burden of CVD. Hypertension is a systolic blood pressure (SBP) of ≥140 mm Hg or diastolic blood pressure (DBP) of ≥90 mm Hg or taking antihypertensive medicine. A higher percentage of men than women have hypertension until age 45 years. From age 45 to 64 years, the percentages are similar; after age 64 years, women have a much higher percentage of hypertension than men have. Also, hypertension is 2 to 3 times more common among women taking oral contraceptives (especially among obese and older women) than in women who do not take them. Among racial groups, the prevalence of hypertension in blacks is among the highest in the world and it is rising. The prevalence of hypertension is 31.8% for African Americans, then 25.3% for American Indians or Alaska natives, 23.3% for whites, and 21% for Hispanics and Asians. Compared with whites, African Americans develop HBP earlier in life and their average BPs are much higher. This results in African Americans having a greater rate of stroke, death due to heart disease, and end-stage kidney disease.

Smoking. In the 40+ years from 1965 to 2004, U.S. smoking rates declined by 50.4% among adults 18 years of age and older. This results in 2008 with 23.1% of men and 18.3% of women being smokers. Nicotine increases the risk of MI and stroke by causing the following: increase in oxygen demand with a concomitant decrease in oxygen supply; an activation of platelets, activation of fibrinogen; and an adverse change in the lipid profile.

Serum Cholesterol. High levels of low-density lipoprotein gradually add to the lipid core of thrombus formation in arteries, which results in MI and stroke. The current cutpoints for cholesterol risk in adults are the following: total cholesterol levels of ≥240 mg/dL are high risk; and levels from 200 to 239 mg/dL are borderline–high risk. The age-adjusted prevalence of total cholesterol levels over 200 mg/dL areas follows: 51.1% of Mexican-American men and 49% of Mexican-American women; 45% of white men and 48.7% of white women; and 40.2% of African American men and 41.8% of African American women.

Obesity. The epidemic of obesity in the United States is well known and is referenced in many chapters of this text. Among Americans ages 20 years and older, the prevalence of overweight or obesity (body mass index [BMI] of ≥25 kg/m² for overweight and ≥30.0 for obesity) is as follows: 74.8% of Mexican-American men and 73% of Mexican-American women; 73.7% of African American men and 77.7% of African American women; and 72.4% of white men and 57.5% of white women.

Type 2 Diabetes Mellitus. The risk of CVD is twofold greater among persons with diabetes mellitus (DM) than without DM. The increased prevalence of DM in the United States is being followed by an increasing prevalence of CVD morbidity and mortality. Diabetes causes damage to the large blood vessels that nourish the brain, heart, and extremi-
ties; this results in stroke, coronary artery disease, and peripheral vascular disease.

About 13% of African Americans 20 years of age and older have DM. Between 11.8% and 13.1% of Mexican-Americans have DM, compared with 6.4% of whites. The most powerful predictor of type 2 DM is obesity, with abdominal (visceral) fat posing a greater risk than lower body obesity poses. Evidence from epidemiologic studies shows a strong genetic factor for DM, but no specific antigen type has yet been identified. In the past, type 2 DM was diagnosed in adults 40 years of age and older, but now we are finding more children with type 2 DM. These children are usually overweight or obese, have a family history of DM, and identify with American Indian, African American, Hispanic, or Asian groups.3

### Subjective Data

<table>
<thead>
<tr>
<th>Examiner Asks</th>
<th>Rationale</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Chest pain</strong></td>
<td>Any chest pain or tightness?</td>
</tr>
<tr>
<td></td>
<td>• Onset: When did it start? How long have you had it this time? Had this</td>
</tr>
<tr>
<td></td>
<td>type of pain before? How often?</td>
</tr>
<tr>
<td></td>
<td>• Location: Where did the pain start? Does the pain radiate to any other</td>
</tr>
<tr>
<td></td>
<td>spot?</td>
</tr>
<tr>
<td></td>
<td>• Character: How would you describe it? Crushing, stabbing, burning,</td>
</tr>
<tr>
<td></td>
<td>viselike? (Allow the person to offer adjectives before you suggest</td>
</tr>
<tr>
<td></td>
<td>them.) (Note if uses clenched fist to describe pain.)</td>
</tr>
<tr>
<td></td>
<td>• Pain brought on by: Activity—what type; rest; emotional upset; after</td>
</tr>
<tr>
<td></td>
<td>eating; during sexual intercourse; with cold weather?</td>
</tr>
<tr>
<td></td>
<td>• Any associated symptoms: Sweating, ashen gray or pale skin, heart skips</td>
</tr>
<tr>
<td></td>
<td>beat, shortness of breath, nausea or vomiting, racing of heart?</td>
</tr>
<tr>
<td></td>
<td>• Pain made worse by moving the arms or neck, breathing, lying flat?</td>
</tr>
<tr>
<td></td>
<td>• Pain relieved by rest or nitroglycerin? How many tablets?</td>
</tr>
<tr>
<td><strong>Dyspnea</strong></td>
<td>Any shortness of breath?</td>
</tr>
<tr>
<td></td>
<td>• What type of activity and how much brings on shortness of breath? How</td>
</tr>
<tr>
<td></td>
<td>much activity brought it on 6 months ago?</td>
</tr>
<tr>
<td></td>
<td>• Onset: Does the shortness of breath come on unexpectedly?</td>
</tr>
<tr>
<td></td>
<td>• Duration: Constant or does it come and go?</td>
</tr>
<tr>
<td></td>
<td>• Seem to be affected by position: Lying down?</td>
</tr>
<tr>
<td></td>
<td>• Awaken you from sleep at night?</td>
</tr>
<tr>
<td><strong>Orthopnea</strong></td>
<td>How many pillows do you use when sleeping or lying down?</td>
</tr>
<tr>
<td></td>
<td>• Does the shortness of breath interfere with activities of daily living?</td>
</tr>
</tbody>
</table>

Dyspnea on exertion (DOE)—quantify exactly (e.g., DOE after walking two level blocks).

Paroxysmal.

Constant or intermittent.

Recumbent.

Paroxysmal nocturnal dyspnea (PND) occurs with heart failure. Lying down increases volume of intrathoracic blood, and the weakened heart cannot accommodate the increased load. Classically, the person awakens after 2 hours of sleep with the perception of needing fresh air.

Orthopnea is the need to assume a more upright position to breathe. Note the exact number of pillows used.

Diaphoresis, cold sweats, pallor, grayness.

Palpitations, dyspnea, nausea, tachycardia, fatigue.

Try to differentiate pain of cardiac versus noncardiac origin.
<table>
<thead>
<tr>
<th>Examiner Asks</th>
<th>Rationale</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>4. Cough.</strong> Do you have a <strong>cough?</strong></td>
<td>Sputum production, mucoid or purulent. Hemoptyis is often a pulmonary disorder but also occurs with mitral stenosis.</td>
</tr>
<tr>
<td>• Duration: How long have you had it?</td>
<td></td>
</tr>
<tr>
<td>• Frequency: Is it related to time of day?</td>
<td></td>
</tr>
<tr>
<td>• Type: Dry, hacking, barky, hoarse, or congested?</td>
<td></td>
</tr>
<tr>
<td>• Do you cough up mucus? Color? Any odor? Blood tinged?</td>
<td></td>
</tr>
<tr>
<td>• Associated with: Activity, position (lying down), anxiety, talking?</td>
<td></td>
</tr>
<tr>
<td>• Does activity make it better or worse (sit, walk, exercise)?</td>
<td></td>
</tr>
<tr>
<td>• Relieved by rest or medication?</td>
<td></td>
</tr>
<tr>
<td><strong>5. Fatigue.</strong> Do you seem to tire easily? Able to keep up with your family and co-workers?</td>
<td>Fatigue from decreased cardiac output is worse in the evening, whereas fatigue from anxiety or depression occurs all day or is worse in the morning.</td>
</tr>
<tr>
<td>• Onset: When did fatigue start? Sudden or gradual? Has any recent change occurred in energy level?</td>
<td>Cyanosis or pallor occurs with myocardial infarction or low cardiac output states as a result of decreased tissue perfusion.</td>
</tr>
<tr>
<td>• Fatigue related to time of day: All day, morning, evening?</td>
<td>Edema is dependent when caused by heart failure.</td>
</tr>
<tr>
<td><strong>6. Cyanosis or pallor.</strong> Ever noted your facial skin turn blue or ashen?</td>
<td>Cardiac edema is worse at evening and better in morning after elevating legs all night.</td>
</tr>
<tr>
<td><strong>7. Edema.</strong> Any swelling of your feet and legs?</td>
<td>Cardiac edema is bilateral; unilateral swelling has a local vein cause.</td>
</tr>
<tr>
<td>• Onset: When did you first notice this? Any recent change?</td>
<td>Nocturia—Recumbency at night promotes fluid reabsorption and excretion; this occurs with heart failure in the person who is ambulatory during the day.</td>
</tr>
<tr>
<td>• What time of day does the swelling occur? Do your shoes feel tight at the end of day?</td>
<td></td>
</tr>
<tr>
<td>• How much swelling would you say there is? Are both legs equally swollen?</td>
<td></td>
</tr>
<tr>
<td>• Does the swelling go away with: Rest, elevation, after a night’s sleep?</td>
<td></td>
</tr>
<tr>
<td>• Any associated symptoms, such as shortness of breath? If so, does the shortness of breath occur before leg swelling or after?</td>
<td></td>
</tr>
<tr>
<td><strong>8. Nocturia.</strong> Do you awaken at night with an urgent need to urinate? How long has this been occurring? Any recent change?</td>
<td></td>
</tr>
<tr>
<td><strong>9. Cardiac history.</strong> Any past history of: Hypertension, elevated cholesterol or triglycerides, heart murmur, congenital heart disease, rheumatic fever or unexplained joint pains as child or youth, recurrent tonsillitis, anemia?</td>
<td></td>
</tr>
<tr>
<td>• Ever had heart disease? When was this? Treated by medication or heart surgery?</td>
<td></td>
</tr>
<tr>
<td>• Last ECG, stress ECG, serum cholesterol measurement, other heart tests?</td>
<td></td>
</tr>
<tr>
<td><strong>10. Family cardiac history.</strong> Any family history of: Hypertension, obesity, diabetes, coronary artery disease (CAD), sudden death at younger age?</td>
<td></td>
</tr>
<tr>
<td><strong>11. Personal habits (cardiac risk factors).</strong></td>
<td></td>
</tr>
</tbody>
</table>
| • Nutrition: Please describe your usual daily diet. (Note if this diet is representative of the basic food groups, the amount of calories, cholesterol,
### Examiner Asks

and any additives such as salt.) What is your usual weight? Has there been any recent change?

- **Smoking:** Do you smoke cigarettes or other tobacco? At what age did you start? How many packs per day? For how many years have you smoked this amount? Have you ever tried to quit? If so, how did this go?

- **Alcohol:** How much alcohol do you usually drink each week, or each day? When was your last drink? What was the number of drinks that episode? Have you ever been told you had a drinking problem?

- **Exercise:** What is your usual amount of exercise each day or week? What type of exercise (state type or sport)? If a sport, what is your usual amount (light, moderate, heavy)?

- **Drugs:** Do you take any antihypertensives, beta-blockers, calcium channel blockers, digoxin, diuretics, aspirin/anticoagulants, over-the-counter or street drugs?

### Rationale

**Risk factors for CAD**—Collect data regarding elevated cholesterol, elevated blood pressure, blood sugar levels above 130 mg/dL or known diabetes mellitus, obesity, cigarette smoking, low activity level, and length of any hormone replacement therapy for postmenopausal women.

### Additional History for Infants

1. **How was the mother’s health during pregnancy:** Any unexplained fever, rubella first trimester, other infection, hypertension, drugs taken?

2. **Have you noted any cyanosis while nursing, crying?** Is the baby able to eat, nurse, or finish bottle without tiring?

3. **Growth:** Has this baby grown as expected by growth charts and about the same as siblings or peers?

4. **Activity:** Were this baby’s motor milestones achieved as expected? Is the baby able to play without tiring? How many naps does the baby take each day? How long does a nap last?

### Additional History for Children

1. **Growth:** Has this child grown as expected by growth charts?

2. **Activity:** Is this child able to keep up with siblings or age mates? Is the child willing or reluctant to go out to play? Is the child able to climb stairs, ride a bike, walk a few blocks? Does the child squat to rest during play or to watch television, or assume a knee-chest position while sleeping? Have you noted “blue spells” during exercise?

3. **Has the child had any unexplained joint pains or unexplained fever?**

4. **Does the child have frequent headaches, nosebleeds?**

5. **Does the child have frequent respiratory infections? How many per year? How are they treated?** Have any of these proved to be streptococcal infections?

6. **Family history:** Does the child have a sibling with heart defect? Is anyone in the child’s family known to have chromosomal abnormalities, such as Down syndrome?
**Additional History for the Pregnant Woman**

1. Have you had any high blood pressure during this or earlier pregnancies?
   - What was your usual blood pressure level before pregnancy? How has your blood pressure been monitored during the pregnancy?
   - If high blood pressure, what treatment has been started?
   - Any associated symptoms: Weight gain, protein in urine, swelling in feet, legs, or face?

2. Have you had any faintness or dizziness with this pregnancy?

**Additional History for the Aging Adult**

1. Do you have any known heart or lung disease: Hypertension, CAD, chronic emphysema, or bronchitis?
   - What efforts to treat this have been started?
   - Usual symptoms changed recently? Does your illness interfere with activities of daily living?

2. Do you take any medications for your illness such as digitalis? Aware of side effects? Have you recently stopped taking your medication? Why?

3. Environment: Does your home have any stairs? How often do you need to climb them? Does this have any effect on activities of daily living?

**OBJECTIVE DATA**

**PREPARATION**

To evaluate the carotid arteries, the person can be sitting up. To assess the jugular veins and the precordium, the person should be supine with the head and chest slightly elevated.

Stand on the person’s right side; this will facilitate your hand placement, viewing of the neck veins, and auscultation of the precordium.

The room must be warm—chilling makes the person uncomfortable, and shivering interferes with heart sounds. Take scrupulous care to ensure quiet; heart sounds are very soft, and any ambient room noise masks them.

Ensure the female’s privacy by keeping her breasts draped. The female’s left breast overrides part of the area you will need to examine. Gently displace the breast upward, or ask the woman to hold it out of the way.

When performing a regional cardiovascular assessment, use this order:
1. Pulse and blood pressure (see Chapter 9)
2. Extremities (see Peripheral Vascular Assessment in Chapter 20)
3. Neck vessels
4. Precordium

The logic of this order is that you will begin observations peripherally and move in toward the heart. For choreography of these steps in the complete physical examination, see Chapter 27.

**EQUIPMENT NEEDED**

- Marking pen
- Small centimeter ruler
- Stethoscope with diaphragm and bell endpieces
- Alcohol wipe (to clean endpiece)

<table>
<thead>
<tr>
<th>Normal Range of Findings</th>
<th>Abnormal Findings</th>
</tr>
</thead>
</table>

**THE NECK VESSELS**

**Palpate the Carotid Artery**

Located central to the heart, the carotid artery yields important information on cardiac function.
Normal Range of Findings

Palpate each carotid artery medial to the sternomastoid muscle in the neck (Fig. 19-16). Avoid excessive pressure on the carotid sinus area higher in the neck; excessive vagal stimulation here could slow down the heart rate, especially in older adults. Take care to palpate gently. Palpate only one carotid artery at a time to avoid compromising arterial blood to the brain.

Feel the contour and amplitude of the pulse. Normally the contour is smooth with a rapid upstroke and slower downstroke, and the normal strength is 2+ or moderate (see Chapter 20). Your findings should be the same bilaterally.

Auscultate the Carotid Artery

For persons middle-aged or older or who show symptoms or signs of cardiovascular disease, auscultate each carotid artery for the presence of a bruit (pronounced bru’-ee) (Fig. 19-17). This is a blowing, swishing sound indicating blood flow turbulence; normally none is present.

Keep the neck in a neutral position. Lightly apply the bell of the stethoscope over the carotid artery at three levels: (1) the angle of the jaw, (2) the midcervical area, and (3) the base of the neck (see Fig. 19-17). Avoid compressing the artery because this could create an artificial bruit, and it could compromise circulation if the carotid artery is already narrowed by atherosclerosis. Ask the person to take a breath, exhale, and hold it briefly while you listen so that tracheal breath sounds do not mask or mimic a carotid artery bruit. (Holding the breath on inhalation will also tense the levator scapulae muscles, which makes it hard to hear the carotids.) Sometimes you can hear normal heart sounds transmitted to the neck; do not confuse these with a bruit.

Abnormal Findings

Carotid sinus hypersensitivity is the condition in which pressure over the carotid sinus leads to a decreased heart rate, decreased BP, and cerebral ischemia with syncope. This may occur in older adults with hypertension or occlusion of the carotid artery.

Diminished pulse feels small and weak (decreased stroke volume).

Increased pulse feels full and strong in hyperkinetic states (see Table 20-1, Variations in Arterial Pulse, on p. 549).

A bruit indicates turbulence due to a local vascular cause, such as atherosclerotic narrowing.

A carotid bruit is audible when the lumen is occluded by $\frac{1}{3}$ to $\frac{1}{2}$. Bruit loudness increases as the atherosclerosis worsens until the lumen is occluded by $\frac{2}{3}$. After that, bruit loudness decreases. When the lumen is completely occluded, the bruit disappears. Thus absence of a bruit does not ensure absence of a carotid lesion.
**Normal Range of Findings**

<table>
<thead>
<tr>
<th>Abnormal Findings</th>
</tr>
</thead>
<tbody>
<tr>
<td>A murmur sounds much the same but is caused by a cardiac disorder. Some aortic valve murmurs (aortic stenosis) radiate to the neck and must be distinguished from a local bruit.</td>
</tr>
</tbody>
</table>

**Inspect the Jugular Venous Pulse**

From the jugular veins you can assess the central venous pressure (CVP) and thus judge the heart’s efficiency as a pump. Stand on the person’s right side because the veins there have a direct route to the heart. Traditionally we have been taught to use the internal jugular vein pulsations for CVP assessment. However, you may use either the external or the internal jugular veins because measurements in both are similar.\(^{27}\) You can see the top of the external jugular vein distention overlying the sternomastoid muscle or the pulsation of the internal jugular vein in the sternal notch.

Position the person supine anywhere from a 30- to a 45-degree angle, wherever you can best see the top of the vein or pulsations. In general, the higher the venous pressure is, the higher the position you need. Remove the pillow to avoid flexing the neck; the head should be in the same plane as the trunk. Turn the person’s head slightly away from the examined side, and direct a strong light tangentially onto the neck to highlight pulsations and shadows.

Note the external jugular veins overlying the sternomastoid muscle. In some persons, the veins are not visible at all, whereas in others they are full in the supine position. As the person is raised to a sitting position, these external jugulars flatten and disappear, usually at 45 degrees.

Now look for pulsations of the internal jugular veins in the area of the suprasternal notch or around the origin of the sternomastoid muscle around the clavicle. You must be able to distinguish internal jugular vein pulsation from that of the carotid artery. It is easy to confuse them because they lie close together. Use the guidelines shown in Table 19-1.

**TABLE 19-1** Characteristics of Jugular Versus Carotid Pulsations

<table>
<thead>
<tr>
<th>Characteristic</th>
<th>Internal Jugular Pulse</th>
<th>Carotid Pulse</th>
</tr>
</thead>
<tbody>
<tr>
<td>1. Location</td>
<td>Lower, more lateral, under or behind the sternomastoid muscle</td>
<td>Higher and medial to this muscle</td>
</tr>
<tr>
<td>2. Quality</td>
<td>Undulant and diffuse, two visible waves per cycle</td>
<td>Brisk and localized, one wave per cycle</td>
</tr>
<tr>
<td>3. Respiration</td>
<td>Varies with respiration; its level descends during inspiration when intrathoracic pressure is decreased</td>
<td>Does not vary</td>
</tr>
<tr>
<td>4. Palpable</td>
<td>No</td>
<td>Yes</td>
</tr>
<tr>
<td>5. Pressure</td>
<td>Light pressure at the base of the neck easily obliterates</td>
<td>No change</td>
</tr>
<tr>
<td>6. Position of person</td>
<td>Level of pulse drops and disappears as the person is brought to a sitting position</td>
<td>Unaffected</td>
</tr>
</tbody>
</table>

Unilateral distention of external jugular veins is due to local cause (kinking or aneurysm).

Full distended external jugular veins above 45 degrees signify increased CVP as with heart failure.
Chapter 19   Heart and Neck Vessels

Normal Range of Findings

Estimate the Jugular Venous Pressure

Think of the jugular veins as a CVP manometer attached directly to the right atrium. You can “read” the CVP at the highest level of pulsations (Fig. 19-18). Use the angle of Louis (sternal angle) as an arbitrary reference point, and compare it with the highest level of the distended vein or venous pulsation.

Hold a vertical ruler on the sternal angle. Align a straight edge on the ruler like a T-square, and adjust the level of the horizontal straight edge to the level of pulsation. Read the level of intersection on the vertical ruler; normal jugular venous pulsation is 2 cm or less above the sternal angle. Also state the person’s position, for example, “internal jugular vein pulsations 3 cm above sternal angle when elevated 30 degrees.”

If you cannot find the internal jugular veins, use the external jugular veins and note the point where they look collapsed. Be aware that the technique of estimating venous pressure is difficult and is not always a reliable predictor of CVP. Consistency in grading among examiners is difficult to achieve.

If venous pressure is elevated or if you suspect heart failure, perform hepatojugular reflux (Fig. 19-19). Position the person comfortably supine, and instruct him or her to breathe quietly through an open mouth. Hold your right hand on the right upper quadrant of the person’s abdomen just below the rib cage. Watch the level of jugular pulsation as you push in with your hand. Exert firm sustained pressure for 30 seconds. This displaces venous blood out of the liver sinusoids and adds its volume to the venous system. If the heart is able to pump this additional volume (i.e., if no elevated CVP is present), the jugular veins will rise for a few seconds and then recede back to previous level.

If heart failure is present, the jugular veins will elevate and stay elevated as long as you push.

THE PRECordial

Inspect the Anterior Chest

Arrange tangential lighting to accentuate any flicker of movement.

Pulsations. You may or may not see the apical impulse, the pulsation created as the left ventricle rotates against the chest wall during systole. When visible, it occupies the fourth or fifth intercostal space, at or inside the midclavicular line. It is easier to see in children and in those with thinner chest walls.

A heave or lift is a sustained forceful thrusting of the ventricle during systole. It occurs with ventricular hypertrophy as a result of increased workload. A right ventricular heave is seen at the sternal border; a left ventricular heave is seen at the apex (see Table 19-8, Abnormal Pulsations on the Precordium).
## Normal Range of Findings
### Palpate the Apical Impulse

(This used to be called the point of maximal impulse, or PMI. Because some abnormal conditions may cause a maximal impulse to be felt elsewhere on the chest, use the term apical impulse specifically for the apex beat.)

Localize the apical impulse precisely by using one finger pad (Fig. 19-20, A). Asking the person to "exhale and then hold it" aids the examiner in locating the pulsation. You may need to roll the person midway to the left to find it; note that this also displaces the apical impulse farther to the left (Fig. 19-20, B).

### Abnormal Findings

- **Cardiac enlargement:**
  - Left ventricular dilation (volume overload) displaces impulse down and to left and increases size more than one space.
  - A **sustained** impulse with increased force and duration but no change in location occurs with left ventricular hypertrophy and no dilation (pressure overload) (see Table 19-8).

  Not palpable with pulmonary emphysema due to overriding lungs.

- A **thrill** is a palpable vibration. It feels like the throat of a purring cat. The thrill signifies turbulent blood flow and accompanies loud murmurs. Absence of a thrill, however, does not necessarily rule out the presence of a murmur.

  Accentuated first and second heart sounds and extra heart sounds also may cause abnormal pulsations.

<table>
<thead>
<tr>
<th>Normal Findings</th>
<th>Abnormal Findings</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Palpate the Apical Impulse</strong></td>
<td><strong>Cardiac enlargement:</strong></td>
</tr>
<tr>
<td><strong>Normal Findings</strong></td>
<td></td>
</tr>
<tr>
<td><strong>Abnormal Findings</strong></td>
<td></td>
</tr>
</tbody>
</table>

**Note:**
- **Location**—The apical impulse should occupy only one interspace, the fourth or fifth, and be at or medial to the midclavicular line
- **Size**—Normally 1 cm × 2 cm
- **Amplitude**—Normally a short, gentle tap
- **Duration**—Short, normally occupies only first half of systole

The apical impulse is palpable in about half of adults. It is not palpable in obese persons or in persons with thick chest walls. With high cardiac output states (anxiety, fever, hyperthyroidism, anemia), the apical impulse increases in amplitude and duration.

### Palpate Across the Precordium

Using the palmar aspects of your four fingers, gently palpate the apex, the left sternal border, and the base, searching for any other pulsations (Fig. 19-21). Normally none occur. If any are present, note the timing. Use the carotid artery pulsation as a guide, or auscultate as you palpate.
Normal Range of Findings

<table>
<thead>
<tr>
<th>Abnormal Findings</th>
</tr>
</thead>
<tbody>
<tr>
<td>Cardiac enlargement is due to increased ventricular volume or wall thickness; it occurs with hypertension, CAD, heart failure, and cardiomyopathy.</td>
</tr>
</tbody>
</table>

### Percussion

Percussion is used to outline the heart’s borders, but it has been displaced by the chest x-ray or echocardiogram. Evidence shows these are more accurate in detecting heart enlargement. When the right ventricle enlarges, it does so in the anteroposterior diameter, which is better seen on x-ray film. Numerous comparison studies show the percussed cardiac border correlates “only moderately” with the true cardiac border. Also, percussion is of limited usefulness with the female breast tissue or in an obese person or a person with a muscular chest wall.

### Auscultation

Identify the auscultatory areas where you will listen. These include the four traditional valve “areas” (Fig. 19-22). The valve areas are not over the actual anatomic locations of the valves but are the sites on the chest wall where sounds produced by the valves are best heard. The sound radiates with the direction of blood flow. The valve areas are:

- Second right interspace—aortic valve area
- Second left interspace—pulmonic valve area
- Left lower sternal border—tricuspid valve area
- Fifth interspace at around left midclavicular line—mitral valve area

Do not limit your auscultation to only four locations. Sounds produced by the valves may be heard all over the precordium. (For this reason, many experts even discourage the naming of the valve areas.) Thus learn to inch your stethoscope in a rough Z pattern, from the base of the heart across and down, then over to the apex. Or start at the apex and work your way up. Include the sites shown in Figure 19-22.
Normal Range of Findings

Recall the characteristics of a good stethoscope (see Chapter 8). Clean the endpieces with an alcohol wipe; you will use both endpieces. Although all heart sounds are low frequency, the diaphragm is for relatively higher pitched sounds and the bell is for relatively lower pitched ones.

Before you begin, alert the person: “I always listen to the heart in a number of places on the chest. Just because I am listening a long time, it does not necessarily mean that something is wrong.”

After you place the stethoscope, try closing your eyes briefly to tune out any distractions. Concentrate, and listen selectively to one sound at a time. Consider that at least two, and perhaps three or four, sounds may be happening in less than 1 second. You cannot process everything at once. Begin with the diaphragm endpiece and use the following routine: (1) note the rate and rhythm, (2) identify S1 and S2, (3) assess S1 and S2 separately, (4) listen for extra heart sounds, and (5) listen for murmurs.

Note the Rate and Rhythm. The rate ranges normally from 50 to 90 beats per minute. (Review the full discussion of the pulse in Chapter 9 and the normal rates across age-groups.) The rhythm should be regular, although sinus arrhythmia occurs normally in young adults and children. With sinus arrhythmia, the rhythm varies with the person’s breathing, increasing at the peak of inspiration and slowing with expiration. Note any other irregular rhythm. If one occurs, check if it has any pattern or if it is totally irregular.

When you notice any irregularity, check for a pulse deficit by auscultating the apical beat while simultaneously palpating the radial pulse. Count a serial measurement (one after the other) of apical beat and radial pulse. Normally, every beat you hear at the apex should perfuse to the periphery and be palpable. The two counts should be identical. When different, subtract the radial rate from the apical and record the remainder as the pulse deficit.

Identify S1 and S2. This is important because S1 is the start of systole and thus serves as the reference point for the timing of all other cardiac sounds. Usually, you can identify S1 instantly because you hear a pair of sounds close together (lub-dup), and S1 is the first of the pair. This guideline works, except in the cases of the tachydysrhythmias (rates >100 per minute). Then the diastolic filling time is shortened, and the beats are too close together to distinguish. Other guidelines to distinguish S1 from S2 are:

- S1 is louder than S2 at the apex; S2 is louder than S1 at the base.
- S1 coincides with the carotid artery pulse. Feel the carotid gently as you auscultate at the apex; the sound you hear as you feel each pulse is S1 (Fig. 19-23).
- S1 coincides with the R wave (the upstroke of the QRS complex) if the person is on an ECG monitor.

Abnormal Findings

Premature beat—an isolated beat is early, or a pattern occurs in which every third or fourth beat sounds early.

Irregularly irregular—no pattern to the sounds; beats come rapidly and at random intervals.

A pulse deficit signals a weak contraction of the ventricles; it occurs with atrial fibrillation, premature beats, and heart failure.
Normal Range of Findings

**Listen to \( S_1 \) and \( S_2 \) Separately.** Note whether each heart sound is normal, accentuated, diminished, or split. Inch your diaphragm across the chest as you do this.

**First Heart Sound (\( S_1 \)).** Caused by closure of the AV valves, \( S_1 \) signals the beginning of systole. You can hear it over the entire precordium, although it is loudest at the apex (Fig. 19-24). (Sometimes the two sounds are equally loud at the apex, because \( S_1 \) is lower pitched than \( S_2 \).)

\[
\begin{align*}
S_1 & \quad \text{S2} \\
\text{LUB} & \quad \text{APLEX} \\
\text{dup} &
\end{align*}
\]

\[\text{19-24}\]

You can hear \( S_1 \) with the diaphragm with the person in any position and equally well in inspiration and expiration. A split \( S_1 \) is normal, but it occurs rarely. A split \( S_1 \) means you are hearing the mitral and tricuspid components separately. It is audible in the tricuspid valve area, the left lower sternal border. The split is very rapid, with the two components only 0.03 second apart.

**Second Heart Sound (\( S_2 \)).** The \( S_2 \) is associated with closure of the semilunar valves. You can hear it with the diaphragm, over the entire precordium, although \( S_2 \) is loudest at the base (Fig. 19-25).

\[
\begin{align*}
S_1 & \quad \text{S2} \\
\text{LUB} & \quad \text{BASE} \\
\text{DUP} &
\end{align*}
\]

\[\text{19-25}\]

**Splitting of \( S_2 \).** A split \( S_2 \) is a normal phenomenon that occurs toward the end of inspiration in some people. Recall that closure of the aortic and pulmonic valves is nearly synchronous. Because of the effects of respiration on the heart described earlier, inspiration separates the timing of the two valves’ closure, and the aortic valve closes 0.06 second before the pulmonic valve. Instead of one DUP, you hear a split sound—T-DUP (Fig. 19-26). During expiration, synchrony returns and the aortic and pulmonic components fuse together. A split \( S_2 \) is heard only in the pulmonic valve area, the second left interspace.

\[
\begin{align*}
\text{EXPIRATION} & \quad \text{INSPIRATION} \\
S_1 & \quad S_2 & \quad S_1 & \quad S_2 \\
\text{A2-P2} & \quad \text{A2 P2} \\
\text{DUP} & \quad \text{T-DUP} &
\end{align*}
\]

\[\text{19-26}\]
Normal Range of Findings

When you first hear the split $S_2$, do not be tempted to ask the person to hold his or her breath so that you can concentrate on the sounds. Breath holding will only equalize ejection times in the right and left sides of the heart and cause the split to go away. Instead, concentrate on the split as you watch the person’s chest rise up and down with breathing. The split $S_2$ occurs about every fourth heartbeat, fading in with inhalation and fading out with exhalation.

Focus on Systole, Then on Diastole, and Listen for any Extra Heart Sounds. Listen with the diaphragm, then switch to the bell, covering all auscultatory areas (Fig. 19-27). Usually these are silent periods. When you do detect an extra heart sound, listen carefully to note its timing and characteristics. During systole, the **midsystolic click** (which is associated with mitral valve prolapse) is the most common extra sound (see Table 19-6). The third and fourth heart sounds occur in diastole; either may be normal or abnormal (see Table 19-7).

**Listen for Murmurs.** A murmur is a blowing, swooshing sound that occurs with turbulent blood flow in the heart or great vessels. Except for the innocent murmurs described, murmurs are abnormal. If you hear a murmur, describe it by indicating these following characteristics:

**Timing.** It is crucial to define the murmur by its occurrence in systole or diastole. You must be able to identify $S_1$ and $S_2$ accurately to do this. Try to further describe the murmur as being in early, mid-, or late systole or diastole; throughout the cardiac event (terned pansystolic or holosystolic/pandiastolic or holodiastolic); and whether it obscures or muffles the heart sounds.

**Loudness.** Describe the intensity in terms of six “grades.” For example, record a grade ii murmur as “ii/ii.”

- **Grade i**— Barely audible, heard only in a quiet room and then with difficulty
- **Grade ii**— Clearly audible, but faint
- **Grade iii**— Moderately loud, easy to hear
- **Grade iv**— Loud, associated with a thrill palpable on the chest wall
- **Grade v**— Very loud, heard with one corner of the stethoscope lifted off the chest wall
- **Grade vi**— Loudest, still heard with entire stethoscope lifted just off the chest wall

Abnormal Findings

- **A fixed split** is unaffected by respiration; the split is always there.
- **A paradoxical split** is the opposite of what you would expect; the sounds fuse on inspiration and split on expiration (see Table 19-5, Variations in Split $S_2$).

- **A pathologic $S_3$** (ventricular gallop) occurs with heart failure and volume overload; a pathologic $S_4$ (atrial gallop) occurs with CAD (see Table 19-7, Diastolic Extra Sounds, for a full description).

Murmurs may be due to congenital defects and acquired valvular defects. Study Tables 19-9 and 19-10 for a complete description.

A systolic murmur may occur with a normal heart or with heart disease; a diastolic murmur always indicates heart disease.
Normal Range of Findings

**Pitch.** Describe the pitch as high, medium, or low. The pitch depends on the pressure and the rate of blood flow producing the murmur.

**Pattern.** The intensity may follow a pattern during the cardiac phase, growing louder (crescendo), tapering off (decrescendo), or increasing to a peak and then decreasing (crescendo-decrescendo, or diamond shaped). Because the whole murmur is just milliseconds long, it takes practice to diagnose any pattern.

**Quality.** Describe the quality as musical, blowing, harsh, or rumbling.

**Location.** Describe the area of maximum intensity of the murmur (where it is best heard) by noting the valve area or intercostal spaces.

**Radiation.** The murmur may be transmitted downstream in the direction of blood flow and may be heard in another place on the precordium, the neck, the back, or the axilla.

**Posture.** Some murmurs disappear or are enhanced by a change in position.

Some murmurs are common in healthy children or adolescents and are termed *innocent* or *functional*. *Innocent* indicates having no valvular or other pathologic cause; *functional* is due to increased blood flow in the heart (e.g., in anemia, fever, pregnancy, hyperthyroidism). The contractile force of the heart is greater in children. This increases blood flow velocity. The increased velocity plus a smaller chest measurement makes an audible murmur.

The innocent murmur is generally soft (grade ii), midsystolic, short, crescendo-decrescendo, and with a vibratory or musical quality (“voooot” sound like fiddle strings). Also, the innocent murmur is heard at the second or third left intercostal space and disappears with sitting, and the young person has no associated signs of cardiac dysfunction.

Although it is important to distinguish innocent murmurs from pathologic ones, it is best to suspect all murmurs as pathologic until they are proved otherwise. Diagnostic tests such as ECG, phonocardiogram, and echocardiogram are needed to establish an accurate diagnosis.

**Change Position.** After auscultating in the supine position, roll the person toward his or her left side. Listen with the bell at the apex for the presence of any diastolic filling sounds (i.e., the $S_3$ or $S_4$) (Fig. 19-28).

Abnormal Findings

The murmur of mitral stenosis is rumbling, whereas that of aortic stenosis is harsh (see Table 19-10).
Normal Range of Findings

Ask the person to sit up, lean forward slightly, and exhale. Listen with the diaphragm firmly pressed at the base, right, and left sides. Check for the soft, high-pitched, early diastolic murmur of aortic or pulmonic regurgitation (Fig. 19-29).

<table>
<thead>
<tr>
<th>Abnormal Findings</th>
</tr>
</thead>
<tbody>
<tr>
<td>Murmur of aortic regurgitation sometimes may be heard only when the person is leaning forward in the sitting position.</td>
</tr>
</tbody>
</table>

**DEVELOPMENTAL COMPETENCE**

**Infants**

The transition from fetal to pulmonic circulation occurs in the immediate newborn period. Fetal shunts normally close within 10 to 15 hours but may take up to 48 hours. Thus you should assess the cardiovascular system during the first 24 hours and again in 2 to 3 days.

Note any extracardiac signs that may reflect heart status (particularly in the skin), liver size, and respiratory status. The skin color should be pink to pinkish brown, depending on the infant’s genetic heritage. If cyanosis occurs, determine its first appearance—at or shortly after birth versus after the neonatal period. Normally, the liver is not enlarged and the respirations are not labored. Also, note the expected parameters of weight gain throughout infancy.

Palpate the apical impulse to determine the size and position of the heart. Because the infant’s heart has a more horizontal placement, expect to palpate the apical impulse at the fourth intercostal space just lateral to the midclavicular line. It may or may not be visible.
Normal Range of Findings

The heart rate is best auscultated because radial pulses are hard to count accurately. Use the small (pediatric size) diaphragm and bell (Fig. 19-30). The heart rate may range from 100 to 180 per minute immediately after birth, then stabilize to an average of 120 to 140 per minute. Infants normally have wide fluctuations with activity, from 170 per minute or more with crying or being active to 70 to 90 per minute with sleeping. Variations are greatest at birth and are even more so with premature babies (see Table 9-3).

<table>
<thead>
<tr>
<th>Abnormal Findings</th>
</tr>
</thead>
<tbody>
<tr>
<td>Persistent tachycardia is &gt;200 per minute in newborns, or &gt;150 per minute in infants.</td>
</tr>
<tr>
<td>Bradycardia is &lt;90 per minute in newborns or &lt;60 in older infants or children. This causes a serious drop in cardiac output because the small muscle mass of their hearts cannot increase stroke volume significantly.</td>
</tr>
</tbody>
</table>

Expect the heart rhythm to have sinus arrhythmia, the phasic speeding up or slowing down with the respiratory cycle.

Rapid rates make it more challenging to evaluate heart sounds. Expect heart sounds to be louder in infants than in adults because of the infant’s thinner chest wall. Also, $S_2$ has a higher pitch and is sharper than $S_1$. Splitting of $S_2$ just after the height of inspiration is common, not at birth, but beginning a few hours after birth.

Murmurs in the immediate newborn period do not necessarily indicate congenital heart disease. Murmurs are relatively common in the first 2 to 3 days because of fetal shunt closure. These murmurs are usually grade i or ii, are systolic, accompany no other signs of cardiac disease, and disappear in 2 to 3 days. The murmur of PDA is a continuous machinery murmur, which disappears by 2 to 3 days. On the other hand, absence of a murmur in the immediate newborn period does not ensure a perfect heart; congenital defects can be present that are not signaled by an early murmur. It is best to listen frequently and to note and describe any murmur according to the characteristics listed on p. 504.

Children

Note any extracardiac or cardiac signs that may indicate heart disease: poor weight gain, developmental delay, persistent tachycardia, tachypnea, dyspnea on exertion, cyanosis, and clubbing. Note that clubbing of fingers and toes usually does not appear until late in the 1st year, even with severe cyanotic defects.
Normal Range of Findings

The apical impulse is sometimes visible in children with thin chest walls. Note any obvious bulge or any heave—these are not normal.

Palpate the apical impulse: in the fourth intercostal space to the left of the midclavicular line until age 4 years; at the fourth interspace at the midclavicular line from age 4 to 6 years; and in the fifth interspace to the right of the midclavicular line at age 7 years (Fig. 19-31).

Abnormal Findings

A precordial bulge to the left of the sternum with a hyperdynamic precordium signals cardiac enlargement. The bulge occurs because the cartilaginous rib cage is more compliant.

A substernal heave occurs with right ventricular enlargement, and an apical heave occurs with left ventricular hypertrophy.

The apical impulse moves laterally with cardiac enlargement.

Thrill (palpable vibration).

The average heart rate slows as the child grows older, although it is still variable with rest or activity (see Table 9-2).

The heart rhythm remains characterized by sinus arrhythmia. Physiologic $S_1$ is common in children (see Table 19-7). It occurs in early diastole, just after $S_2$, and is a dull soft sound that is best heard at the apex.

A venous hum—due to turbulence of blood flow in the jugular venous system—is common in healthy children and has no pathologic significance. It is a continuous, low-pitched, soft hum that is heard throughout the cycle, although it is loudest in diastole. Listen with the bell over the supraclavicular fossa at the medial third of the clavicle, especially on the right, or over the upper anterior chest.

The venous hum is usually not affected by respiration, may sound louder when the child stands, and is easily obliterated by occluding the jugular veins in the neck with your fingers.

Heart murmurs that are innocent (or functional) in origin are very common through childhood. Some authors say they have a 30% occurrence, and some authors say nearly all children may demonstrate a murmur at some time. Most innocent murmurs have these characteristics: soft, relatively short systolic ejection murmur; medium pitch; vibratory; best heard at the left lower sternal or midsternal border, with no radiation to the apex, base, or back.

For the child whose murmur has been shown to be innocent, it is very important that the parents understand this completely. They need to believe that this murmur is just a "noise" and has no pathologic significance. Otherwise, the parents may become overprotective and limit activity for the child, which may result in the child developing a negative self-concept.

This latter maneuver helps differentiate the venous hum from other cardiac murmurs (e.g., PDA).

Distinguish innocent murmurs from pathologic ones. This may involve referral to another examiner or the performance of diagnostic tests such as the ECG or ultrasonography.
Normal Range of Findings

The Pregnant Woman
The vital signs usually yield an increase in resting pulse rate of 10 to 15 beats per minute and a drop in blood pressure from the normal prepregnancy level. The BP decreases to its lowest point during the second trimester and then slowly rises during the third trimester. The BP varies with position. It is usually lowest in left lateral recumbent position, a bit higher when supine, and highest when sitting.10

Inspection of the skin often shows a mild hyperemia in light-skinned women because the increased cutaneous blood flow tries to eliminate the excess heat generated by the increased metabolism. Palpation of the apical impulse is higher and lateral compared with the normal position, as the enlarging uterus elevates the diaphragm and displaces the heart up and to the left and rotates it on its long axis.

Auscultation of the heart sounds shows changes caused by the increased blood volume and workload:

• Heart sounds
  Exaggerated splitting of $S_1$ and increased loudness of $S_1$
  A loud, easily heard $S_3$
• Heart murmurs
  A systolic murmur in 90%, which disappears soon after delivery
  A soft, diastolic murmur heard transiently in 19%
  A continuous murmur from breast vasculature in 10%.10

The last-mentioned murmur is termed a mammary souffle (pronounced soof’ f’l), which occurs near term or when the mother is lactating; it is due to increased blood flow through the internal mammary artery. The murmur is heard in the second, third, or fourth intercostal space; it is continuous, although it is accentuated in systole. You can obliterate it by pressure with the stethoscope or one finger lateral to the murmur.

The ECG has no changes except for a slight left axis deviation due to the change in the heart’s position.

The Aging Adult
A gradual rise in systolic blood pressure is common with aging; the diastolic blood pressure stays fairly constant with a resulting widening of pulse pressure. Some older adults experience orthostatic hypotension, a sudden drop in blood pressure when rising to sit or stand.

Use caution in palpating and auscultating the carotid artery. Avoid pressure in the carotid sinus area, which could cause a reflex slowing of the heart rate. Also, pressure on the carotid artery could compromise circulation if the artery is already narrowed by atherosclerosis.

When measuring jugular venous pressure, view the right internal jugular vein. The aorta stiffens, dilates, and elongates with aging, which may compress the left neck veins and obscure pulsations on the left side.15a

The chest often increases in anteroposterior diameter with aging. This makes it more difficult to palpate the apical impulse and to hear the splitting of $S_2$. The $S_3$ often occurs in older people with no known cardiac disease. Systolic murmurs are common, occurring in over 50% of aging people.15a

Occasional premature ectopic beats are common and do not necessarily indicate underlying heart disease. When in doubt, obtain an ECG. However, consider that the ECG records for only one isolated minute in time and may need to be supplemented by a test of 24-hour ambulatory heart monitoring.

Abnormal Findings

Suspect pregnancy-induced hypertension with a sustained rise of 30 mm Hg systolic or 15 mm Hg diastolic under basal conditions.

Murmurs of aortic valve disease cannot be obliterated.

The $S_3$ is associated with heart failure and is always abnormal over age 35 years (see Table 19-7).
When someone complains of chest pain or pain radiating down the left arm, we think heart attack. After all, these are the symptoms that typically occur, aren’t they? Well, yes and no. They are the most “typical” symptoms men have when having a myocardial infarction (MI), but not women. For women, symptoms can be quite different. A woman’s “atypical” symptoms may be one of the reasons that more women are dying from heart disease than men these days. According to the Women’s Heart Foundation, almost a third of women experience no chest pain at all when having a heart attack. Instead, 71% of women report flu-like symptoms, including extreme fatigue, for up to a month before the attack. Women are more likely to feel a hot or cold burning sensation or a tenderness to touch in their back, shoulders, arms, or jaw—not sharp pain. Women’s symptoms often include nausea, vomiting, indigestion, and shortness of breath, which are easy to attribute to something other than the heart. The evidence now shows that women tend to minimize their symptoms or attribute them to something else. This may be due to a lack of awareness.

The Heart Truth® is a national awareness and prevention campaign about heart disease in women sponsored by the National Heart, Lung, and Blood Institute (NHLBI). The campaign includes three components: (1) professional education, (2) patient education, and (3) public awareness. At The Heart Truth® website, health professionals can access both clinical and patient education resources. Of particular interest are the clinical assessment tools, including a Risk Status, LDL, and Drug-Therapy Guide; a 10-year heart attack calculator; and a body mass index (BMI) calculator, which are either available online or as an applications for a pocket PC. For patients, there is the Heart Truth E-zine, the NHLBI quarterly electronic publication that provides new information about heart disease research and heart-healthy recipes. Patients can also download the The Healthy Heart Handbook for Women.
OBJECTIVE

Neck: Carotids 2+ and = bilaterally. Internal jugular vein pulsations present when supine and disappear when elevated to a 45° position.

Precordium: Inspection. No visible pulsations, no heave or lift.

Palpation: Apical impulse in 5th ics at left midclavicular line, no thrill.

Auscultation: Rate 68 beats per minute, rhythm regular, S1-S2 are normal, not diminished or accentuated, no S3, no S4 or other extra sounds, no murmurs.

ASSESSMENT

Neck vessels healthy by inspection and auscultation
Heart sounds normal

Focused Assessment: Clinical Case Study

Mr. N.V. is a 53-year-old white male woodcutter admitted to the CCU at University Medical Center (UMC) with chest pain.

SUBJECTIVE

1 year PTA—N.V. admitted to UMC with crushing substernal chest pain, radiating to L shoulder, accompanied by nausea, vomiting, diaphoresis.

Diagnosed as MI, hospitalized 7 days, discharged with nitroglycerin prn for anginal pain.

Did not return to work. Activity included walking 1 mile/day, hunting. Had occasional episodes of chest pain with exercise, relieved by rest.

1 day PTA—had increasing frequency of chest pain, about every 2 hours, lasting few minutes, saw pain as warning to go to MD.

Day of admission—severe substernal chest pain (“like someone sitting on my chest”) unrelieved by rest. Saw personal MD, while in office had episode of chest pain as last year’s, accompanied by diaphoresis, no N & V or SOB, relieved by 1 nitroglycerin. Transferred to UMC by paramedics. No further pain since admission 2 hours ago.

Family hx—mother died of MI at age 57.

Personal habits—smokes 1 ½ pack cigarettes daily × 34 years, no alcohol, diet—trying to limit fat and fried food, still high in added salt.

OBJECTIVE

Extremities: Skin pink, no cyanosis. Upper extrem.—capillary refill sluggish, no clubbing. Lower extrem.—no edema, no hair growth 10 cm below knee bilaterally.

Pulses—

Carotid 2+ 2+ 2+ 2+ 2+ 2+ 2+ 0 0 1+ all = bilaterally

B/P R arm 104/66 mm Hg

Neck: External jugulars flat. Internal jugular pulsations present when supine and absent when elevated to 45°.

Precordium: Inspection. Apical impulse visible 5th ics, 7 cm left of midsternal line, no heave.

Palpation: Apical impulse palpable in 5th and 6th ics. No thrill.

Auscultation: Apical rate 92 bpm regular, S1-S2 are normal, not diminished or accentuated, no S3 or S4, grade iii/vi systolic murmur present at left lower sternal border.

ASSESSMENT

Substernal chest pain
Systolic murmur
Ineffective tissue perfusion R/T interruption in flow
Decreased cardiac output R/T reduction in stroke volume
Decreased cardiac output occurs when the heart fails as a pump, and the circulation becomes backed up and congested. Signs and symptoms of heart failure come from two basic mechanisms: (1) the heart’s inability to pump enough blood to meet the metabolic demands of the body; and (2) the kidney’s compensatory mechanisms of abnormal retention of sodium and water to compensate for the decreased cardiac output. This increases blood volume and venous return, which causes further congestion.

Onset of heart failure may be: (1) acute, as following a myocardial infarction when direct damage to the heart’s contracting ability has occurred; or (2) chronic, as with hypertension, when the ventricles must pump against chronically increased pressure.

**TABLE 19-2 Clinical Portrait of Heart Failure**

<table>
<thead>
<tr>
<th>Abnormal Finding</th>
<th>Clinical Description</th>
</tr>
</thead>
<tbody>
<tr>
<td>Dilated pupils, a sympathetic nervous system response</td>
<td>Anxiety, gasping from pulmonary congestion</td>
</tr>
<tr>
<td>Skin pale, gray, or cyanotic</td>
<td>Falling O₂ saturation</td>
</tr>
<tr>
<td>Dyspnea, SOBOE is early symptom from pulmonary congestion</td>
<td>Confusion, unconsciousness from decreased O₂ to brain</td>
</tr>
<tr>
<td>Orthopnea, cannot breathe unless sitting up</td>
<td>Jugular vein distention from venous congestion</td>
</tr>
<tr>
<td>Crackles, wheeze are adventitious breath sounds</td>
<td>Infarct, may be cause of decreased cardiac output</td>
</tr>
<tr>
<td>Cough, frothy pink or white sputum</td>
<td>Fatigue, weakness from decreased cardiac output</td>
</tr>
<tr>
<td>Decreased blood pressure stimulates sympathetic nervous system, which acts on heart to increase rate and increase force of contraction</td>
<td>S₃ gallop, tachycardia</td>
</tr>
<tr>
<td>Nausea and vomiting as peristalsis slows and bile and fluids back up into stomach</td>
<td>Enlarged spleen and liver from venous congestion, which causes pressure on breathing</td>
</tr>
<tr>
<td>Ascites, fluid in peritoneal cavity</td>
<td>Decreased urine output as kidneys compensate for decreased CO by retaining sodium and H₂O</td>
</tr>
<tr>
<td>Dependent, pitting edema in sacrum, legs</td>
<td>Weak pulse</td>
</tr>
<tr>
<td>Cool, moist skin as peripheral vasoconstriction shunts blood to vital organs</td>
<td></td>
</tr>
</tbody>
</table>
### TABLE 19-3 Variations in S₁

The intensity of S₁ depends on three factors: (1) position of AV valve at the start of systole, (2) structure of the valve leaflets, and (3) how quickly pressure rises in the ventricle.

<table>
<thead>
<tr>
<th>Factor</th>
<th>Examples</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Loud (Accentuated) S₁</strong>&lt;br&gt; S₁ S₂</td>
<td>1. Position of AV valve at start of systole—wide open and no time to drift together&lt;br&gt; Hyperkinetic states where blood velocity is increased: exercise, fever, anemia, hyperthyroidism&lt;br&gt; 2. Change in valve structure—calcification of valve, needs increasing ventricular pressure to close the valve against increased atrial pressure&lt;br&gt; Mitral stenosis with leaflets still mobile</td>
</tr>
<tr>
<td><strong>Faint (Diminished) S₁</strong>&lt;br&gt; S₁ S₂</td>
<td>1. Position of AV valve—delayed conduction from atria to ventricles. Mitral valve drifts shut before ventricular contraction closes it&lt;br&gt; First-degree heart block (prolonged PR interval)&lt;br&gt; 2. Change in valve structure—extreme calcification, which limits mobility&lt;br&gt; Mitral insufficiency&lt;br&gt; 3. More forceful atrial contraction into noncompliant ventricle; delays or diminishes ventricular contraction&lt;br&gt; Severe hypertension—systemic or pulmonary</td>
</tr>
<tr>
<td><strong>Varying Intensity of S₁</strong>&lt;br&gt; S₁ S₂</td>
<td>1. Position of AV valve varies before closing from beat to beat&lt;br&gt; Atrial fibrillation—irregularly irregular rhythm&lt;br&gt; 2. Atria and ventricles beat independently&lt;br&gt; Complete heart block with changing PR interval</td>
</tr>
<tr>
<td><strong>Split S₁</strong>&lt;br&gt; S₁ S₂</td>
<td>Mitral and tricuspid components are heard separately&lt;br&gt; Normal but uncommon</td>
</tr>
</tbody>
</table>
### TABLE 19-4 | Variations in S₂

<table>
<thead>
<tr>
<th>Condition</th>
<th>Example</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Accentuated S₂</strong></td>
<td></td>
</tr>
<tr>
<td>1. Higher closing pressure</td>
<td>Systemic hypertension, ringing or booming S₂</td>
</tr>
<tr>
<td>2. Exercise and excitement increase pressure in aorta</td>
<td></td>
</tr>
<tr>
<td>3. Pulmonary hypertension</td>
<td>Mitral stenosis, heart failure</td>
</tr>
<tr>
<td>4. Semilunar valves calcified but still mobile</td>
<td>Aortic or pulmonic stenosis</td>
</tr>
<tr>
<td><strong>Diminished S₂</strong></td>
<td></td>
</tr>
<tr>
<td>1. A fall in systemic blood pressure causes a decrease in valve strength</td>
<td>Shock</td>
</tr>
<tr>
<td>2. Semilunar valves thickened and calcified, with decreased mobility</td>
<td>Aortic or pulmonic stenosis</td>
</tr>
</tbody>
</table>

### TABLE 19-5 | Variations in Split S₂

**Normal Splitting**

**Expiration**

<table>
<thead>
<tr>
<th>S₁</th>
</tr>
</thead>
<tbody>
<tr>
<td>S₂</td>
</tr>
</tbody>
</table>

**Inspiration**

<table>
<thead>
<tr>
<th>A₂-P₂</th>
</tr>
</thead>
</table>

<table>
<thead>
<tr>
<th>Condition</th>
<th>Example</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Fixed Split</strong></td>
<td></td>
</tr>
<tr>
<td><strong>Expiration</strong></td>
<td>A fixed split is unaffected by respiration; the split is always there.</td>
</tr>
<tr>
<td><strong>Inspiration</strong></td>
<td>A₂-P₂</td>
</tr>
<tr>
<td></td>
<td></td>
</tr>
<tr>
<td><strong>Paradoxical Split</strong></td>
<td></td>
</tr>
<tr>
<td><strong>Expiration</strong></td>
<td>Conditions that delay aortic valve closure cause the opposite of a normal split. In inspiration, P₂ is normally delayed so with a paradoxical split, the sounds fuse. In expiration, you hear the split, in the order of P₂A₂.</td>
</tr>
<tr>
<td><strong>Inspiration</strong></td>
<td>P₂-A₂</td>
</tr>
<tr>
<td></td>
<td></td>
</tr>
<tr>
<td><strong>Wide Split</strong></td>
<td></td>
</tr>
<tr>
<td><strong>Expiration</strong></td>
<td>When the right ventricle has delayed electrical activation, the split is very wide on inspiration and is still there on expiration.</td>
</tr>
<tr>
<td><strong>Inspiration</strong></td>
<td>A₂-P₂</td>
</tr>
</tbody>
</table>

**Abnormal Findings**

- **Atrial septal defect**
- **Right ventricular failure**
- **Aortic stenosis**
- **Left bundle branch block**
- **Patent ductus arteriosus**
- **Right bundle branch block** (which delays P₂)
TABLE 19-6  Systolic Extra Sounds

<table>
<thead>
<tr>
<th>Early systolic:</th>
<th>Mid-/late systolic:</th>
</tr>
</thead>
<tbody>
<tr>
<td>Ejection click</td>
<td>Midsystolic (mitral) click</td>
</tr>
<tr>
<td>Aortic prosthetic valve sounds</td>
<td></td>
</tr>
</tbody>
</table>

**EXPIRATION**

Aortic ejection click (apex and base)

Mid-/late systolic:

Midsystolic (mitral) click

Pulmonic ejection click (base only)

---

**Ejection Click**

The ejection click occurs early in systole at the start of ejection because it results from opening of the semilunar valves. Normally, the SL valves open silently, but in the presence of stenosis (e.g., aortic stenosis, pulmonic stenosis), their opening makes a sound. It is short and high pitched, with a click quality, and is heard better with the diaphragm.

The aortic ejection click is heard at the second right interspace and apex and may be loudest at the apex. Its intensity does not change with respiration. The pulmonic ejection click is best heard in the second left interspace and often grows softer with inspiration.

---

**Aortic Prosthetic Valve Sounds**

As a sequela of modern technologic intervention for heart problems, some people now have *iatrogenically* induced heart sounds. The opening of a mechanical aortic ball-in-cage prosthesis produces an early systolic sound. This sound is less intense with a tilting disk prosthesis and is absent with a biologic tissue prosthesis (e.g., porcine).

---

**Midsystolic Click**

Although it is systolic, this is not an ejection click. It is associated with mitral valve prolapse, in which the mitral valve leaflets not only close with contraction but balloon back up into the left atrium. During ballooning, the sudden tensing of the valve leaflets and the chordae tendineae creates the click.

The sound occurs in mid- to late systole and is short and high pitched, with a click quality. It is best heard with the diaphragm, at the apex, but also may be heard at the left lower sternal border. The click usually is followed by a systolic murmur. The click and murmur move with postural change; when the person assumes a squatting position, the click may move closer to S₂, and the murmur may sound louder and delayed. The Valsalva maneuver also moves the click closer to S₂.
TABLE 19-7  Diastolic Extra Sounds

<table>
<thead>
<tr>
<th>Diastolic extra Sounds</th>
<th>Early diastole:</th>
<th>Mid-diastole:</th>
<th>Late diastole:</th>
</tr>
</thead>
<tbody>
<tr>
<td>Opening snap</td>
<td></td>
<td>Third heart sound</td>
<td>Fourth heart sound</td>
</tr>
<tr>
<td>Mitral prosthetic valve sound</td>
<td></td>
<td>Summation sound (S₃ + S₄)</td>
<td>Pacemaker-induced sound</td>
</tr>
</tbody>
</table>

**Opening Snap**

Normally the opening of the AV valves is silent. In the presence of stenosis, increasingly higher atrial pressure is required to open the valve. The deformed valve opens with a noise: the opening snap. It is sharp and high pitched, with a snapping quality. It sounds after S₂ and is best heard with the diaphragm at the third or fourth left interspace at the sternal border, less well at the apex.

The opening snap usually is not an isolated sound. As a sign of mitral stenosis, the opening snap usually ushers in the low-pitched diastolic rumbling murmur of that condition.

**Mitral Prosthetic Valve Sound**

An iatrogenic sound, the opening of a ball-in-cage mitral prosthesis gives an early diastolic sound: an opening click just after S₂. It is loud, is heard over the whole precordium, and is loudest at the apex and left lower sternal border.

**Third Heart Sound**

The S₃ is a ventricular filling sound. It occurs in early diastole during the rapid filling phase. Your hearing quickly accommodates to the S₃, so it is best heard when you listen initially. It sounds after S₂ but later than an opening snap would be. It is a dull, soft sound, and it is low pitched, like “distant thunder.” It is heard best in a quiet room, at the apex, with the bell held lightly (just enough to form a seal), and with the person in the left lateral position.

The S₃ can be confused with a split S₂. Use these guidelines to distinguish the S₃:

- **Location**—The S₃ is heard at the apex or left lower sternal border; the split S₂ at the base.
- **Respiratory variation**—The S₃ does not vary in timing with respirations; the split S₂ does.
- **Pitch**—The S₃ is lower pitched; the pitch of the split S₂ stays the same.

The S₃ may be normal (physiologic) or abnormal (pathologic). The physiologic S₃ is heard frequently in children and young adults; it occasionally may persist after age 40 years, especially in women. The normal S₃ usually disappears when the person sits up.

In adults, the S₃ is usually abnormal. The pathologic S₃ is also called a ventricular gallop or an S₃ gallop, and it persists when sitting up. The S₃ indicates decreased compliance of the ventricles, as in heart failure. The S₃ may be the earliest sign of heart failure. The S₃ may originate from either the left or the right ventricle; a left-sided S₃ is heard at the apex in the left lateral position, and a right-sided S₃ is heard at the left lower sternal border with the person supine and is louder in inspiration.

The S₃ occurs also with conditions of volume overload, such as mitral regurgitation and aortic or tricuspid regurgitation. The S₃ is also found in high cardiac output states in the absence of heart disease, such as hyperthyroidism, anemia, and pregnancy. When the primary condition is corrected, the gallop disappears.

Continued
**Fourth Heart Sound**

The $S_4$ is a ventricular filling sound. It occurs when the atra contract late in diastole. It is heard immediately before $S_1$. This is a very soft sound, of very low pitch. You need a good bell, and you must listen for it. It is heard best at the apex, with the person in left lateral position.

A **physiologic** $S_4$ may occur in adults older than 40 or 50 years with no evidence of cardiovascular disease, especially after exercise.

A **pathologic** $S_4$ is termed an atrial gallop or an $S_4$ gallop. It occurs with decreased compliance of the ventricle (e.g., coronary artery disease, cardiomyopathy) and with systolic overload (afterload), including outflow obstruction to the ventricle (aortic stenosis) and systemic hypertension. A left-sided $S_4$ occurs with these conditions. It is heard best at the apex, in the left lateral position.

A right-sided $S_4$ is less common. It is heard at the left lower sternal border and may increase with inspiration. It occurs with pulmonary stenosis or pulmonary hypertension.

**Summation Sound**

When both the pathologic $S_3$ and $S_4$ are present, a quadruple rhythm is heard. Often, in cases of cardiac stress, one response is tachycardia. During rapid rates, the diastolic filling time shortens and the $S_3$ and $S_4$ move closer together. They sound superimposed in mid-diastole, and you hear one loud, prolonged, summed sound, often louder than either $S_1$ or $S_2$.

**EXTRACARDIAC SOUNDS**

**Pericardial Friction Rub**

Inflammation of the pericardium gives rise to a friction rub. The sound is high pitched and scratchy, like sandpaper being rubbed. It is best heard with the diaphragm, with the person sitting up and leaning forward, and with the breath held in expiration.

A friction rub can be heard any place on the precordium but usually is best heard at the apex and left lower sternal border, places where the pericardium comes in close contact with the chest wall. Timing may be systolic and diastolic. The friction rub of pericarditis is common during the 1st week after a myocardial infarction and may last only a few hours.
TABLE 19-8  Abnormal Pulsations on the Precordium

**Base**
A **thrill** in the second and third right interspaces occurs with severe aortic stenosis and systemic hypertension.
A **thrill** in the second and third left interspaces occurs with pulmonic stenosis and pulmonic hypertension.

**Left Sternal Border**
A **lift (heave)** occurs with right ventricular hypertrophy, as found in pulmonic valve disease, pulmonic hypertension, and chronic lung disease. You feel a diffuse lifting impulse during systole at the left lower sternal border. It may be associated with retraction at the apex because the left ventricle is rotated posteriorly by the enlarged right ventricle.

**Apex**
Cardiac enlargement displaces the apical impulse laterally and over a wider area when left ventricular hypertrophy and dilation are present. This is **volume overload**, as in mitral regurgitation, aortic regurgitation, and left-to-right shunts.

**Apex**
The apical impulse is increased in force and duration but is not necessarily displaced to the left when left ventricular hypertrophy occurs alone without dilation. This is **pressure overload**, as found in aortic stenosis or systemic hypertension.

---

TABLE 19-9  Congenital Heart Defects

<table>
<thead>
<tr>
<th>Description</th>
<th>Clinical Data</th>
</tr>
</thead>
<tbody>
<tr>
<td>Patent Ductus Arteriosus (PDA)</td>
<td>Persistence of the channel joining left pulmonary artery to aorta. This is normal in the fetus and usually closes spontaneously within hours of birth.</td>
</tr>
<tr>
<td></td>
<td>S: Usually no symptoms in early childhood; growth and development are normal.</td>
</tr>
<tr>
<td></td>
<td>O: Blood pressure has wide pulse pressure and bounding peripheral pulses from rapid runoff of blood into low-resistance pulmonary bed during diastole. Thrill often palpable at left upper sternal border. The continuous murmur heard in systole and diastole is called a <em>machinery murmur</em>.</td>
</tr>
</tbody>
</table>

Images © Pat Thomas, 2006.
**Atrial Septal Defect (ASD)**

Abnormal opening in the atrial septum, resulting usually in left-to-right shunt and causing large increase in pulmonary blood flow.

*S*: Defect is remarkably well tolerated. Symptoms in infant are rare; growth and development normal. Children and young adults have mild fatigue and DOE.

*O*: Sternal lift often present. \( S_2 \) has fixed split, with \( P_2 \) often louder than \( A_2 \). Murmur is systolic, ejection, medium pitch, best heard at base in second left interspace. Murmur caused not by shunt itself but by increased blood flow through pulmonic valve.

**Ventricular Septal Defect (VSD)**

Abnormal opening in septum between the ventricles, usually subaortic area. The size and exact position vary considerably.

*S*: Small defects are asymptomatic. Infants with large defects have poor growth, slow weight gain; later look pale, thin, delicate. May have feeding problems; DOE; frequent respiratory infections; and when the condition is severe, heart failure.

*O*: Loud, harsh holosystolic murmur, best heard at left lower sternal border, may be accompanied by thrill. Large defects also have soft diastolic murmur at apex (mitral flow murmur) due to increased blood flow through mitral valve.

**Tetralogy of Fallot**

Four components: (1) right ventricular outflow stenosis, (2) VSD, (3) right ventricular hypertrophy, and (4) overriding aorta. *Result*: shunts a lot of venous blood directly into aorta away from pulmonary system, so blood never gets oxygenated.

*S*: Severe cyanosis, not in first months of life but develops as infant grows and RV outflow (i.e., pulmonic) stenosis gets worse. Cyanosis with crying and exertion at first, then at rest. Uses squatting posture after starts walking. DOE common. Development is slowed.

*O*: Thrill palpable at left lower sternal border. \( S_1 \) normal; \( S_2 \) has \( A_2 \) loud and \( P_2 \) diminished or absent. Murmur is systolic, loud, crescendo-decrescendo.

**Coarctation of the Aorta**

Severe narrowing of descending aorta, usually at the junction of the ductus arteriosus and the aortic arch, just distal to the origin of the left subclavian artery. Results in increased workload on left ventricle.

Associated with defects of aortic valve in most cases, as well as associated patent ductus arteriosus; and associated ventricular septal defect.

*S*: In infants with associated lesions or symptoms, diagnosis occurs in first few months as symptoms of heart failure develop. For asymptomatic children and adolescents, growth and development are normal. Diagnosis usually incidental due to blood pressure findings. Adolescents may complain of vague lower extremity cramping that is worse with exercise.

*O*: Upper extremity hypertension over 20 mm Hg higher than lower extremity measures is a hallmark of coarctation. Another important sign is absent or greatly diminished femoral pulses. A systolic murmur is heard best at the left sternal border, radiating to the back.

---

**TABLE 19-9**  
**Congenital Heart Defects—cont’d**

<table>
<thead>
<tr>
<th>Description</th>
<th>Clinical Data</th>
</tr>
</thead>
<tbody>
<tr>
<td>Atrial Septal Defect (ASD)</td>
<td>Abnormal opening in the atrial septum, resulting usually in left-to-right shunt and causing large increase in pulmonary blood flow.</td>
</tr>
<tr>
<td>Ventricular Septal Defect (VSD)</td>
<td>Abnormal opening in septum between the ventricles, usually subaortic area. The size and exact position vary considerably.</td>
</tr>
<tr>
<td>Tetralogy of Fallot</td>
<td>Four components: (1) right ventricular outflow stenosis, (2) VSD, (3) right ventricular hypertrophy, and (4) overriding aorta. <em>Result</em>: shunts a lot of venous blood directly into aorta away from pulmonary system, so blood never gets oxygenated.</td>
</tr>
<tr>
<td>Coarctation of the Aorta</td>
<td>Severe narrowing of descending aorta, usually at the junction of the ductus arteriosus and the aortic arch, just distal to the origin of the left subclavian artery. Results in increased workload on left ventricle. Associated with defects of aortic valve in most cases, as well as associated patent ductus arteriosus; and associated ventricular septal defect.</td>
</tr>
</tbody>
</table>

*S*, Subjective data; *O*, objective data.  
Images © Pat Thomas, 2006.
TABLE 19-10  Murmurs Due to Valvular Defects

<table>
<thead>
<tr>
<th>Murmurs Due to Valvular Defects</th>
</tr>
</thead>
<tbody>
<tr>
<td>Midsystolic Ejection Murmurs</td>
</tr>
<tr>
<td>Due to forward flow through semilunar valves</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Description</th>
<th>Clinical Data</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Aortic Stenosis</strong></td>
<td><strong>S</strong>: Fatigue, DOE, palpitation, dizziness, fainting, anginal pain. <strong>O</strong>: Pallor, slow diminished radial pulse, low blood pressure, and auscultatory gap are common. Apical impulse sustained and displaced to left. Thrill in systole over second and third right interspaces and right side of neck. <strong>S</strong>₁ normal, often ejection click present, often paradoxical split <strong>S</strong>₂, <strong>S</strong>₄ present with LV hypertrophy. Murmur: Loud, harsh, midsystolic, crescendo-decrescendo, loudest at second right interspace, radiates widely to side of neck, down left sternal border, or apex.</td>
</tr>
<tr>
<td><strong>Pulmonic Stenosis</strong></td>
<td><strong>O</strong>: Thrill in systole at second and third left interspace, ejection click often present after <strong>S</strong>₁, diminished <strong>S</strong>₂ and usually with wide split, <strong>S</strong>₄ common with RV hypertrophy. Murmur: Systolic, medium pitch, coarse, crescendo-decrescendo (diamond shape), best heard at second left interspace, radiates to the left and neck.</td>
</tr>
</tbody>
</table>

S, Subjective data; O, objective data.
Midsystolic ejection Murmurs
Due to forward flow through semilunar valves

<table>
<thead>
<tr>
<th>S1</th>
<th>S2</th>
<th>S1</th>
<th>S2</th>
<th>S1</th>
<th>S2</th>
</tr>
</thead>
</table>

SYSTOLE DIASTOLE

Description Clinical Data

Pansystolic regurgitant Murmurs
Due to backward flow of blood from area of higher pressure to one of lower pressure

<table>
<thead>
<tr>
<th>S1</th>
<th>S2</th>
<th>S1</th>
<th>S2</th>
<th>S1</th>
<th>S2</th>
</tr>
</thead>
</table>

**Mitral Regurgitation**

Stream of blood regurgitates back into LA during systole through incompetent mitral valve. In diastole, blood passes back into LV again along with new flow; results in LV dilation and hypertrophy.

S: Fatigue, palpitation, orthopnea, PND.
O: Thrill in systole at apex. Lift at apex. Apical impulse displaced down and to left. S1 diminished, S2 accentuated, S3 at apex often present.
Murmur: Pansystolic, often loud, blowing, best heard at apex, radiates well to left axilla.

**Tricuspid Regurgitation**

Backflow of blood through incompetent tricuspid valve into RA.

O: Engorged pulsating neck veins, liver enlarged. Lift at sternum if RV hypertrophy present, often thrill at left lower sternal border.
Murmur: Soft, blowing, pansystolic, best heard at left lower sternal border, increases with inspiration.

**TABLE 19-10 Murmurs Due to Valvular Defects—cont’d**

<table>
<thead>
<tr>
<th>S, Subjective data; O, objective data.</th>
<th>Images © Pat Thomas, 2006. Continued</th>
</tr>
</thead>
</table>

**Abnormal Findings**
TABLE 19-10  Murmurs Due to Valvular Defects—cont’d

Diastolic Rumbles of AV Valves
Filling murmurs at low pressures, best heard with bell lightly touching skin

<table>
<thead>
<tr>
<th>Description</th>
<th>Clinical Data</th>
</tr>
</thead>
</table>
| *Mitral Stenosis* | Calcified mitral valve will not open properly, impedes forward flow of blood into LV during diastole. Results in LA enlarged and LA pressure increased.  
S: Fatigue, palpitations, DOE, orthopnea, occasional PND or pulmonary edema.  
O: Diminished, often irregular arterial pulse. Lift at apex, diastolic thrill common at apex. $S_1$ accentuated; opening snap after $S_2$ heard over wide area of precordium, followed by murmur. Murmur: Low-pitched diastolic rumble, best heard at apex, with person in left lateral position; does not radiate. |
| *Tricuspid Stenosis* | Calcification of tricuspid valve impedes forward flow into RV during diastole.  
O: Diminished arterial pulse, jugular venous pulse prominent.  
Murmur: Diastolic rumble; best heard at left lower sternal border; louder in inspiration. |

$S$, Subjective data; $O$, objective data.  
Images © Pat Thomas, 2006.  
Continued
Early Diastolic Murmurs
Due to SL valve incompetence

Aortic Regurgitation
Stream of blood regurgitates back through incompetent aortic valve into LV during diastole. LV dilation and hypertrophy due to increased LV stroke volume. Rapid ejection of large stroke volume into poorly filled aorta, then rapid runoff in diastole as part of blood pushed back into LV.

S: Only minor symptoms for many years, then rapid deterioration: DOE, PND, angina, dizziness.
O: Bounding “water-hammer” pulse in carotid, brachial, and femoral arteries. Blood pressure has wide pulse pressure. Pulsations in cervical and suprasternal area, apical impulse displaced to left and down, apical impulse feels brief. Murmur starts almost simultaneously with S2: soft high pitched, blowing diastolic, decrescendo, best heard at third left interspace at base, as person sits up and leans forward, radiates down.

Pulmonic Regurgitation
Backflow of blood through incompetent pulmonic valve, from pulmonary artery to RV.

Murmur has same timing and characteristics as that of aortic regurgitation, and is hard to distinguish on physical examination.
BIBLIOGRAPHY