

HEART AND NECK VESSEL ASSESSMENT

● STRUCTURE AND FUNCTION

The cardiovascular system is a highly complex system that includes the heart and a closed system of blood vessels. To collect accurate data and correctly interpret that data, the examiner must have an understanding of the structure and function of the heart, the great vessels, the electrical conduction system of the heart, the cardiac cycle, the production of heart sounds, cardiac output, and the neck vessels. This information helps the examiner to differentiate between normal and abnormal findings as they relate to the cardiovascular system.

HEART AND GREAT VESSELS

The heart is a hollow, muscular, four-chambered (left and right atria and left and right ventricles) organ located in the middle of the thoracic cavity between the lungs in the space called the *mediastinum*. It is about the size of a clenched fist and weighs approximately 255 g (9 oz) in women and 310 g (10.9 oz) in men. The heart extends vertically from the left second to the left fifth intercostal space (ICS) and horizontally from the right edge of the sternum to the left midclavicular line (MCL). The heart can be described as an inverted cone. The upper portion, near the left second ICS, is the base and the lower portion, near the left fifth ICS and the left MCL, is the apex. The anterior chest area that overlies the heart and great vessels is called the *precordium* (Fig. 18-1). The right side of the heart pumps blood to the lungs for gas exchange (pulmonary circulation); the left side of the heart pumps blood to all other parts of the body (systemic circulation).

The large veins and arteries leading directly to and away from the heart are referred to as the *great vessels*. The *superior and inferior vena cava* return blood to

the right atrium from the upper and lower torso respectively. The *pulmonary artery* exits the right ventricle, bifurcates, and carries blood to the lungs. The *pulmonary veins* (two from each lung) return oxygenated blood to the left atrium. The *aorta* transports oxygenated blood from the left ventricle to the body (Fig. 18-2).

Heart Chambers and Valves

The heart consists of four chambers or cavities: two upper chambers, the *right and left atria*, and two lower chambers, the *right and left ventricles*. The right and left sides of the heart are separated by a partition called the *septum*. The thin-walled atria receive blood returning to the heart and pump blood into the ventricles. The thicker-walled ventricles pump blood out of the heart. The left ventricle is thicker than the right ventricle because the left side of the heart has a greater workload.

The entrance and exit of each ventricle are protected by one-way valves that direct the flow of blood through the heart. The *atrioventricular* (AV) valves are located at the entrance into the ventricles. There are two AV valves: the tricuspid valve and the bicuspid (mitral) valve. The tricuspid valve is composed of three cusps or flaps and is located between the right atrium and the right ventricle; the bicuspid (mitral) valve is composed of two cusps or flaps and is located between the left atrium and the left ventricle. Collagen fibers, called *chordae tendineae*, anchor the AV valve flaps to papillary muscles within the ventricles.

Open AV valves allow blood to flow from the atria into the ventricles. However, as the ventricles begin to contract, the AV valves snap shut, preventing the regurgitation of blood into the atria. The valves are prevented from blowing open in the reverse direction (i.e., toward the atria) by their secure anchors to the papillary muscles of the ventricular wall. The *semilunar valves* are located

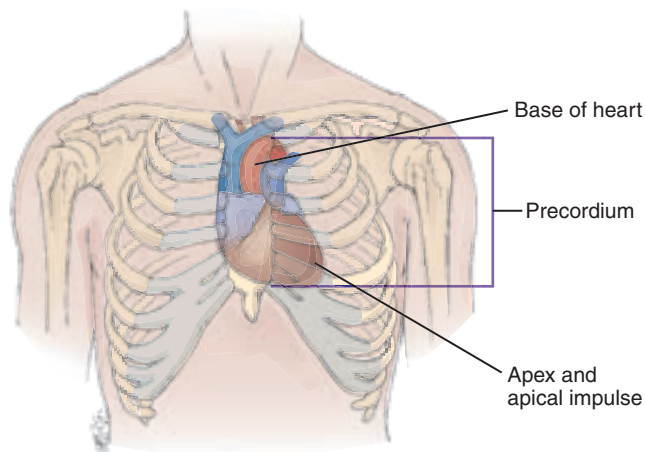


Figure 18-1 The heart and major blood vessels lie centrally in the chest behind the protective sternum.

at the exit of each ventricle at the beginning of the great vessels. Each valve has three cusps or flaps that look like half-moons, hence the name “semilunar.” There are two semilunar valves: the pulmonic valve is located at the entrance of the pulmonary artery as it exits the right ventricle and the aortic valve is located at the beginning of the ascending aorta as it exits the left ventricle. These valves are open during ventricular contraction and close from the pressure of blood when the ventricles relax. Blood is thus prevented from flowing backward into the relaxed ventricles (see Fig. 18-2).

Heart Covering and Walls

The *pericardium* is a tough, inextensible, loose-fitting, fibroserous sac that attaches to the great vessels and, thereby, surrounds the heart. A serous membrane lining, the *parietal pericardium*, secretes a small amount of peri-

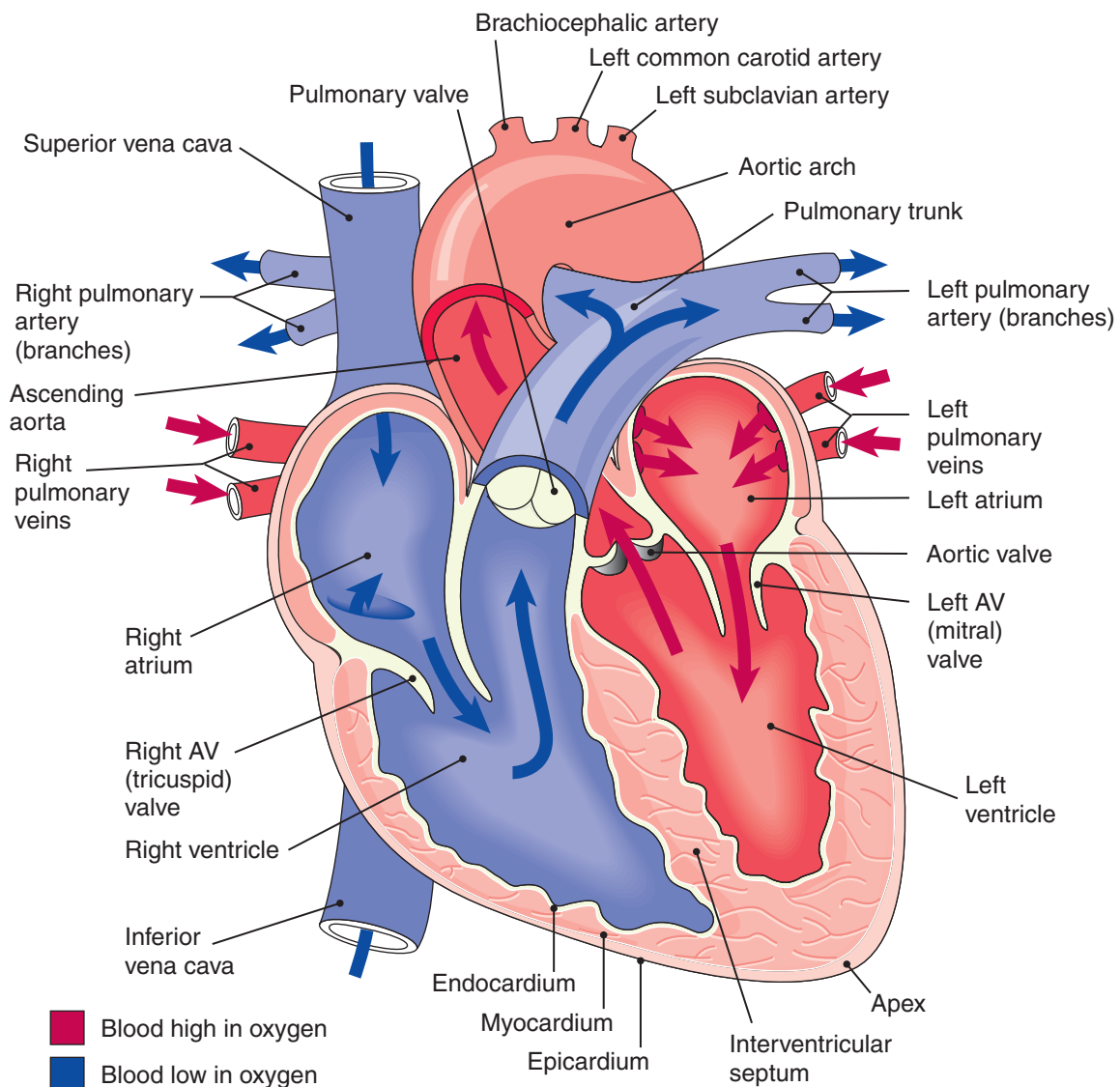


Figure 18-2 Heart chambers, valves, and direction of circulatory flow.

cardial fluid that allows for smooth, friction-free movement of the heart. This same type of serous membrane covers the outer surface of the heart and is known as the *epicardium*. The *myocardium* is the thickest layer of the heart and is made up of contractile cardiac muscle cells. The *endocardium* is a thin layer of endothelial tissue that forms the innermost layer of the heart and is continuous with the endothelial lining of blood vessels (see Fig. 18-2).

ELECTRICAL CONDUCTION OF THE HEART

Cardiac muscle cells have a unique inherent ability. They can spontaneously generate an electrical impulse and conduct it through the heart. The generation and conduction of electrical impulses by specialized sections of the myocardium regulate the events associated with the filling and emptying of the cardiac chambers. The process is called the *cardiac cycle* (see description below).

Pathways

The *sinoatrial (SA) node* (or sinus node) is located on the posterior wall of the right atrium near the junction of the superior and inferior vena cava. The SA node, with inherent rhythmicity, generates impulses (at a rate of 60

to 100 per minute) that are conducted over both atria, causing them to contract simultaneously and send blood into the ventricles. The current, initiated by the SA node, is conducted across the atria to the *AV node* located in the lower interatrial septum (Fig. 18-3). The AV node slightly delays incoming electrical impulses from the atria then relays the impulse to the AV bundle (bundle of His) in the upper interventricular septum. The electrical impulse then travels down the right and left bundle branches and the Purkinje fibers in the myocardium of both ventricles, causing them to contract almost simultaneously. Although the SA node functions as the “pacemaker of the heart,” this activity shifts to other areas of the conduction system, such as the Bundle of His (with an inherent discharge of 40 to 60 per minute), if the SA node cannot function.

Electrical Activity

Electrical impulses, which are generated by the SA node and travel throughout the cardiac conduction circuit, can be detected on the surface of the skin. This electrical activity can be measured and recorded by electrocardiography (ECG, aka EKG), which records the depolarization and repolarization of the cardiac muscle. The phases of the ECG are known as P, Q, R, S, and T. Display 18-1 describes the phases of the ECG.

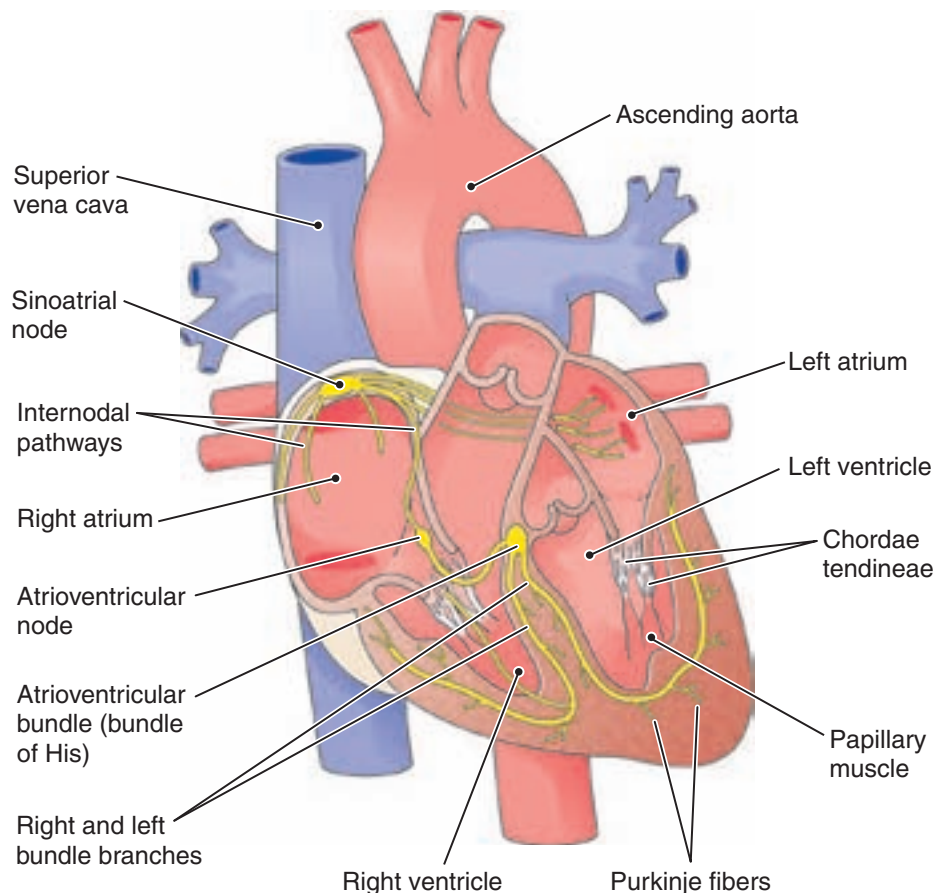
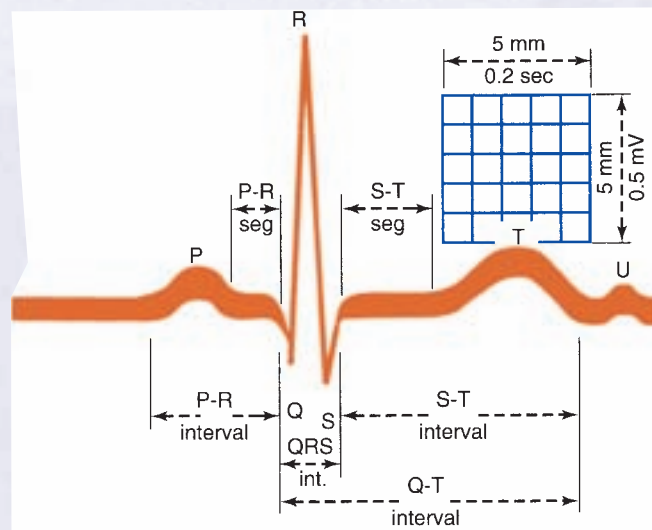


Figure 18-3 The electrical conduction system of the heart begins with impulse generated by the sinoatrial node (green) and circuted continuously over the heart.

DISPLAY 18-1 PHASES OF THE ELECTROCARDIOGRAM

The phases of the electrocardiogram (ECG), which records depolarization and repolarization of the heart, are assigned letters: P, Q, R, S, and T.



- **P wave:** Atrial depolarization; conduction of the impulse throughout the atria.
- **PR interval:** Time from the beginning of the atrial depolarization to the beginning of ventricular depolarization, that is, from the beginning of the P wave to the beginning of the QRS complex.
- **QRS complex:** Ventricular depolarization (also atrial repolarization); conduction of the impulse throughout the ventricles, which then triggers contraction of the ventricles; measured from the beginning of the Q wave to the end of the S wave.
- **ST segment:** Period between ventricular depolarization and the beginning of ventricular repolarization.
- **T wave:** Ventricular repolarization; the ventricles return to a resting state.
- **QT interval:** Total time for ventricular depolarization and repolarization, that is, from the beginning of the Q wave to the end of the T wave; the QT interval varies with heart rate.
- **U wave:** May or may not be present; if it is present, it follows the T wave and represents the final phase of ventricular repolarization.

THE CARDIAC CYCLE

The cardiac cycle refers to the filling and emptying of the heart's chambers. The cardiac cycle has two phases: diastole (relaxation of the ventricles, known as filling) and systole (contraction of the ventricles, known as emptying). Diastole endures for approximately two-thirds of the cardiac cycle and systole is the remaining one-third (Fig. 18-4).

Diastole

During ventricular diastole, the AV valves are open and the ventricles are relaxed. This causes higher pressure in the atria than in the ventricles. Therefore, blood rushes

through the atria into the ventricles. This early, rapid, passive filling is called *early or protodiastolic filling*. This is followed by a period of slow passive filling. Finally, near the end of ventricular diastole, the atria contract and complete the emptying of blood out of the upper chambers by propelling it into the ventricles. This final active filling phase is called *presystole*, *atrial systole*, or sometimes the "*atrial kick*." This action raises left ventricular pressure.

Systole

The filling phases during diastole result in a large amount of blood in the ventricles, causing the pressure in the ventricles to be higher than in the atria. This causes the AV valves

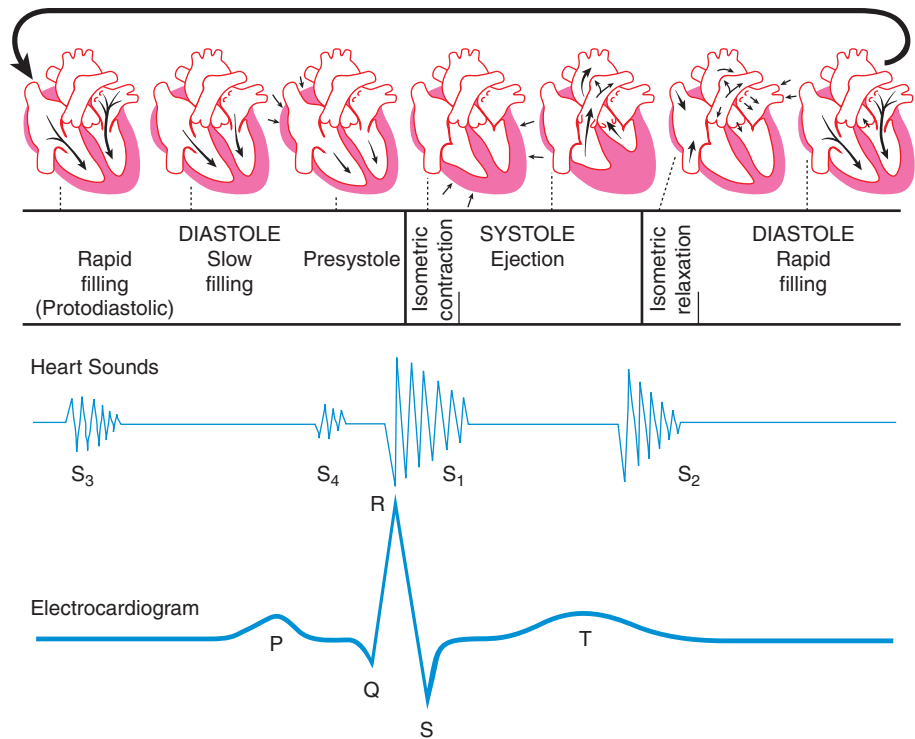


Figure 18-4 The cardiac cycle consists of filling and ejection. Heart sounds S_2 , S_3 , and S_4 are associated with diastole, while S_1 is associated with systole. The electrical activity of the heart is measured throughout diastole and systole by electrocardiography.

(mitral and tricuspid) to shut. Closure of the AV valves produces the first heart sound (S_1), which is the beginning of systole. This valve closure also prevents blood from flowing backward (a process known as *regurgitation*) into the atria during ventricular contraction.

At this point in systole, all four valves are closed and the ventricles contract (isometric contraction). There is now high pressure inside the ventricles, causing the aortic valve to open on the left side of the heart and the pulmonic valve to open on the right side of the heart. Blood is ejected rapidly through these valves. With ventricular emptying, the ventricular pressure falls and the semilunar valves close. This closure produces the second heart sound (S_2), which signals the end of systole. After closure of the semilunar valves, the ventricles relax. Atrial pressure is now higher than the ventricular pressure, causing the AV valves to open and diastolic filling to begin again.

HEART SOUNDS

Heart sounds are produced by valve closure, as described above. The opening of valves is silent. Normal heart sounds, characterized as “lub dubb” (S_1 and S_2), and, occasionally, extra heart sounds and murmurs can be auscultated with a stethoscope over the precordium, the area of the anterior chest overlying the heart and great vessels.

Normal Heart Sounds

The first heart sound (S_1) is the result of closure of the AV valves: the mitral and tricuspid valves. As mentioned previously, S_1 correlates with the beginning of systole (see

Display 18-2 for more information about S_1 and variations of S_1). S_1 (“lub”) is usually heard as one sound but may be heard as two sounds (see also Fig. 18-4). If heard as two sounds, the first component represents mitral valve closure (M_1), and the second component represents tricuspid closure (T_1). M_1 occurs first because of increased pressure on the left side of the heart and because of the route of myocardial depolarization. S_1 may be heard over the entire precordium but is heard best at the apex (left MCL, fifth ICS).

The second heart sound (S_2) results from closure of the semilunar valves (aortic and pulmonic) and correlates with the beginning of diastole. S_2 (“dubb”) is also usually heard as one sound but may be heard as two sounds. If S_2 is heard as two sounds, the first component represents aortic valve closure (A_2) and the second component represents pulmonic valve closure (P_2). A_2 occurs first because of increased pressure on the left side of the heart and because of the route of myocardial depolarization. If S_2 is heard as two distinct sounds, it is called a *split* S_2 . A splitting of S_2 may be exaggerated during inspiration and disappear during expiration. S_2 is heard best at the base of the heart. See Display 18-3 for more information about variations of S_2 .

Extra Heart Sounds

S_3 and S_4 are referred to as diastolic filling sounds or extra heart sounds, which result from ventricular vibration secondary to rapid ventricular filling. If present, S_3 can be heard early in diastole, after S_2 (see Fig. 18-4). S_4 also results

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DISPLAY 18-2 UNDERSTANDING NORMAL S_1 SOUNDS AND VARIATIONS

S_1 , which is the first heart sound, is produced by the atrioventricular (AV) closing. S_1 (the “lub” portion of “lub dubb”) correlates with the beginning of systole.

The intensity of S_1 depends on the position of the mitral valve at the start of systole, the structure of the valve leaflets, and how quickly pressure rises in the ventricles. All of these factors influence the speed and amount of closure the valve experiences, which, in turn, determine the amount of sound produced.

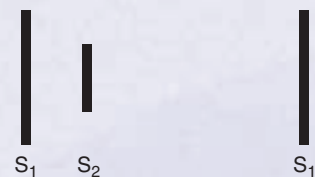
Clinical Tip: Normal variations in S_1 are heard at the base and the apex of the heart. S_1 is softer at the base and louder at the apex of the heart. An S_1 may be split along the lower left sternal border, where the tricuspid component of the sound, usually too faint to be heard, can be auscultated. A split S_1 heard over the apex may be an S_4 .

Accentuated S_1

An accentuated S_1 sound is louder than an S_2 . This occurs when the mitral valve is wide open and closes quickly. Examples include

- Hyperkinetic states in which blood velocity increases such as fever, anemia, and hyperthyroidism
- Mitral stenosis in which the leaflets are still mobile but increased ventricular pressure is needed to close the valve

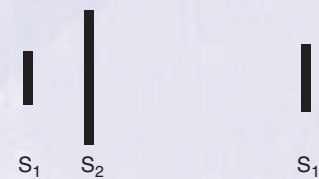
1st Cardiac Cycle Beginning of Next Cardiac Cycle



Diminished S_1

Sometimes the S_1 sound is softer than the S_2 sound. This occurs when the mitral valve is not fully open at the time of ventricular contraction and valve closing. Examples include

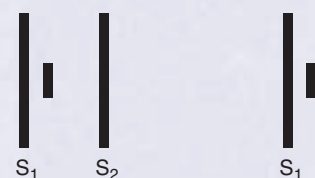
- Delayed conduction from the atria to the ventricles as in first-degree heart block, which allows the mitral valve to drift closed before ventricular contraction closes it
- Mitral insufficiency in which extreme calcification of the valve limits mobility
- Delayed or diminished ventricular contraction arising from forceful atrial contraction into a noncompliant ventricle as in severe pulmonary or systemic hypertension.



Split S_1

As named, a split S_1 occurs as a split sound. This occurs when the left and right ventricles contract at different times (asynchronous ventricular contraction). Examples include

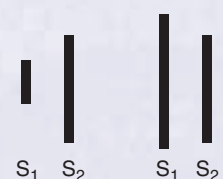
- Conduction delaying the cardiac impulse to one of the ventricles as in bundle branch block
- Ventricular ectopy in which the impulse starts in one ventricle, contracting it first, and then spreading to the second ventricle



Varying S_1

This occurs when the mitral valve is in different positions when contraction occurs. Examples include

- Rhythms in which the atria and ventricles are beating independently of each other
- Totally irregular rhythm such as atrial fibrillation



DISPLAY 18-3 VARIATIONS IN S_2

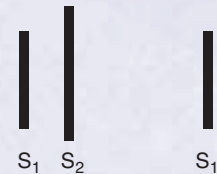
The S_2 sound depends on the closure of the aortic and the pulmonic valves. Closure of the pulmonic valve is delayed by inspiration, resulting in a split S_2 sound. The components of the split sound are referred to as A_2 (aortic valve sound) and P_2 (pulmonic valve sound). If either sound is absent, no split sounds are heard. The A_2 sound is heard best over the second right intercostal space. P_2 is normally softer than A_2 .

Accentuated S_2

An accentuated S_2 means that S_2 is louder than S_1 . This occurs in conditions in which the aortic or pulmonic valve has a higher closing pressure. Examples include

- Increased pressure in the aorta from exercise, excitement, or systemic hypertension (a booming S_2 is heard with systemic hypertension)
- Increased pressure in the pulmonary vasculature, which may occur with mitral stenosis or congestive heart failure
- Calcification of the semilunar valve in which the valve is still mobile as in pulmonic or aortic stenosis

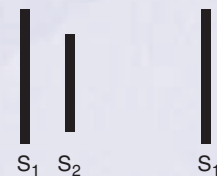
1st Cardiac Cycle Beginning of Next Cardiac Cycle



Diminished S_2

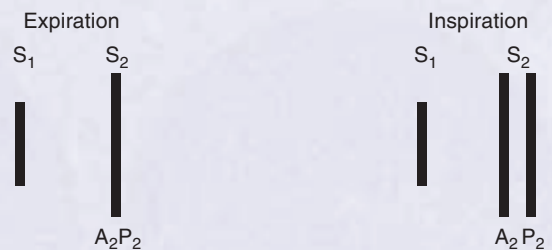
A diminished S_2 means that S_2 is softer than S_1 . This occurs in conditions in which the aortic or pulmonic valves have decreased mobility. Examples include

- Decreased systemic blood pressure, which weakens the valves, as in shock
- Aortic or pulmonic stenosis in which the valves are thickened and calcified, with decreased mobility



Normal (Physiologic) Split S_2

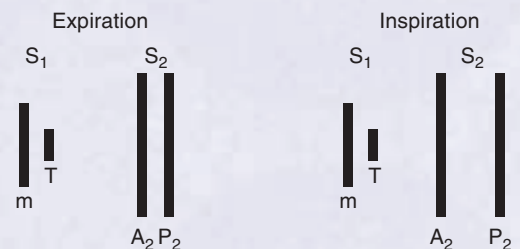
A normal split S_2 can be heard over the second or third left intercostal space. It is usually heard best during inspiration and disappears during expiration. Over the aortic area and apex, the pulmonic component of S_2 is usually too faint to be heard and S_2 is a single sound resulting from aortic valve closure. In some patients, S_2 may not become single on expiration unless the patient sits up. Splitting that does not disappear during expiration is suggestive of heart disease.



Wide Split S_2

This is an increase in the usual splitting that persists throughout the entire respiratory cycle and widens on expiration. It occurs when there is delayed electrical activation of the right ventricle. Example includes

- Right bundle branch block, which delays pulmonic valve closing

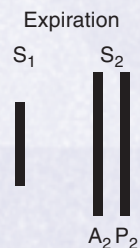
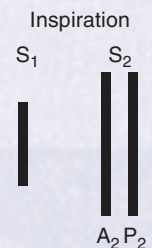


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DISPLAY 18-3 VARIATIONS IN S_2 Continued**Fixed Split S_2**

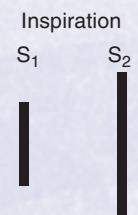
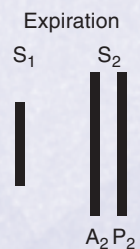
This is a wide splitting that does not vary with respiration. It occurs when there is delayed closure of one of the valves. Example includes

- Atrial septal defect and right ventricular failure, which delay pulmonic valve closing

1st Cardiac Cycle**Beginning of Next Cardiac Cycle****Reversed Split S_2**

This is a split S_2 that appears on expiration and disappears on inspiration—also known as paradoxical split. It occurs when closure of the aortic valve is abnormally delayed, causing A_2 to follow P_2 in expiration. Normal inspiratory delay of P_2 makes the split disappear during inspiration. Example includes

- Left bundle branch block

**Accentuated A_2**

An accentuated A_2 is loud over the right, second intercostal space. This occurs with increased pressure as in systemic hypertension and aortic root dilation because of the closer position of the aortic valve to the chest wall.

Diminished A_2

A diminished A_2 is soft or absent over the right, second intercostal space. This occurs with immobility of the aortic valve in calcific aortic stenosis.

Accentuated P_2

An accentuated P_2 is louder than or equal to an A_2 sound. This occurs with pulmonary hypertension, dilated pulmonary artery, and atrial septal defect. A wide split S_2 , heard even at the apex, indicates an accentuated P_2 .

Diminished P_2

A soft or absent P_2 sound occurs with an increased anteroposterior diameter of the chest (barrel chest), which is associated with aging, pulmonic stenosis, or COPD (chronic obstructive pulmonary disease).

from ventricular vibration but, contrary to S_3 , the vibration is secondary to ventricular resistance (noncompliance) during atrial contraction. If present, S_4 can be heard late in diastole, just before S_1 (see Fig. 18-4). S_3 is often termed *ventricular gallop*, and S_4 is called *atrial gallop*. Extra heart sounds are described further in the Physical Assessment section of the text and in Display 18-4.

Murmurs

Blood normally flows silently through the heart. There are conditions, however, that can create turbulent blood flow in which a swooshing or blowing sound may be auscultated over the precordium. Conditions that contribute to turbulent blood flow include (1) increased blood velocity,

DISPLAY 18-4 AUSCULTATING HEART SOUNDS

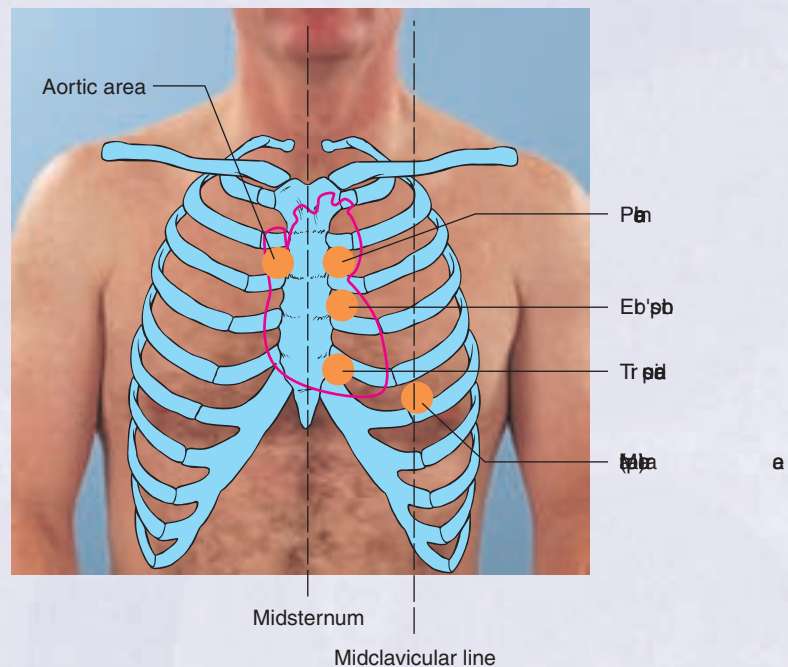
Most nurses need many hours of practice in auscultating heart sounds to assess a client's health status and interpret findings proficiently and confidently. Practitioners may be able to recognize an abnormal heart sound but may have difficulty determining what and where it is exactly. Continued exposure and experience increase one's ability to determine the exact nature and characteristics of abnormal heart sounds. An added difficulty involves palpation, particularly of the apical impulse in clients who are obese or barrel chested. These conditions increase the distance from the apex of the heart to the precordium.

Where to Auscultate

Heart sounds can be auscultated in the traditional five areas on the precordium, which is the anterior surface of the body overlying the heart and great vessels. The traditional areas include the aortic area, the pulmonic area, Erb's point, the tricuspid area, and the mitral or apical area. The four valve areas do not reflect the anatomic location of the valves. Rather, they reflect the way in which heart sounds radiate to the chest wall. Sounds always travel in the direction of blood flow. For example, sounds that originate in the tricuspid valve are usually best heard along the left lower sternal border at the fourth or fifth intercostal space.

Traditional Areas of Auscultation

- Aortic area: Second intercostal space at the right sternal border—the base of the heart
- Pulmonic area: Second or third intercostal space at the left sternal border—the base of the heart
- Erb's point: Third to fifth intercostal space at the left sternal border
- Mitral (apical): Fifth intercostal space near the left midclavicular line—the apex of the heart
- Tricuspid area: Fourth or fifth intercostal space at the left lower sternal border



Alternative Areas

In reality, the areas described above overlap extensively and sounds produced by the valves can be heard all over the precordium. Therefore, it is important to listen to more than just five specific points on the precordium. Keep the fact of overlap in mind and use the names of the chambers instead of Erb's point, mitral, and tricuspid areas when auscultating over the precordium. "Alternative" (versus the traditional) areas of auscultation overlap and are not as discrete as the traditional areas. The alternative areas are the aortic area, pulmonic area, left atrial area, right atrial area, left ventricular area, and right ventricular area.

Cover the entire precordium. As you auscultate in all areas, concentrate on systematically moving the stethoscope from left to right across the entire heart area from the base to the apex (top to bottom) or from the apex to the base (bottom to top).

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DISPLAY 18-4 AUSCULTATING HEART SOUNDS *Continued*

Alternative Areas of Auscultation

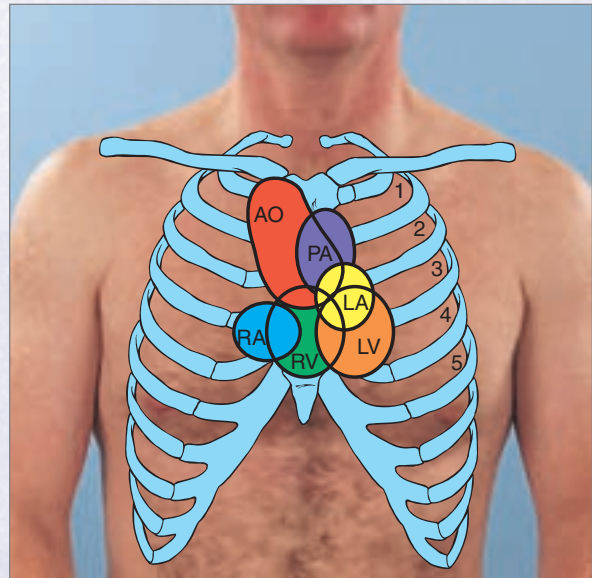
- Aortic area: Right second intercostal space to apex of heart
- Pulmonic area: second and third left intercostal spaces close to sternum but may be higher or lower
- Left atrial area: Second to fourth intercostal space at the left sternal border
- Right atrial area: Third to fifth intercostal space at the right sternal border
- Left ventricular area: Second to fifth intercostal spaces, extending from the left sternal border to the left midclavicular line
- Right ventricular area: Second to fifth intercostal spaces, centered over the sternum

How to Auscultate

Position yourself on the client's right side. The client should be supine with the upper trunk elevated 30 degrees. Use the diaphragm of the stethoscope to auscultate all areas of the precordium for high-pitched sounds. Use the bell of the stethoscope to detect (differentiate) low-pitched sounds or gallops. The diaphragm should be applied firmly to the chest, whereas the bell should be applied lightly.

Focus on one sound at a time as you auscultate each area of the precordium. Start by listening to the heart's rate and rhythm. Then identify the first and second heart sounds, concentrate on each heart sound individually, listen for extra heart sounds, listen for murmurs, and finally listen with the client in different positions.

Clinical Tip: Closing your eyes reduces visual stimuli and distractions and may enhance your ability to concentrate on auditory stimuli.



- (2) structural valve defects, (3) valve malfunction, and (4) abnormal chamber openings (e.g., septal defect).

CARDIAC OUTPUT

Cardiac output (CO) is the amount of blood pumped by the ventricles during a given period of time (usually 1 min) and is determined by the stroke volume (SV) multiplied by the heart rate (HR): $SV \times HR = CO$. The normal adult cardiac output is 5 to 6 L/min.

Stroke Volume

Stroke volume is the amount of blood pumped from the heart with each contraction (stroke volume from the left ventricle is usually 70 mL). Stroke volume is influenced by several factors:

- The degree of stretch of the heart muscle up to a critical length before contraction (preload); the greater the preload, the greater the stroke volume.

This holds true unless the heart muscle is stretched so much that it cannot contract effectively.

- The pressure against which the heart muscle has to eject blood during contraction (afterload); increased afterload results in decreased stroke volume.
- Synergy of contraction (i.e., the uniform, synchronized contraction of the myocardium); conditions that cause an asynchronous contraction decrease stroke volume.
- Compliance or distensibility of the ventricles; decreased compliance decreases stroke volume.
- Contractility or the force of contractions of the myocardium under given loading conditions; increased contractility increases stroke volume.

Although cardiac muscle has an innate pattern of contractility, cardiac activity is also mediated by the autonomic nervous system to respond to changing needs. The sympathetic impulses increase heart rate and, therefore, cardiac output. The parasympathetic impulses, which travel

to the heart by the vagus nerve, decrease the heart rate and, therefore, decrease cardiac output.

NECK VESSELS

Assessment of the cardiovascular system includes evaluation of the vessels of the neck: the carotid artery and the jugular veins (Fig. 18-5). Assessment of the pulses of these vessels reflects the integrity of the heart muscle.

Carotid Artery Pulse

The right and left common carotid arteries extend from the brachiocephalic trunk and the aortic arch and are located in the groove between the trachea and the right and left sternocleidomastoid muscles. Slightly below the mandible, each bifurcates into an internal and external carotid artery. They supply the neck and head, including the brain, with oxygenated blood. The carotid artery pulse is a centrally located arterial pulse. Because it is close to the heart, the pressure wave pulsation coincides closely with ventricular systole. The carotid arterial pulse is good for assessing amplitude and contour of the pulse wave. The pulse should normally have a smooth, rapid upstroke that occurs in early systole and a more gradual downstroke.

Jugular Venous Pulse and Pressure

There are two sets of jugular veins: internal and external. The internal jugular veins lie deep and medial to the sternocleidomastoid muscle. The external jugular veins are

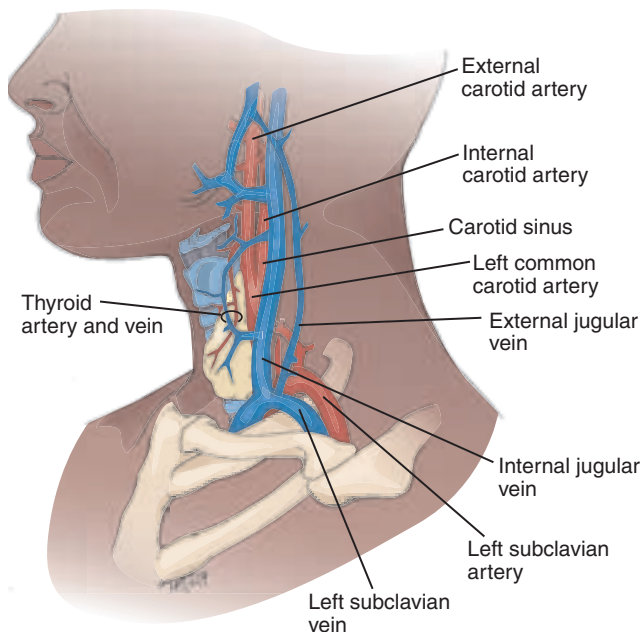


Figure 18-5 Major neck vessels, including the carotid arteries and jugular veins.

more superficial; they lie lateral to the sternocleidomastoid muscle and above the clavicle. The jugular veins return blood to the heart from the head and neck by way of the superior vena cava.

Assessment of the jugular venous pulse is important for determining the hemodynamics of the right side of the heart. The level of the jugular venous pressure reflects right atrial (central venous) pressure and, usually, right ventricular diastolic filling pressure. Right-sided heart failure raises pressure and volume, thus raising jugular venous pressure.

Decreased jugular venous pressure occurs with reduced left ventricular output or reduced blood volume. The right internal jugular vein is most directly connected to the right atrium and provides the best assessment of pressure changes. Components of the jugular venous pulse follow:

- a wave—reflects rise in atrial pressure that occurs with atrial contraction
- x descent—reflects right atrial relaxation and descent of the atrial floor during ventricular systole
- v wave—reflects right atrial filling, increased volume, and increased atrial pressure
- y descent—reflects right atrial emptying into the right ventricle and decreased atrial pressure

Figure 18-6 illustrates the jugular venous pulse.

HEALTH ASSESSMENT

COLLECTING SUBJECTIVE DATA: THE NURSING HEALTH HISTORY

Subjective data collected about the heart and neck vessels helps the nurse to identify abnormal conditions that may affect the client's ability to perform activities of daily living and to fulfill his role and responsibilities. Data collection

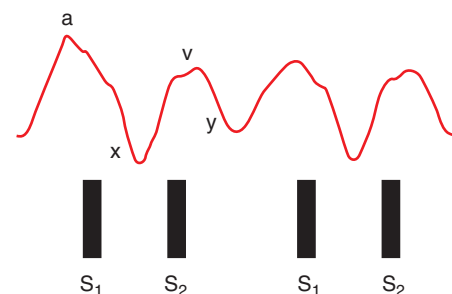


Figure 18-6 Jugular venous pulse wave reflects pressure levels in the heart.

also provides information on the client's risk for cardiovascular disease and helps to identify area where health education is needed. The client may not be aware of the significant role that health promotion activities can play in preventing cardiovascular disease.

When compiling the nursing history of current complaints or symptoms, personal and family history, and lifestyle and health practices, remember to thoroughly explore signs and symptoms that the client brings to your attention either intentionally or inadvertently.

HISTORY OF PRESENT HEALTH CONCERN

Use the **COLDSPA** mnemonic as a guideline for information to collect. In addition, the following questions help elicit important information.

C.O.L.D.S.P.A

Character: Describe the sign or symptom. How does it feel, look, sound, smell, and so forth?

Onset: When did it begin?

Location: Where is it? Does it radiate?

Duration: How long does it last? Does it recur?

Severity: How bad is it?

Pattern: What makes it better? What makes it worse?

Associated Factors: What other symptoms occur with it?

QUESTION

RATIONALE

Chest Pain and Palpitations

Do you experience chest pain? When did it start? Describe the type of pain, location, radiation, duration, and how often you experience the pain. Rate the pain on a scale of 0 to 10, with 10 being the worst possible pain. Does activity make the pain worse? Did you have perspiration (diaphoresis) with the chest pain?

Chest pain can be cardiac, pulmonary, muscular, or gastrointestinal in origin. Angina (cardiac chest pain) is usually described as a sensation of squeezing around the heart; a steady, severe pain; and a sense of pressure. It may radiate to the left shoulder and down the left arm or to the jaw. Diaphoresis and pain worsened by activity are usually related to cardiac chest pain.

Do you experience palpitations?

Palpitations may occur with an abnormality of the heart's conduction system or during the heart's attempt to increase cardiac output by increasing the heart rate. Palpitations may cause the client to feel anxious.

Other Symptoms

Do you tire easily? Do you experience fatigue? Describe when the fatigue started. Was it sudden or gradual? Do you notice it at any particular time of day?

Fatigue may result from compromised cardiac output. Fatigue related to decreased cardiac output is worse in the evening or as the day progresses.

Do you have difficulty breathing or shortness of breath (dyspnea)?

Dyspnea may result from congestive heart failure, pulmonary disorders, coronary artery disease, myocardial ischemia, and myocardial infarction. Dyspnea may occur at rest, during sleep, or with mild, moderate, or extreme exertion.

Do you wake up at night with an urgent need to urinate (nocturia)? How many times a night?

Increased renal perfusion during periods of rest or recumbency may cause nocturia. Decreased frequency may be related to decreased cardiac output.

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QUESTION <i>Continued</i>	RATIONALE <i>Continued</i>
Do you experience dizziness?	Dizziness may indicate decreased blood flow to the brain due to myocardial damage; however, there are several other causes for dizziness such as inner ear syndromes, decreased cerebral circulation, and hypotension. Dizziness may put the client at risk for falls.
Do you experience swelling (edema) in your feet, ankles, or legs?	Edema of the lower extremities may occur as a result of heart failure.
Do you have frequent heart burn? When does it occur? What relieves it? How often do you experience it?	Cardiac pain may be overlooked or misinterpreted as gastrointestinal problems. Gastrointestinal pain may occur after meals and is relieved with antacids, whereas cardiac pain may occur anytime, is not relieved with antacids, and worsens with activity.

●●➤ PAST HEALTH HISTORY

QUESTION	RATIONALE
Have you been diagnosed with a heart defect or a murmur?	Congenital or acquired defects affect the heart's ability to pump, decreasing the oxygen supply to the tissues.
Have you ever had rheumatic fever?	Approximately 40% of people with rheumatic fever develop rheumatic carditis. Rheumatic carditis develops after exposure to group A beta-hemolytic streptococci and results in inflammation of all layers of the heart, impairing contraction and valvular function.
Have you ever had heart surgery or cardiac balloon interventions?	Previous heart surgery may change the heart sounds heard during auscultation. Surgery and cardiac balloon interventions indicate prior cardiac compromise.
Have you ever had an electrocardiogram (ECG)? When was the last one performed? Do you know the results?	A prior ECG allows the health care team to evaluate for any changes in cardiac conduction or previous myocardial infarction.
Have you ever had a blood test called a lipid profile? Based on your last test, do you know what your cholesterol levels were?	Dyslipidemia presents the greatest risk for the developing coronary artery disease. Elevated cholesterol levels have been linked to the development of atherosclerosis (Libby, Schoenbeck, Mach, Selwyn & Ganz, 1998).
Do you take medications or use other treatments for heart disease? How often do you take them? Why do you take them?	Clients may have medications prescribed for heart disease but may not take them regularly. Clients may skip taking their diuretics because of having to urinate frequently. Beta-blockers may be omitted because of the adverse effects on sexual energy. Education about medications may be needed.

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QUESTION *Continued***RATIONALE** *Continued*

Do you monitor your own heart rate or blood pressure?

Self-monitoring of heart rate or blood pressure is recommended if the client is taking cardiotoxic or antihypertensive medications respectively. A demonstration is necessary to ensure appropriate technique.

●●► FAMILY HISTORY

QUESTION**RATIONALE**

Is there a history of hypertension, myocardial infarction (MI), coronary heart disease (CHD), elevated cholesterol levels, or diabetes mellitus (DM) in your family?

A genetic predisposition to these risk factors increases a client's chance for development of heart disease.

●●► LIFESTYLE AND HEALTH PRACTICES

QUESTION**RATIONALE**

Do you smoke? How many packs of cigarettes per day and for how many years?

Cigarette smoking greatly increases the risk of heart disease (see Risk Factors—Coronary Heart Disease).

What type of stress do you have in your life? How do you cope with it?

Stress has been identified as a possible risk factor for heart disease.

Describe what you usually eat in a 24-hour period.

An elevated cholesterol level increases the chance of fatty plaque formation in the coronary vessels.

How much alcohol do you consume each day/week?

Excessive intake of alcohol has been linked to hypertension.

Do you exercise? What type of exercise and how often?

A sedentary lifestyle is a known modifiable risk factor contributing to heart disease. Aerobic exercise three times per week for 30 min is more beneficial than anaerobic exercise or sporadic exercise in preventing heart disease.

Describe your daily activities. How are they different from your routine 5 or 10 years ago? Does fatigue, chest pain, or shortness of breath limit your ability to perform daily activities? Describe. Are you able to care for yourself?

Heart disease may impede the ability to perform daily activities. Exertional dyspnea or fatigue may indicate heart failure. An inability to complete activities of daily living may necessitate a referral for home care.

Has your heart disease had any effect on your sexual activity?

Many clients with heart disease are afraid that sexual activity will precipitate chest pain. If the client can walk one block or climb two flights of stairs without experiencing symptoms, it is generally acceptable for the

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QUESTION *Continued*

How many pillows do you use to sleep at night? Do you get up to urinate during the night? Do you feel rested in the morning?

How important is having a healthy heart to your ability to feel good about yourself and your appearance? What fears about heart disease do you have?

RATIONALE *Continued*

client to engage in sexual intercourse. Nitroglycerin can be taken before intercourse as a prophylactic for chest pain. In addition, the side-lying position for sexual intercourse may reduce the workload on the heart.

If heart function is compromised, cardiac output to the kidneys is reduced during episodes of activity. At rest, cardiac output increases, as does glomerular filtration and urinary output. Orthopnea (the inability to breathe while supine) and nocturia may indicate heart failure. In addition, these two conditions may also impede the ability to get adequate rest.

A person's feeling of self-worth may depend on his or her ability to perform usual daily activities and fulfill his or her usual roles.

RISK FACTORS

CORONARY HEART DISEASE

Overview

The World Health Organization (WHO) reports that global death rates in 2002 from cardiovascular diseases numbered 16.7 million/year, of which 7.2 million were from coronary heart disease (CHD). According to the American Heart Association (2001), CHD is the single largest killer of Americans, both men and women. In 1998, a total of 459,841 deaths in the United States were from CHD. Moreover, about 12.4 million people alive today have a history of heart attack, chest pain, or both. The rates are declining, however. There was a 28.4% decline in CHD deaths between 1988 and 1998. However, in 2001 about 1.1 million Americans had a new or recurrent coronary attack, with more than 40% dying as a result. Of those who died, 85% were age 65 or older and 80% of deaths in those under age 65 occurred during the first attack. The lifetime risk of developing CHD after age 40 is 49% for men and 32% for women. About 25% of men and 38% of women will die within 1 year after an initial recognized heart attack.

Risk Factors (AHA, 2001; WHO, 2004; except as noted)

- Age: Male over age 45; female over age 55 (postmenopausal or ovaries removed and not on estrogen replacement therapy)
- Family history: Father or brother had heart attack before age 55; mother or sister before age 65; close relative had stroke
- Cigarette smoking or exposure to second-hand smoke

Risk Reduction Teaching Tips

Young Clients (Misra, 2000)

- Learn about heart and related diseases.
- Maintain ideal body weight.
- Exercise regularly.
- Avoid smoking and chewing tobacco.
- Eat a balanced diet.

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Risk Factors (AHA, 2001; WHO, 2004; except as noted)

- Cholesterol or high-density lipoprotein (HDL) levels: Total cholesterol >240 mg/dL; HDL <35 mg/dL
- Blood pressure above 140/90
- Limited physical activity: Fewer than 30 minutes of moderate activity most days
- Body weight: 20 or more pounds overweight; upper body adiposity (Azevedo, Ramos, vonHafe & Barros, 1999)
- Diabetes or fasting blood glucose level ≥ 126 mg/dL
- Dietary intake low in antioxidants, especially fruit (Eichholzer, Luthy, Gutzwiller & Stahelin, 2001)
- Low-grade systemic infection/inflammation (elevated C-reactive protein; Rifai & Ridker, 2001)
- Low birth weight (Leeson, Kattenhorn, Morley, Lucas & Deanfield, 2001)
- Stress: Psychological/emotional or physical stress; family relationship stresses; burnout; and daily hassles, especially in women (Hallmen, Burell, Setterlind, Oden & Lisspirs, 2001)



Risk Reduction Teaching Tips

Adult Clients

- Have blood pressure checked regularly.
- Exercise regularly (three to five times per week for 20 to 30 minutes)
- Avoid smoking or stop smoking cigarettes; avoid second-hand smoke.
- Eat a well-balanced diet: low in cholesterol and saturated fats, high in fruits and vegetables with moderate amounts of salt and sugar.
- Have regular medical checkups.
- Maintain a healthy weight; lose weight if overweight or obese.
- Learn about heart disease and the signs of heart attack.
 - Uncomfortable pressure, fullness, squeezing, or pain in the center of the chest that lasts for more than a few minutes
 - Pain spreading to the shoulders, neck, or arms
 - Chest discomfort with lightheadedness, fainting, sweating, nausea, or shortness of breath

Postmenopausal Clients

- Learn about estrogen replacement therapy.
- Maintain controlled blood glucose levels.
- Take antihypertensive medications if prescribed.
- Minimize stress levels whenever possible.



Cultural Considerations

According to the AHA (2001), among American adults age 20 and older, the estimated age-adjusted (2000 standard) prevalence of CHD is 6.9% for non-Hispanic white men and 5.4% for women; 7.1% for non-Hispanic black men and 9% for women; 7.2% for Mexican-American men and 6.8% for women. For heart attack (myocardial infarction), the prevalence is 5.2% for non-Hispanic white men and 2% for women; 4.3% for non-Hispanic black men and 3.3% for women; and 4.1% for Mexican-American men and 1.9% for women.

Racial differences in incidence of CHD have both genetic and environmental components. For example, African Americans have higher HDL levels but higher lifestyle risk factors than white Americans. However, both groups have a similar CHD frequency. Blacks also have been noted to have higher rates of hypertension than whites, and Hispanics have lower rates than either of the other two groups in the United States. How much of the variation is due to genetic and how much to cultural and lifestyle differences is not known. Overfield (1995) notes that hypertension in blacks is clinically and biochemically different from that in whites. Blood pressure correlates with darker skin color, which may be due to the role

of melanin as a reservoir for heavy metals such as sodium. Comparisons of blacks and whites of higher education and socioeconomic levels indicate little difference in hypertension rates (Overfield, 1995). However, rates for blacks remain high and for black women the rates are rising. Compared with U.S. white women, hypertension in U.S. black women has a higher incidence, earlier onset, and longer duration and results in higher mortality, which remains among the highest rate in the industrialized world (Gillum, 1996).

Teaching is needed for individuals, families, and communities because of the widespread nature of CHD in developed countries. Immigrants need instruction on avoiding lifestyle changes that can increase their risks. It has been suggested that the best method for preventing CHD in the United States is a population-based approach, especially educating children to adopt and maintain healthy lifestyles (Berenson & Pickoff, 1995).

COLLECTING OBJECTIVE DATA: PHYSICAL EXAMINATION

A major purpose of this examination is to identify any sign of heart disease and thereby initiate early referral and treatment. Since 1900, cardiovascular disease (CVD) has been the number one killer in the United States every year except 1918 and more than 2,600 Americans die of CVD every day, for an average of one death every 33 seconds (AHA, 2000). Some 60.8 million Americans have one or more types of CVD (AHA, 2001). The National Cholesterol Education Project (NCEP) recommends that all adults age 20 years or older have their total cholesterol and HDL cholesterol levels checked at least once every 5 years.

Assessment of the heart and neck vessels is an essential part of the total cardiovascular examination. It is important to remember that additional data gathered during assessment of the blood pressure, skin, nails, head, thorax and lungs, and peripheral pulses all play a part in the complete cardiovascular assessment. These additional assessment areas are covered in Chapters 7, 8, 9, 12, and 19.

The part of the cardiovascular assessment covered in this chapter involves inspection, palpation, and auscultation of the neck and anterior chest area (precordium). Inspection is a fairly easy skill to acquire. However, auscultation requires a lot of practice to develop expert proficiency. Novice practitioners may be able to recognize an abnormal heart sound but may have difficulty determining what and where it is exactly. Continued exposure and experience increase the practitioner's ability to determine the exact nature and characteristics of abnormal heart sounds. In addition, it may be difficult to palpate the apical impulse in clients who are obese or barrel chested because these conditions increase the distance from the apex of the heart to the precordium.

Heart and neck vessel assessment skills are useful to the nurse in all types of health care settings, including acute, clinical, and home health care.

Clinical Tip: When performing a total body system examination (see Chapter 26), it is often convenient to assess the heart and neck vessels immediately after assessment of the thorax and lungs.

Preparing the Client

Prepare clients for the examination by explaining that they will need to expose the anterior chest. Female clients may keep their breasts covered and may simply hold the left breast out of the way when necessary. Explain to the client that she will need to assume several different positions for this examination. Auscultation and palpation of the neck vessels and inspection, palpation, and auscultation of the precordium are performed with the client in the supine position with the head elevated to about 30 degrees. The client will be asked to assume a left lateral position for palpation of the apical impulse if the examiner is having trouble locating the pulse with the client in the supine position. In addition, the client will be asked to assume a left lateral and a sitting-up and leaning-forward position so the examiner can auscultate for the presence of any abnormal heart sounds. These positions may bring out an abnormal sound not detected with the client in the supine position. Make sure you explain to the client that you will be listening to the heart in a number of places and that this does not necessarily mean that anything is wrong. Provide the client with as much modesty as possible during the examination, describe the steps of the examination, and answer any questions the client may have. These actions will help to ease any client anxiety.

Equipment

- Stethoscope with a bell and diaphragm
- Small pillow
- Penlight or movable examination light
- Watch with second hand
- Centimeter rulers (two)

Physical Assessment

Remember these key points during examination:

- Understand the anatomy and function of the heart and major coronary vessels to identify and interpret heart sounds and electrocardiograms accurately.
- Know normal variations of the cardiovascular system in the elderly client.

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PHYSICAL ASSESSMENT

Assessment Procedure

Normal Findings


Abnormal Findings

Neck Vessels

Inspection

Observe the jugular venous pulse.

Inspect the jugular venous pulse by standing on the right side of the client. The client should be in a supine position with the torso elevated 30 to 45 degrees. Make sure the head and torso are on the same plane. Ask the client to turn the head slightly to the left. Shine a tangential light source onto the neck to increase visualization of pulsations as well as shadows. Next inspect the suprasternal notch or the area around the clavicles for pulsations of the internal jugular veins.

 **Clinical Tip:** Be careful not to confuse pulsations of the carotid arteries with pulsations of the internal jugular veins.

Evaluate jugular venous pressure.

Evaluate jugular venous pressure by watching for distention of the jugular vein. It is normal for the jugular veins to be visible when the client is supine; to evaluate jugular vein distention, position the client in a supine position with the head of the bed elevated 30, 45, 60, and 90 degrees. At each increase of the elevation, have the client's head turned slightly away from the side being evaluated. Using tangential lighting, observe for distention, protrusion, or bulging.

Note: In acute care settings, invasive cardiac monitors (pulmonary artery catheters) are used for precisely measuring pressures.

The jugular venous pulse is not normally visible with the client sitting upright. This position fully distends the vein, and pulsations may or may not be discernible.

The jugular vein should not be distended, bulging, or protruding at 45 degrees or greater.

Fully distended jugular veins with the client's torso elevated more than 45 degrees indicate increased central venous pressure that may be the result of right ventricular failure, pulmonary hypertension, pulmonary emboli, or cardiac tamponade.

Distention, bulging, or protrusion at 45, 60, or 90 degrees may indicate right-sided heart failure. Document at which positions (45, 60, and/or 90 degrees) you observe distention.

Clients with obstructive pulmonary disease may have elevated venous pressure only during expiration.

An inspiratory increase in venous pressure, called Kussmaul's sign, may occur in clients with severe constrictive pericarditis.


continued

Assessment Procedure	Normal Findings	Abnormal Findings
<p>Auscultation and Palpation</p> <p>Auscultate the carotid arteries. Auscultate the carotid arteries if the client is middle-aged or older or if you suspect cardiovascular disease. Place the bell of the stethoscope over the carotid artery and ask the client to hold his or her breath for a moment so breath sounds do not conceal any vascular sounds (Fig. 18-7).</p> <p>Clinical Tip: Always auscultate the carotid arteries before palpating because palpation may increase or slow the heart rate, therefore, changing the strength of the carotid impulse heard.</p> <p>Palpate the carotid arteries. Palpate each carotid artery alternately by placing the pads of the index and middle fingers medial to the sternocleidomastoid muscle on the neck (Fig. 18-8). Note amplitude and contour of the pulse, elasticity of the artery, and any thrills.</p> <p>Clinical Tip: If you detect occlusion during auscultation, palpate very</p>	<p>No blowing or swishing or other sounds are heard.</p> <p>Pulses are equally strong; a 2+ or normal with no variation in strength from beat to beat. Contour is normally smooth and rapid on the upstroke and slower and less abrupt on the downstroke. Arteries are elastic and no thrills are noted.</p> <p>The strength of the pulse is evaluated on a scale from 0 to 4 as follows:</p> <p>Pulse Amplitude Scale 0 = Absent 1+ = Weak 2+ = Normal 3+ = Increased 4+ = Bounding</p>	<p>A bruit, a blowing or swishing sound caused by turbulent blood flow through a narrowed vessel, is indicative of occlusive arterial disease. However, if the artery is more than two-thirds occluded, a bruit may not be heard.</p> <p>Pulse inequality may indicate arterial constriction or occlusion in one carotid.</p> <p>Weak pulses may indicate hypovolemia, shock, or decreased cardiac output.</p> <p>A bounding, firm pulse may indicate hypervolemia or increased cardiac output.</p> <p>Variations in strength from beat to beat or with respiration are abnormal and may indicate a variety of problems (Abnormal Findings 18-1).</p> <p>A delayed upstroke may indicate aortic stenosis.</p>



Figure 18-7 Auscultating the carotid artery.
(© B. Proud.)

continued

Assessment Procedure	Normal Findings	Abnormal Findings
<p>lightly to avoid blocking circulation or triggering vagal stimulation and bradycardia, hypotension, or even cardiac arrest.</p> <p>Palpate the carotid arteries individually because bilateral palpation could result in reduced cerebral blood flow.</p> <p> Be cautious with older clients because atherosclerosis may have caused obstruction and compression may easily block circulation.</p>		<p>Loss of elasticity may indicate arteriosclerosis. Thrills may indicate a narrowing of the artery.</p>

Heart (Precordium)

Inspection

Inspect pulsations. With the client in supine position with the head of the bed elevated between 30 and 45 degrees, stand on the client's right side and look for the apical impulse and any abnormal pulsations.

The apical impulse may or may not be visible. If apparent, it would be in the mitral area (left midclavicular line, fourth or fifth intercostal space). The apical impulse is a result of the left ventricle moving outward during systole.

Pulsations, which may also be called heaves or lifts, other than the apical pulsation are considered abnormal and should be evaluated. A heave or lift may occur as the result of an enlarged ventricle from an overload of work. Abnormal Findings 18-2 describes abnormal ventricular impulses.


 **Clinical Tip:** The apical impulse was originally called the point of maximal impulse (PMI). However, this term is not used any more because a maximal impulse may occur in other areas of the precordium as a result of abnormal conditions.



Figure 18-8 Palpating the carotid artery.
(© B. Proud.)

continued


Assessment Procedure	Normal Findings	Abnormal Findings
<p>Palpation</p> <p>Palpate the apical impulse. Remain on the client's right side and ask the client to remain supine. Use the palmar surfaces of your hand to palpate the apical impulse in the mitral area (fourth or fifth intercostal space at the midclavicular line) (Fig. 18-9A). After locating the pulse, use one finger pad for more accurate palpation (see Fig. 18-9B).</p> <p>Clinical Tip: If this pulsation cannot be palpated, have the client assume a left lateral position. This displaces the heart toward the left chest wall and relocates the apical impulse farther to the left.</p> <p>Palpate for abnormal pulsations. Use your palmar surfaces to palpate the apex, left sternal border, and base.</p> <p>Auscultation</p> <p>Auscultate heart rate and rhythm. Follow the guidelines given in Display 18-4. Place the diaphragm of the</p>	<p>The apical impulse is palpated in the mitral area and may be the size of a nickel (1 to 2 cm). Amplitude is usually small—like a gentle tap. The duration is brief, lasting through the first two-thirds of systole and often less. In obese clients or clients with large breasts, the apical impulse may not be palpable.</p> <p> In older clients the apical impulse may be difficult to palpate because of increased anteroposterior chest diameter.</p> <p>No pulsations or vibrations are palpated in the areas of the apex, left sternal border, or base.</p> <p>Rate should be 60 to 100 beats per minute with regular rhythm. A regularly irregular rhythm, such as</p>	<p>The apical impulse may be impossible to palpate in clients with pulmonary emphysema. If the apical impulse is larger than 1 to 2 cm, displaced, more forceful, or of longer duration, suspect cardiac enlargement.</p> <p>A thrill, which feels similar to a purring cat, or a pulsation is usually associated with a grade IV or higher murmur.</p> <p>Bradycardia (less than 60 beats/min) or tachycardia (more than 100 beats/min) may result in decreased cardiac</p>



Figure 18-9 Locate the apical impulse with the palmar surface (A), then palpate the apical impulse with the fingerpad (B). (© B. Proud.)

Assessment Procedure	Normal Findings	Abnormal Findings
<p>stethoscope at the apex and listen closely to the rate and rhythm of the apical impulse.</p>	<p>sinus arrhythmia when the heart rate increases with inspiration and decreases with expiration, may be normal in young adults. Normally the pulse rate in females is 5 to 10 beats per minute faster than in males. Pulse rates do not differ by race or age in adults (Overfield, 1995).</p>	<p>output. Clients with regular irregular rhythms (i.e., premature atrial contraction or premature ventricular contractions) and irregular rhythms (i.e., atrial fibrillation and atrial flutter with varying block) should be referred for further evaluation. These types of irregular patterns may predispose the client to decreased cardiac output, heart failure, or emboli (see Abnormal Findings 18-3).</p>
<p>If you detect an irregular rhythm, auscultate for a pulse rate deficit. This is done by palpating the radial pulse while you auscultate the apical pulse. Count for a full minute.</p>	<p>The radial and apical pulse rates should be identical.</p>	<p>A pulse deficit (difference between the apical and peripheral/radial pulses) may indicate atrial fibrillation, atrial flutter, premature ventricular contractions, and varying degrees of heart block.</p>
<p>Auscultate to identify S₁ and S₂. Auscultate the first heart sound (S₁ or “lub”) and the second heart sound (S₂ or “dubb”). Remember these two sounds make up the cardiac cycle of systole and diastole. S₁ starts systole, and S₂ starts diastole. The space, or systolic pause, between S₁ and S₂ is of short duration (thus S₁ and S₂ occur very close together), whereas the space, or diastolic pause, between S₂ and the start of another S₁ is of longer duration.</p>	<p>S₁ corresponds with each carotid pulsation and is loudest at the apex of the heart. S₂ immediately follows after S₁ and is loudest at the base of the heart.</p>	<p>See Displays 18-2 and 18-3.</p>
<p> Clinical Tip: If you are experiencing difficulty differentiating S₁ from S₂, palpate the carotid pulse: the harsh sound that occurs with the carotid pulse is S₁ (Fig. 18-10).</p>		
<p>Listen to S₁. Use the diaphragm of the stethoscope to best hear S₁ (Fig. 18-11).</p>	<p>A distinct sound is heard in each area but loudest at the apex. May become softer with inspiration. A split S₁ may be heard normally in young adults at the left lateral sternal border.</p>	<p>Accentuated, diminished, varying, or split S₁ are all abnormal findings (see Display 18-2).</p>

continued

Assessment Procedure	Normal Findings	Abnormal Findings
<p>Listen to S₂. Use the diaphragm of the stethoscope. Ask the client to breath regularly.</p> <p>Clinical Tip: Do not ask the client to hold his or her breath. Breath holding will cause any normal or abnormal split to subside.</p> <p>Auscultate for extra heart sounds. Use the diaphragm first then the bell to auscultate over the entire heart area. Note the characteristics (e.g., location, timing) of any extra sound heard. Auscultate during the systolic pause (space heard between S₁ and S₂).</p> <p>Auscultate during the diastolic pause (space heard between end of S₂ and the next S₁).</p> <p>Clinical Tip: While auscultating, keep in mind that development of a pathologic S₃ may be the earliest sign of heart failure.</p>	<p>Distinct sound is heard in each area but is loudest at the base. A split S₂ (into two distinct sounds of its components—A₂ and P₂) is normal and termed <i>physiologic splitting</i>. It is usually heard late in inspiration at the second or third left interspaces (see Display 18-3).</p> <p>Normally no sounds are heard.</p> <p>Normally no sounds are heard. A physiologic S₃ heart sound is a benign finding commonly heard at the beginning of the diastolic pause in children, adolescents, and young adults. It is rare after age 40. The physiologic S₃ usually subsides upon standing or sitting up. A physiologic S₄ heart sound may be heard near the end of diastole in well-conditioned athletes and in adults older than age 40 or 50 with no evidence of heart disease, especially after exercise.</p>	<p>Any split S₂ heard in expiration is abnormal. The abnormal split can be one of three types: wide, fixed, or reversed.</p> <p>Ejection sounds or clicks (e.g., a mid-systolic click associated with mitral valve prolapse). A friction rub may also be heard during the systolic pause. Abnormal Findings 18-4 provides a full description of the extra heart sounds (normal and abnormal) of systole and diastole.</p> <p>A pathologic S₃ (ventricular gallop) may be heard with ischemic heart disease, hyperkinetic states (e.g., anemia), or restrictive myocardial disease.</p> <p>A pathologic S₄ (atrial gallop) toward the left side of the precordium may be heard with coronary artery disease, hypertensive heart disease, cardiomyopathy, and aortic stenosis. A pathologic S₄ toward the right side of the precordium may be heard with pulmonary hypertension and pulmonic stenosis.</p> <p>S₃ and S₄ pathologic sounds together create a quadruple rhythm, which is called a <i>summation gallop</i>. Opening snaps occur early in diastole and</p>



Figure 18-10 Palpating the carotid pulse while auscultating S₁ and S₂. (© B. Proud.)

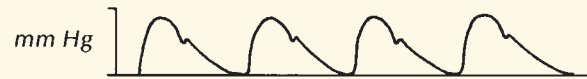


Figure 18-11 Auscultating S₁. (© B. Proud.)

Assessment Procedure	Normal Findings	Abnormal Findings
<p>Auscultate for murmurs. A murmur is a swishing sound caused by turbulent blood flow through the heart valves or great vessels. Auscultate for murmurs across the entire heart area. Use the diaphragm and the bell of the stethoscope in all areas of auscultation because murmurs have a variety of pitches. Also auscultate with the client in different positions because some murmurs occur or subside according to the client's position (see "Position Changes for Auscultation" immediately below).</p> <p>Auscultate in with the client assuming other positions. Ask the client to assume a left lateral position. Use the bell of the stethoscope and listen at the apex of the heart.</p> <p>Ask the client to sit up, lean forward, and exhale. Use the diaphragm of the stethoscope and listen over the apex and along the left sternal border (Fig. 18-12).</p>	<p>Normally no murmurs are heard. However, innocent and physiologic midsystolic murmurs may be present in a healthy heart.</p> <p>S_1 and S_2 heart sounds are normally present.</p> <p>S_1 and S_2 heart sounds are normally present.</p>	<p>indicate mitral valve stenosis. A friction rub may also be heard during the diastolic pause (see Abnormal Findings 18-4).</p> <p>Pathologic midsystolic, pansystolic, and diastolic murmurs. Abnormal Findings 18-5 describes pathologic murmurs.</p> <p>An S_3 or S_4 heart sound or a murmur of mitral stenosis that was not detected with the client in the supine position may be revealed when the client assumes the left lateral position.</p> <p>Murmur of aortic regurgitation may be detected when the client assumes this position.</p>
		
<p>Figure 18-12 Auscultating at left sternal border with client sitting up, leaning forward, and exhaling. (© B. Proud.)</p>		

ABNORMAL FINDINGS 18-1**ABNORMAL ARTERIAL PULSE AND PRESSURE WAVES**

A normal pulse, represented below, has a smooth, rounded wave with a notch on the descending slope. The pulse should feel strong and regular. The notch is not palpable. The pulse pressure (the difference between the systolic and diastolic pressure) is 30 to 40 mmHg. Pulse pressure may be measured in waveforms, which are produced when a pulmonary artery catheter is used to evaluate arterial pressure.

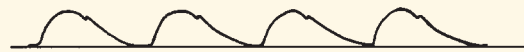


The arterial pressure waveform consists of five parts: Anacrotic limb, systolic peak, dicrotic limb, dicrotic notch, and end diastole. The initial upstroke, or anacrotic limb, occurs as blood is rapidly ejected from the ventricle through the open aortic valve into the aorta. The anacrotic limb ends at the systolic peak, the waveform's highest point. Arterial pressure falls as the blood continues into the peripheral vessels and the waveform turns downward forming the dicrotic limb. When the pressure in the ventricle is less than the pressure in the aortic root, the aortic valve closes and a small notch (dicrotic notch) appears on the waveform. The closing of the aortic notch is the beginning of diastole. The pressure continues to fall in the aortic root until it reaches its lowest point, seen on the waveform as the diastolic peak.

Changes in circulation and heart rhythm affect the pulse and its waveform. Listed below are some of the variations you may find.

Small, Weak Pulse**Characteristics**

- Diminished pulse pressure
- Weak and small on palpation
- Slow upstroke
- Prolonged systolic peak

**Causes**

- Conditions causing a decreased stroke volume
- Heart failure
- Hypovolemia
- Severe aortic stenosis
- Conditions causing increased peripheral resistance
- Hypothermia
- Severe congestive heart failure

Large, Bounding Pulse**Characteristics**

- Increased pulse pressure
- Strong and bounding on palpation
- Rapid rise and fall with a brief systolic peak

**Causes**

- Conditions that cause an increased stroke volume or decreased peripheral resistance
- Fever
- Anemia
- Hyperthyroidism
- Aortic regurgitation
- Patent ductus arteriosus
- Conditions resulting in increased stroke volume due to decreased heart rate
- Bradycardia
- Complete heart block
- Conditions resulting in decreased compliance of the aortic walls
- Aging
- Atherosclerosis

continued

ABNORMAL FINDINGS 18-1**ABNORMAL ARTERIAL PULSE AND PRESSURE WAVES** *Continued***Bisferiens Pulse****Characteristics**

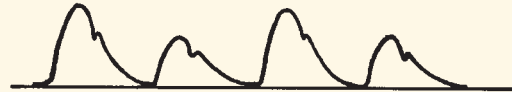
- Double systolic peak

Causes

- Pure aortic regurgitation
- Combined aortic stenosis and regurgitation
- Hypertrophic cardiomyopathy

**Pulsus Alternans****Characteristics**

- Regular rhythm
- Changes in amplitude (or strength) from beat to beat (you may need a sphygmomanometer to detect the difference)

**Causes**

- Left ventricular failure (usually accompanied by an S₃ sound on the left)

Bigeminal Pulse**Characteristics**

- Regular, irregular rhythm (one normal beat followed by a premature contraction)
- Alternates in amplitude (one strong pulse followed by a quick, weaker one)

**Causes**

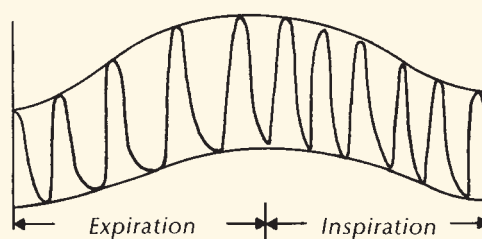
- Premature ventricular contractions

Paradoxical Pulse**Characteristics**

- Palpable decrease in pulse amplitude on quiet inspiration
- Pulse becomes stronger with expiration
- You may need a sphygmomanometer to detect the change (the systolic pressure will decrease by more than 10 mmHg during inspiration)

Causes

- Pericardial tamponade
- Constrictive pericarditis
- Obstructive lung disease

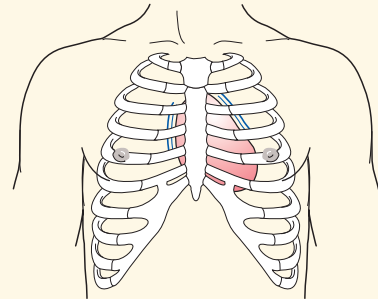


ABNORMAL FINDINGS 18-2**VENTRICULAR IMPULSES**

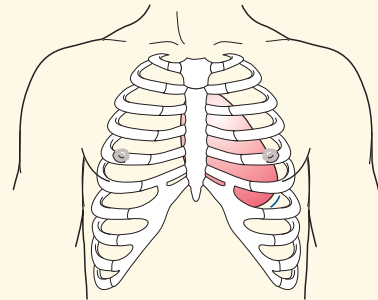
Assessment of the chest may reveal abnormalities or variations of the ventricular impulse, signs of hypertension, hypertrophy, volume overload, and pressure overload. Some of the abnormalities or variations include the following:

Lift

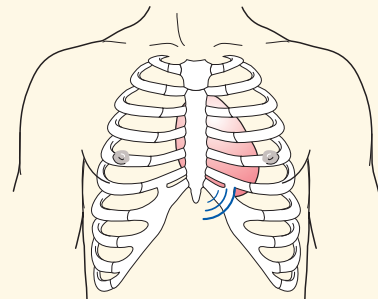
A diffuse lifting left during systole at the left lower sternal border, a lift or heave is associated with right ventricular hypertrophy caused by pulmonic valve disease, pulmonic hypertension, and chronic lung disease. You may also see retraction at the apex, from the posterior rotation of the left ventricle caused by the oversized right ventricle.

**Thrill**

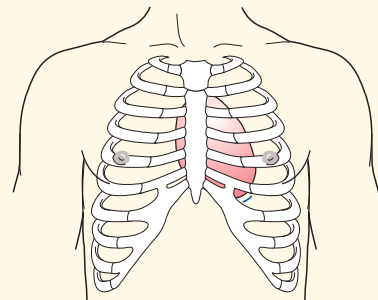
A thrill is palpated over the second and third intercostal space; a thrill may indicate severe aortic stenosis and systemic hypertension. A thrill palpated over the second and third left intercostal spaces may indicate pulmonic stenosis and pulmonic hypertension.

**Accentuated Apical Impulse**

A sign of pressure overload, the accentuated apical impulse has increased force and duration but is not usually displaced in left ventricular hypertrophy without dilatation associated with aortic stenosis or systemic hypertension.

**Laterally Displaced Apical Impulse**

A sign of volume overload, an apical impulse displaced laterally and found over a wider area is the result of ventricular hypertrophy and dilatation associated with mitral regurgitation, aortic regurgitation, or left-to-right shunts.



ABNORMAL FINDINGS 18-3

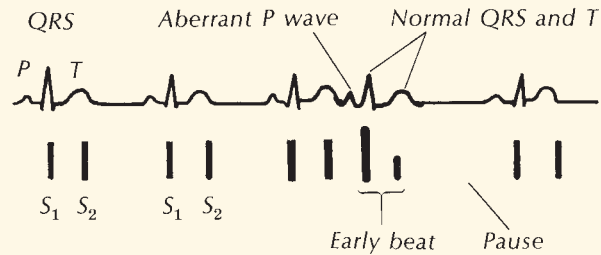
ABNORMAL HEART RHYTHMS

Changes in the heart rhythm alter the sounds heard on auscultation.

Premature Atrial or Junctional Contractions

These beats occur earlier than the next expected beat and are followed by a pause. The rhythm resumes with the next beat.

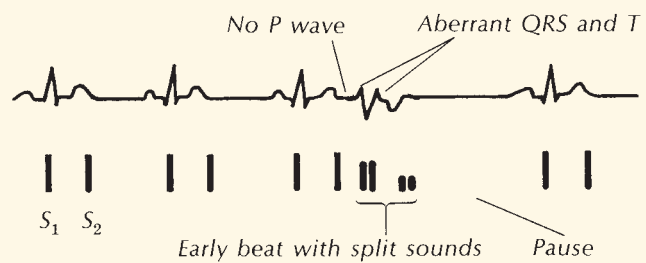
Auscultation Tip: The early beat has an S_1 of different intensity and a diminished S_2 . S_1 and S_2 are otherwise similar to normal beats.



Premature Ventricular Contractions

These beats occur earlier than the next expected beat and are followed by a pause. The rhythm resumes with the next beat.

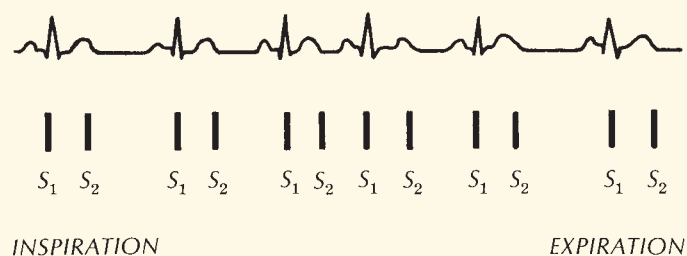
Auscultation Tip: The early beat has an S_1 of different intensity and a diminished S_2 . Both sounds are usually split.



Sinus Arrhythmia

With this dysrhythmia, the heart rate speeds up and slows down in a cycle, usually becoming faster with inhalation and slower with expiration.

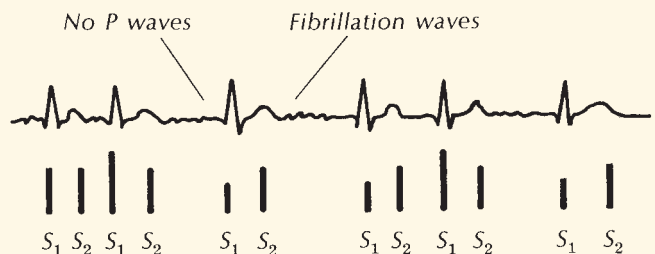
Auscultation Tip: S_1 and S_2 sounds are usually normal. The S_1 may vary with the heart rate.



Atrial Fibrillation and Atrial Flutter with Varying Ventricular Response

With this dysrhythmia, ventricular contraction occurs irregularly. At times, short runs of the irregular rhythm may appear regularly.

Auscultation Tip: S_1 varies in intensity.



ABNORMAL FINDINGS 18-4 EXTRA HEART SOUNDS

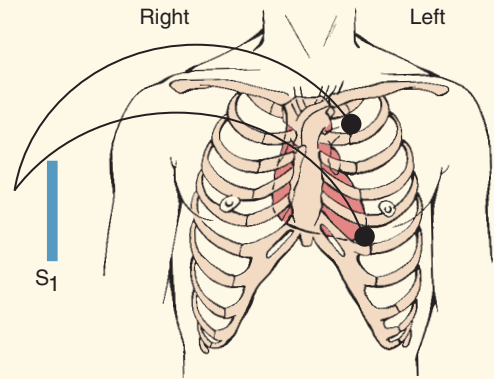
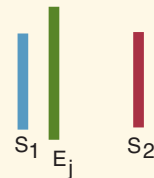
Additional heart sounds can be classified by their timing in the cardiac cycle. The presence of the sound during systole or diastole helps in its identification. Some sounds extend into both systole and diastole.

Extra Heart Sounds During Systole—Clicks

High-frequency sounds heard just after S_1 (ejection clicks) are produced by a functioning but diseased valve. clicks can occur in early or mid-to-late systole and are best heard through the diaphragm of the stethoscope.

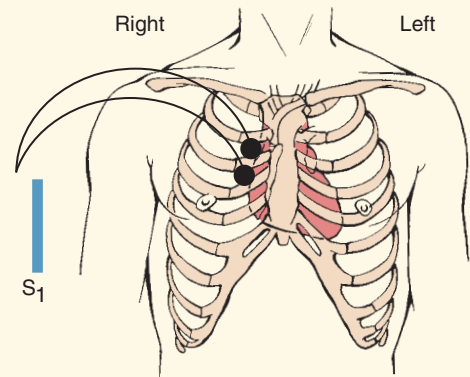
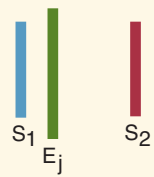
Aortic Ejection Click

Heard during early systole at the second right intercostal space and apex, the aortic ejection click occurs with the opening of the aortic valve and does not change with respiration.



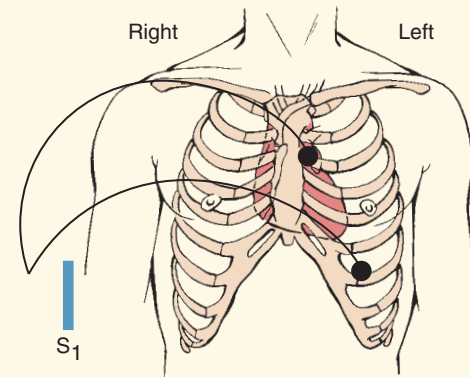
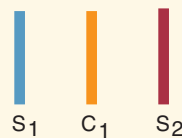
Pulmonic Ejection Click

Best heard at the second left intercostal space during early systole, the pulmonic ejection click often becomes softer with inspiration.



Midsystolic Click

Heard in middle or late systole, a midsystolic click can be heard over the mitral or apical area and is the result of mitral valve leaflet prolapse during left ventricular emptying. A late systolic murmur typically follows, indicating mild mitral regurgitation.



continued

ABNORMAL FINDINGS 18-4 EXTRA HEART SOUNDS *Continued*

Extra Heart Sounds During Diastole

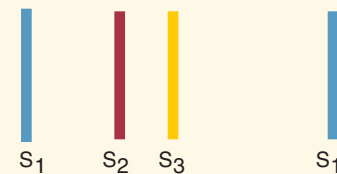
Opening Snap

Occurring in early diastole, an opening snap (OS) is heard with the opening of a stenotic or stiff mitral valve. Heard throughout the whole precordium, it does not vary with respirations. Often mistaken for a split S_2 or an S_3 , the opening snap occurs earlier in diastole and has a higher pitch than an S_3 .



S_3 (Third Heart Sound)

Also called a ventricular gallop, the S_3 has a low frequency and is heard best using the bell of the stethoscope at the apical area or lower right ventricular area of the chest with the patient in the left lateral position. The sound is often accentuated during inspiration and has the rhythm of the word “Ken-tuc-ky.” S_3 is the result of vibrations caused by the blood hitting the ventricular wall during rapid ventricular filling.



The S_3 can be a normal finding in young children, people with a high cardiac output, and in the third trimester of pregnancy. It is rarely normal in people older than age 40 years and is usually associated with decreased myocardial contractility, myocardial failure, congestive heart failure, and volume overload of the ventricle from valvular disease.

S_4 (Fourth Heart Sound)

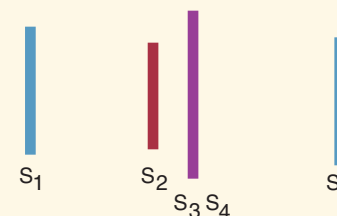
Also called an atrial gallop, S_4 is a low-frequency sound occurring at the end of diastole when the atria contract. It is caused by vibrations from blood flowing rapidly into the ventricles after atrial contraction. S_4 has the rhythm of the word “Ten-nes-see” and may increase during inspiration. It is best heard with the bell of the stethoscope over the apical area with the patient in a supine or left lateral position and is never heard in the absence of atrial contraction.



The S_4 can be a normal sound in trained athletes and some older patients, especially after exercise. However, it is usually an abnormal finding and is associated with coronary artery disease, hypertension, aortic and pulmonic stenosis, and acute myocardial infarction.

Summation Gallop

The simultaneous occurrence of S_3 and S_4 is called a summation gallop. It is brought about by rapid heart rates in which diastolic filling time is shortened, moving S_3 and S_4 closer together, resulting in one prolonged sound. Summation gallop is associated with severe congestive heart disease.

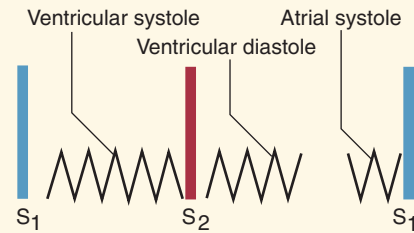


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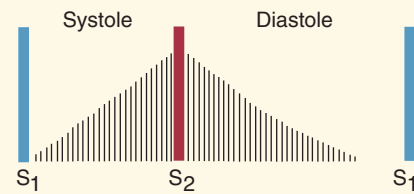
ABNORMAL FINDINGS 18-4**EXTRA HEART SOUNDS** *Continued***Extra Heart Sounds in Both Systole and Diastole****Pericardial Friction Rub**

Usually heard best in the third intercostal space to the left of the sternum, a pericardial friction rub is caused by inflammation of the pericardial sac. A high-pitched, scratchy, scraping sound, the rub may increase with exhalation and when the patient leans forward. For best results, use the diaphragm of the stethoscope and have the patient sit up, lean forward, exhale, and hold his or her breath.

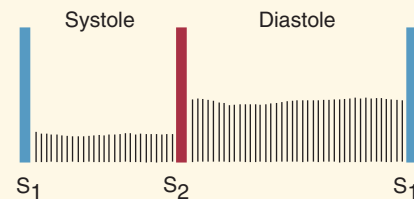
The pericardial friction rub can have up to three components: atrial systole, ventricular systole, and ventricular diastole. These components are associated with cardiac movement. The first two components are usually present. If only one component is present, the rub may be confused with a murmur. Friction rubs are commonly heard during the first week after a myocardial infarction. If a significant pericardial effusion is present, S_1 and S_2 sounds will be distant.

**Patent Ductus Arteriosus**

Patent ductus arteriosus (PDA) is a congenital anomaly that leaves an open channel between the aorta and pulmonary artery. Found over the second left intercostal space, the murmur of PDA may radiate to the left clavicle. It is classified as a continuous murmur because it extends through systole and into part of diastole. It has a medium pitch and a harsh, machinery-like sound. The murmur is loudest in late systole, obscures S_2 , fades in diastole, and often has a silent interval in late diastole.

**Venous Hum**

Common in children, a venous hum is a benign sound caused by turbulence of blood in the jugular veins. It is heard above the medial third of the clavicles, especially on the right, and may radiate to the first and second intercostal spaces. A low-pitched sound, it is often described as a humming or roaring continuous murmur without a silent interval and is loudest in diastole. A venous hum can be obliterated by putting pressure on the jugular veins.



ABNORMAL FINDINGS 18-5 HEART MURMURS

Heart murmurs are typically characterized by turbulent blood flow, which creates a swooshing or blowing sound over the precordium. When listening to the heart, be alert for this turbulence and keep the characteristics of heart murmurs in mind.

Characteristics

Heart murmurs are assessed according to various characteristics, which include timing, intensity, pitch, quality, shape or pattern, location, transmission, and ventilation and position.

Timing

A murmur can occur during systole or diastole. In addition to determining when it occurs, it is important to determine where it occurs, because a systolic murmur can be present in a healthy heart whereas a diastolic murmur always indicates heart disease. Systolic murmurs can be divided into three categories: midsystolic, pansystolic, and late systolic. Diastolic murmurs can be divided into three categories: early diastolic, mid-diastolic, and late diastolic.

Intensity

Six grades describe the intensity of a murmur.

Grade 1: Very faint, heard only after the listener has “tuned in”; may not be heard in all positions

Grade 2: Quiet but heard immediately on placing the stethoscope on the chest

Grade 3: Moderately loud

Grade 4: Loud*

Grade 5: Very loud, may be heard with a stethoscope partly off the chest*

Grade 6: May be heard with the stethoscope entirely off the chest*

Pitch

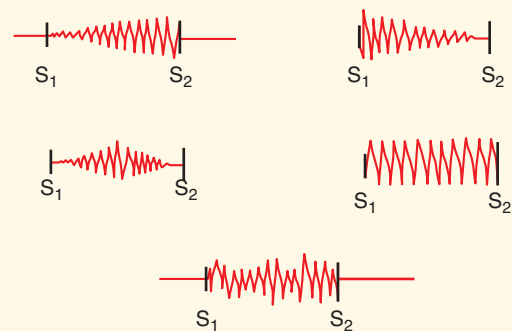
Murmurs can assume a high, medium, or low pitch.

Quality

The sound murmurs make has been described as blowing, rushing, roaring, rumbling, harsh, or musical.

Shape or Pattern

The shape of a murmur is determined by its intensity from beginning to end. There are four different categories of shape: crescendo (growing louder), decrescendo (growing softer), crescendo-decrescendo (growing louder and then growing softer), and plateau (staying the same throughout).



Location

Determine where you can best hear the murmur; this is the point where the murmur originates. Try to be as exact as possible in describing its location. Use the heart landmarks in your description (e.g., the second intercostal space at the left sternal border).

Transmission

The murmur may be felt in areas other than the point of origin. If you determine where the murmur transmits, you can determine the direction of blood flow and the intensity of the murmur.

Ventilation and Position

Determine if the murmur is affected by inspiration, expiration, or a change in body position.

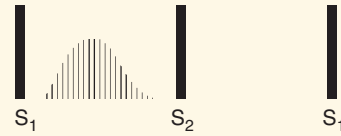
Midsystolic Murmurs

The most common type of heart murmurs, midsystolic murmurs occur during ventricular ejection and can be innocent, physiologic, or pathologic. They have a crescendo-decrescendo shape and usually peak near midsystole and stop before S₂.

continued

ABNORMAL FINDINGS 18-5**HEART MURMURS** *Continued***Innocent Murmur**

Not associated with any physical abnormality, innocent murmurs occur when the ejection of blood into the aorta is turbulent. Very common in children and young adults, they may also be heard in older people with no evidence of cardiovascular disease. A patient may have an innocent murmur and another kind of murmur.



Location: Second to fourth left intercostal spaces between the left sternal border and the apex

Radiation: Little radiation

Intensity: Grade 1 to 2

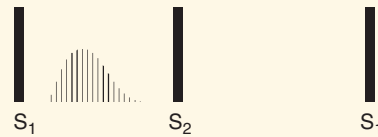
Pitch: Medium

Quality: Variable

Position: Usually disappear when the patient sits

Physiologic Murmur

Caused by a temporary increase in blood flow, a physiologic murmur can occur with anemia, pregnancy, fever, and hyperthyroidism.



Location: Second to fourth left intercostal spaces between the left sternal border and the apex

Radiation: Little radiation

Intensity: Grade 1 to 2

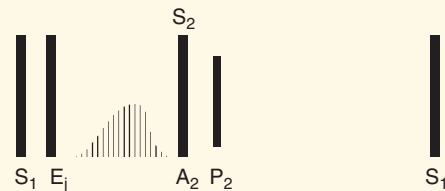
Pitch: Medium

Quality: Harsh

Murmur of Pulmonic Stenosis

A pathologic murmur, the murmur of pulmonic stenosis occurs from impeded flow across the pulmonic valve and increased right ventricular afterload. Often occurring as a congenital anomaly, the murmur is commonly found in children. Pathologic changes in flow across the valve, as in atrial septal defect, may also mimic this condition.

With severe pulmonic stenosis, the S₂ is widely split and P₂ is diminished. An early pulmonic ejection sound is also common. A right-sided S₄ may also be present, and the right ventricular impulse is often stronger and may be prolonged.



Location: Second and third intercostal spaces

Radiation: Toward the left shoulder and neck

Intensity: Soft to loud (may be associated with a thrill if loud)

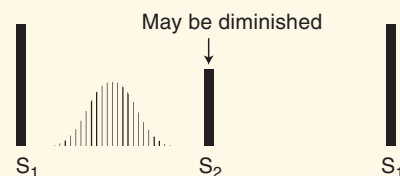
Pitch: Medium

Quality: Harsh

Position: Loudest during inspiration

Murmur of Aortic Stenosis

The murmur of aortic stenosis occurs when stenosis of the aortic valve impedes blood flow across the valve and increases left ventricular afterload. Aortic stenosis may result from a congenital anomaly, rheumatic disease, or a degenerative process. Conditions that may mimic this murmur include aortic sclerosis, a bicuspid aortic valve, a dilated aorta, or any condition that mimics the flow across the valve, such as aortic regurgitation.



continued

ABNORMAL FINDINGS 18-5

HEART MURMURS *Continued*

If valvular disease is severe, A_2 may be delayed, resulting in an unsplit S_2 or a paradoxical split S_2 . An S_4 may occur as a result of decreased left ventricular compliance. An aortic ejection sound, if present, suggests a congenital cause.

Location: Right second intercostal space

Radiation: May radiate to the neck and down the left sternal border to the apex

Intensity: Usually loud, with a thrill

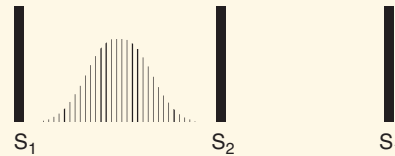
Pitch: Medium

Quality: Harsh, may be musical at the apex

Position: Heard best with the patient sitting and leaning forward, loudest during expiration

Murmur of Hypertrophic Cardiomyopathy

Caused by unusually rapid ejection of blood from the left ventricle during systole, the murmur of cardiac hypertrophy results from massive hypertrophy of the ventricular muscle. There may be a coexisting obstruction to blood flow. If there is an accompanying distortion of the mitral valve, mitral regurgitation may result. The patient may also have an S_3 and an S_4 . There may be a sustained apical impulse with two palpable components.



Location: Third and fourth left intercostal space, decreases with squatting, increases with straining down

Intensity: Variable

Pitch: Medium

Quality: Harsh

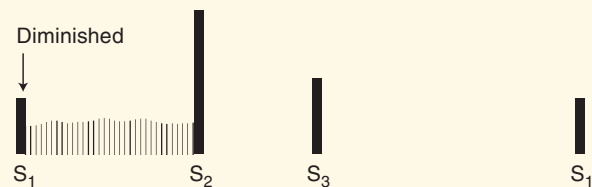
Pansystolic Murmurs

Occurring when blood flows from a chamber with high pressure to a chamber of low pressure through an orifice that should be closed, pansystolic murmurs are pathologic. Also called *holosystolic murmur*, these murmurs begin with S_1 and continue through systole to S_2 .

Murmur of Mitral Regurgitation

Occurring when the mitral valve fails to close fully in systole, the murmur of mitral regurgitation is the result of blood flowing from the left ventricle back into the left atrium. Volume overload occurs in the left ventricle, causing dilatation and hypertrophy.

The S_1 sound is often decreased, and the apical impulse is stronger and may be prolonged. Left ventricular volume overload should be suspected if an apical S_3 is heard.



Location: Apex

Radiation: To the left axilla, less often to the left sternal border

Intensity: Soft to loud, an apical thrill is associated with loud murmurs

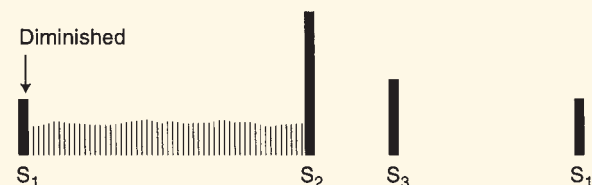
Pitch: Medium to high

Quality: Blowing

Position: Heard best with patient in the left lateral decubitus position, does not become louder with inspiration

Murmur of Tricuspid Regurgitation

Blood flowing from the right ventricle back into the right atrium over a tricuspid valve that has not fully closed causes the murmur of tricuspid regurgitation. Right ventricular failure with dilatation is the most common cause and usually results from pulmonary hypertension or left ventricular failure.



continued

ABNORMAL FINDINGS 18-5**HEART MURMURS** *Continued*

With this murmur, the right ventricular impulse is stronger and may be prolonged. There may be an S_3 along the lower left sternal border and the jugular venous pressure is often elevated with visible v waves.

Location: Lower left sternal border

Radiation: To the right of the sternum, to the xiphoid area, and sometimes to the midclavicular line; there is no radiation to the axilla

Intensity: Variable

Pitch: Medium to high

Quality: Blowing

Position: May increase slightly with inspiration

Ventricular Septal Defect

A congenital abnormality in which blood flows from the left ventricle into the right ventricle through a hole in the septum, a ventricular septal defect causes a loud murmur that obscures the A_2 sound. Other findings vary depending on the severity of the defect and any associated lesions.

Location: Third, fourth, and fifth left intercostal space

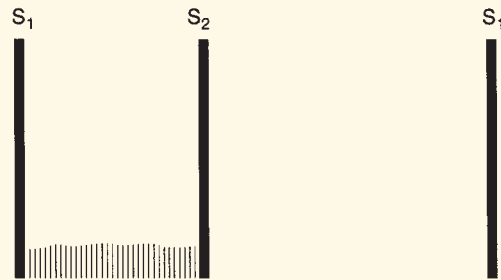
Radiation: Often wide

Intensity: Very loud, with a thrill

Pitch: High

Quality: Harsh

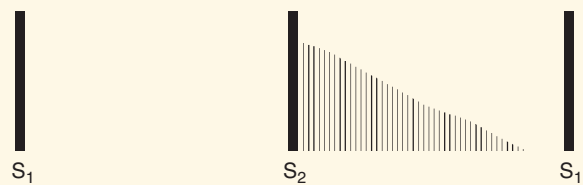
Position: Increase with exercise

**Diastolic Murmurs**

Usually indicative of heart disease, diastolic murmurs occur in two types. Early decrescendo diastolic murmurs indicate flow through an incompetent semilunar valve, commonly the aortic valve. Rumbling diastolic murmurs in mid- or late diastole indicate valve stenosis, usually of the mitral valve.

Aortic Regurgitation

Occurring when the leaflets of the aortic valve fail to close completely, the murmur of aortic regurgitation is the result of blood flowing from the aorta back into the left ventricle. This results in left ventricular volume overload. An ejection sound also may be present. Severe regurgitation should be suspected if an S_3 or S_4 is also present. The apical impulse becomes displaced downward and laterally with a widened diameter and increased duration. As the pulse pressure increases, the arterial pulses are often large and bounding.



Location: Second to fourth left intercostal space

Radiation: May radiate to the apex or left sternal border

Intensity: Grade 1 to 3

Pitch: High

Quality: Blowing, sometime mistaken for breath sounds

Position: Heard best with the patient sitting, leaning forward. Have the patient exhale and then hold his or her breath.

continued

ABNORMAL FINDINGS 18-5

HEART MURMURS *Continued***Murmur of Mitral Stenosis**

The murmur of mitral stenosis is the result of blood flow across a diseased mitral valve. Thickened, stiff, distorted leaflets are usually the result of rheumatic fever. The murmur is loud during mid-diastole as the ventricle fills rapidly, grows quiet, and becomes loud again immediately before systole, as the atria contract. In patients with atrial fibrillation, the second half of the murmur is absent because of the lack of atrial contraction.

The patient also has a loud S_1 , which may be palpable at the apex. There is often an opening snap (OS) after S_2 . P_2 becomes loud and the right ventricular impulse becomes palpable if pulmonary hypertension develops.

Location: Apex

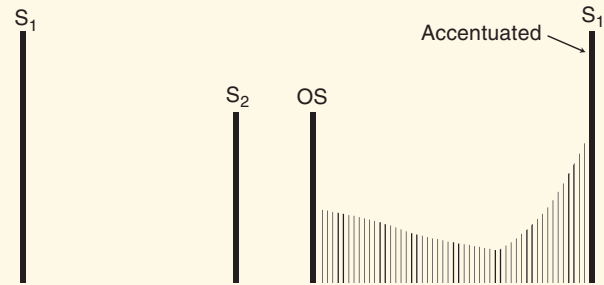
Radiation: Little or none

Intensity: Grade I to 4

Pitch: Low

Quality: Rumbling

Position: Best heard with the bell exactly on the apex and the patient turned to a left lateral position. Mild exercise and listening during exhalation also make the murmur easier to hear.



VALIDATING AND DOCUMENTING FINDINGS

Validate the heart and neck vessel assessment data that you have collected. This is necessary to verify that the data are reliable and accurate. Document the assessment data following the health care facility or agency policy.

Sample of Subjective Data



No chest pain, dyspnea, dizziness, or palpitations. No previous history of cardiovascular disease. Denies rheumatic fever. No current medications or treatments. Denies family history of hypertension, myocardial infarction, coronary heart disease, high cholesterol levels, or diabetes mellitus. Client has never had an ECG. States he needs to exercise more and consume less fat. Client does not monitor own pulse or blood pressure. Denies the use of tobacco. Sleeps 6 to 8 h per night. Feels rested after sleep. States that job can be somewhat stressful.

Sample of Objective Data



Carotid pulse equal bilaterally, 2+, elastic. No bruits auscultated over carotids. Jugular venous pulsation disappears when upright. Jugular venous pressure $\times 2$ cm. No visible pulsations, heaves, or lifts on precordium. Apical impulse palpated in the fifth ICS at the left MCL, approximately the size of a nickel, with no thrill. Apical heart rate auscultated, 70 beats/min, regular rhythm, S_1 heard best at apex, S_2 heard best at base. No S_3 or S_4 auscultated. No splitting of heart sounds, snaps, clicks, or murmurs noted.

ANALYSIS OF DATA

After collecting subjective and objective data pertaining to the heart and neck vessels, identify abnormal findings and client strengths. Then cluster the data to reveal any significant patterns or abnormalities. These data may be

used to make clinical judgments about the status of the client's heart and neck vessels.

DIAGNOSTIC REASONING: POSSIBLE CONCLUSIONS

Selected Nursing Diagnoses

The following is a listing of selected nursing diagnoses that you may identify when analyzing data for this part of the assessment.

Wellness Diagnoses

- Readiness for enhanced cardiac output
- Health-Seeking Behavior: Desired information on exercise and low-fat diet

Risk Diagnoses

- Risk for Sexual Dysfunction related to misinformation or lack of knowledge regarding sexual activity and heart disease
- Risk for Ineffective Denial related to smoking and obesity

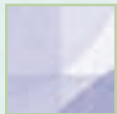
Actual Diagnoses

- Fatigue related to decreased cardiac output
- Activity Intolerance related to compromised oxygen transport secondary to heart failure
- Acute Pain: Cardiac related to an inequality between oxygen supply and demand
- Anxiety
- Ineffective Tissue Perfusion: Cardiac related to impaired circulation

Selective Collaborative Problems

After grouping the data, you may see various collaborative problems emerge. Remember that collaborative problems differ from nursing diagnoses in that they cannot be prevented by nursing interventions. However, these physiologic complications of medical conditions can be detected and monitored by the nurse. In addition, the nurse can use physician- and nurse-prescribed interventions to minimize the complications of these problems. The nurse may also have to refer the client in such situations for further treatment of the problem. Following is a list of collaborative problems that may be identified when assessing the heart

DIAGNOSTIC REASONING: CASE STUDY



The case study demonstrates how to analyze thoracic and lung assessment data for a specific client. The critical thinking exercises included in the study guide/lab manual and interactive product that complement this text also offer opportunities to analyze assessment data.

Malcolm Winchester is being admitted to the coronary care unit (CCU) with a diagnosis of hypertension, angina, R/O MI (myocardial infarction). He is a tall, slender black man who looks younger than his stated age of 45. He is in no acute distress. Mr. Winchester says, "I don't know why they brought me here—I guess my wife panicked and called 911. I have these pains all of the time, but my doc said they were from my high blood pressure. I don't hurt now."

His wife arrives, looking pale and anxious. "I don't know what to do with him. I work so hard to keep him healthy, but he goes out to that fast food place and eats burgers and fries. I'm so tired of dealing with him when he won't help himself." Mr. Winchester grins and says, "I just got to have my junk

food! That low-fat, low-salt diet my doctor put me on is impossible."

Physical assessment reveals BP 210/110 right arm reclining and 200/108 left arm reclining, pulse 88 regular and strong, respirations 16 regular and moderately shallow, temperature 36.5°C (97.7°F). His apical beat is also 88 and strong; heart sounds: S₁ and S₂ with no murmurs and clicks, but an S₄ is noted. Evaluation of the thorax reveals no heaves or visible pulsation. Neck veins are flat at >45 degrees and no carotid bruits noted. Skin is warm and dry, dark brown with pink nail beds, palms, and oral mucous membranes. Pedal pulses strong; 1+ ankle edema present.

The following concept map illustrates the diagnostic reasoning process.

1) Identify abnormal findings and client strengths

Subjective Data

- "I don't know why they brought me here."
- "I have these pains all of the time"
- Denies pain at this time
- Has to have junk food—low fat, low-salt diet "impossible"
- Wife: "Don't know what to do with him"
- Wife: "He eats hamburgers and french fries and forgets to take medication"
- Wife: "Tired of dealing with him when he won't help himself"

Objective Data

- Admitted with angina, R/O MI
- BP 210/110 right arm reclining and 200/108 left arm reclining
- S₄ heart sound
- 1 + pedal edema

2) Identify cue clusters

- BP 210/110 and 200/108
- S₄ heart sound

- Confirms eating junk food
- Finds low-fat, low-salt diet "impossible"

- "I have pains all the time"
- Denies pain at this time

- Wife: "Don't know what to do with him ... tired of dealing with him when he won't help himself"
- Called 911 when he had pain

3) Draw inferences

Dangerously high blood pressure with concurrent atrial gallop seen with hypertension. Refer to physician

Chooses not to follow special diet. Unable to tolerate special diet

Not experiencing pain currently but has history of pain related to hypertension

Wife, who perceives herself as a caregiver is frustrated and anxious about client's ill health and noncompliance—possibly burned out

4) List possible nursing diagnoses

Ineffective Health Maintenance r/t choice not to follow prescribed dietary treatment of hypertension

Ineffective Therapeutic Regimen Management r/t intolerance of therapeutic diet and knowledge deficit of alternative strategies for managing hypertension

Risk for Acute Pain: Acute pain (angina) r/t knowledge deficit of management strategies

Caregiver Role Strain r/t frustration with client's non-compliant behavior and possible anxiety over seriousness of symptoms

Ineffective Family Coping r/t strain on family from client's illness

5) Check for defining characteristics

Major: Reports unhealthful practices (e.g., high-fat, high-salt diet)
Minor: None, except possibly compulsive behavior regarding diet ("has to have my junk food")

Major: Verbalizes dislike of and difficulty with integration of prescribed regimen (diet) for treatment of illness
Minor: Verbalizes he did not include treatment in daily routine

Major: Reports pain "all the time" but not at this time
Minor: None

Major: None
Minor: Possibly implied apprehension about the future for care receiver's health. Also possibly depressed feelings and anger.

Major: None specific
Minor: None specific

6) Confirm or rule out diagnoses

Accept diagnosis because it meets defining characteristics and is validated by client

Confirm, because diagnosis meets defining characteristics

Accept this diagnosis because it is a risk diagnosis

Data are insufficient to accept this diagnosis, although it is certainly a risk diagnosis given the wife's verbalization of frustration

Rule out diagnosis because it does not meet the major defining characteristic. More data are needed

7) Document conclusions

Nursing diagnoses that are appropriate for this client include:

- Ineffective Health Maintenance r/t choice not to follow dietary treatment of hypertension
- Ineffective Therapeutic Regimen Management r/t intolerance of therapeutic diet and knowledge deficit of alternative strategies for managing hypertension
- Risk for Acute Pain: acute pain (angina) r/t knowledge deficit of management strategies

Potential collaborative problems including the following:

- PC: Cerebrovascular accident
- PC: Retinal hemorrhage
- PC: Myocardial infarction
- PC: Heart failure
- PC: Renal failure

and neck vessels. These problems are worded as Potential Complications (or PC) followed by the problem.

- PC: Decreased cardiac output
- PC: Dysrhythmias
- PC: Hypertension
- PC: Congestive heart failure
- PC: Angina
- PC: Cerebrovascular accident
- PC: Cerebral hemorrhage
- PC: Renal failure

Medical Problems

Once the data are grouped, certain signs and symptoms may become evident and may require medical diagnosis and treatment. Referral to a primary care provider is necessary.

References and Selected Readings

- Appel, L. J., Moore, T. J., et al. (1997). A clinical trial of the effects of dietary patterns on blood pressure. *New England Journal of Medicine*, 336, 1–17.
- Archbold, R. A., Barakat, K., Magee, P., & Curzen, N. (2001). Screening for carotid artery disease before cardiac surgery: Is current clinical practice evidence based? *Clinical Cardiology*, 24(1), 26–32.
- Barett, et al. (2004). Mastering cardiac murmurs: The power of repetition. *Chest*, 126, 470–475.
- Carabello, B. A., & Crawford, J. (1997). Valvular heart disease. *New England Journal of Medicine*, 337, 32.
- Chizner, M. (2003). The diagnosis of heart disease. *Disease-a month*, 48(1), 7–98.
- Davidson, L. J., Bennett, S. E., Hamera, E. K., & Raines, B. K. (2004). What constitutes advanced assessment? *Journal of Nursing Education*, 43(9), 421–425.
- Daviglus, M. L., Stamler, J., Pirzada, A., Yan, L. L., Garside, D. B., Liu, K., Wade-Dyer, A. R., Lloyd-Jones, D. M., & Greenland, P. (2004). Favorable cardiac risk profile in young women and low risk of cardiovascular and all-cause mortality. *Journal of the American Medical Association*, 292(13), 1588–1592.
- Dulak, S. B. (2004). Hands-on help: Assessing heart sounds. *RN*, 67(8), 241–244.
- Fabius, D. B. (2000). Solving the mystery of heart murmurs. *Nursing 2000*, 30(7), 39–44.
- Gillett, M., Davis, W. A., Jackson, D., Bruce, D. G., Davis, T. M., & Fremantle, D. (2003). Prospective evaluation of carotid bruit as a predictor of first sign of type 2 diabetes: The Fremantle Diabetes Study. *Stroke*, 34(9), 2145–2151.
- Jolobe, O. M. P. (2001). Systolic murmurs and aortic stenosis. *Quarterly Journal of Medicine*, 94(1), 49.
- Kirton, C. A. (1997). Assessing a heart murmur. *Nursing 1997*, 27(9), 51.
- Kirton, C. A. (2000). Physical assessment. Assessing normal heart sounds. *Nursing 2000*, 30(2), 52–54.
- Loveridge, M. (2003). Acquiring percussion and auscultation skills through experiential learning. *Emergency Nurse*, 11(6), 31–37.
- Ludwig, L. M. (1998). Cardiovascular assessment for home healthcare nurses. Part 1. Assessing blood pressure and cardiac function. *Home Healthcare Nurse*, 16(8), 547–554.
- Marshall, K. G. (1998). More techniques of auscultation: General principles of murmurs. *Patient Care*, 9(2), S1–S5.
- Mehta, M. (2003). Assessing cardiovascular status. *Nursing 2003*, 33(1), 56–58.
- Nirav, J., Mehta, M., & Ijaz, A. (2003). Third heart sound: Genesis and clinical importance. *International Journal of Cardiology*, 97(2), 183–186.
- Wasserman, A. (2000). Chest pain. Is it life-threatening—or benign? *Consultant*, 40(7), 1204–1208.
- Welsby, P. D., et al. (2003). The stethoscope: Some preliminary investigations. *Postgraduate Medical Journal*, 79, 695–698.
- American Heart Association (AHA). (2001). *Coronary heart disease*. Available online. Author.
- . (1999). *Risk factor assessment for heart attack or stroke*. Available online. Author.
- Azevedo, A., Ramos, E., vonHafe, P., & Barros, H. (1999). Upper-body adiposity and risk of myocardial infarction. *Journal of Cardiovascular Risk*, 6(5), 321–325.
- Berenson, G., & Pickoff, A. (1995). Preventive cardiology and its potential influence on the early natural history of adult heart diseases: The Bogalusa Heart Study and the Heart Smart Program. *American Journal of the Medical Sciences*, 310(Suppl. 1), S1333–S1338.
- Eichholzer, M., Luthy, J., Gutzwiller, F., & Stahelin, H. (2001). The role of folate, antioxidant vitamins and other constituents in fruit and vegetables in the prevention of cardiovascular disease: The epidemiological evidence. *International Journal for Vitamin and Nutrition Research*, 71(1), 5–17.
- Gillum, R. (1996). Epidemiology of hypertension in African American women. *American Heart Journal*, 131, 385–395.
- Hallmen, T., Burell, G., Setterlind, S., Oden, A., & Lisspers, J. (2001). Psychosocial risk factors for coronary heart disease, their importance compared with other risk factors and gender differences in sensitivity. *Journal of Cardiovascular Risk*, 8(1), 39–49.
- Leeson, C. P., Kattenhorn, M., Morley, R., Lucas, A., & Deanfield, J. (2001). Impact of low birth weight and cardiovascular risk factors on endothelial function in early adult life. *Circulation*, 103(9), 1264–1268.
- Libby, P., Schoenbeck, V., Mach, F., Selwyn, A., & Ganz, P. (1998). Current concepts in cardiovascular pathology: The role of LDL cholesterol in plaque rupture and stabilization. *American Journal of Medicine*, 104(24), 145–185.
- Misra, A. (2000). Risk factors for atherosclerosis in young individuals. *Journal of Cardiovascular Risk*, 7(3), 215–219.
- Overfield, T. (1995). *Biological variation in health and illness: Race, age, and sex differences* (2nd ed.). Boca Raton, FL: CRC Press.
- Rifai, N. M., & Ridker, P. M. (2001). High sensitivity C-reactive protein: A novel and promising marker of coronary heart disease. *Clinical Chemistry*, 47(3), 403–411.
- W.H.O. (2004). Types of cardiovascular disease. Retrieved November, 2004 from http://www.who.int/cardiovascular_diseases/resources/atlas/

