Assessing the Cardiovascular and Lymphatic Systems

LEARNING OUTCOMES
1. Describe the anatomy, physiology, and functions of the cardiovascular and lymphatic systems.
2. Describe normal variations in cardiovascular assessment findings for the older adult.
3. Give examples of genetic disorders of the cardiovascular system.
4. Identify specific topics for consideration during a health history assessment interview of the patient with cardiovascular or lymphatic disorders.
5. Explain techniques used to assess cardiovascular and lymphatic structure and function.
6. Identify manifestations of impaired cardiovascular structure and functions.

CLINICAL COMPETENCIES
1. Complete a health history for patients having alterations in the structure and functions of the cardiovascular or lymphatic systems.
2. Conduct and document a physical assessment of cardiovascular and lymphatic status.
3. Assess an ECG strip and identify normal rhythm and cardiac events and abnormal cardiac rhythm.
4. Monitor the results of diagnostic tests and communicate abnormal findings within the interprofessional team.

MAJOR CHAPTER CONCEPTS
- Correct structure and function of the cardiovascular and lymphatic systems are vital to the transport of oxygen and carbon dioxide throughout the body and for the return of excess tissue fluids back to the bloodstream.
- Manifestations of dysfunction, injury, and disorders affecting the cardiovascular and lymphatic systems may be detected during a general health assessment as well as during focused cardiovascular and lymphatic system assessments.

KEY TERMS
- apical impulse, 849
- cardiac index (CI), 830
- cardiac output (CO), 829
- dysrhythmia, 851
- heave, 849
- hemorrhage, 838
- ischemic, 829
- Korotkoff’s sounds, 854
- lift, 849
- lymphadenopathy, 858
- lymphedema, 854
- orthostatic hypotension, 854
- retraction, 849
- thrill, 851
- thrust, 849

EQUIPMENT NEEDED
- Stethoscope with a diaphragm and a bell
- Blood pressure cuff
- Good light source
- Watch with a second hand
- Centimeter ruler
- Tape measure
- Doppler ultrasound device (if needed) and transducer gel

The cardiovascular system is comprised of the heart (the system’s pump), the peripheral vascular system (a network of arteries, veins, and capillaries), and the hematologic system (blood and blood components). The lymphatic system (the lymph, lymph nodes, and spleen) is a special vascular system that helps maintain sufficient blood volume in the cardiovascular system by picking up excess tissue fluid and returning it to the bloodstream.

The heart beats an average of 80 times per minute, or once every 0.86 second, every minute of an individual’s life. As the heart ejects blood with each beat, a closed system of blood vessels transports oxygenated blood to all body organs and tissues and then returns deoxygenated blood to the heart for reoxygenation in the lungs. Deficits in the structure or function of the cardiovascular and lymphatic system may adversely affect all body tissues and may affect self-care, mobility, comfort, self-concept, sexuality, and role performance.
THE HEART
The heart is a hollow, cone-shaped organ approximately the size of an adult man’s fist. Beating from 60 to 100 beats each minute for a lifetime, it moves more than 1800 gallons of blood each day (Huether & McCance, 2011). Located in the mediastinum of the thoracic cavity, between the vertebral column and the sternum, the heart is flanked laterally by the lungs. The heart weighs less than 0.5 kg (1 lb) in a normal healthy adult. Two-thirds of the heart mass lies to the left of the sternum; the upper base lies beneath the second rib, and the pointed apex is approximate with the fifth intercostal space, midpoint to the clavicle (Figure 29–1).

The heart is covered by the pericardium, a double layer of fibroserous membrane (Figure 29–2). The pericardium encases the heart and anchors it to surrounding structures, forming the pericardial sac. The snug fit of the pericardium prevents the heart from overfilling with blood. The outermost layer is the parietal pericardium; the visceral pericardium (or epicardium) adheres to the heart surface. The small space between the visceral and parietal layers of the pericardium is called the pericardial cavity. Ten to 30 mL of a serous lubricating fluid produced in this space cushions the heart as it beats.

The heart wall consists of three layers of tissue: the epicardium, the myocardium, and the endocardium (refer to Figure 29–2). The epicardium covers the entire heart and great vessels, and then folds over to form the parietal layer that lines the pericardium and adheres to the heart surface. The myocardium, the middle layer of the heart wall, consists of specialized cardiac muscle cells (myofibrils) that provide the bulk of contractile heart muscle. The endocardium is a thin three-layer membrane that lines the inside of the heart’s chambers and great vessels.

Chambers and Valves of the Heart
The heart has two upper atria and two lower ventricles. They are separated longitudinally by the interventricular septum (Figure 29–3). The right atrium receives deoxygenated blood from the veins of the body: The superior vena cava returns blood from the body area above the diaphragm, the inferior vena cava returns blood from the body below the diaphragm, and the coronary sinus drains blood from the heart. The left atrium receives freshly oxygenated blood from the lungs through the pulmonary veins. The right ventricle receives deoxygenated blood from the right atrium and pumps it through the pulmonary artery to the pulmonary capillary bed for oxygenation. The newly oxygenated blood then travels through the pulmonary veins to the left atrium. Blood enters the left atrium and crosses the mitral (bicuspid) valve into the left ventricle. Blood is then pumped out of the aorta to the arterial circulation.

The heart’s chambers are each separated by a valve that allows unidirectional blood flow to the next chamber or great vessel (refer to Figure 29–3). The atria are separated from the ventricles by the two atrioventricular (AV) valves; the tricuspid valve is on the right side, and the bicuspid (or mitral) valve is on the left. The flaps of each of these valves are anchored to the papillary muscles of the ventricles by the chordae tendineae. These structures control the movement of the AV valves to prevent backflow of blood. The ventricles are connected

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**Figure 29–1** Location of the heart in the mediastinum of the thorax. A, Relationship of the heart to the sternum, ribs, and diaphragm. B, Cross-sectional view showing relative position of the heart in the thorax. C, Relationship of the heart and great vessels to the lungs.
to their great vessels by the semilunar valves. On the right, the pulmonary (pulmonic) valve joins the right ventricle with the pulmonary artery. On the left, the aortic valve joins the left ventricle to the aorta. Closure of the AV valves at the onset of contraction (systole) produces the first heart sound, or S₁ (characterized by the syllable lub); closure of the semilunar valves at the onset of relaxation (diastole) produces the second heart sound, or S₂ (characterized by the syllable lub).

**Systemic, Pulmonary, and Coronary Circulation**

Because each side of the heart both receives and ejects blood, the heart is often described as a double pump. Blood enters the right atrium and moves to the pulmonary bed at almost the exact same time that blood is entering the left atrium. The circulatory system has two parts: the systemic circulation (a high-pressure system), which supplies blood to all other body tissues, and the pulmonary circulation (a low-pressure system). The systemic circulation consists of the left side of the heart, the aorta and its branches, the capillaries that supply the brain and peripheral tissues, the systemic venous system, and the vena cava. The pulmonary circulation consists of the right side of the heart, the pulmonary artery, the pulmonary capillaries, and the pulmonary vein. Pulmonary circulation begins with the right side of the heart. Deoxygenated blood from the venous system enters the right atrium through two large veins, the superior and inferior venae cavae, and is transported to the lungs via the pulmonary artery.
and its branches (Figure 29–4). After oxygen and carbon dioxide are exchanged in the pulmonary capillaries, oxygen-rich blood returns to the left atrium through several pulmonary veins. Blood is then pumped out of the left ventricle through the aorta and its major branches to supply all body tissues by the systemic circulation.

Oxygen is supplied to the heart muscle by its own network of vessels through the coronary circulation. The left and right coronary arteries originate at the base of the aorta and branch out to encircle the myocardium (Figure 29–5A), supplying it with blood, oxygen, and nutrients. The left main coronary artery divides to form the anterior descending and circumflex arteries. The anterior descending artery supplies the anterior interventricular septum and the left ventricle. The circumflex branch supplies the left lateral wall of the left ventricle. The right coronary artery supplies the right ventricle and forms the posterior descending artery. The posterior descending artery supplies the posterior portion of the heart. While ventricular contraction delivers blood through the pulmonary circulation and the systemic circulation, it is during ventricular relaxation that the coronary arteries fill with oxygenated blood. After the blood perfuses the heart muscle, the cardiac veins drain the blood into the coronary sinus, which empties into the right atrium of the heart (Figure 29–5B). Blood flow through the coronary arteries is regulated by several factors. Aortic pressure is the primary factor. Other factors include the heart rate (most flow occurs during diastole, when the muscle is relaxed), metabolic activity of the heart, and blood vessel tone (constriction).
The Cardiac Cycle and Cardiac Output

The contraction and relaxation of the heart constitute one heartbeat and this process is called the cardiac cycle (Figure 29–6). Ventricular filling is followed by ventricular systole, a phase during which the ventricles contract and eject blood into the pulmonary and systemic circuits. Systole is followed by a relaxation phase known as diastole, during which the ventricles refill, the atria contract, and the myocardium is perfused. Normally, the complete cardiac cycle occurs about 70 to 80 times per minute, measured as the heart rate (HR).

During diastole, the volume in the ventricles is increased to about 120 mL (the end-diastolic volume), and at the end of systole, about 50 mL of blood remains in the ventricles (the end-systolic volume). The difference between the end-diastolic volume and the end-systolic volume is called the stroke volume (SV). Stroke volume ranges from 60 to 100 mL/beat and averages about 70 mL/beat in an adult. The ejection fraction is the stroke volume divided by the end-diastolic volume and represents the fraction or percent of the diastolic volume that is ejected from the heart during systole (Huether & McCance, 2011). For example, an end-diastolic volume of 120 mL divided by a stroke volume of 80 mL equals an ejection fraction of 66%. The normal ejection fraction ranges from 50% to 70%.

The cardiac output (CO) is the amount of blood pumped by the ventricles into the pulmonary and systemic circulations in 1 minute. Multiplying the HR by the SV determines the cardiac output: \[ \text{HR} \times \text{SV} = \text{CO}. \] The average adult CO ranges from 4 to 8 L/min. Cardiac output is an indicator of how well the heart is functioning as a pump. If the heart cannot pump effectively, CO and tissue perfusion are decreased. Body tissues that do not receive enough blood and oxygen (carried in the blood on hemoglobin) become ischemic (deprived of oxygen). If the tissues do not receive enough blood flow to maintain the functions of the cells, the cells die, resulting in necrosis (infarction).

Activity level, metabolic rate, physiologic and psychologic stress responses, age, and body size all influence CO. In addition, CO is determined by the interaction of four major factors: heart rate, contractility, preload, and afterload. Changes in each of these variables influence CO intrinsically, and each can be manipulated to affect CO.

The heart’s ability to respond to the body’s changing need for CO is called cardiac reserve.

HEART RATE

Heart rate is affected by both direct and indirect autonomic nervous system stimulation. Direct stimulation is accomplished through the innervation of the heart muscle by sympathetic and parasympathetic nerves. The sympathetic nervous system increases the heart rate, whereas the parasympathetic vagal tone slows the heart rate. Reflex regulation of the heart rate in response to systemic blood pressure also occurs through activation of baroreceptors (pressure receptors) located in the carotid sinus, aortic arch, venae cavae, and pulmonary veins.

If heart rate increases, CO increases (up to a point), even if there is no change in stroke volume. However, rapid heart rates decrease the amount of time available for ventricular filling during diastole. Cardiac output then falls because decreased filling time decreases stroke volume. Coronary artery perfusion also decreases because the coronary arteries fill primarily during diastole. Cardiac output decreases during bradycardia if stroke volume stays the same, because the number of cardiac cycles is decreased.

CONTRACTILITY

Contractility is the ability of the cardiac muscle fibers to shorten. Poor contractility of the heart muscle reduces the forward flow of blood from the heart, increases the ventricular pressures from accumulation of blood volume, and reduces CO. Increased contractility may stress the heart by increasing the SV in pathologic conditions.

PRELOAD

Preload is the amount of cardiac muscle fiber tension, or stretch, that exists at the end of diastole, just before contraction of the ventricles. Preload is influenced by venous return (volume) and the compliance of the ventricles (resulting pressure). Preload is based on ventricular end-diastolic volume (VEDV) and ventricular end-diastolic pressure (VEDP). It is related to the total volume of blood in the ventricles. The greater the volume, the greater the stretch of the cardiac muscle fibers, and the greater the force with which the fibers contract to accomplish

Figure 29–6 The cardiac cycle has three events: (1) ventricular filling in mid-to-late diastole, (2) ventricular systole, and (3) isovolumetric relaxation in early diastole.
emptying. This principle is called Starling’s law of the heart. Disorders such as renal disease and congestive heart failure result in sodium and water retention and increased preload. Vasoconstriction also increases venous return and preload.

**MEMORY CUE**

This mechanism has a physiologic limit. Just as continuous overstretching of a rubber band causes the band to relax and lose its ability to recoil, overstretching of the cardiac muscle fibers eventually results in ineffective contraction.

Too little circulating blood volume results in a decreased venous return and therefore a decreased preload. A decreased preload reduces stroke volume and thus cardiac output. Decreased preload may result from hemorrhage or misdistribution of blood volume, as occurs in third spacing (see Chapter 11).

**AFTERLOAD**

Afterload is the force the ventricles must overcome to eject their blood volume. It is the pressure in the arterial system ahead of the ventricles. The right ventricle must generate enough tension to open the pulmonary valve and eject its volume into the low-pressure pulmonary arteries. Right ventricle afterload is measured as pulmonary vascular resistance (PVR). The left ventricle, in contrast, ejects its load by overcoming the pressure behind the aortic valve. Afterload of the left ventricle is measured as systemic vascular resistance (SVR). Arterial pressures are much higher than pulmonary pressures; thus, the left ventricle has to work much harder than the right ventricle.

Alterations in vascular tone affect afterload and ventricular work. As the pulmonary or arterial blood pressure increases (e.g., through vasoconstriction), PVR and/or SVR increases, and the work of the ventricles increases. As workload increases, consumption of myocardial oxygen also increases. A compromised heart cannot effectively meet this increased oxygen demand, and a vicious cycle ensues. By contrast, a very low afterload decreases the forward flow of blood into the systemic circulation and the coronary arteries.

**CLINICAL INDICATORS OF CARDIAC OUTPUT**

For many critically ill patients, invasive hemodynamic monitoring catheters are used to measure CO in quantifiable numbers. However, advanced technology is not the only way to identify and assess compromised blood flow. Because CO perfuses the body’s tissues, clinical indicators of low CO may be manifested by changes in organ function that result from compromised blood flow. For example, a decrease in blood flow to the brain presents as a change in level of consciousness. Other manifestations of decreased CO are discussed in Chapters 11 and 30.

Cardiac index (CI) is the CO adjusted for the patient’s body size, also called the patient’s body surface area (BSA). Because it takes into account the patient’s BSA, the cardiac index provides more meaningful data about the heart’s ability to perfuse the tissues and therefore is a more accurate indicator of the effectiveness of the circulation than the CO.

BSA is stated in square meters (m²), and cardiac index is calculated as CO divided by BSA. Cardiac measurements are considered adequate when they fall within the range of 2.5 to 4.2 L/min/m². For example, two patients have a CO of 4 L/min. This parameter is within normal limits. However, one patient is 157 cm (5 ft, 2 in.) tall and weighs 54.5 kg (120 lb), with a BSA of 1.54 m². This patient’s cardiac index is 4 ÷ 1.54, or 2.6 L/min/m². The second patient is 188 cm (6 ft, 2 in.) tall and weighs 81.7 kg (280 lb), with a BSA of 2.52 m². This patient’s cardiac index is 4 ÷ 2.52, or 1.6 L/min/m². The cardiac index results show that the same CO of 4 L/min is adequate for the first patient but grossly inadequate for the second patient.

**The Conduction System of the Heart**

The cardiac cycle is perpetuated by a complex electrical circuit commonly known as the intrinsic conduction system of the heart. Cardiac muscle cells possess an inherent characteristic of self-excitation, which enables them to initiate and transmit impulses independent of a stimulus. However, specialized areas of myocardial cells typically exert a controlling influence in this electrical pathway.

One of these specialized areas is the sinoatrial (SA) node, located at the junction of the superior vena cava and right atrium (Figure 29–7). The SA node acts as the normal “pacemaker” of the heart, usually generating an impulse 60 to 100 times per minute. This impulse travels across the atria via internodal pathways to the AV node, in the floor of the interatrial septum. The very small functional fibers of the AV node slow the impulse, slightly delaying its transmission to the ventricles. It then passes through the bundle of His at the atrioventricular junction and continues down the interventricular septum through the right and left bundle branches and out to the Purkinje fibers in the ventricular muscle walls.

This path of electrical transmission produces a series of changes in ion concentration across the membrane of each cardiac muscle cell. The electrical stimulus increases the permeability of the cell membrane, creating an action potential (electrical potential). The result is an exchange of sodium, potassium, and calcium ions across the cell membrane, which changes the intracellular electrical charge to a positive state. This process of depolarization results in myocardial contraction. As the ion exchange reverses and the cell returns to its resting state of electronegativity, the cell is repolarized, and cardiac muscle relaxes. The cellular action potential serves as the basis for electrocardiography (ECG), a diagnostic test of cardiac function.

**The Peripheral Vascular System**

The two components of the peripheral vascular system are the arterial network and the venous network. The arterial network begins with the major arteries that branch from the aorta. The major arteries of the systemic circulation are illustrated in Figure 29–8. These major arteries branch into successively smaller arteries, which in turn subdivide into the smallest of the arterial vessels, called arterioles. The smallest arterioles feed into beds of hairlike capillaries in the body’s organs and tissues.

In the capillary beds, oxygen and nutrients are exchanged for metabolic wastes, and deoxygenated blood moves back to the heart through venules, the smallest vessels of the venous network. Venules join the smallest of veins, which in turn join larger and larger veins. The blood transported by the veins empties into the superior and
inferior venae cavae entering the right side of the heart. The major veins of the systemic circulation are shown in Figure 29–9.

**Structure of Blood Vessels**

The structure of blood vessels reflects their different functions within the circulatory system (Figure 29–10). Except for the tiniest vessels, blood vessel walls have three layers: the tunica intima, the tunica media, and the tunica adventitia. The tunica intima, the innermost layer, is made of endothelium that provides a slick surface to facilitate the flow of blood. In arteries, the middle layer, or tunica media, is made of smooth muscle and is thicker than the tunica media of veins. This makes arteries more elastic than veins and allows the arteries to alternately expand and recoil as the heart contracts and relaxes with each beat, producing a pressure wave that can be felt as a pulse over an artery. The smaller arterioles are less elastic than arteries but contain more smooth muscle, which promotes their constriction and dilation. In fact, arterioles exert the major control over arterial blood pressure. The tunica adventitia, or outermost layer, is made of connective tissue and serves to protect and anchor the vessel. Veins have a thicker tunica adventitia than do arteries.

Blood in the veins travels at a much lower pressure than does blood in the arteries. Veins have thinner walls, a larger lumen, and greater capacity, and many are supplied with valves that help blood flow against gravity back to the heart. The “milking” action of skeletal muscle contraction supports venous return. When skeletal muscles contract against veins, the valves proximal to the contraction open, and blood is propelled toward the heart. The abdominal and thoracic pressure changes that occur with breathing also propel blood toward the heart.

The tiny capillaries, which connect the arterioles and venules, contain only one thin layer of tunica intima that is permeable to the gases and molecules exchanged between blood and tissue cells. Capillaries typically are found in interwoven networks. They filter and shunt blood from precapillary arterioles to postcapillary venules.

**Arterial Circulation**

The factors that affect arterial circulation are blood flow, peripheral vascular resistance, and blood pressure. Blood flow refers to the volume of blood transported in a vessel, in an organ, or throughout the entire circulation over a given period of time. It is commonly expressed as liters or milliliters per minute or cubic centimeters per second.

Peripheral vascular resistance (PVR) refers to the opposing forces or impedance to blood flow as the arterial channels become more and more distant from the heart. Peripheral vascular resistance is determined by three factors:

- **Blood viscosity**: The greater the viscosity, or thickness, of the blood, the greater its resistance to moving and flowing.
- **Length of the vessel**: The longer the vessel, the greater the resistance to blood flow.
- **Diameter of the vessel**: The smaller the diameter of a vessel, the greater the friction against the walls of the vessel and, thus, the greater the impedance to blood flow.

Blood pressure (BP) is the force exerted against the walls of the arteries by the blood as it is pumped from the heart. It is most accurately referred to as mean arterial pressure (MAP). The highest pressure exerted against the arterial walls at the peak of ventricular contraction (systole) is called the systolic BP. The lowest pressure exerted during ventricular relaxation (diastole) is the diastolic BP.

Mean arterial blood pressure is regulated mainly by cardiac output and peripheral vascular resistance, as represented in this formula: $MAP = CO \times PVR$. For clinical use, the MAP may be estimated by calculating the diastolic blood pressure plus one-third of the pulse pressure (the difference between the systolic and diastolic blood pressure).
Figure 29–8  Major arteries of the systemic circulation.
Figure 29-9  Major veins of the systemic circulation.
Factors Influencing Arterial Blood Pressure

Blood flow, peripheral vascular resistance, and BP, which influence arterial circulation, are in turn influenced by various factors, as follows:

- The sympathetic and parasympathetic nervous systems are the primary mechanisms that regulate BP. Stimulation of the sympathetic nervous system exerts a major effect on peripheral resistance by causing vasoconstriction of the arterioles, thereby increasing BP. Parasympathetic stimulation causes vasodilation of the arterioles, lowering BP.
- Baroreceptors and chemoreceptors in the aortic arch, carotid sinus, and other large vessels are sensitive to pressure and chemical changes and cause reflex sympathetic stimulation, resulting in vasoconstriction, increased heart rate, and increased BP.
- The kidneys help maintain BP by excreting or conserving sodium and water. When BP decreases, the kidneys initiate the renin-angiotensin mechanism. This stimulates vasoconstriction, resulting in the release of the hormone aldosterone from the adrenal cortex, increasing sodium ion reabsorption and water retention. In addition, pituitary release of antidiuretic hormone (ADH) promotes renal reabsorption of water. The net result is an increase in blood volume and a consequent increase in CO and BP.
- Temperatures may affect peripheral resistance: Cold causes vasoconstriction, whereas warmth produces vasodilation.
- Many chemicals, hormones, and drugs influence BP by affecting CO and/or PVR. For example, epinephrine causes vasoconstriction and increased heart rate; prostaglandins dilate blood vessel diameter (by relaxing vascular smooth muscle); endothelin, a chemical released by the inner lining of vessels, is a potent vasoconstrictor; nicotine causes vasoconstriction; and alcohol and histamine cause vasodilation.
- Dietary factors such as intake of salt, saturated fats, and cholesterol elevate BP by affecting blood volume and vessel diameter.
- Race, gender, age, weight, time of day, position, exercise, and emotional state may also affect BP. These factors influence the arterial pressure. Systemic venous pressure, though it is much lower, is also influenced by such factors as blood volume, venous tone, and right atrial pressure.

The Lymphatic System

The structures of the lymphatic system include the lymph, lymph nodes, spleen, thymus, tonsils, and the Peyer’s patches of the small intestine. Lymph nodes are small aggregates of specialized cells that assist the immune system by removing foreign material, infectious organisms, and tumor cells from lymph. Lymph nodes are distributed along the lymphatic vessels, forming clusters in certain body regions such as the neck, axilla, and groin (see Figure 29–11).

The spleen, the largest lymphoid organ, is in the upper left quadrant of the abdomen under the thorax. The main function of the spleen is to filter the blood by breaking down old red blood cells and storing or releasing to the liver their by-products (such as iron). The spleen also synthesizes lymphocytes, stores platelets for blood clotting, and serves as a reservoir of blood. The thymus gland is in the lower throat and is most active in childhood, producing hormones (such as thymosin) that facilitate the immune action of lymphocytes.

The tonsils of the pharynx and Peyer’s patches of the small intestine are lymphoid organs that protect the upper respiratory and digestive tracts from foreign pathogens.

The lymphatic vessels, or lymphatics, form a network around the arterial and venous channels and interweave at the capillary beds. They collect and drain excess tissue fluid, called lymph, that leaks from the cardiovascular system and accumulates at the venous end of the capillary bed. The lymphatics return this fluid to the heart through a one-way system of lymphatic venules and veins that eventually drain...
into the right lymphatic duct and left thoracic duct, both of which empty into their respective subclavian veins. Lymphatics are a low-pressure system without a pump; their fluid transport depends on the rhythmic contraction of their smooth muscle and the muscular and respiratory movements that assist venous circulation.

The Hematologic System

Blood consists of plasma, solutes (e.g., proteins, electrolytes, and organic constituents), red blood cells, white blood cells, and platelets (which are fragments of cells). The hematopoietic (blood-forming) system includes the bone marrow (myeloid) tissues, where blood cells form, and the lymphoid tissues of the lymph nodes, where white blood cells mature and circulate. All blood cells originate from cells in the bone marrow called stem cells, or hemocytoblasts. The origin of the cellular components of blood is illustrated in Figure 29–12. Normal laboratory values for blood components are found in Table 29–1.

Regulatory mechanisms cause stem cells to differentiate into families of parent cells, each of which gives rise to one of the formed elements of the blood (red blood cells, platelets, and white blood cells). The functions of blood include transporting oxygen, nutrients, hormones, and metabolic wastes; protecting against invasion of pathogens; maintaining blood coagulation; and regulating fluids, electrolytes, acids, bases, and body temperature.

Red Blood Cells

Red blood cells (RBCs), or erythrocytes, are the most common type of blood cell. They are shaped like biconcave disks (Figure 29–13). This unique shape increases the surface area of the cell and allows the cell to pass through very small capillaries without disrupting the cell membrane. RBCs and the hemoglobin molecules they contain transport oxygen to body tissues. Hemoglobin also binds with some carbon dioxide, carrying it to the lungs for excretion. Abnormal numbers of RBCs, changes in their size and shape, or altered hemoglobin content or structure can adversely affect health. Anemia, the most common RBC disorder, is an abnormally low RBC count or reduced hemoglobin content. Polycythemia is an abnormally high RBC count.
Hemoglobin, synthesized within the RBC, is the oxygen-carrying protein. It consists of the heme molecule and globin, a protein molecule. Globin is made of four polypeptide chains—two alpha chains and two beta chains (Figure 29–14). Each of the four polypeptide chains has a heme unit that contains an iron atom. The iron atom binds reversibly with oxygen, allowing it to transport oxygen as oxyhemoglobin to the cells. The rate of synthesis depends on the availability of iron. The size, color, and shape of stained RBCs also

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**Figure 29–12**  Blood cell formation from stem cells. Regulatory factors control the differentiation of stem cells into blasts. Each of the five kinds of blasts is committed to producing one type of mature blood cell. Erythroblasts, for example, can differentiate only into RBCs; megakaryoblasts can differentiate only into platelets.
Assessing the Cardiovascular and Lymphatic Systems

### Complete Blood Count (CBC)

<table>
<thead>
<tr>
<th>Component</th>
<th>Purpose</th>
<th>Normal Values</th>
</tr>
</thead>
<tbody>
<tr>
<td>Hemoglobin (Hb)</td>
<td>Measures the capacity of the hemoglobin to carry gases.</td>
<td>Women: 12–16 g/dL, Men: 13.5–18 g/dL</td>
</tr>
<tr>
<td>Hematocrit (Hct)</td>
<td>Measures packed cell volume of RBCs, expressed as a percent of the total blood volume.</td>
<td>Women: 38%–47%, Men: 40%–54%</td>
</tr>
<tr>
<td>Total RBC count</td>
<td>Counts number of circulating RBCs.</td>
<td>Women: 4–5 × 10⁶/μL, Men: 4.5–6 × 10⁶/μL</td>
</tr>
<tr>
<td>Red cell indices: MCV 10⁶</td>
<td>Determines relative size of MCV (mean corpuscular volume).</td>
<td>82–98 fl</td>
</tr>
<tr>
<td>MCH</td>
<td>Measures average weight of Hb/RBC (MCH = mean corpuscular hemoglobin).</td>
<td>27–29 pg</td>
</tr>
<tr>
<td>MCHC</td>
<td>Evaluates RBC saturation with Hb (MCHC = mean corpuscular hemoglobin concentration).</td>
<td>32%–36%</td>
</tr>
<tr>
<td>WBC count</td>
<td>Measures total number of leukocytes (total count) and whether each kind of WBC is present in proper proportion (differential).</td>
<td>Total WBC count: 4000–11,000/μL (4–11 × 10⁹/L) WBC differential: neutrophils: 50%–70%; eosinophils: 2%–4%; basophils: 0%–2%; lymphocytes: 20%–40%; monocytes: 4%–8%</td>
</tr>
<tr>
<td>Platelets</td>
<td>Measures number of platelets available to maintain clotting functions.</td>
<td>150,000–400,000/μL (150–400 × 10⁹/L)</td>
</tr>
</tbody>
</table>

**Figure 29–13**  
Top and side view of a red blood cell (erythrocyte). Note the distinctive biconcave shape.

may be analyzed. RBCs may be normocytic (normal size), smaller than normal (microcytic), or larger than normal (macrocytic). Their color may be normal (normochromic) or diminished (hypochromic).

**RED BLOOD CELL PRODUCTION AND REGULATION**

In adults, RBC production (erythropoiesis) (Figure 29–15) begins in the red bone marrow of the vertebrae, sternum, ribs, and pelvis, and is completed in the blood or spleen. Erythroblasts begin forming hemoglobin while they are in the bone marrow, a process that continues throughout the RBC life span. The cells enter the circulation as reticulocytes, which fully mature in about 48 hours. The complete sequence from stem cell to RBC takes 3 to 5 days.

The stimulus for increased RBC production is tissue hypoxia. The hormone erythropoietin is released by the kidneys in response to hypoxia. It stimulates the bone marrow to produce RBCs. However, the process of RBC production takes about 5 days to maximize. During periods of increased RBC production, the percentage of reticulocytes (immature RBCs) in the blood exceeds that of mature cells.

**RED BLOOD CELL DESTRUCTION**

RBCs have a life span of about 120 days. Old or damaged RBCs are lysed (destroyed) by phagocytes in the spleen, liver, bone marrow, and lymph nodes. The process of RBC destruction is called hemolysis. Phagocytes save and reuse amino acids and iron from heme units in the lysed RBCs. Most of the heme unit is converted to bilirubin, an orange-yellow pigment that is removed from the blood by
the liver and excreted in the bile. During disease processes causing increased hemolysis or impaired liver function, bilirubin accumulates in the serum, causing jaundice, a yellowish appearance of the skin and sclera.

**White Blood Cells**

White blood cells (WBCs), or leukocytes, originate from hemopoietic stem cells in the bone marrow and differentiate into the various types of white blood cells. They are a part of the body’s defense against microorganisms. Leukocytosis is a higher-than-normal WBC count; leukopenia is a WBC count that is lower than normal.

The two basic types of WBCs are granular leukocytes (or granulocytes) and nongranular leukocytes. Stimulated by granulocyte-macrophage colony-stimulating factor (GM-CSF) and granulocyte colony-stimulating factor (G-CSF), granulocytes mature fully in the bone marrow before being released into the bloodstream. The three types of granulocytes are as follows:

- Neutrophils (also called polymorphonuclear [PMN] or segmented [segs] leukocytes) are active phagocytes, the first cells to arrive at a site of injury. Their numbers increase during inflammation. Immature forms of neutrophils (bands) are released during inflammation or infections, and are referred to as having a shift to the left (so named because immature cell frequencies appear on the left side of the graph) on a differential blood count. Neutrophils have a life span of only about 10 hours and are constantly being replaced.
- Eosinophils are found in large numbers in the mucosa of the intestines and lungs. Their numbers increase during allergic reactions and parasitic infections.
- Basophils contain histamine, heparin, and other inflammatory mediators. Basophils increase in numbers during allergic and inflammatory reactions.

Nongranular WBCs (agranulocytes) include the monocytes and lymphocytes. They enter the bloodstream before final maturaton. These cells are an active part of the inflammatory and immune responses and are discussed in Chapters 12 and 13.

**Platelets**

Platelets (thrombocytes) produce ATP and release mediators required for clotting. Platelets are formed in the bone marrow as pinched-off portions of large megakaryocytes. Platelet production is controlled by thrombopoietin, a protein produced by the liver, kidney, smooth muscle, and bone marrow. The number of circulating platelets controls thrombopoietin release. Platelets live up to 10 days in circulation. An excess of platelets is thrombocytosis. A deficit of platelets is thrombocytopenia.

**Hemostasis**

Platelet and coagulation disorders affect hemostasis (control of bleeding). Hemostasis is a series of complex interactions between platelets and clotting mechanisms that maintain a relatively steady state of blood volume, BP, and blood flow through injured vessels. The five stages of hemostasis are (1) vessel spasm, (2) formation of the platelet plug, (3) development of an insoluble fibrin clot, (4) clot retraction, and (5) clot dissolution.

**VESSEL SPASM**
When a blood vessel is damaged, thromboxane A₂ (TXA₂) is released from platelets and cells, causing vessel spasm. This spasm constricts the damaged vessel for about 1 minute, reducing blood flow.

**FORMATION OF THE PLATELET PLUG**
Platelets attracted to the damaged vessel wall change from smooth disks to spiny spheres. Receptors on the activated platelets bind with von Willebrand’s factor (a protein molecule) and exposed collagen fibers at the site of injury to form the platelet plug (Figure 29–16). The platelets release adenosine diphosphate (ADP) and TXA₂ to activate nearby platelets, adhering them to the developing plug. Activation of the clotting pathway on the platelet surface converts fibrinogen to fibrin. Fibrin, in turn, forms a meshwork that binds the platelets and other blood cells to form a stable plug.

**DEVELOPMENT OF THE FIBRIN CLOT**
The process of coagulation creates a meshwork of fibrin strands that cements the blood components to form an insoluble clot. Coagulation requires many interactive reactions and two clotting pathways (Figure 29–17). The slower intrinsic pathway is activated when blood contacts collagen in the injured vessel wall; the faster extrinsic pathway is activated when blood is exposed to tissues. The final outcome of both pathways is fibrin clot formation. Each procoagulation substance is activated in sequence; the activation of one coagulation factor activates another in turn. Table 29–2 lists known factors, their origin, and their function or pathway. A deficiency of one or more factors or inappropriate inactivation of any factor alters normal coagulation.

**CLOT RETRACTION**
After the clot is stabilized (within about 30 minutes), trapped platelets contract. Platelet contraction squeezes the fibrin strands, pulling the broken portions of the ruptured blood vessel closer together.
promote plasminogen activator release. The liver and endothelium also produce fibrinolytic inhibitors.

**ASSESSING CARDIOVASCULAR AND LYMPHATIC FUNCTION**

Cardiovascular function is assessed by findings from diagnostic tests, a health assessment interview to collect subjective data, and a physical assessment to collect objective data.

**Diagnostic Tests**

The results of diagnostic tests of cardiac function are used to support the diagnosis of a specific disease, to provide information to identify or modify the appropriate medications or therapy used to treat the disease, and to help the interprofessional team monitor the patient’s responses to treatment and nursing care interventions. Diagnostic tests to assess the structures and functions of the heart are described on page 841. More information is included in the discussion of specific disorders in Chapters 30 through 33.

Regardless of the type of diagnostic test, the nurse is responsible for explaining the procedure and any special preparation needed, ensuring the consent form is signed (if necessary), supporting the patient during the examination as necessary, documenting the procedure as appropriate, and monitoring the results of the test. The nurse is responsible for postprocedure care and patient teaching for self-care at home.

**Genetic Considerations**

When conducting a health assessment interview and physical assessment, it is important for the nurse to consider genetic influences on the health of the adult. Ask about family members with health problems affecting the cardiovascular system, such as high BP, high cholesterol levels, leukemia, or early-onset CAD. Depending on the racial and ethnic background of the patient, ask about any family members with sickle cell disease or thalassemia. During the physical assessment, assess for any manifestations that might indicate a genetic disorder (see the Genetic Considerations box on page 845). If data are found to indicate genetic risk factors or alterations, ask about genetic testing and refer for appropriate genetic counseling and evaluation. Chapter 8 provides further information about genetics in medical-surgical nursing.

**The Health Assessment Interview**

A health assessment interview to determine problems with cardiovascular or lymphatic structure and function may be conducted during a health screening, may focus on a chief complaint (such as chest pain or leg pain when walking), or may be part of a complete health assessment. If the patient has a problem with cardiovascular or lymphatic function, analyze its onset, characteristics, course, severity, precipitating and relieving factors, and any associated symptoms, noting the timing and circumstances. For example, ask the patient the following:

- What is the location of the chest pain you experienced? Did it move up to your jaw or into your left arm?
- Describe the type of activity that brings on your chest pain.
- Does the leg pain occur only with activities such as walking, or during rest or sleep?
- Have you felt light-headed during the times your heart is racing?

**Figure 29–16** Platelet plug formation and blood clotting. This flow diagram summarizes the events leading to fibrin clot formation. PF₃ (blue arrow) released from damaged tissue combines with other clotting factors to release prothrombin activator, the first step of coagulation. Second, prothrombin is converted into thrombin. Finally, thrombin transforms soluble fibrinogen into insoluble fibrin (red arrow) to form a clot.
Figure 29-17  • Clot formation. Both the slower intrinsic pathway and the more rapid extrinsic pathway activate Factor X. Factor X then combines with other factors to form prothrombin activator. Prothrombin activator transforms prothrombin into thrombin, which then transforms fibrinogen into long fibrin strands. Thrombin also activates Factor XIII, which draws the fibrin strands together into a dense meshwork. The complete process of clot formation occurs within 3 to 6 minutes after blood vessel damage.

<table>
<thead>
<tr>
<th>Factor</th>
<th>Name</th>
<th>Function or Pathway</th>
</tr>
</thead>
<tbody>
<tr>
<td>I</td>
<td>Fibrinogen</td>
<td>Converted to fibrin strands</td>
</tr>
<tr>
<td>II</td>
<td>Prothrombin</td>
<td>Converted to thrombin</td>
</tr>
<tr>
<td>III</td>
<td>Thromboplastin</td>
<td>Catalyzes conversion of thrombin</td>
</tr>
<tr>
<td>IV</td>
<td>Calcium ions</td>
<td>Needed for all steps of coagulation</td>
</tr>
<tr>
<td>V</td>
<td>Proaccelerin</td>
<td>Extrinsic/intrinsic pathways</td>
</tr>
<tr>
<td>VII</td>
<td>Serum prothrombin conversion accelerator</td>
<td>Extrinsic pathway</td>
</tr>
<tr>
<td>VIII</td>
<td>Antihemophilic factor</td>
<td>Intrinsic pathway</td>
</tr>
<tr>
<td>IX</td>
<td>Plasma prothrombin component</td>
<td>Intrinsic pathway</td>
</tr>
<tr>
<td>X</td>
<td>Stuart factor</td>
<td>Extrinsic/intrinsic pathways</td>
</tr>
<tr>
<td>XI</td>
<td>Plasma prothrombin antecedent</td>
<td>Intrinsic pathway</td>
</tr>
<tr>
<td>XII</td>
<td>Hageman factor</td>
<td>Intrinsic pathway</td>
</tr>
<tr>
<td>XIII</td>
<td>Fibrin stabilizing factor</td>
<td>Cross-links fibrin strands to form insoluble clot</td>
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## Chapter 29 • Assessing the Cardiovascular and Lymphatic Systems

### THE HEART AND PERIPHERAL VASCULAR SYSTEM

<table>
<thead>
<tr>
<th>Name of Test</th>
<th>Purpose and Description</th>
<th>Related Nursing Interventions</th>
</tr>
</thead>
<tbody>
<tr>
<td>Blood pool imaging (gated scan or multigated acquisition scan [MUGA])</td>
<td>This test is useful for evaluation of cardiac status following myocardial infarction and congestive heart failure and effectiveness of cardiac medications. Also used to evaluate left ventricular (LV) function during rest and exercise. Following IV injection of technetium-99m pertechnetate, sequential evaluation of the heart can be performed for several hours. It can be done at the patient’s bedside.</td>
<td>No special preparation is needed. Inform the patient that food and fluids should not be taken for 6–8 h before the test. Assess for allergies to seafood, iodine, or iodine contrast dyes. If an allergic response to the dye is possible, antihistamines (such as Benadryl) or steroids may be administered the evening before and the morning of the test. Assess for use of aspirin or NSAIDs (risk of bleeding). Viagra (risk of heart problems), or history of kidney disease (dye used may be toxic to the kidneys). Take and record vital signs, including peripheral pulses. Explain that the patient is positioned on a padded table that tilts. A local anesthetic is used at the site of catheter insertion. ECG leads are applied and vital signs are monitored during the procedure. The patient lies supine and is asked to cough and deep breathe frequently. The procedure takes 0.5–3 h. After the procedure, monitor vital signs every 15 min for the first hour and then every 30 min until stable. Assess cardiac rhythm and rate for alterations. Assess peripheral pulses distal to the insertion site. Assess for chest heaviness, dyspnea, level of consciousness, and abdominal or groin pain. Monitor catheter insertion site for bleeding or hematoma. Administer pain medications as prescribed. Instruct patient to increase fluid intake and restrict activity as ordered. See information in this table for treadmill test. Instruct the patient to avoid intake of caffeine (including chocolate) for 12–24 h before having a test with dipyridamole Cardiolite.</td>
</tr>
<tr>
<td>Cardiac catheterization (coronary angiography, coronary arteriography)</td>
<td>A cardiac catheterization may be performed to identify coronary artery disease (CAD) or cardiac valvular disease, to determine pulmonary artery or heart chamber pressures, to obtain a myocardial biopsy, to evaluate artificial valves, or to perform angioplasty or stent an area of CAD. The test is performed by inserting a long catheter into a vein or artery (depending on whether the right side or the left side of the heart is being examined) in the arm or leg. Using fluoroscopy, the catheter is then threaded to the heart chambers or coronary arteries or both. Contrast dye is injected and heart structures are visualized and heart activity filmed. The test is done in the hospital for diagnosis and before heart surgery. <strong>Right cardiac catheterization:</strong> The catheter is inserted into the brachial, subclavian, internal jugular or femoral vein and then threaded through the inferior vena cava into the right atrium to the pulmonary artery. Pressures are measured at each site and blood samples can be obtained for the right side of the heart. The functions of the tricuspid and pulmonary valves can be observed. <strong>Left cardiac catheterization:</strong> The catheter is inserted into the radial, brachial, or femoral artery and advanced retrograde through the aorta to the coronary arteries and/or left ventricle. The patency of the coronary arteries and/or functions of the aortic and mitral valves and left ventricle can be observed.</td>
<td></td>
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<tr>
<td>Cardiolite scan</td>
<td>This test is used to evaluate blood flow in different parts of the heart. Cardiolite (technetium-99m sestamibi) is injected IV. In pharmacologic stress scans, dipyridamole (Persantine) or adenosine is injected to increase blood flow to coronary arteries. Additional pharmacologic stress scans can use dobutamine or arbutamine for their positive inotropic properties. These scans may be done in conjunction with a treadmill test.</td>
<td></td>
</tr>
<tr>
<td>Cardiac computed tomography (CT) scan</td>
<td>A cardiac CT scan may be conducted to visualize the heart anatomy or coronary circulation or to quantify early calcium deposits in coronary arteries. The calcium score screening heart scan is used to evaluate risk for future coronary artery disease and coronary artery bypass graft patency. It does not require injection of IV iodine. If calcium is present, a score is generated that estimates the extent of coronary artery disease. A negative test does not rule out the potential for soft plaque atherosclerosis.</td>
<td>Assess for allergy to iodine or seafood if contrast medium is to be administered. If allergy exists, follow orders for times and types of medications. For IV contrast studies, instruct patient not to eat or drink for 4 h prior to the test. Assess medications: oral hypoglycemic agents are contraindicated for use with iodinated contrast. Request patient remove hairpins, earrings, and dentures. No special preparation is needed.</td>
</tr>
<tr>
<td>Chest x-ray</td>
<td>An x-ray of the thorax can illustrate the contours, placement, and chambers of the heart. It may be done to identify heart displacement or hypertrophy, or fluid in the pericardial sac.</td>
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(continued)
### DIAGNOSTIC TESTS of the Cardiovascular and Lymphatic System (continued)

<table>
<thead>
<tr>
<th>Name of Test</th>
<th>Purpose and Description</th>
<th>Related Nursing Interventions</th>
</tr>
</thead>
<tbody>
<tr>
<td>Echocardiogram</td>
<td>Echocardiograms use a transducer to record waves that are bounced off the heart, and to record the direction and flow of blood through the heart in audio and graphic data. An M (motion)-mode echocardiogram records the motion, wall thickness, and chamber size of the heart. A 2-D echocardiogram provides a cross-sectional view of the heart. Spectral Doppler records blood flow through the chambers and septal wall defects. Color Doppler detects blood flow through the heart, valve function, and presence of shunting. Three-dimensional echocardiography combines 2-D echocardiography and ultrasound technology to evaluate the speed and direction of blood flow through the heart, which can identify pathology such as leaky valves. Four-dimensional echocardiography provides a moving picture of the 3D echo. Stress echocardiography combines a treadmill test with ultrasound images to evaluate segmental function and wall motion. If the patient is not physically able to exercise, IV dobutamine may be administered and ultrasound images taken.</td>
<td>No special preparation is needed; see related nursing care for the patient having a treadmill test for a stress echocardiogram.</td>
</tr>
<tr>
<td>Electrocardiogram (ECG)</td>
<td>See Boxes 29–1 and 29–2.</td>
<td>No special preparation is needed. Recommend a low-fat meal the evening prior to the test, then no food for 8–12 h. Instruct the patient to have no alcohol intake for 24 h prior to the test. Assess medications. Blood lipids may be increased by thyroxine, estrogens, aspirin, antibiotics (tetracycline and neomycin), nicotinic acid, heparin, and colchicine.</td>
</tr>
<tr>
<td>Lipids</td>
<td>Blood lipids are cholesterol, triglycerides, and phospholipids. They circulate bound to proteins, and so are known as lipoproteins. Lipids are measured to evaluate risk for CAD and to monitor effectiveness of anticholesterol medications. Normal values: Cholesterol: &lt; 200 mg/dL. Triglycerides: &lt; 150 mg/dL. HDL: Optimal: &gt; 60 mg/dL. Men: &gt; 40 mg/dL. Women: &gt; 50 mg/dL. LDL: &lt; 100 mg/dL. (Note: Normal values may vary by laboratory.)</td>
<td>Assess for any metallic implants (such as clips on brain aneurysms, pacemaker, body piercing, tattoos, and shrapnel). If present, notify physician. Remove transdermal medication patches (both OTC and prescribed) unless otherwise ordered. Replace the patch following the procedure. Tell the patient to inform the staff about the patch when making the appointment and when completing the admission information. Ask if patient is pregnant; if so the test is not performed. Ask about claustrophobia; if a problem exists, request patient to ask the referring physician for a relaxing medication prior to the MRI. If the patient is very claustrophobic, obese, or confused, an open MRI may be used. A contrast agent (gadolinium) may be used, especially in those allergic to dyes used in CT scanning.</td>
</tr>
<tr>
<td>Magnetic resonance imaging (MRI)</td>
<td>An MRI may be used to identify and locate areas of myocardial infarction, perfusion of the heart, and patency of coronary arteries after coronary grafts, and to evaluate pericarditis and cardiac tumors.</td>
<td>Instruct the patient to not eat food or drink fluids other than water after midnight or during the test. Tell the patient to discontinue beta-blockers, calcium channel blockers, and ACE inhibitors for 36 h prior to the test. If hospitalized, do not administer nitrates for 6 h prior to the test.</td>
</tr>
<tr>
<td>Nuclear dobutamine stress test</td>
<td>Dobutamine is an adrenergic drug that increases myocardial contractility, heart rate, and systolic BP, which increases coronary oxygen consumption and thus increases coronary blood flow. The test is conducted in two parts: resting and stress. The test usually takes about 3.5–4 h.</td>
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**DIAGNOSTIC TESTS** of the Cardiovascular and Lymphatic System (continued)

<table>
<thead>
<tr>
<th>Name of Test</th>
<th>Purpose and Description</th>
<th>Related Nursing Interventions</th>
</tr>
</thead>
<tbody>
<tr>
<td>Nuclear dipyridamole</td>
<td>This test is used when the patient is not physically able to walk on a treadmill. Dipyridamole (Persantine), given IV, dilates the coronary arteries and increases myocardial blood flow. Coronary arteries that are narrowed from CAD cannot dilate to increase myocardial perfusion.</td>
<td></td>
</tr>
<tr>
<td>(Persantine) stress test</td>
<td>This procedure is performed in the hospital to remove fluid from the pericardial sac for diagnostic or therapeutic purposes. It may also be done as an emergency procedure for the patient with cardiac tamponade (which may result in death). After local anesthetic, a large-gauge (16 to 18) needle is inserted to the left of the xiphoid process into the pericardial sac and excess fluid is withdrawn (Figure 29–18).</td>
<td></td>
</tr>
<tr>
<td>Pericardiocentesis</td>
<td></td>
<td>Patient is NPO after midnight except for water. Food, fluids, and drugs that contain caffeine should be avoided for 24 h prior to the test, as should decaffeinated fluids. Some drugs, such as theophylline preparations, are discontinued for 36 h prior to the test. Take and record baseline vital signs. Assess for history of cardiac problems. Explain to patient the need to remain still during the procedure, that the procedure takes about 30 min, and that a local anesthetic will be used at the needle insertion site. Monitor the ECG during and after the procedure and report abnormal findings to the physician. Monitor vital signs after the procedure as ordered. Notify the physician of changes in cardiac rhythm, BP, heart rate, or level of consciousness. If the procedure is to treat an emergency, monitor central venous pressure (CVP) and BP closely. As the effusion is relieved, CVP will decrease and BP will increase.</td>
</tr>
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</table>

**Figure 29–18** Pericardiocentesis.

- Positron emission tomography (PET)
  - Following injection of a radionuclide, two scans are performed (resting and chemically induced stress) and the resulting images are compared for myocardial perfusion and myocardial metabolic function. A stress test (treadmill) may be a part of the test. If the myocardium is ischemic or damaged, the images will be different. Normally, the images will be the same.

- Thallium/technetium stress test (myocardial imaging perfusion test, cardiac blood pool imaging)
  - **Thallium stress test**: Thallium-201, a radioisotope that accumulates in myocardial cells, is used during the stress test to evaluate myocardial perfusion. Second scans are done 2–3 h later when the heart is at rest; this is to differentiate between an ischemic area and an infarcted or scarred area of myocardium.
  - **Exercise technetium perfusion test**: Technetium-99m-laced compounds are administered and a scan is done to evaluate cardiac perfusion, wall motion, and ejection fraction. This is probably the most useful noninvasive test to diagnose and monitor CAD.

- Treadmill test (stress test)
  - Stress testing is based on the theory that coronary artery disease results in depression of the ST segment with exercise. Depression of the ST segment and depression or inversion of the T wave indicates myocardial ischemia. When the patient is walking on a treadmill machine, the work rate of the heart is changed every 3 min for 15 min by increasing the speed and degree of incline by 3% each time. Patients exercise until they are fatigued, develop symptoms, or reach their maximum predicted heart rate.

Assess the patient’s blood glucose: For accurate metabolic activity images, the blood glucose level must be between 60 and 140 mg/dL. If exercise is included in the test, instruct the patient to be NPO and avoid smoking and caffeine for 24 h prior to the test.

Assess medications; those that affect the BP or heart rate should be discontinued for 24–36 h prior to the test (unless the test is being done to monitor the effectiveness of the medications). See treadmill test for other interventions.

Assess the patient’s blood glucose: For accurate metabolic activity images, the blood glucose level must be between 60 and 140 mg/dL. If exercise is included in the test, instruct the patient to be NPO and avoid smoking and caffeine for 24 h prior to the test.

Ask the patient to wear comfortable shoes, and to avoid food, fluids, and smoking for 2–3 h before the test. Assess for events that contraindicate the tests: recent myocardial infarction; severe, unstable angina; controlled dysrhythmias; congestive heart failure; or recent pulmonary embolism.

(continued)
### DIAGNOSTIC TESTS of the Cardiovascular and Lymphatic System (continued)

<table>
<thead>
<tr>
<th>Name of Test</th>
<th>Purpose and Description</th>
<th>Related Nursing Interventions</th>
</tr>
</thead>
<tbody>
<tr>
<td>Transesophageal echocardiography (TEE)</td>
<td>A TEE allows visualization of adjacent cardiac and extracardiac structures to identify or monitor mitral and aortic valve pathology, left atrium intracardiac thrombus, acute dissection of the aorta, endocarditis, perioperative left ventricular function, and intracardiac repairs during surgery. A transducer (probe) attached to an endoscope is inserted into the esophagus, and images are taken. Concurrent IV contrast medium, Doppler ultrasound, and color flow imaging may be used.</td>
<td>Instruct the patient to not eat or drink fluids for 4 h before the test. Explain that a sedative will be given before the test. Take and record vital signs.</td>
</tr>
<tr>
<td>Abdominal or thoracic CT scan</td>
<td>A radiologic study used to assess the liver or spleen and enlarged lymph nodes in the mediastinum.</td>
<td>Tell the patient not to eat or drink for 4 h before the test. Oral hypoglycemic agents should not be taken if iodinated contrast is used. Assess for allergy to iodine products and notify physician if allergy is found. Administer oral contrast as prescribed. Following the test, tell the patient to increase oral intake of fluids to help flush out the dye, and to report any allergic reactions to the dye (such as skin rash, headache, vomiting, or kidney dysfunction).</td>
</tr>
<tr>
<td>Lymph node biopsy</td>
<td>A lymph node biopsy is done to obtain tissue for histologic examination for diagnosis and treatment. It may be open (performed in the operating room) or closed (needle) by needle aspiration of tissue from a lymph node.</td>
<td>Use sterile technique when changing dressings.</td>
</tr>
<tr>
<td>Lymphangiography (lymphangiogram)</td>
<td>This is an x-ray examination of the lymphatic vessels and lymph nodes, used to assess metastasis of the lymph nodes, to identify malignant lymphoma, and to identify the cause of lymphedema. An iodine contrast substance is injected at various sites and fluoroscopy is used to visualize lymphatic filling.</td>
<td>Ask the patient about allergies to seafood, iodine, or contrast medium used in a previous x-ray test. Tell the patient that the blue contrast dye discolors the urine and possibly the skin for a few days. Take and record vital signs. Ask patient to void before the test. After the test, monitor for dyspnea, pain, and hypotension; assess incision sites for manifestations of infection, and assess for leg edema. Elevate lower extremities as indicated.</td>
</tr>
<tr>
<td>Magnetic resonance imaging (MRI)—liver, spleen, lymph nodes</td>
<td>A radiologic study used to visualize the liver, spleen, and lymph nodes. It does not require injection of contrast medium.</td>
<td>Assess for any metallic implants (such as clips on brain aneurysms, pacemaker, body piercing, tattoos, and shrapnel). If present, notify physician. Remove transdermal medication patches (both OTC and prescribed) unless otherwise ordered (FDA, 2009). Replace the patch following the procedure. Tell the patient to inform the staff about the patch when making the appointment and when completing the admission information. Ask if patient is pregnant; if so the test is not performed. Ask about claustrophobia; if a problem exists, request patient to ask the referring physician for a relaxing medication prior to the MRI. If the patient is very claustrophobic, obese, or confused, an open MRI may be used.</td>
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GENETIC CONSIDERATIONS

Examples of Cardiovascular Disorders

- Familial hypercholesterolemia is a single-gene disorder that results in atherosclerosis and CAD, which may occur at an earlier age than in the general population (i.e., before age 55 in men and age 65 in women). However, increased cholesterol levels may also be inherited and are a risk factor for CAD in both men and women.
- Marfan’s syndrome is an autosomal-dominant inherited disorder that affects the skeleton, the eyes, and the cardiovascular system. The cardiovascular effects are a dilation of the proximal aorta and aortic dissection associated with degeneration of the elastic fibers in the tunica media of the aorta. There may also be thoracic aortic aneurysms.
- Hypertrophic cardiomyopathy is a disease of the sarcomere proteins. More than 100 different mutations in 10 genes encoding contractile sarcomeres have been identified.
- Long QT syndrome (LQTS) is an inherited genetic disorder that results from structural abnormalities of the sodium, potassium, and calcium channels in the heart, leading to dysrhythmias. This can result in unconsciousness, and may cause sudden cardiac death in teenagers and young adults when exposed to stressors ranging from exercise to loud sounds.
- Sickle cell disease is the most common inherited blood disorder in the United States, affecting 1 in 500 people of African descent. It is characterized by episodes of pain, chronic hemolytic anemia, and severe infections.
- Gaucher disease, more common in descendants of Eastern European Jewish people, is an inherited illness caused by a gene mutation. The gene is responsible for an enzyme that breaks down a specific fat. When the fat is not broken down, it accumulates in the liver, spleen, and bone marrow, causing pain, fatigue, jaundice, bone damage, anemia, and even death.
- Hemophilia A is a hereditary blood disorder, primarily affecting males, characterized by a deficiency of the blood clotting factor, Factor VIII. Abnormal bleeding results.
- Chronic myeloid leukemia (CML), a cancer of blood cells, is characterized by replacement of bone marrow with malignant, leukemic cells. Leukemic cells also circulate in the blood, causing enlargement of the spleen, liver, and other organs. This leukemia is the result of chromosomal abnormality called the Philadelphia chromosome.
- Thalassemia, an inherited disease of faulty hemoglobin synthesis, is more often found in descendants of people living near the Mediterranean Sea, Africa, the Middle East, and Asia. It comprises a group of disorders that range from very mild blood abnormalities to severe or fatal anemia.
The electrocardiogram (ECG) is a graphic record of the heart’s activity. Electrodes applied to the body surface are used to obtain a graphic representation of cardiac electrical activity. These electrodes detect the magnitude and direction of electrical currents produced in the heart. They attach to the electrocardiograph by an insulated wire called a lead. The electrocardiograph converts the electrical impulses it receives into a series of waveforms that represent cardiac depolarization and repolarization. Placement of electrodes on different parts of the body allows different views of this electrical activity, much like turning the head while holding a camera provides different views of the scenery. ECG waveforms and patterns are examined to detect dysrhythmias as well as myocardial damage, the effects of drugs, and electrolyte imbalances.

ECG waveforms reflect the direction of electrical flow in relation to a positive electrode. Current flowing toward the positive electrode produces an upward (positive) waveform; current flowing away from the positive electrode produces a downward (negative) waveform. Current flowing perpendicular to the positive pole produces a biphasic (both positive and negative) waveform. Absence of electrical activity, called the isoelectric line, is represented by a straight line.

ECG waveforms are recorded by a heated stylus on heat-sensitive paper. The paper is marked at standard intervals that represent time and voltage or amplitude (see Figure 1). Each small box is 1 mm². The recording speed of the standard ECG is 25 mm/second, so each small box represents 0.04 second. Five small boxes horizontally and vertically make one large box, equivalent to 0.20 second. Five large boxes represent 1 full second. Measured vertically, each small box represents 0.1 mV.

Both bipolar and unipolar leads are used in recording the ECG. A bipolar lead uses two electrodes of opposite polarity (negative and positive). In a unipolar lead, one positive electrode and a negative reference point at the center of the heart are used. The electrical potential between the two monitoring points is graphically recorded as the ECG waveform.

The heart can be viewed from both the frontal plane and the horizontal plane (see Figure 2). Each plane provides a unique perspective of the heart muscle. The frontal plane is an imaginary cut through the body that views the heart from top to bottom (superior–inferior) and side to side (right–left). This perspective of the heart is analogous to a paper doll cutout. It provides information about the inferior and lateral walls of the heart. The horizontal plane is a cross-sectional view of the heart from front to back (anterior–posterior) and side to side (right–left). Information regarding the anterior, septal, and lateral walls of the heart, as well as the posterior wall, is obtained from this view.

A standard 12-lead ECG provides a simultaneous recording of six limb leads and six precordial leads (see Figure 3). The limb leads provide information about the heart in the frontal plane and
include three bipolar leads (I, II, III) and three unipolar leads (aVR, aVL, and aVF). The bipolar limb leads measure electrical activity between a negative lead on one extremity and a positive lead on another. The unipolar limb leads (called augmented leads) measure the electrical activity between a single positive electrode on the center of the heart and a positive electrode on the chest wall.

The precordial leads, also known as chest leads or V leads, view the heart in the horizontal plane. They include six unipolar leads (V1, V2, V3, V4, V5, and V6), which measure electrical activity between the center of the heart and a positive electrode on the chest wall. The cardiac cycle is depicted as a series of waveforms, the P, Q, R, S, and T waves (see Figure 4).

- The P wave represents atrial depolarization and contraction. The impulse is from the sinus node. The P wave precedes the QRS complex and is normally smooth, round, and upright. P waves may be absent when the SA node is not acting as the pacemaker. Atrial repolarization occurs during ventricular depolarization and usually is not seen on the ECG.
- The PR interval represents the time required for the sinus impulse to travel to the AV node and into the Purkinje fibers. This interval is measured from beginning of P wave to beginning of QRS complex. If no Q wave is seen, the beginning of the R wave is used. The PR interval is normally 0.12 to 0.20 second (up to 0.24 second is considered normal in patients over age 65). PR intervals greater than 0.20 second indicate a delay in conduction from the SA node to the ventricles.
- The QRS complex represents ventricular depolarization and contraction. The QRS complex includes three separate waves: The Q wave is the first negative deflection, the R wave is the positive or upright deflection, and the S wave is the first negative deflection after the R wave. Not all QRS complexes have all three waves; nonetheless, the complex is called a QRS complex. The normal duration of a QRS complex is from 0.06 to 0.10 second. QRS complexes greater than 0.10 second indicate delays in transmitting the impulse through the ventricular conduction system.
- The ST segment signifies the beginning of ventricular repolarization. The ST segment, the period from the end of the QRS complex to the beginning of the T wave, should be isoelectric. An abnormal ST segment is displaced (elevated or depressed) from the isoelectric line.
- The T wave represents ventricular repolarization. It normally has a smooth, rounded shape that is usually less than 10 mm tall. It usually points in the same direction as the QRS complex. Abnormalities of the T wave may indicate myocardial ischemia or injury, or electrolyte imbalances.
- The QT interval is measured from the beginning of the QRS complex to the end of the T wave. It represents the total time of ventricular depolarization and repolarization. Its duration varies with gender, age, and heart rate; usually, it is 0.32 to 0.44 second long. Prolonged QT intervals indicate a prolonged relative refractory period and a greater risk of dysrhythmias. Shortened QT intervals may result from medications or electrolyte imbalances.
- The U wave is not normally seen. It is thought to signify repolarization of the terminal Purkinje fibers. If present, the U wave follows the same direction as the T wave. It is most commonly seen in hypokalemia.
The interview begins by exploring the patient’s chief complaint (e.g., chest pain, leg pain, or fatigue). Describe the patient’s chest pain or leg pain in terms of location, quality or character, timing, setting or precipitating factors, severity, aggravating and relieving factors, and associated symptoms (Table 29–3).

Explore the patient’s medical history for any cardiovascular disorders such as angina, heart attack, congestive heart failure (CHF), stroke, hypertension (HTN), peripheral vascular disease (PVD), or other chronic illnesses (such as diabetes or bleeding disorders). Ask the patient about previous heart surgery or illnesses, such as rheumatic fever, scarlet fever, or recurrent streptococcal throat infections, and radiation treatment for breast cancer. Review the patient’s family history for CAD, HTN, stroke, hyperlipidemia, diabetes, congenital heart disease, or sudden death.

Ask the patient about past or present occurrence of cardiovascular symptoms, such as chest pain, shortness of breath, difficulty breathing, cough, palpitations, fatigue, light-headedness or dizziness, fainting, heart murmur, blood clots, leg cramps or swelling, changes in skin color or temperature, varicose veins, or edema. Because cardiovascular function affects all other body systems, a full history may need to explore other related systems, such as respiratory function. Ask about past or present bleeding (from the nose, gums or mouth, or rectum) as well as associated symptoms (such as pallor, dizziness, fatigue), lymph node changes (swelling, pain, heat), swelling of extremities, and recurrent infections.

Review the patient’s personal habits and nutritional history, including body weight; eating patterns; dietary intake of fats, salt, fluids; dietary restrictions; hypersensitivities or intolerances to food or medication; and the use of caffeine and alcohol. If the patient uses tobacco products, ask about type (cigarettes, pipe, cigars, snuff), duration, amount, and efforts to quit. If the patient uses street drugs, ask about type, method of intake (e.g., inhaled or injected), duration of use, and efforts to quit. Include questions about the patient’s activity level and tolerance, recreational activities, and relaxation habits. Assess the patient’s sleep patterns for interruptions in sleep due to dyspnea, cough, discomfort, urination, or stress. Ask how many pillows the patient uses when sleeping.

It is important to consider socioeconomic factors that may precipitate or aggravate circulatory problems, such as inadequate clothing, shoes, or shelter; and occupational levels such as prolonged sitting or standing, exposure to radiation, or extremes of temperature. Lifestyle, including intravenous drug use or sexual practices, may be significant in determining the risk for diseases associated with bleeding and impaired lymphatic function.

### BOX 29–2 Interpreting an ECG

Interpreting an ECG strip to determine the cardiac rhythm is a skill that takes practice to learn and master. Many methods are used to analyze ECGs. It is important to use a consistent method for ECG analysis. Identifying and interpreting complex dysrhythmias requires advanced skills and knowledge obtained through further education. One method follows:

- **Step 1: Determine rate.** Assess heart rate. Use P waves to determine the atrial rate and R waves for the ventricular rate. Several approaches can determine the heart rate:
  - Count the number of complexes in a 6-second rhythm strip (the top margin of ECG paper is marked at 3-second intervals), and multiply by 10. This provides an estimate of the rate and is particularly valuable if rhythms are irregular.
  - Count the number of large boxes between two consecutive complexes, and divide by 300 (the number of large boxes in 1 minute). For example, there are 6 large boxes between two R waves; 300 divided by 6 equals a ventricular rate of 50 bpm. Memorize the following sequence for rapid rate determination: 300, 150, 100, 75, 60, 50, 43. One large box between complexes equals a rate of 300; two, a rate of 150; three, a rate of 100; and so on.
  - Count the number of small boxes between two consecutive complexes, and divide 1500 (the number of small boxes in 1 minute) by this number. For example, there are 19 small boxes between two R waves; 1500 divided by 19 equals a ventricular rate of 79 bpm. This is the most precise measurement of heart rate.
- **Step 2: Determine regularity.** Regularity is the consistency with which the P waves or QRS complexes occur. In a regular rhythm, all waves occur at a consistent rate. Rhythm regularity is determined by measuring the interval between consecutive waves. Place one point of an ECG caliper (a measuring device) on the peak of the P wave (for atrial rhythm) or the R wave (for ventricular rhythm). Adjust the other point to the peak of the next wave, P to P or R to R. Keeping the calipers set at this distance, evaluate intervals between consecutive waves. The rhythm is regular if all caliper points fall on succeeding wave peaks. Alternately, use a strip of blank paper on top of the ECG strip, marking the peaks of two or three consecutive waves.
- **Step 3: Assess P wave.** The presence or absence of P waves helps determine the origin of the rhythm. All the P waves should be alike in size and shape (morphology). If P waves are not seen or they differ in shape, the rhythm may not originate in the sinus node.
- **Step 4: Assess P to QRS relationship.** Determine the relationship between P waves and QRS complexes. There should be one and only one P wave for every QRS complex, because the normal stimulus for ventricular contraction originates in the sinus node.
- **Step 5: Determine interval durations.** To evaluate impulse transmission through the cardiac conduction system, measure the PR interval, QRS duration, and QT interval. To measure, count the number of small boxes from the beginning of the interval to the end, and multiply by 0.04 second. Then determine whether the interval duration is within its normal limits. For example, the PR interval is 3.5 small boxes wide, or 0.14 second. This is within the normal limits of 0.12 to 0.20 second. This interval should be consistent, not varying from beat to beat. A PR interval greater than 0.20 second or one that varies from beat to beat is abnormal.
  
  The QRS complex duration is normally between 0.06 and 0.10 second. A QRS complex greater than 0.12 second indicates delayed ventricular conduction.
  
  The QT interval is normally 0.32 to 0.44 second. It varies inversely with the heart rate: The faster the heart rate, the shorter the QT interval. As a general rule, the QT interval should be no more than half the previous R–R interval. A prolonged QT interval indicates a prolonged relative refractory period of the heart.
- **Step 6: Identify abnormalities.** Note the presence and frequency of ectopic (extra) beats, deviation of the ST segment above or below the baseline, and abnormalities in waveform shape and duration.
**Physical Assessment**

Physical assessment of cardiovascular function may be performed either as part of a total assessment or alone for patients with suspected or known problems with heart, peripheral vascular, lymphatic, or hematologic function. The patient may sit or lie in the supine position. Before beginning the assessment, collect all required equipment and explain the techniques to the patient to decrease anxiety.

Assess the heart through inspection, palpation, and auscultation over the precordium (the area of the chest wall overlying the heart) (Figure 29–19). Movements over the precordium may be more easily seen with tangential lighting (in which the light is directed at a right angle to the area being observed, producing shadows). A quiet environment is essential to hear and assess heart sounds accurately. Assess the heart and thorax for the following:

- **The apical impulse** is a normal, visible pulsation (thrust) in the area of the midclavicular line in the left fifth intercostal space. It can be seen on inspection in about half of the adult population. (The apical impulse was previously called the point of maximal impulse [PMI] but this term is no longer used because a maximal impulse may occur in other areas of the precordium as a result of abnormal conditions.)
- **Retraction** is a pulling in of the tissue of the precordium; a slight retraction just medial to the midclavicular line at the area of the apical impulse is normal and is more likely to be visible in thin patients.
- Pulsations (other than the normal apical pulsations), which may be called **heaves** or **lifts**, are considered abnormal. They may occur as the result of an enlarged ventricle.

The techniques used to assess the peripheral vascular and lymphatic systems include inspecting the skin for such changes as edema, ulcerations, or alterations in color and temperature; auscultating BP; and palpating the major pulse points of the body (Figure 29–20).

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**TABLE 29–3 Assessing Chest Pain**

<table>
<thead>
<tr>
<th>Characteristic</th>
<th>Examples</th>
</tr>
</thead>
<tbody>
<tr>
<td>Location</td>
<td>Subternal, precordial, jaw (more common in women), back (more common in women)</td>
</tr>
<tr>
<td></td>
<td>Radiation to neck, jaw, shoulder, arm, back between the shoulders</td>
</tr>
<tr>
<td>Character/quality</td>
<td>Pressure; tightness; crushing, burning, or aching quality; heaviness; dullness; “heartburn” or indigestion</td>
</tr>
<tr>
<td>Timing: onset, duration, and frequency</td>
<td>Onset: Sudden or gradual? Duration: How many minutes does the pain last? Frequency: Is the pain continuous or periodic?</td>
</tr>
<tr>
<td>Setting/precipitating factors</td>
<td>Awake, at rest, sleep interrupted? With activity? With eating, exertion, exercise, elimination, emotional upset?</td>
</tr>
<tr>
<td>Intensity/severity</td>
<td>Can range from 0 (no pain) to 10 (worst pain ever felt)</td>
</tr>
<tr>
<td>Aggravating factors</td>
<td>Activity, breathing, temperature</td>
</tr>
<tr>
<td>Relieving factors</td>
<td>Medication (nitroglycerin, antacid), rest; there may be no relieving factors</td>
</tr>
<tr>
<td>Associated symptoms</td>
<td>Fatigue, shortness of breath (more common in women), palpitations, nausea and vomiting (more common in women), sweating, anxiety, light-headedness or dizziness</td>
</tr>
</tbody>
</table>
and lymph nodes. The 5 Ps of peripheral vascular disease include pulselessness, pallor, pain, paresthesias, and paralysis. The patient may be assessed in the supine, sitting, and standing positions. Physical assessment of the lymphatic system, using inspection and palpation, is usually integrated into the assessment of other body systems. For example, the tonsils are inspected with the pharynx during the head and neck assessment; the regional lymph nodes are evaluated with corresponding body regions (e.g., occipital, auricular, and cervical nodes are evaluated with assessment of the head and neck, axillary nodes with assessment of the breast or thorax, epitrochlear node with assessment of the peripheral vascular exam of the arms, and inguinal nodes with assessment of the abdomen); and the spleen can be palpated during the abdominal assessment. Normal age-related findings for the older adult are summarized in the Nursing Care of the Older Adult box.

### NURSING CARE OF THE OLDER ADULT

#### Age-Related Cardiovascular Changes

<table>
<thead>
<tr>
<th>Age-Related Change</th>
<th>Significance</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Myocardium:</strong></td>
<td></td>
</tr>
<tr>
<td>- Efficiency and contractility.</td>
<td>- Decreased CO when under physiologic stress with resulting tachycardia that lasts longer. The person may require rest time between physical activities.</td>
</tr>
<tr>
<td><strong>Sinus Node:</strong></td>
<td></td>
</tr>
<tr>
<td>- Increase in thickness of shell surrounding the node, and a decrease in the number of pacemaker cells throughout the conduction system.</td>
<td>- Stroke volume may increase to compensate for tachycardia, leading to increased BP.</td>
</tr>
<tr>
<td><strong>Left ventricle:</strong></td>
<td></td>
</tr>
<tr>
<td>- Slight hypertrophy, prolonged isometric contraction phase and relaxation time; time for diastolic filling and systolic emptying cycle.</td>
<td>- BP increases to compensate for increased peripheral resistance and decreased CO.</td>
</tr>
<tr>
<td><strong>Valves and blood vessels:</strong></td>
<td></td>
</tr>
<tr>
<td>- Aorta is elongated and dilated, valves are thicker and more rigid, and resistance to peripheral blood flow increases by 1% per year.</td>
<td>- Baroreceptor response decreases.</td>
</tr>
<tr>
<td><strong>Bone marrow:</strong></td>
<td></td>
</tr>
<tr>
<td>- Ability of bone marrow to respond to need for increased RBCs, WBCs, and platelets.</td>
<td>- Anemia may result.</td>
</tr>
<tr>
<td><strong>Blood vessels:</strong></td>
<td></td>
</tr>
<tr>
<td>- Tunica intima: fibrosis, calcium and lipid accumulation, cellular proliferation.</td>
<td>- As a result of age-related changes, the systolic BP rises. Decreased arterial elasticity results in vascular changes in the heart, kidneys, and pituitary gland. Decreased baroreceptor function results in postural hypotension. Vessels in the head, neck, and extremities are more prominent.</td>
</tr>
<tr>
<td>- Tunica media: thins, elastin fibers calcify; increase in calcium results in stiffening. Baroreceptor function is impaired and peripheral resistance increases.</td>
<td>- Inefficient vasoconstriction, decreased CO, and reduced muscle mass and subcutaneous tissue lead to a reduced ability to respond to cold temperatures.</td>
</tr>
<tr>
<td><strong>Immune system:</strong></td>
<td></td>
</tr>
<tr>
<td>- Impaired function of B and T lymphocytes.</td>
<td>- With a decrease in BP and changes in blood vessel walls, tissue perfusion may be inadequate, leading to edema, inflammation, pressure ulcers, and changes in effects of medications.</td>
</tr>
</tbody>
</table>

- Production of antibodies.

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**SAMPLE DOCUMENTATION**

**Assessment of Cardiac Function**

56-year-old male admitted to cardiac critical care unit from ED to rule out myocardial infarction. States he has pain in the middle of his chest that is “like a heavy pressure”; 6 on a 10-point scale. Skin cool, slightly moist. BP 190/94 mmHg right arm and 186/92 mmHg left arm (both reclining). Apical pulse 92 bpm, regular and strong. No pulse deficit. Respiration 28/min. Apical impulse nonpalpable, no visible heaves or thrusts. S1 and S2 auscultated without murmurs or clicks. S4 noted.
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Cardiovascular Assessments

Technique/Normal Findings  Abnormal Findings

Apical Impulse Assessment

First using the palmar surface and then repeating with finger pads, palpate the precordium for symmetry of movement and the apical impulse for location, size, amplitude, and duration. Refer to Figure 29–19 for the sequence for palpation. To locate the apical impulse, ask the patient to assume a left lateral recumbent position. Simultaneous palpation of the carotid pulse may also be helpful. The apical impulse is not palpable in all patients. The apical impulse may be palpated in the mitral area, and has only a brief small amplitude.

• An enlarged or displaced heart is associated with an apical impulse lateral to the midclavicular line (MCL) or below the fifth left intercostal space (ICS).
• Increased size, amplitude, and duration of the apical impulse are associated with left ventricular volume overload (increased afterload) in conditions such as HTN and aortic stenosis, and with pressure overload (increased preload) in conditions such as aortic or mitral regurgitation.
• Increased amplitude alone may occur with hyperkinetic states, such as anxiety, hyperthyroidism, and anemia.
• Decreased amplitude is associated with a dilated heart in cardiomyopathy.
• Displacement alone may also occur with dextrocardia, diaphragmatic hernia, gastric distention, or chronic lung disease.
• A thrill (a palpable vibration over the precordium or an artery) may accompany severe valve stenosis.
• A marked increase in amplitude of the apical impulse at the right ventricular area occurs with right ventricular volume overload in atrial septal defect.
• An increase in amplitude and duration occurs with right ventricular pressure overload in pulmonic stenosis and pulmonary hypertension. A lift or heave may also be seen in these conditions (and in chronic lung disease).
• A palpable thrill in this area occurs with ventricular septal defect.
• Right ventricular enlargement may produce a downward pulsation against the fingertips.
• An accentuated pulsation at the pulmonary area may be present in hyperkinetic states.
• A prominent pulsation reflects increased flow or dilation of the pulmonary artery.
• A thrill may be associated with aortic or pulmonary stenosis, aortic stenosis, pulmonary HTN, or atrial septal defect.
• Increased pulsation at the aortic area may suggest aortic aneurysm.
• A palpable second heart sound (S₂) may be noted with systemic HTN.

Palpate the subxiphoid area with the index and middle finger. No pulsations or vibrations should be palpated.

Cardiac Rate and Rhythm Assessment

Auscultate heart rate (Figure 29–21). The heart rate should be 60 to 100 beats per minute (bpm) with regular rhythm.

• A heart rate exceeding 100 bpm is tachycardia. A heart rate less than 60 bpm is bradycardia.

Simultaneously palpate the radial pulse while listening to the apical pulse. The radial and apical pulses should be equal.

Auscultate heart rhythm. The heart rhythm should be regular.

• If the radial pulse falls behind the apical rate, the patient has a pulse deficit, indicating weak, ineffective contractions of the left ventricle.
• Dysrhythmias (abnormal heart rate or rhythm) may be regular or irregular in rhythm; their rates may be slow or fast. Irregular rhythms may occur in a pattern (e.g., an early beat every second beat, called bigeminy), sporadically, or with frequency and disorganization (e.g., atrial fibrillation). A pattern of gradual increase and decrease in heart rate that is within the normal heart rate and that correlates with inspiration and expiration is called sinus arrhythmia.

(continued)
### Cardiovascular Assessments (continued)

**Heart Sounds Assessment**

See guidelines for cardiac auscultation in Box 29–3.

**Box 29–3 Guidelines for Cardiac Auscultation**

1. Locate the major auscultatory areas on the precordium (refer to Figure 29–21).
2. Choose a sequence of listening. Either begin from the apex and move upward along the sternal border to the base, or begin at the base and move downward to the apex. One suggested sequence is shown in Figure 29–21.
3. Listen first with the patient in the sitting or supine position. Then ask the patient to lie on the left side, and focus on the apex. Lastly, ask the patient to sit up and lean forward. These position changes bring the heart closer to the chest wall and enhance auscultation. Carry out the following steps when the patient assumes each of these positions:
   a. First, auscultate each area with the diaphragm of the stethoscope to listen for high-pitched sounds: $S_1$, $S_2$, murmurs, pericardial friction rubs.
   b. Next, auscultate each area with the bell of the stethoscope to listen for lower-pitched sounds: $S_3$, $S_4$, murmurs.
   c. Listen for the effect of respirations on each sound; while the patient is sitting up and leaning forward, ask him or her to exhale and hold the breath while you listen to heart sounds.

<table>
<thead>
<tr>
<th>Technique/Normal Findings</th>
<th>Abnormal Findings</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Heart Sounds Assessment</strong></td>
<td></td>
</tr>
<tr>
<td><strong>Identify $S_1$ (first heart sound) and note its intensity. At each auscultatory area, listen for several cardiac cycles. $S_1$ is loudest at the apex of the heart.</strong></td>
<td>- An accentuated $S_1$ occurs with tachycardia, states in which CO is high (fever, anxiety, exercise, anemia, hyperthyroidism), complete heart block, and mitral stenosis.</td>
</tr>
<tr>
<td><strong>Listen for splitting of $S_2$. Splitting of $S_1$ may occur during inspiration.</strong></td>
<td>- A diminished $S_1$ occurs with first-degree heart block, mitral regurgitation, CHF, CAD, and pulmonary or systemic HTN. The intensity is also decreased with obesity, emphysema, and pericardial effusion. Varying intensity of $S_1$ occurs with complete heart block and grossly irregular rhythms.</td>
</tr>
<tr>
<td><strong>Identify $S_2$ (second heart sound) and note its intensity. $S_2$ immediately follows $S_1$ and is loudest at the base of the heart.</strong></td>
<td>- Abnormal splitting of $S_1$ may be heard with right bundle branch block and premature ventricular contractions.</td>
</tr>
<tr>
<td><strong>Listen for splitting of $S_3$. No splitting of $S_2$ should be heard.</strong></td>
<td>- An accentuated $S_2$ may be heard with HTN, exercise, excitement, and conditions of pulmonary HTN such as CHF and cor pulmonale.</td>
</tr>
<tr>
<td><strong>Identify extra heart sounds in systole. Extra heart sounds are not present in systole.</strong></td>
<td>- A diminished $S_2$ occurs with aortic stenosis, a fall in systolic BP (shock), and increased anteroposterior chest diameter.</td>
</tr>
<tr>
<td><strong>Identify the presence of extra heart sounds in diastole. Extra heart sounds are not present in diastole.</strong></td>
<td>- Wide splitting of $S_2$ is associated with delayed emptying of the right ventricle, resulting in delayed pulmonary valve closure (e.g., mitral regurgitation, pulmonary stenosis, and right bundle branch block).</td>
</tr>
<tr>
<td><strong>Murmur Assessment</strong></td>
<td>- Fixed splitting occurs when right ventricular output is greater than left ventricular output and pulmonary valve closure is delayed (e.g., with atrial septal defect and right ventricular failure).</td>
</tr>
<tr>
<td>Identify any murmurs. Note location, timing, presence during systole or diastole, and intensity. Use the following scale to grade murmurs: I = Barely heard II = Quietly heard III = Clearly heard</td>
<td>- Paradoxical splitting occurs when closure of the aortic valve is delayed (e.g., left bundle branch block).</td>
</tr>
<tr>
<td><strong>Midsystolic murmurs are heard with semilunar valve disease (e.g., aortic and pulmonary stenosis) and with hypertrophic cardiomyopathy.</strong></td>
<td>- Ejection sounds (or clicks) result from the opening of deformed semilunar valves (e.g., aortic and pulmonary stenosis).</td>
</tr>
<tr>
<td><strong>Pansystolic (holosystolic) murmurs are heard with AV valve disease (e.g., mitral and tricuspid regurgitation, ventricular septal defect).</strong></td>
<td>- A midsystolic click is heard with mitral valve prolapse (MVP).</td>
</tr>
<tr>
<td><strong>A late systolic murmur is heard with MVP.</strong></td>
<td>- An opening snap results from the opening of a semitonic mitral stenotic valve.</td>
</tr>
<tr>
<td><strong>Early diastolic murmurs occur with regurgitant flow across incompetent semilunar valves (e.g., aortic regurgitation).</strong></td>
<td>- A pathologic $S_2$ (a third heart sound that immediately follows $S_2$, called a ventricular gallop) results from myocardial failure and ventricular volume overload (e.g., CHF, mitral or tricuspid regurgitation).</td>
</tr>
<tr>
<td></td>
<td>- An $S_3$ (a fourth heart sound that immediately precedes $S_1$, called an atrial gallop) results from increased resistance to ventricular filling after atrial contraction (e.g., HTN, CAD, aortic stenosis, and cardiomyopathy).</td>
</tr>
<tr>
<td></td>
<td>- A combined $S_3$ and $S_4$ is called a summation gallop and occurs with severe CHF.</td>
</tr>
<tr>
<td></td>
<td>- A pericardial friction rub results from inflammation of the pericardial sac, as with pericarditis.</td>
</tr>
</tbody>
</table>
Cardiovascular Assessments (continued)

<table>
<thead>
<tr>
<th>Technique/Normal Findings</th>
<th>Abnormal Findings</th>
</tr>
</thead>
<tbody>
<tr>
<td>IV = Loud</td>
<td>• Mid-diastolic and presystolic murmurs, such as with mitral stenosis, occur with turbulent flow across the AV valves.</td>
</tr>
<tr>
<td>V  = Very loud</td>
<td>• Continuous murmurs throughout systole and all or part of diastole occur with patent ductus arteriosus.</td>
</tr>
<tr>
<td>VI = Loudest; may be heard with stethoscope off the chest. A thrill may accompany murmurs of grade IV to grade VI.</td>
<td>Note pattern/shape, crescendo, decrescendo, and radiation/transmission (to axilla, neck). No murmurs should be heard.</td>
</tr>
</tbody>
</table>

**Blood Pressure and Pulse Pressure Assessment**

See Box 29-4 for blood pressure measurement guidelines.

**BOX 29-4 Guidelines for Blood Pressure Assessment**

**REVIEW OF KOROTKOFF’S SOUNDS**

The first sound heard is the systolic pressure; at least two consecutive sounds should be clear. If the sound disappears and then is heard again 10 to 15 mm later, an auscultatory gap is present; this may be a normal variant, or it may be associated with hypertension. The first diastolic sound is heard as a muffling of the Korotkoff’s sound and is considered the best approximation of the true diastolic pressure. The second diastolic sound is the level at which sounds are no longer heard.

The American Heart Association recommends documenting all three readings when measuring BP, for example, 120/72/64 mmHg. If only two readings are documented, the systolic and the second diastolic pressure are taken, for example, 120/64 mmHg.

**TECHNIQUE REMINDERS**

• Choose a cuff of an appropriate size: The cuff should snugly cover two-thirds of the upper arm, and the bladder should completely encircle the arm. The bladder should be centered over the brachial artery, with the lower edge 2 to 3 cm above the antecubital space.

• The patient’s arm should be slightly flexed and supported (on a table or by the examiner) at heart level.

• To determine how high to inflate the cuff, palpate the brachial pulse, and inflate the cuff to the point on the manometer at which the pulse is no longer felt; then, add 30 mmHg to this reading, and use the sum as the target for inflation. Wait 15 seconds before reinflating the cuff to auscultate the BP.

• To recheck a BP, wait at least 30 seconds before attempting another inflation.

• Always inflate the cuff completely, and then deflate it. Once deflation begins, allow it to continue; do not try to reinflate the cuff if the first systolic sound is not heard or if the cuff inadvertently deflates.

• The bell of the stethoscope more effectively transmits the low-pitched sounds of BP.

**SOURCES OF ERROR**

• Falsely high readings can occur if the cuff is too small, too loose, or if the patient supports his or her own arm.

• Falsely low readings can occur if a standard cuff is used on a patient with thin arms.

• Inadequate inflation may result in underestimation of the systolic pressure or overestimation of the diastolic pressure if an auscultatory gap is present.

• Rapid deflation and repeated or slow inflations (causing venous congestion) can lead to underestimation of the systolic BP and overestimation of the diastolic BP.

**FACTORS ALTERING BLOOD PRESSURE**

• A change from the horizontal to upright position causes a slight decrease (5 to 10 mm) in systolic BP; the diastolic BP remains unchanged or rises slightly.

• BP taken in the arm is lower when the patient is standing.

• If the BP is taken with the patient in the lateral recumbent position, a lower BP reading may be obtained in both arms; this is especially apparent in the right arm with the patient in the left lateral position.

• Factors that increase BP include exercise, caffeine, tobacco use, cold environment, eating a large meal, painful stimuli, and emotions.

• Factors that lower BP include sleep (by 20 mmHg) and very fast, slow, or irregular heart rates.

• BP tends to be higher in taller or heavier patients.

• Legs should not be crossed when sitting, lying, or standing during BP measurement.

**ALTERNATIVE METHODS OF BLOOD PRESSURE MEASUREMENT**

• The palpatory method may be necessary if severe hypotension is present and the BP is inaudible. Palpate the brachial pulse, and inflate the cuff 30 mm above the point where the pulse disappears; deflate the cuff, and note the point on the manometer where the pulse becomes palpable again. Record this as the palpatory systolic BP.

• Leg BP measurement may be needed when there is injury of the arms or to rule out coarctation of the aorta or aortic insufficiency when arm diastolic BP is over 90 mmHg. Place the patient in the prone or supine position with the leg slightly flexed. Place a large leg cuff on the thigh with the bladder centered over the popliteal artery. Place the bell of the stethoscope over the popliteal space. Normal leg systolic BP is higher than arm BP; diastolic BP should be equal to or lower than arm BP. Abnormally low leg BP occurs with aortic insufficiency and coarctation of the aorta.
Cardiovascular Assessments (continued)

**Technique/Normal Findings**

Auscultate BP in each arm with the patient seated. The normal BP is considered to be <120/80 mmHg.

Auscultate BP in each arm with the patient standing. If orthostatic changes occur, measure the BP with the patient supine, legs dangling, and again with the patient standing, 1–3 min apart. A decrease of systolic BP is expected, but should be <10 mmHg; diastolic BP should not drop on standing.

Observe the pulse pressure. The pulse pressure is the difference between the systolic and diastolic BP. For example, if the BP is 140/80 mmHg, the pulse pressure is 60. A normal pulse pressure is one-third the systolic measurement.

**Skin Assessment**

Inspect the color of the skin. The skin color should be appropriate to the patient’s age and race.

- Pallor reflects constriction of peripheral blood flow (e.g., due to syncope or shock) or decreased circulating oxyhemoglobin (e.g., due to hemorrhage or anemia).
- Central cyanosis of the lips, earlobes, oral mucosa, and tongue suggests chronic cardiopulmonary disease. (See Box 29–5 for abnormal findings associated with chronic cardiopulmonary disease.)

**BOX 29–5 Abnormal Findings Associated with Peripheral Vascular and Lymphatic Assessment**

- Pallor is an absence of color of the skin. The degree of pallor depends on the patient’s normal skin color and health status. Dark skin may appear ashen or have a yellowish tinge.
- Cyanosis is a bluish discoloration of the skin and mucous membranes in people with light skin. In people with dark skin, cyanosis may be difficult to observe. Inspect the nail beds and conjunctiva.
- Edema is an abnormal accumulation of fluid in the interstitial spaces of body tissues. It is often most apparent in the lower extremities.
- Varicose veins are tortuous and dilated veins that have incompetent valves. The saphenous veins of the legs are most commonly affected.
- Enlarged lymph nodes result from infection or malignancy.
- Atrophic changes are changes in the size or activity of body tissues as a result of pathology or injury. Decreased blood flow and oxygenation of the lower extremities often cause atrophic changes of loss of hair, thickened toenails, changes in pigmentation, and ulcersation.
- Gangrene is the necrosis (or death) of tissue, most often the result of loss of blood supply and infection. Gangrene often begins in the most distal of the tissues of the extremities.
- Pressure ulcers, also called decubitus ulcers or bed sores, are the result of ischemia and hypoxia of tissue following prolonged pressure. These ulcers often are located over bony prominences. If untreated, the tissue changes proceed from red skin to deep, crater-like ulcers.
- Lymphangitis (inflammation of a lymphatic vessel) may produce a red streak with induration (hardness) following the course of the lymphatic collecting duct; infected skin lesions may be present, particularly between the digits.
- Lymphedema (swelling due to lymphatic obstruction) occurs with congenital lymphatic anomaly (Milroy's disease) or with trauma to the regional lymphatic ducts from surgery or metastasis (e.g., arm lymphedema after radical mastectomy with axillary node removal).
- Edema of lymphatic origin is usually not pitting, and the skin may be thickened; one example is the taut swelling of the face and body that occurs with myxedema, associated with hypothyroidism.

Inspect the skin of the extremities and over the regional lymph nodes, noting any edema, erythema, red streaks, or skin lesions. There should be no edema, redness, or lesions over the regional lymph nodes.
Cardiovascular Assessments (continued)

**Artery and Vein Assessment**

Palpate the temporal arteries. *There should be no redness, swelling, nodules, or variations in pulse amplitude.*

Inspect and palpate the carotid arteries. Note symmetry, the pulse rate, rhythm, volume, and amplitude. *Note any variation with respiration. Describe all pulses as increased, normal, diminished, or absent.*

Scales ranging from 0 to 4+ are sometimes used as follows:

- 0 = Absent
- 1+ = Diminished
- 2+ = Normal
- 3+ = Increased
- 4+ =Bounding

Pulse waveforms are shown in Figure 29–22. The carotid pulses should be bilaterally equal in rate, rhythm, volume, and amplitude.

- Redness, swelling, nodularity, and variations in pulse amplitude may occur with temporal arteritis.

- A unilateral pulsating bulge is seen with a tortuous or kinked carotid artery.

- Alterations in pulse rate or rhythm are due to cardiac dysrhythmias.

- An absent pulse indicates arterial occlusion.

- A hypokinetic (weak) pulse is associated with decreased stroke volume (Figure 29–22B). This may be due to congestive heart failure (CHF), aortic stenosis, or hypovolemia; to increased peripheral resistance, which may result from cold temperatures; or to arterial narrowing, commonly found with atherosclerosis.

- A hyperkinetic (bounding) pulse occurs with increased stroke volume and/or decreased peripheral resistance (Figure 29–22C). This may result from states in which CO is high or from aortic regurgitation. It also may occur with anemia, hyperthyroidism, bradycardia, or reduced compliance, as with atherosclerosis.

- A bigeminal pulse is marked by decreased amplitude of every second beat (Figure 29–22D). This may be due to premature contractions (usually ventricular).

- Pulsus alternans is a regular pulse with alternating strong and weak beats (Figure 29–22E). This may be due to left ventricular failure and severe HTN.

- A murmuring or blowing sound heard over stenosed peripheral vessels is known as a bruit. A bruit heard over the middle to upper carotid artery suggests atherosclerosis.

- An increase in jugular venous pressure (JVP) over 3 cm and located above the sternal angle reflects increased right atrial pressure. This occurs with right ventricular failure or, less commonly, with constrictive pericarditis, tricuspid stenosis, and superior vena cavae obstruction.

- A decrease in venous pressure reflects reduced left ventricular output or blood volume.

- Unilateral neck vein distention suggests local compression or anatomic anomaly.

- A rise in the column of neck vein distention over 1 cm with liver compression indicates right heart failure.

(continued)
### Cardiovascular Assessments (continued)

#### Upper Extremity Assessment

**Technique/Normal Findings**

- Inspect and palpate the arms and hands, noting size and symmetry, skin color, and temperature. **Arms and hands should be symmetrical in size and shape, warm, and of appropriate skin color.**

**Abnormal Findings**

- Unilateral swelling with venous prominence occurs with venous obstruction.
- Cyanosis of the nail beds reflects chronic cardiopulmonary disease.
- Unilateral swelling with venous prominence occurs with venous obstruction.
- Cold temperature of the hands and fingers occurs with vasoconstriction.
- Capillary refill that takes less than 3 sec reflects circulatory compromise, such as hypovolemia or anemia.

**Palpate the nail beds for capillary refill.** (Apply pressure to the patient’s fingertip. Watch for blanching of the nail beds. Release the pressure. Note the time it takes for capillary refill, indicated by the return of pink color on release of the pressure.) **Capillary refill should be less than 2 seconds (i.e., immediate).**

**Assess venous pattern and pressure.** (Elevate one of the patient’s arms over the head for a few seconds. Slowly lower the arm. Observe the filling of the patient’s hand veins.) **Hand veins should fill equally and immediately.**

- Distention of hand veins at elevations over 9 cm above heart level reflects an increase in systemic venous pressure.

#### BOX 29–6 Assessing Jugular Venous Pressure

When a patient with normal venous pressure lies in the supine position, full neck veins are normally visible, but as the head of the bed is elevated, the pulsations disappear. In the patient with greatly elevated venous pressure, visible pulsations of the jugular vein are present even in the upright position. To conduct the inspection, do the following:

1. Remove clothing from the patient’s neck and chest. Elevate the head of the bed 30 to 45 degrees, and turn the patient’s head to the opposite side. Shine a light tangentially across the neck to increase shadows. If the external jugular veins are distended, they will be visible vertically between the mandible and outer clavicle.
2. If jugular distention is present, assess the jugular venous pressure by measuring from the highest point of visible distention to the sternal angle (the point at which the clavicles meet) on both sides of the neck (see the accompanying figure). Bilateral measurements above 3 cm are considered elevated and indicate increased venous pressure; distention on only one side may indicate obstruction.

Palpate the radial and brachial pulses. Note rate, rhythm, volume amplitude, symmetry, and variations with respiration. (Refer to Figure 29–22 for pulse patterns.) **Radial and brachial pulses should have equal and normal rate, be strong, and not vary with respirations.**

- Alterations in pulse rate or rhythm are due to cardiac dysrhythmias (such as atrial fibrillation, atrial flutter, and premature ventricular contractions). A pulse rate over 100 bpm is tachycardia; a pulse rate below 60 bpm is bradycardia.
- A pulse deficit (slower radial rate than apical rate) occurs with dysrhythmias and CHF.
- Irregularities of rhythm produce early beats and pauses (skipped beats) in the pulse, which may be regular in pattern, sporadic, or grossly irregular.
- Diminished or absent radial pulses may be due to thromboangiitis obliterans (Buerger’s disease) or acute arterial occlusion.
- A weak and thready pulse, often with tachycardia, reflects decreased CO.
- A bounding pulse occurs with hyperkinetic states and atherosclerosis.
- Unequal pulses between extremities suggest arterial narrowing or obstruction on one side.
- In sinus dysrhythmia (a normal variant, especially in young adults), the pulse rate increases with inspiration and decreases with expiration.
### Cardiovascular Assessments (continued)

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<tr>
<th>Technique/Normal Findings</th>
<th>Abnormal Findings</th>
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| If arterial insufficiency is suspected, palpate the ulnar pulse and perform the Allen test:  
  • Have the patient make a tight fist.  
  • Compress both the radial and ulnar arteries.  
  • Have the patient open the hand to a slightly flexed position.  
  • Observe for pallor and manifestations of pain.  
  • Release the ulnar artery and observe for the return of pink color within 3–5 sec.  
  • Repeat the procedure on the radial artery.  
  • Color should return within 3–5 sec in both the ulnar and the radial arteries. | • The normal ulnar artery may or may not have a palpable pulse.  
• Persistent pallor with the Allen test suggests ulnar artery occlusion. |

#### Lower Extremity Assessment

Inspect and palpate each leg, noting size, shape, and symmetry; arterial pattern; skin color, temperature, and texture; hair pattern; pigmentation; rashes; ulcers, sensation; and capillary refill. **Legs should be symmetric in size and shape, arterial pattern, appropriate color, warm, without lesions. Capillary refill on toenails should be immediate.**

With the patient supine, assess the venous pattern of the legs. Repeat with the patient standing. **Venous pattern on both legs should be symmetric, and there should be no edema, cyanosis, or lesions.**

Palpate the femoral, popliteal, posterior tibial, and dorsalis pedis pulses for volume, amplitude, and symmetry (refer to Figure 29–20). **All lower extremity pulses should be strong and equal in amplitude.**

If pulses are diminished, observe for postural color changes. Elevate both legs 60 degrees, and observe the color of the soles of the feet. Have the patient sit and dangle the legs; note the return of color to the feet.

If arterial insufficiency is suspected, auscultate the femoral arteries. **No bruits should be heard.**

Inspect and gently palpate the calves. **There should be no redness or swelling, heat, or pain in the calves of the legs.**

Inspect and palpate for edema. Use your thumb to compress the dorsum of the patient's foot, around the ankles, and along the tibia (Figure 29–23A). A depression in the skin that does not immediately refill is called pitting edema. **Normally, there is no edema.**

Edema can be graded on a scale from 1+ to 4+ (Figure 29–23B):  
1+ (−2-mm depression): no visible change in the leg; slight pitting  
2+ (−4-mm depression): no marked change in the shape of the leg; pitting slightly deeper  
3+ (−6-mm depression): leg visibly swollen; pitting deep  
4+ (−8-mm depression): leg very swollen; pitting very deep

**• Edema may be caused by disease of the cardiovascular system such as CHF; by renal, hepatic, or lymphatic problems; or by infection.  
• Venous distention suggests venous insufficiency or incompetence.**

(continued)
Cardiovascular Assessments (continued)

Technique/Normal Findings

Abdominal Assessment

Inspect and palpate the abdominal aorta. Note size, width, and any visible pulsations or bulging. Abdominal aorta should be of appropriate size without visible pulsations or bulging.

1. A pulsating mass in the upper abdomen suggests an aortic aneurysm, particularly in the older adult.
2. An aorta greater than 2.5–3 cm in width reflects pathologic dilation, most likely due to arteriosclerosis.

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Auscultation sites of the abdominal aorta and its branches.

- Abdominal bruits reflect turbulent blood flow associated with partial arterial occlusion.
- A bruit heard over the aorta suggests an aneurysm.
- A bruit heard over the epigastrium and radiating laterally, especially with HTN, suggests renal artery stenosis.
- Bruits heard in the lower abdominal quadrants suggest partial occlusion of the iliac arteries.

Lymph Node Assessment

Palpate the regional lymph nodes of the head and neck, axillae, arms, and groin. Use firm, circular movements of the finger pads and note size, shape, symmetry, consistency, delineation, mobility, tenderness, sensation, and condition of overlying skin. Nodes should not be enlarged or painful.

- Lymphadenopathy refers to the enlargement of lymph nodes (over 1 cm) with or without tenderness. It may be caused by inflammation, infection, or malignancy of the nodes or the regions drained by the nodes.
- Lymph node enlargement with tenderness suggests inflammation (lymphadenitis). With bacterial infection, the nodes may be warm and matted with localized swelling.
- Malignant or metastatic nodes may be hard, indicating lymphoma; rubbery, indicating Hodgkin’s disease; or fixed to adjacent structures. Usually they are not tender.
- Ear infections and scalp and facial lesions, such as acne, may cause enlargement of the preauricular and cervical nodes.
- Anterior cervical nodes are enlarged and infected with streptococcal pharyngitis and mononucleosis.
### Cardiovascular Assessments (continued)

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<td>• Lymphadenitis of the cervical and submandibular nodes occurs with herpes simplex lesions.</td>
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<td>• Enlargement of supraclavicular nodes, especially the left, is highly suggestive of metastatic disease from abdominal and thoracic cancer.</td>
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<td>• Axillary lymphadenopathy is associated with breast cancer.</td>
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<td>• Lesions of the genitals may produce enlargement of the inguinal nodes.</td>
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<tr>
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<td>• Persistent generalized lymphadenopathy is associated with acquired immunodeficiency syndrome (AIDS) and AIDS-related complex.</td>
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### Spleen Assessment

Palpate for the spleen, in the upper left quadrant of the abdomen. **The spleen is normally not palpable.**

A palpable spleen in the left upper abdominal quadrant of an adult may indicate abnormal enlargement (splenomegaly) and may be associated with cancer, blood dyscrasias, and viral infection, such as mononucleosis.

Percuss for splenic dullness in the lowest left intercostal space (ICS) at the anterior axillary line or in the 9th to 10th ICS at the midaxillary line (Figure 29–25). **Normally, tympany is heard.**

A dull percussion note in the lowest left ICS at the anterior axillary line or below the tenth rib at the midaxillary line suggests splenic enlargement.

### CHAPTER HIGHLIGHTS

- Normal anatomy, physiology, and functions of the heart, blood vessels, and lymphatic system are the basis for assessment.
- Both general health and focused cardiovascular and lymphatic system assessments can detect dysfunction, injury, and disorders.
- Assessment of the cardiovascular and lymphatic systems includes diagnostic tests, genetic considerations, a health interview, and a physical assessment.

### TEST YOURSELF NCLEX-RN® REVIEW

1. A patient who is hemorrhaging has decreased preload. What physiologic effect should the nurse expect to occur with this patient?
   1. increased afterload
   2. decreased cardiac output
   3. decreased action potential
   4. increased ejection fraction

2. The nurse is preparing to assess a patient who is experiencing chest pain. Which question should the nurse ask to learn more information about the intensity of the pain?
   1. “Did the pain move into your left arm?”
   2. “Was the pain a pressure, a burning, or tightness?”
   3. “Was your pain relieved by resting or worse when you were busy?”
   4. “On a scale of 0 (no pain) to 10 (worst pain), what number is your pain?”
3. The nurse is preparing to assess a patient’s apical impulse. Which anatomic location should the nurse use to make this assessment?
   1. right nipple line, any intercostal space
   2. left substernal line, sixth intercostal space
   3. left midclavicular line, fifth intercostal space
   4. right midaxillary line, second intercostal space

4. The nurse assesses a patient’s heart rate as being 50 beats per minute. How should the nurse document this finding?
   1. bradycardia
   2. tachycardia
   3. hypotension
   4. hypertension

5. A patient’s laboratory values indicate a low red blood cell count. What subjective data should the nurse expect to assess that is consistent with this data?
   1. fatigue
   2. nausea
   3. chest pain
   4. sore throat

6. A patient is being admitted for a low platelet count. Which findings should the nurse expect when conducting a physical assessment of this patient?
   1. varicose veins
   2. excessive bruising
   3. enlarged lymph nodes
   4. changes in pulse pressure

7. The nurse determines that an older patient would benefit from interventions to address peripheral vascular resistance. What manifestations did the nurse assess in this patient? (Select all that apply.)
   1. joint pain
   2. sunken eyeballs
   3. distant bowel sounds
   4. elevated blood pressure
   5. lower extremity fatigue

8. The nurse is preparing to assess a patient’s carotid arteries. Which techniques should the nurse use for this assessment? (Select all that apply.)
   1. Palpate for pulse rate.
   2. Inspect for pulsations.
   3. Auscultate for rhythm.
   4. Percuss for arterial wall density.
   5. Palpate deeply for arterial wall integrity.

9. During the physical examination of a patient’s abdomen, the nurse auscultates a blowing sound over the aorta. How should the nurse document this finding?
   1. bruit
   2. dysrhythmia
   3. bigeminal pulse
   4. hypokinetic pulse

10. A patient has been admitted with severe leg pain. The limb is cyanotic, cool to the touch, and peripheral pulses are absent. What should the nurse do first after this assessment?
    1. Document the findings.
    2. Teach relaxation techniques.
    3. Notify the physician immediately.
    4. Ask how long the limb has been hurting.

See Test Yourself answers in Appendix B.

BIBLIOGRAPHY


